CONTENTS

This Issue in the Journal
3 A summary of the original articles featured in this issue

Editorial
6 The New Zealand health system after 75 years: let’s stop and smell the roses
Toni Ashton

Original Articles
9 Severe *Clostridium difficile* infection in New Zealand associated with an emerging strain, PCR-ribotype 244
Mary De Almeida, Helen Heffernan, Anne Dervan, Sarah Bakker, Joshua Freeman, Hasan Bhally, Susan Taylor, Thomas Riley, Sally Roberts

15 Contribution by primary health nurses and general practitioners to the Diabetes Annual Review (Get Checked) programme in Auckland, New Zealand
Barbara Daly, Timothy Kenealy, Bruce Arroll, Nicolette Sheridan, Robert Scragg

27 Registered nurse assessment and treatment of skin sepsis in New Zealand schools: the development of protocols
Alison Vogel, Diana Lennon, Sarah Gray, Elizabeth Farrell, Philippa Anderson

39 Malignant hypertension: a preventable emergency
Walter van der Merwe, Veronica van der Merwe

46 Can we improve the prevention and detection of congenital abnormalities? An audit of early pregnancy care in New Zealand
Nicola Arroll, Cindy Farquhar, Lynn Sadler, Peter Stone, Vicki Masson

Review Article
57 IgE-mediated food allergy—diagnosis and management in New Zealand children
J Sinclair, S Brothers, P Jackson, T Stanley, M Ang, P Brown, A Craig, A Daniell, C Doocey, S Hoare, S Lester, P McIlroy, G Ostring, D Purvis, J Sanders, R Smiley, M Sutherland, T Townend, J Wilde, G Williams
Viewpoint

68 Questions about New Zealand’s health system in 2013, its 75th anniversary year
Robin Gauld

Clinical Correspondence

75 Scalp involvement by Sarcoptes scabiei var hominis resembling seborrhoeic dermatitis in two immunocompromised patients with systemic lupus erythematosus
Paul Jarrett, Antonia Birry

79 Medical image. A rare complication of cytarabine therapy
Praveen Ramakrishnan Geethakumari, Sreejith Nair

81 Medical image. Short metacarpal and metatarsals
Gopal Chandra Ghosh, Bhimarey Kategary, Arnab Sarkar, Krishnarpan Chatterjee, Brijesh Sharma

83 Medical image. Systemic retinoids for recurrent keratoacanthomas
Faisal Ali, John T Lear

Letters

86 The Legacy of Percy Pease
Kiarash Taghavi, James K Hamill

89 RMO patient safety forums in New Zealand: agents for change
Lucie Collinson, Katie Thorne, Stephen Dee, Kate MacIntyre, Grant Pidgeon

100 Years Ago in the NZMJ

93 Coroner's inquest

Methuselah

94 Selected excerpts from Methuselah

Erratum

96 Lessons from the February 2011 Christchurch Earthquake for the training and preparation of Post Graduate Year 1 Doctors (Dale C Sheehan, John Thwaites, Blair York, Jaejin Lee)
NZMJ
This Issue in the Journal

Severe *Clostridium difficile* infection in New Zealand associated with an emerging strain, PCR-ribotype 244
Mary De Almeida, Helen Heffernan, Anne Dervan, Sarah Bakker, Joshua Freeman, Hasan Bhally, Susan Taylor, Thomas Riley, Sally Roberts

PCR-ribotype 244 is a newly recognised strain of *Clostridium difficile* (*C. difficile*) in New Zealand. *C. difficile* is a bacteria commonly associated with antibiotic-associated diarrhoea. A case-control study of hospitalised patients in Auckland found that cases infected with *C. difficile* PCR-ribotype 244 were more likely to have severe disease than patients infected with other strains.

Contribution by primary health nurses and general practitioners to the Diabetes Annual Review (Get Checked) programme in Auckland, New Zealand
Barbara Daly, Timothy Kenealy, Bruce Arroll, Nicolette Sheridan, Robert Scragg

The 'Get Checked' annual diabetes review programme was successful in engaging practice and community-based specialist nurses in the community management of diabetes and has revealed positive relationships between nurses and doctors, extended roles for nurses and the importance of engaging nurses in the design of health care programmes. Nurses involved in the programme undertake a large proportion of all patient annual reviews at their practice and major nursing roles include measuring blood pressure, weighing patients, undertaking foot examinations and giving health promotional advice. DHBs need to continue to acknowledge the valuable contribution made by the largest health professional workforce and ensure primary health care nurses are involved in developing an effective replacement ‘Get Checked’ programme.

Registered nurse assessment and treatment of skin sepsis in New Zealand schools: the development of protocols
Alison Vogel, Diana Lennon, Sarah Gray, Elizabeth Farrell, Philippa Anderson

Skin infections are common problem in children and many children are admitted to hospital for treatment. Nurse led clinics in schools are being developed and tested as a way of providing early treatment that is easy to access. We assessed the available evidence to develop practical protocols for nurses to use to assess and treat skin infections in schools.
Malignant hypertension: a preventable emergency
Walter van der Merwe, Veronica van der Merwe

18 out of 565 (3.2%) consecutive patients seen in a difficult hypertension (high blood pressure) clinic presented with malignant hypertension. Malignant hypertension means blood pressure which is so high that the patient is at immediate risk of severe organ damage (blindness, stroke, kidney failure etc) and even death. All cases were thoroughly investigated and all were determined to have a background of “essential” hypertension—this is the common type of high blood pressure which has no demonstrable underlying cause. All the patients survived, but most were left with a degree of chronic kidney disease. All cases of malignant hypertension could have been prevented with adequate blood pressure treatment, but most were not on blood pressure medication at the time of presentation. An annual blood pressure check in all adults to detect those with elevated pressures requiring treatment is one of the most cost-effective public health measures.

Can we improve the prevention and detection of congenital abnormalities? An audit of early pregnancy care in New Zealand
Nicola Arroll, Cindy Farquhar, Lynn Sadler, Peter Stone, Vicki Masson

This paper describes an audit of 137 perinatal deaths due to congenital cardiovascular, central nervous system or chromosomal abnormality in 2010 that were identified prospectively. We assessed the care these women received by reviewing their clinical notes and ultrasound scans. Most women (83%) were seen early in pregnancy (before 14 weeks gestation), in the majority of cases by their general practitioner. There was a delay (between two and 33 weeks) for 21% of these women before they registered with their maternity provider. Only 7% of women were reported as having taken folic acid supplements before they became pregnant. Folic acid needs to be taken before pregnancy and during the first trimester to reduce the risk of neural tube defects which form during the first month of pregnancy. While not all abnormalities can be detected early in pregnancy, delay in accessing antenatal care and failure by health care providers to offer screening early were the most common reasons for delay in diagnosis of screen detectable abnormalities in this audit.

IgE-mediated food allergy—diagnosis and management in New Zealand children ((review article))
J Sinclair, S Brothers, P Jackson, T Stanley, M Ang, P Brown, A Craig, A Daniell, C Doocey, S Hoare, S Lester, P McIlroy, G Ostring, D Purvis, J Sanders, R Smiley, M Sutherland, T Townend, J Wilde, G Williams

Food allergy is a common problem for New Zealand children, and needs to be accurately diagnosed. Management includes avoiding specific foods, and managing the risk of accidental food exposure. With time many food allergies resolve, children need ongoing follow-up to work out when this occurs.
Questions about New Zealand’s health system in 2013, its 75th anniversary year ((viewpoint article))
Robin Gauld

This year, 2013, is a significant occasion for the New Zealand health system for it marks the 75th anniversary of the laying of its foundations. Such an anniversary is a time for celebration but also for reflection on whether we have achieved the goals sought in the passage, on 14 September 1938, of the Social Security Act under the government of Michael Joseph Savage. It is also a time for debate around how well our present health system functions and performs in the light of the 1938 Act’s aims. This article, therefore, reflects on the goals of the Social Security Act for health care, whether we have achieved them and what the key barriers to this have been. It also asks whether we should recommit to the original 1938 aims for health care, or develop a new set of principles that reflect how the New Zealand health system is presently structured and performs.
The New Zealand health system after 75 years: let’s stop and smell the roses

Toni Ashton

As Professor Robin Gauld discusses in an article in this edition of the NZMJ, this year is the 75th anniversary of the 1938 Social Security Act, which laid the foundations for the New Zealand public health system as it is today. Birthdays are usually a time for celebration, as well as a time to reflect on both the past and the future. So what do we have to celebrate?

According to the Minister of Health: “The New Zealand health system is performing well…Almost 90% of New Zealanders report that they are in good health…Life expectancy continues to rise. In 2010, life expectancy at birth in New Zealand stood at 81.0 years, more than 1 year higher than the OECD average of 79.8 years.”1

Other indicators of improving health status include: infant mortality has fallen to a record low (at 4.2 deaths per 1000 live births),2 93% of children under 2 years of age are now fully vaccinated,3 and smoking rates continue to decline, especially amongst youth.4

Of course, many other factors have contributed to these improvements in health status other than the health system. So what other indicators do we have of the performance of the health system? Three domains that are commonly used to assess health system performance are access (including timeliness), quality, and cost (or value for money).

Universal access to free, high quality care in public hospitals for everyone with an urgent or essential need is certainly a cause for celebration. Timely access to non-urgent care is also rapidly improving, with the Minister of Health’s target for elective surgical procedures increasing the numbers of operations being performed in public hospitals and reducing surgical waiting times.1

Between November 2008 and April 2012, an additional 1078 full-time equivalent (FTE) doctors and 2445 more nursing FTEs were employed in the public health system, bringing the number of FTE doctors in New Zealand public hospitals to 7008 and nurses to 20,781,5 and thus facilitating further expansion of services provided by the district health boards (DHBs).

Gauld discusses some problems of access to primary medical care, especially in relation to copayments for general practitioner (GP) services. Yet timeliness and physical access to primary health services are generally very good, with 84% of people reporting that they are able to see a GP within 24 hours.4

Unlike many countries, almost all practices have a practice nurse which reduces the pressure on GPs, expands the scope of services provided, and keeps the costs down for some patients.
Access to after-hours care has also improved significantly in recent years. In a 2012 survey, only 72 (6%) of 1152 patients attending a GP considered that the opening hours were too restricted while 75% knew how to get after-hours access if required.6

With regard to quality, one legacy of the series of health reforms that have been implemented in New Zealand over the past two decades has been the emergence of a culture of quality throughout the system. Many agencies and organisations have contributed to this shift including DHBs (many of which have developed strong quality improvement programmes), the Office of the Health and Disability Commissioner, Independent Practitioner Associations (IPAs), Primary Health Organisations (PHOs), and most recently, the Health Quality and Safety Commission.

Resultant improvements in the quality of our health services are too numerous to mention here but, to the extent that they have improved the experiences of patients, are surely worthy of celebration.

As far as cost is concerned, New Zealand spends an average of US$3182 per person per year compared with US$3800 in Australia and US$3322 for the OECD average.7 While our lower expenditure largely reflects the fact that NZ’s GDP is lower than many OECD countries, the coverage and quality of our health system is comparable to that of many wealthier countries suggesting that we get pretty good value for money.

Here it is opportune to acknowledge the sterling work done by New Zealand’s Pharmaceutical Management Agency (Pharmac) in keeping the prices of pharmaceuticals down over the past 20 years. Analysis by the Commonwealth Fund indicated that, in 2006/07, the prices in New Zealand of 30 commonly prescribed drugs were around two-thirds of the average price of 9 other countries, and one-third of the average price paid in the United States.8 In 2011, annual expenditure per capita on pharmaceuticals in New Zealand was just US$298 compared with the OECD average of US$495.50.6

Another cause for celebration is that, unlike many other OECD countries, New Zealand governments have continued to invest tax funds into the health system. The public share of total expenditure has been slowly increasing in recent years and now accounts for about 83% of total expenditure compared with the an average of 77% across all OECD countries.6 This has resulted in a concomitant fall in the share of health expenditure that is accounted for by out-of-pocket payments from 17% of total health expenditure in 2001 to 10.9% in 2011. This is the fourth lowest in the OECD, with only the United Kingdom, France and the Netherlands reporting lower shares of expenditure paid out-of-pocket.

Perhaps the greatest cause for celebration is the mere fact that a collectively-funded, universal public health system exists in New Zealand, and is likely to continue to do so for many years into the future. Clearly there is still room for improvement in some areas—and Gauld discusses a number of these. There will also be ongoing challenges, especially in terms of how to manage the burgeoning cost of long-term chronic conditions such as diabetes and dementia and the need to continue reducing ethnic inequalities.
But we can be grateful that we have in place a health system that is well-placed to
plan services and develop processes for managing these challenges. On behalf of all
New Zealanders, thank you Michael Joseph Savage!

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Severe *Clostridium difficile* infection in New Zealand associated with an emerging strain, PCR-ribotype 244

Mary N De Almeida, Helen Heffernan, Anne Dervan, Sarah Bakker, Joshua T Freeman, Hasan Bhally, Susan L Taylor, Thomas V Riley, Sally A Roberts

**Abstract**

**Aim** To compare disease severity and clinical outcome of *Clostridium difficile* infection (CDI) due to PCR-ribotype (RT) 244 with CDI due to other strains present in Auckland.

**Method** A retrospective, case-control study was conducted. Ten cases with CDI due to RT 244 were compared with 20 controls infected with other *C. difficile* strains. RT 244 isolates were further analysed for antimicrobial susceptibility, binary toxin genes and mutations in the *tcdC* gene.

**Results** Cases were significantly more likely to have severe disease than controls (OR 9.33; \(p=0.015\)). 50% of cases had community-associated CDI compared with 15% of controls (\(p=0.078\)). All RT 244 isolates produced binary toxin and had a single-base pair deletion in *tcdC* at position 117.

**Conclusion** *C. difficile* RT 244 is a newly recognised strain in New Zealand. It shares several features that characterise RT 027. Given its propensity to cause severe community-associated disease, a heightened awareness of this strain is needed to ensure early testing in patients admitted from the community with identified risk factors for CDI.

*Clostridium difficile* infection (CDI) is the commonest cause of healthcare-associated diarrhoea. In Europe and North America the incidence and severity of *Clostridium difficile* infection (CDI) has increased over the past decade.\(^1\) This increase has been attributed largely to the emergence of so called “hypervirulent” strains such as PCR-ribotype (RT) 027.\(^2\) However, to date these strains have remained very uncommon in New Zealand.\(^3,4\)

Recently a cluster of “presumptive 027” cases was identified in Melbourne, Australia (personal communication, Dr R. Stuart, Monash Medical Centre, 2012). On further testing the isolates were not confirmed as RT 027 but rather a ribotype new to Australia designated RT 244. Preliminary investigations showed that compared to other strains, infection with RT 244 was associated with severe community-onset disease and higher mortality. In New Zealand a newly recognised strain of *C. difficile*, identified during a national survey in November 2011 was compared to RT 244 and the ribotyping patterns were indistinguishable.

We sought to compare disease severity and clinical outcome of CDI due to RT 244 with CDI due to other *C. difficile* strains circulating during the same time period. We further characterised RT 244 by performing antimicrobial susceptibility testing.
against moxifloxacin, clindamycin and metronidazole, and determining the presence of the binary toxin genes and mutations in the \textit{tcdC} gene.

**Methods**

A retrospective, matched case-control study was undertaken. The cases were 10 patients with CDI due to RT 244 who were hospitalised in the Auckland region of New Zealand between October 2011 and May 2012. Each case was matched for age (±10 years) and gender with two controls. Controls were drawn from patients within the Auckland region who had \textit{C. difficile} other than RT 244 isolated from stool.

Data collected included: demographics; comorbidities; hospitalisation in the preceding 6 months; location at symptom onset; antibiotic exposure during 4 weeks prior to symptom onset; and exposure to H\textsubscript{2} antagonists, proton-pump inhibitors or chemotherapy. For each patient, the Charlson Co-morbidity Index was calculated.\textsuperscript{5} Disease severity, treatment regimen, disease recurrence and all-cause 30-day mortality were also determined.

CDI was defined as the presence of diarrhoea and a positive stool \textit{C. difficile} toxin result. Each patient was classified as having either community or healthcare-associated disease using surveillance definitions.\textsuperscript{5} Community-associated (CA-CDI) was defined as CDI symptom onset in the community, or within 48 hours after admission to a healthcare facility, provided symptom onset was more than 12 weeks after the last discharge from a healthcare facility.

Severe CDI was defined as an episode of CDI with one or more signs of severe colitis including: fever (temperature $>38.5$ °C); rigors; haemodynamic instability; signs of ileus; marked leukocytosis (leukocyte count $>15 \times 10^9$ x/L); marked left shift (band neutrophils $>20\%$ of leukocytes); rise in creatinine ($>50\%$ above baseline); pseudomembranous colitis and abnormal imaging including distension of large intestine; colonic wall thickening including low-attenuation mural thickening; pericolonic fat stranding; and ascites not explained by other causes.\textsuperscript{7} These markers could not be attributable to a concomitant disease.

CDI recurrence was considered to have occurred if there was reappearance of symptoms after initial improvement, or if there was a further positive toxin result within 8 weeks of the last positive toxin result in combination with a prescription for metronidazole.

Stool specimens were inoculated onto chromogenic \textit{C. difficile} agar (ChromID\textsuperscript{TM} \textit{C. difficile}, BioMérieux, Marcy L’Etoile, France) and colonies with typical appearance were identified by routine laboratory methods. PCR-ribotyping was performed by a standard method using capillary-gel electrophoresis.\textsuperscript{6} PCR for binary toxin genes and \textit{tcdC} gene sequencing for detection of \textit{tcdC} gene mutations was performed as previously described.\textsuperscript{9,10}

Antimicrobial susceptibility testing of the RT 244 isolates was performed using metronidazole M.I.C.E strips (Oxoid, Thermo Fisher Scientific Inc, Hampshire, United Kingdom) and moxifloxacin, clindamycin and vancomycin Etests (BioMerieux, Marcy L’Etoile, France).

The study was assessed by the Northern Region Ethics Committee and formal approval was not considered necessary.

**Statistical analysis**—Continuous and categorical variables were compared using the Student $t$ test and Fisher exact test, respectively. $P<0.05$ was considered significant.

**Results**

Compared with controls, cases with RT 244 causing CDI were significantly more likely to have severe disease (OR 9.33; 95\% confidence interval 1.27-82.59; $p=0.015$). In addition, 50\% of cases had CA-CDI compared with 15\% of controls, although this difference was not significant ($p=0.078$). There were no significant differences between the cases and controls in comorbidities, antibiotic exposure in the previous 4 weeks, the rate of disease recurrence and 30-day all-cause mortality (Table 1).
Table 1. Comparison of cases with *Clostridium difficile* PCR-ribotype 244 infection and controls infected with other *C. difficile* strains

<table>
<thead>
<tr>
<th>Variables</th>
<th>Cases with RT-244 (n=10)</th>
<th>Controls(^a) (n=20)</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean CCI</td>
<td>3.5</td>
<td>5.45</td>
<td></td>
<td>0.095</td>
</tr>
<tr>
<td>Risk factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotic therapy(^b)</td>
<td>7 (70%)</td>
<td>19 (95%)</td>
<td>0.123 (0.004–1.76)</td>
<td>0.095</td>
</tr>
<tr>
<td>PPI/H2 antagonist</td>
<td>5 (50%)</td>
<td>16 (80%)</td>
<td>0.25 (0.03–1.70)</td>
<td>0.115</td>
</tr>
<tr>
<td>Chemotherapy</td>
<td>0</td>
<td>1 (5%)</td>
<td>–</td>
<td>1.000</td>
</tr>
<tr>
<td>CA-CDI</td>
<td>5 (50%)</td>
<td>3 (15%)</td>
<td>5.67 (0.76–48.23)</td>
<td>0.078</td>
</tr>
<tr>
<td>Outcome</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe disease</td>
<td>7 (70%)</td>
<td>4 (20%)</td>
<td>9.33 (1.27–82.59)</td>
<td>0.015</td>
</tr>
<tr>
<td>Recurrence</td>
<td>4 (40%)</td>
<td>3 (15%)</td>
<td>3.78 (0.49–31.85)</td>
<td>0.181</td>
</tr>
<tr>
<td>30-day all-cause mortality</td>
<td>1 (10%)</td>
<td>3 (15%)</td>
<td>0.63 (0.02–8.9)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

Abbreviations: OR, odds ratio; CI, confidence interval; CCI, Charlson comorbidity index; PPI, proton-pump inhibitor; CA-CDI, community-associated *C. difficile* infection.

\(^a\) PCR-ribotypes among controls included 002 (3), 014 (5), 020 (1), 070 (3), NZ 11/12 (1), NZ 11/14 (1), NZ 11/16 (1), NZ 11/17 (2), NZ 11/24 (1), NZ 11/25 (1) and NZ 11/30 (1). (Strains that did not match the European Centre for Disease Prevention and Control’s set of reference strains were assigned New Zealand PCR-ribotype strain number prefixed with ‘NZ11’).

\(^b\) Antibiotic therapy in 4 weeks prior to symptom onset.

The three cases that underwent endoscopy all had macroscopic evidence of pseudomembranous colitis. In comparison, the only control patient that underwent endoscopy did not have macroscopic evidence of pseudomembranous colitis although histological evidence of pseudomembranous colitis was apparent following colectomy for toxic megacolon.

All 10 *C. difficile* RT 244 isolates were susceptible to moxifloxacin (minimum inhibitory concentration (MIC) 1 mg/L) and metronidazole (MIC 0.25–0.5 mg/L) by CLSI criteria.\(^1\)\(^1\) The clindamycin MICs ranged from 2 to 4 mg/L (the breakpoint for resistance is \(\geq 8\) mg/L). All isolates were susceptible to vancomycin (MIC 1 mg/L) by EUCAST criteria.\(^1\)\(^2\) All 10 isolates carried the binary toxin genes and had a 1-base pair deletion in the *tcdC* regulatory gene at position 117. No additional deletions or insertions were found within the *tcdC* gene.

**Discussion**

This study reports on the characteristics of *C. difficile* infection caused by RT 244 strains. This is a newly recognised strain in New Zealand. In our study, patients infected with RT 244 were more likely to present with severe disease (p=0.015) and there was a trend towards community-associated disease (p=0.078) similar to the findings in the Australian cohort.

Traditionally, CDI has been considered a hospital-acquired infection and testing of patients presenting with community-onset diarrhoea for CDI is not standard practice in New Zealand. These findings provide further support to suggestions that CDI should be considered in the differential diagnosis of adult patients presenting with community-onset diarrhoea, in whom routine enteric pathogens have been excluded and, in particular, for patients with severe diarrhoea and who have had a recent course of antibiotics.
This strain has features similar to *C. difficile* RT 027, in particular, the presence of binary toxin and a 1-base pair deletion at position 117 in the *tcdC* gene. Indeed, whole genome sequencing undertaken at Oxford University suggests that these two strains have descended from a common ancestor (Prof TV Riley et al., unpublished data, 2012). However, RT 244 lacks the 18-base pair *tcdC* deletion characteristic of RT 027. Another major difference between the recent epidemic RT 027 and RT 244 is susceptibility to moxifloxacin; epidemic RT 027 isolates are typically resistant whereas RT 244 isolates are susceptible.

Testing algorithms for CDI differ between hospitals in New Zealand. Laboratories that use the GeneXpert *C. difficile* PCR assay are likely to detect RT 244 as the assay offers presumptive identification of RT 027 based on the presence of binary toxin genes and a 1-base pair deletion in the *tcdC* gene at position 117. However, most testing algorithms limit the use of the GeneXpert assay to testing of samples that have glutamate dehydrogenase, but not toxin, detected by an enzyme immunoassay. With this algorithm, infections caused by the RT 244 strain may go undetected, unless other circumstances, such as severe disease, trigger further testing.

This study has a number of limitations. It was retrospective and the quality of documentation in the clinical records was variable. There were only 10 cases and this may have limited our ability to show statistical difference between the cases and the controls. Regardless, our findings highlight the need for an increased awareness of *C. difficile* RT 244 and its potential risks, given its similarity to RT 027, and support calls for enhanced measures for its diagnosis in our patient population.

The source of this newly emerging strain is not known. It belongs to the same clonal lineage as RT 027 and has the potential to cause severe disease with significant morbidity and mortality in patients with and without comorbidities. Severe disease can be defined in a number of ways.

A recent study in which severe disease was defined as intensive care admission, interventional surgery or death within 30 days of diagnosis, failed to show an association between specific ribotypes and severe infection. White cell count and albumin level at presentation were the most clinically relevant predictors of severe disease in that study.

We used a definition for severe disease based on clinical and laboratory findings which may explain the association between infection with RT 244 and disease severity. Also, there was a trend towards community-associated disease suggesting that our patients were less likely to have some of the risk factors traditionally associated with CDI.

Further work needs to be done to determine the reservoirs of this strain. It is important that we focus on infection prevention and control measures that should reduce the introduction and transmission of this strain at least in healthcare settings.
Competing interests: None identified.

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Acknowledgements: We thank staff of the Microbiology Department at LabPlus for performing anaerobic culture and antimicrobial susceptibility testing, and ESR for molecular analysis of isolates.

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Contribution by primary health nurses and general practitioners to the Diabetes Annual Review (Get Checked) programme in Auckland, New Zealand

Barbara Daly, Timothy Kenealy, Bruce Arroll, Nicolette Sheridan, Robert Scragg

Abstract

Aim To describe primary health care (practice and specialist) nurses involvement in the government-funded annual diabetes review 'Get Checked' programme and the division of care between nurses and general practitioners in Auckland, New Zealand.

Method Of the total 911 practice and specialist nurses identified and working in the greater Auckland region, 276 (30%) were randomly selected and invited to undertake a self-administered questionnaire and telephone interview in 2006–8.

Results An 86% response rate was achieved. Over 60% of practice nurses and over half of specialist nurses participate in 'Get Checked' reviews. Of those nurses, 40% of practice and 70% specialist nurses, reported completing over half of the total number of 'Get Checked' reviews at their practice. Of the nurses sampled who work in general practice (n=198), 38% reported that 'nurses mostly complete' the reviews, 45% stated that 'nurses and doctors equally complete' them and 17% reported that only 'doctors' did so. For the nurses who reported that 'nurses and doctors equally complete' the reviews (n=89), most nurses undertake blood pressure measurements (90%), weigh patients (88%), give lifestyle advice (87%), examine patient’s feet (73%), and 44% carried out the complete review of the patients they consult.

Conclusion These findings show the 'Get Checked' programme was successful in engaging practice and community-based specialist nurses in the community management of diabetes and has revealed positive relationships between nurses and doctors, extended roles for nurses and the importance of engaging nurses in the design of health care programmes.

The increasing incidence of type 2 diabetes in New Zealand (NZ), and the inability to continue to meet the health care needs of large numbers of people with diabetes-related complications within specialist secondary health care services, initiated a Government-funded diabetes annual review (DAR) 'Get Checked' programme in 2000.

A parallel development, the Primary Health Care (PHC) Strategy (2001), encouraged the development of not-for-profit Primary Health Organisations (PHOs), and following European and United Kingdom (UK) trends, aimed to increase PHC diabetes services and provide systematic care for all diabetes patients.

Funding was provided for general practices through District Health Boards (DHBs) and PHOs, on a fee-for-service basis. Either general practitioners (GPs) or practice-based PHC nurses were able to carry out the 'Get Checked' review, or its components, and check lists were used to encourage a comprehensive review of patients and for
reimbursement purposes.\(^2\) This created an opportunity and the expectation that practice-based nurses would expand their role and capacity in the community management of diabetes.\(^6\)

In 2010, almost a quarter of all 42,334 registered nurses who are in NZ were working in a community or rural setting with 45\% employed as practice nurses (PN), who are the largest group of PHC nurses.\(^7\) PN predominantly work in general practice, although a small proportion work in Accident and Medical Clinics.\(^8\)

The main two groups of specialist nurses (SN) who provide community-based diabetes care are diabetes nurse specialists (DNS), who predominantly work in hospital-based outpatient services, and chronic care management (CCM) nurses who mainly work for independent for-profit community-based PHC providers who are usually GPs.

In addition to the 'Get Checked' programme, funding was provided for 'Care Plus' through PHOs from 2004, designed for patients with high needs and chronic care conditions that offered patients 2 hours of free care every 6 months.\(^9,10\) 'Care Plus' was generally also paid on a fee-for-service basis through PHOs, via a capitation funding for 5\% of the practice population being enrolled at any one time.\(^10\)

Large international intervention trials have reported improvements in the clinical management of blood glucose levels (BGLs) and major cardiovascular (CV) risk factors reduces microvascular complications\(^11\) and all diabetes-related complications, respectively.\(^12-16\)

This representative cross-sectional survey reports on the participation of practice-based PHC nurses in the ‘Get Checked' review programme, in the largest urban area in NZ.

The aim of this paper is to describe PN and SN participation in the DAR 'Get Checked' programme and the division of care between GPs and practice-based PHC nurses in Auckland, and quantify their contribution as originally outlined in the PHC Strategy.

Methods
A cross-sectional survey of community-based PHC nurses, working in the greater Auckland region, involved in the management of diabetes, including the ‘Get Checked’ annual review, was conducted between September, 2006 and February, 2008. The recruitment of the PHC nurses and methods have been described previously,\(^8\) but briefly, we randomly sampled 26\% of all PN, district nurses, DNS and CCM nurses who completed a postal self-administered questionnaire (n=284) and telephone interview (n=287).

Overall an 86\% response rate was achieved. Biographical characteristics of the nurses have been reported previously.\(^8\) Of the total sample 54\% of SN and 92\% of PN worked in general practices, 8\% of PN in Accident and Medical Clinics and 22\% of SNs worked in other community health care settings.\(^8\)

This report has been restricted to 210 PN and 21 SN because district nurses (n=49) and DNS who work in hospital outpatients clinics (n=7) do not participate in the ‘Get Checked' programme. Ethics approval was granted from the Northern Regional Ethics Committee (NTX/05/10/128).

The questionnaire contained questions about the nurse’s practice or clinic, the numbers of patients, and diabetes patients enrolled, and general participation by nurses and GPs in the DAR ‘Get Checked' programme. For example, participants were asked about their personal involvement in the programme, what percentage of the ‘Get Checked' reviews they carry out at their practice, and the support from the practice they received for the programme.
Respondents who did not participate in the programme were asked why, with the following responses categories: 'doctors carry out the Get Checked assessments'; 'lack of time'; 'lack of knowledge' and 'other'. Nurses who indicated they personally participated were also asked 'who mostly carries out the annual Get Checked diabetes assessments at your practice or service', with the following possible responses: 'doctor'; 'nurse' or 'both doctor and nurse equally'. Additional questions were asked on specific aspects carried out by the nurses from respondents who indicated 'doctors and nurses' equally carried out the review at their practice.

All 231 nurses completed the telephone interview, including the two PN who did not return the questionnaire. At the end of the interview, one day within the past week that each nurse had worked was randomly selected, and additional questions were asked about the number of diabetes patients consulted on this day, including 'Get Checked' reviews. Further patient details and information were gathered on the assessments, care and health promotional advice diabetes patients received during the consultation.

For statistical analyses, nurses were categorised by PN or SN, the latter included the DNS (n=12) and CCM (n=9) nurses who were combined for analyses due to their small numbers. Standard univariate methods were used for analysing categorical outcome data, using PROC FREQ in SAS version 9.2 (SAS Institute, Cary, North Carolina, 2008) and PROC MULTILOG in SUDAAN (version 10 Research Triangle Institute, 2008) to correct for the clustering effects by nurses who had carried out more than one diabetes 'Get Checked' review. P-values from either Fisher or Pearson exact tests were selected when over 20% of expected cell numbers were <5.

Results

Table 1 outlines the biographical details of the 229 PN and SN who completed and returned the questionnaire. Most were female and 47% were aged over 50 years, and significantly more PN were European New Zealanders compared with SN (Table 1). Information regarding the number of patients, including those with diabetes, registered at each nurse’s practice or service, was known by 80% of respondents, although only 43% were able to access the appropriate database for this information.

Of the nurses who reported the number of patients registered at their practice or service, 97% reported up to 22,000 patients. Three nurses worked in very large practices or services (30,000 to 78,000) and one SN worked for the large PHO, 'ProCare', with 800,000 patients registered (not included in analyses). More PN (40%) worked in practices with 100 to 300 diabetes patients registered; while 46% of SN worked in practices or services with over 300 diabetes patients.

Table 2 shows the proportion of SN and PN who reported participating in the 'Get Checked' reviews at their practice and the division of 'Get Checked' reviews between GPs and nurses. Of the nurses sampled, over 60% personally carry out 'Get Checked' reviews or aspects of them and in addition, 70% and 40% of SN and PN respectively carry out over 50% of all 'Get Checked' reviews at their associated general practice.

Of the 75 PN who do not participate in the 'Get Checked' reviews, 43% reported that designated nurses complete these reviews and 39% stated that only doctors carry out the reviews; while most SN stated the reviews were outside of their specialist roles.
Table 1. Biographical details of practice and specialist nurses (n=229) – sex, age, ethnicity and the number of patients registered at their practice or service

<table>
<thead>
<tr>
<th>Variable and level</th>
<th>Total</th>
<th>Type of Nurse</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Practice nurses</td>
<td>Specialist nurses</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>99</td>
<td>99</td>
<td>100</td>
</tr>
<tr>
<td>Male</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25–40</td>
<td>19</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>41–50</td>
<td>34</td>
<td>34</td>
<td>33</td>
</tr>
<tr>
<td>≥51</td>
<td>47</td>
<td>46</td>
<td>57</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ European</td>
<td>74</td>
<td>76</td>
<td>52</td>
</tr>
<tr>
<td>Asian</td>
<td>8</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Pacific Island</td>
<td>6</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>3</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Māori</td>
<td>3</td>
<td>1</td>
<td>24</td>
</tr>
<tr>
<td>*Other</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Number of diabetes patients registered at practice or clinic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of patients registered</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9–3000</td>
<td>22</td>
<td>21</td>
<td>36</td>
</tr>
<tr>
<td>3001–7000</td>
<td>43</td>
<td>44</td>
<td>27</td>
</tr>
<tr>
<td>7001–78,000*</td>
<td>35</td>
<td>35</td>
<td>36</td>
</tr>
<tr>
<td>Number of diabetes patients</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2–100</td>
<td>26</td>
<td>26</td>
<td>23</td>
</tr>
<tr>
<td>101–300</td>
<td>39</td>
<td>40</td>
<td>31</td>
</tr>
<tr>
<td>301–4200*</td>
<td>35</td>
<td>34</td>
<td>46</td>
</tr>
<tr>
<td>Source of information</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Database</td>
<td>57</td>
<td>56</td>
<td>64</td>
</tr>
<tr>
<td>Estimated</td>
<td>43</td>
<td>44</td>
<td>36</td>
</tr>
</tbody>
</table>

Abbreviations: No, number.
P-value showing significance of variation in percentages in subgroups, from the Chi-squared value and either Fisher or Pearson exact tests were selected.

*Other–Australia (n=4), North America (n=4), Europe (n=2), South Africa (n=1) and Middle East (n=1).

#Three Primary Health Care Organisations with >30,000 patients registered – one with 800,000 patients was excluded from the analyses.

Most SN and half of the 175 PN who participated in the 'Get Checked' programme reported receiving a 'lot of support' from their practice while 20% received 'little or no support' (Table 2). Reasons for the lack of support mirrored the reasons given above as to why respondents did not participate in the programme, along with the additional reason that patients were enrolled in other 'special' assessment programmes (data not shown).

Of the 186 PN whose practices were involved in 'Get Checked' reviews, 44% reported that doctors and nurses equally carry out these reviews and 38% stated that nurses mostly carry out these reviews—twice the proportion than doctors. Further, of the 89 nurses who reported that doctors and nurses working at their practice equally carry out 'Get Checked' reviews, 44% reported nurses carry out complete reviews and a large proportion weighed patients, measured blood pressure, carry out foot examinations and gave lifestyle advice (Table 2).
Of those nurses, 26 reported other activities performed by nurses that included: testing of capillary blood glucose and visual acuity; antenatal and postnatal care; writing 'Green Scripts', organised appropriate referrals, educated patients on test results and medication (including insulin), and promoted smoking cessation – separate data not shown.

Table 2. PN and SN who participate in the 'Get Checked' programme and the proportion of general practitioners and nurses who equally carried out reviews and specific activity undertaken by each (n=223)

<table>
<thead>
<tr>
<th>Variable and level</th>
<th>Total n=236</th>
<th>Type of Nurse</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Practice Nurses n=208</td>
<td>Specialist Nurses n=28</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Nurses who participate in Get Checked</td>
<td>n=223</td>
<td>n=206</td>
<td>n=17</td>
</tr>
<tr>
<td>Percentage of Get Checked carried out by nurses &lt;5%</td>
<td>n=134</td>
<td>n=124</td>
<td>n=10</td>
</tr>
<tr>
<td>5–25%</td>
<td>18</td>
<td>19</td>
<td>10</td>
</tr>
<tr>
<td>26–50%</td>
<td>19</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>&gt;50%</td>
<td>20</td>
<td>21</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>43</td>
<td>40</td>
<td>70</td>
</tr>
<tr>
<td>Support for nurses for Get Checked</td>
<td>n=188</td>
<td>n=175</td>
<td>n=13</td>
</tr>
<tr>
<td>A lot</td>
<td>53</td>
<td>51</td>
<td>85</td>
</tr>
<tr>
<td>Some</td>
<td>28</td>
<td>29</td>
<td>15</td>
</tr>
<tr>
<td>A little</td>
<td>13</td>
<td>14</td>
<td>0</td>
</tr>
<tr>
<td>None</td>
<td>6</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Reasons for not participating</td>
<td>n=79</td>
<td>n=75</td>
<td>n=4</td>
</tr>
<tr>
<td>Doctors only do Get Checked</td>
<td>37</td>
<td>39</td>
<td>0</td>
</tr>
<tr>
<td>Lack of time</td>
<td>11</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Lack of knowledge</td>
<td>6</td>
<td>7</td>
<td>0</td>
</tr>
</tbody>
</table>

General practitioners & practice-based nurses involved in Get Checked programme

| Who mostly did Get Checked?                           | n=198       | n=186         | n=12    |      |
| Both Doctor & Nurses equally (89)                 | 45          | 44            | 67      | 0.16 |
| Nurses                                            | 38          | 38            | 33      |      |
| Doctors                                          | 17          | 18            | 0       |      |
| Aspects that nurses performed when doctors & nurses equally reviewed patients | n=89        | n=81          | n=8     |      |
| Complete review                                   | 44          | 41            | 75      | 0.13 |
| Weight                                            | 88          | 89            | 75      | 0.26 |
| Blood pressure                                    | 90          | 90            | 88      | 0.59 |
| Feet checked                                      | 73          | 72            | 88      | 0.44 |
| Lifestyle advice                                  | 87          | 86            | 88      | 0.93 |

Abbreviations: Drs, doctors; Pts, patients.
P-value showing significance of variation in percentages in subgroups, from the Chi-squared value and either Fisher or Pearson exact tests were selected.

Table 3 reports on the type of consultation 79 (38%) PN and 12 (57%) SN carried out among the 196 diabetes patients consulted on the randomly selected day. The majority
of consultations were follow-up visits (61%), 26% were special programme consultations, and PN completed 80% of the total 'Get Checked' reviews (Table 3).

Table 3. Proportion of practice and specialist nurses undertaking 'Get Checked' reviews on a randomly selected day (n=230).

<table>
<thead>
<tr>
<th>Variable and level</th>
<th>Total</th>
<th>Type of Nurse</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total sample of nurses</td>
<td>n=230</td>
<td>n=91 (40%)</td>
</tr>
<tr>
<td></td>
<td>Total nurses consulting sampled patients</td>
<td>n=196</td>
<td>n=153 (78%)</td>
</tr>
<tr>
<td>Type of consultation</td>
<td>n %</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Follow-up</td>
<td>120 61</td>
<td>93 61</td>
<td>27 63</td>
</tr>
<tr>
<td>Get Checked</td>
<td>22 11</td>
<td>16 10</td>
<td>6 14</td>
</tr>
<tr>
<td>Care Plus</td>
<td>30 15</td>
<td>24 16</td>
<td>6 14</td>
</tr>
<tr>
<td>Other&lt;sup&gt;a&lt;/sup&gt;</td>
<td>24 12</td>
<td>20 13</td>
<td>4 9</td>
</tr>
<tr>
<td>Number of nurses undertaking Get Checked</td>
<td>n=18</td>
<td>n=16</td>
<td>n=2</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>1</td>
<td>5</td>
<td>10 5</td>
<td>1 4</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>5 2</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>0.5</td>
<td>1 0.5</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>0</td>
<td>1 4</td>
</tr>
<tr>
<td>Number of Get Checked consultations</td>
<td>n=30</td>
<td>n=24</td>
<td>n=6</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>1</td>
<td>5</td>
<td>7</td>
<td>1 5</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>0</td>
<td>8</td>
</tr>
</tbody>
</table>

*Data not collected on: 7 patients consulted by one PN; 2 of 3 and 1 of 2 patients consulted by two other PN; 28 patients consulted by one ophthalmology DSN and 5 patients consulted by one CCM nurse.

Table 4 compares assessments and care received by the 30 patients undergoing a 'Get Checked' review with 120 patients undergoing the usual follow-up consultation by PN and SN. Significantly more patients undergoing the 'Get Checked' review were weighed, had their blood pressure measured and received foot examinations and advice on foot protection, compared with patients attending follow-up consultations.

Of the five patients who used tobacco, only one wished to stop but was not advised on nicotine replacement therapy, and two patients were to be followed up by their GPs.
## Table 4. Assessments and care received by patients undergoing the Get Checked annual reviews compared with patients undergoing 'usual care' follow-up consultations (n=150)

<table>
<thead>
<tr>
<th>Variable and level</th>
<th>Get Checked (n=30)</th>
<th>Follow-up (n=120)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total N %</td>
<td>n %</td>
<td></td>
</tr>
<tr>
<td><strong>Nursing care &amp; activities</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure measured</td>
<td>30 100</td>
<td>120 70</td>
<td>0.0006</td>
</tr>
<tr>
<td>Patient weighed</td>
<td>30 97</td>
<td>120 58</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Feet examined</td>
<td>30 87</td>
<td>120 33</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Microfilament test</td>
<td>26 96</td>
<td>39 56</td>
<td>0.0005</td>
</tr>
<tr>
<td>Foot advice received</td>
<td>30 43</td>
<td>119 19</td>
<td>0.006</td>
</tr>
<tr>
<td>Exercise advice received</td>
<td>30 83</td>
<td>120 69</td>
<td>0.12</td>
</tr>
<tr>
<td>Green Scripts received</td>
<td>7</td>
<td>3</td>
<td>0.26*</td>
</tr>
<tr>
<td>Diet advice received</td>
<td>30 77</td>
<td>120 73</td>
<td>0.64</td>
</tr>
<tr>
<td>Capillary blood glucose test</td>
<td>30 47</td>
<td>118 46</td>
<td>0.93</td>
</tr>
<tr>
<td>Patients to be telephoned</td>
<td>30 23</td>
<td>119 33</td>
<td>0.32</td>
</tr>
<tr>
<td><strong>Patient documented information</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking status recorded</td>
<td>30 100</td>
<td>119 97</td>
<td>0.60*</td>
</tr>
<tr>
<td>Tobacco users</td>
<td>5 17</td>
<td>16 13</td>
<td>0.60*</td>
</tr>
<tr>
<td>Asked about stopping</td>
<td>1 20</td>
<td>3 19</td>
<td>0.15*</td>
</tr>
<tr>
<td>Referral for NRT</td>
<td>0</td>
<td>67</td>
<td>–</td>
</tr>
<tr>
<td>Quitline/Community service</td>
<td>40*</td>
<td>67</td>
<td>–</td>
</tr>
<tr>
<td>Medications known</td>
<td>100</td>
<td>118 99</td>
<td>1.00*</td>
</tr>
<tr>
<td>Patients prescribed:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Statin</td>
<td>30 67</td>
<td>117 68</td>
<td>0.86</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>63</td>
<td>59</td>
<td>0.66</td>
</tr>
<tr>
<td>Metformin</td>
<td>60 117</td>
<td>67</td>
<td>0.49</td>
</tr>
<tr>
<td>Insulin</td>
<td>23 118</td>
<td>24</td>
<td>0.96</td>
</tr>
<tr>
<td>Sulphonylurea</td>
<td>23 117</td>
<td>32</td>
<td>0.38</td>
</tr>
<tr>
<td>HbA1c recorded</td>
<td>30 97</td>
<td>120 80</td>
<td>0.03*</td>
</tr>
<tr>
<td>Total Cholesterol recorded</td>
<td>30 83</td>
<td>120 58</td>
<td>0.01*</td>
</tr>
<tr>
<td>BMI or height &amp; weight recorded</td>
<td>30 40</td>
<td>120 8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Patient management by practice</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular appointments</td>
<td>≤3 months since last</td>
<td>30 97</td>
<td>120 97</td>
</tr>
<tr>
<td>Blood test ≤3 months</td>
<td>30 87</td>
<td>116 84</td>
<td>0.90</td>
</tr>
<tr>
<td>Retinal Screen ≤2 years</td>
<td>82</td>
<td>72</td>
<td>–</td>
</tr>
<tr>
<td>Over 2 years</td>
<td>27 7</td>
<td>113 6</td>
<td>0.45*</td>
</tr>
<tr>
<td>Not known / no data</td>
<td>11</td>
<td>22</td>
<td>–</td>
</tr>
<tr>
<td>Microalbuminuria &lt; 3 months</td>
<td>30 80</td>
<td>119 65</td>
<td>0.48*</td>
</tr>
</tbody>
</table>

P value showing significance of variation in percentages in subgroups, from the Chi-squared value using either Fisher or Pearson* exact tests and Chi-squared test not performed with small cell numbers.

*Planned phone call to one patient by GP and discussion planned with another patient during next consultation.

### Discussion

This is a quantitative report of participation by PHC nurses in the DAR 'Get Checked' programme for a representative sample of practice-based PN and SN from the wider Auckland region and evaluates aspects of the PHC Strategy (2001). Most PN and SN reported having at least 100 diabetes patients registered at their practice or clinic with a higher median number compared with 123 general practices surveyed in UK.17
Most consultations by PN and SN were follow-up appointments; 38% of PN on average consult at least one diabetes patient a day, and 26% of all PN diabetes consultations were special programme consultations. Over 60% of the PN and SNs were involved in the 'Get Checked' programme.

Of the PN sampled, 40% carried out over 50% of all 'Get Checked' reviews at their practice, far greater than the 8% of PN who reported completing diabetes reviews at South Link-based practices in the South Island, but comparable to PN in Nottingham who participated in DAR in 46% of practices for patients with type 2 diabetes.

Of the 30 patients who had 'Get Checked' consultations on the randomly sampled day, major nursing roles included measuring blood pressures, weighing patients, undertaking comprehensive foot examinations and giving health promotional advice.

PN respondents reported that twice the proportion of nurses compared with GPs mostly carried out complete 'Get Checked' reviews, and that a further 45% did so equally with GPs, despite the historic tendency for the scope of PN to be moulded around that of GPs.

Although a quarter of all PN consultations were special programme ('Get Checked' or 'Care Plus' consultations, another NZ survey reported significantly fewer ethnic minority patients participated in 'Get Checked', particularly Māori patients, and another reported a high attendance rate, although disparities existed for those who attended related services such as retinal screening especially for Pacific Island patients.

In contrast, another survey reporting on 13,281 diabetes patients found no ethnic differences in foot screening in those who participated in 'Get Checked'. The nursing profession recognises the importance of building Maori and Pacific nursing workforce capacity as it is commonly believed that ethnic concordance will contribute to addressing ethnic minority inequity in access and treatment to PHC services.

The 'Get Checked' programme has been one of the central activities enabling PN to take a more active and semi-autonomous role in general practice over the last 10 years. The large proportion of PN involved in the 'Get Checked' programme demonstrates the success of funding reimbursements, increasing the capacity of PN providing community-based diabetes care and fulfils one of the principal aims of the PHC Strategy in attempting to provide PHC for all, as described by Finlayson et al.

Following the lead of the UK and Europe, the introduction of the Government-funded 'Get Checked' review programme provided a systematic review process, allowing for evaluation, comparison, increased patient documentation and ultimately improving patient outcomes. Improvements have been reported in mean blood pressure, cholesterol levels, and albumin:creatinine ratio, smoking rates and an increase in prescriptions for hypoglycaemic medication, ACE inhibitors and statins.

Results from this survey document the division of labour between GPs and practice-based nurses and comparisons are made with 'follow-up' consultations in general practice. Patients however, may receive care from GPs they do not receive from nurses, such as adjusting medication to improve BGLs, blood pressure and lipid profiles.
A strength of this survey was the very high response rate and representative comprehensive cross-sectional sample of practice-based PN and SN in the largest urban area in NZ. Potential bias by PN and SN over reporting on their own, and reporting GP participation and the respective division of care in the 'Get Checked' programme is a limitation of this survey.

Despite this, information gathered on actual 'Get Checked' reviews carried out by the participants on a randomly selected day that each nurse had worked, correlated with self-reported participation by PN and SN. A further limitation is the self-identification of SN as there is no national standard or criteria for this specialist nursing role, although most SN have undertaken formal tertiary post-registration education. 8

Most PN participate in the 'Get Checked' review programme and completed a large proportion of the 'Get Checked' reviews at their associated practice and felt supported. The findings from this survey show the Government-funded DAR 'Get Checked' programme has been successful in growing the capacity of PN in the community management of diabetes and they have developed collegial working relationships with GP in most practices in Auckland. Despite this, the 'Get Checked' programme has been criticised for rewarding completed consultations rather than improving patient outcomes and ended in 2012. 28

Not all aspects of the review have been comprehensively evaluated and findings from this survey show indirect benefits for patients regarding nursing-focused care (foot examinations, blood pressure measurements and health promotion advice). Despite this, tobacco use—associated with the poorest outcomes—has not been systematically targeted by nurses during 'Get Checked' consultations and this is not reported on in a recent 'Get Checked' audit review. 28

PHC based-diabetes care in NZ has followed similar international trends 4,5 with targeted funding for primary-care based DAR and now aims to tie reimbursement with improvements in patient outcomes. 29

Although the 'Get Checked' programme has ended, funding has been retained for diabetes services, through the new Diabetes Care Improvement Package, 29 and regions have been given the opportunity to re-design existing services to maximise benefits for patients such as basing reimbursement on improved clinical indicators and risk factors for diabetes-related complications. 29

DHBs need to continue to acknowledge the valuable contribution made by the largest professional PHC workforce and ensure PHC nurses are involved in developing an effective replacement DAR programme and that their workforce capacity and capability are fully realised.

In the future it is expected PHC nurses will consolidate and continue to expand their role in the community management of diabetes. Following changes in the Medicines Act of 1981 30 and the 2003 Health Practitioners Competence Assurance Act, 31 it is expected a greater proportion of PHC nurses will attain advanced roles as DNS and nurse practitioners with prescribing rights, as recently successfully piloted in four sites by DNS in NZ. 32

Further legislative changes are expected to allow fully qualified and nationally recognised DNS to prescribe the full array of medication required for managing
patients with diabetes \(^{33}\) and parallels changes occurring in the UK, US, Canada and Australia. \(^{34}\)

**Competing interests:** Nil.

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**References:**


Registered nurse assessment and treatment of skin sepsis in New Zealand schools: the development of protocols

Alison M Vogel, Diana R Lennon, Sarah Gray, Elizabeth Farrell, Philippa Anderson

Abstract

Background Skin infection is the commonest medical cause of hospitalisation in school children. Disadvantaged children, usually Māori or Pacific, have high rates of preventable diseases.

Aim To improve access to early treatment for skin infections using nurse-led school clinics in South Auckland, including provision of antibiotics under delegated standing orders.

Method Evidence-based protocols for the recognition and treatment of skin sepsis were developed following a literature search. A training package was developed for health professionals involved and outcome data were collected from a pilot study in which the protocols were trialled.

Results An algorithm for diagnosis of skin infections was adapted from Steer et al (Bull World Health Organ. 2009;87:173–9). Fusidic acid ointment was recommended as first-line treatment for localised impetigo. Twice daily oral cephalexin was recommended for extensive impetigo and cellulitis, for palatability and simplicity of dosing. Fifty-six episodes of skin infection received treatment under standing orders in the first 15 weeks of the pilot study.

Conclusion Robust evidence to determine optimal choice, dosage and duration of antibiotic therapy for skin sepsis in children is lacking. The algorithms described are consistent with available evidence and provide a pragmatic approach for use in registered nurse (RN)-led school clinics.

Disadvantaged children, usually Māori or Pacific, suffer disproportionately from preventable diseases. There are with issues with access to healthcare and uptake of preventive measures. This translates into high hospitalisation rates for infectious diseases. Skin infection is the most common medical cause of hospitalisation in school-aged children in Counties Manukau District Health Board (CMDHB) and skin infection rates are increasing in New Zealand and internationally. In 2003 New Zealand (NZ) cellulitis hospital admission rates were just under double those reported in Australia and the USA. There are significant ethnic disparities in admission rates, and these disparities have been increasing. CMDHB Māori and Pacific 5–9 year olds had hospital admission rates of 7.3 and 9.5/1000 respectively compared with 1.7/1000 for other ethnicities in 2010 (personal communication, Dean Papa, CMDHB).

There are estimated to be up to 14 times as many cases of skin infection seen in primary care as are admitted to hospital. Skin infection can be associated with serious
sequelae e.g. post-streptococcal glomerulonephritis (PSGN).\textsuperscript{10,11} The Ministry of Health (MOH) has made a commitment to reduce Acute Rheumatic Fever (ARF) rates in Māori and Pacific to Pākeha (NZ European) levels by 2020.\textsuperscript{12–14}

A recent meta-analysis has concluded that school-based clinics can reduce the incidence of ARF by 60%.\textsuperscript{15} The National Heart Foundation has published a NZ guideline for the primary prevention of ARF in which it is recommended that school-based sore throat clinics be considered when rates of ARF exceed 50/100,000 in the population.\textsuperscript{16} On this basis the Ministry of Health has funded sore throat services in schools (year 1–8) in very high risk areas.\textsuperscript{13}

Almost half of NZ rheumatic fever cases occur in CMDHB.\textsuperscript{17} A Health Research Council Feasibility Study grant was used to fund a pilot study in CMDHB to assess the acceptability and feasibility of expanding registered nurse (RN)-led school sore throat clinics to include treatment of skin infections under delegated standing orders.

In New Zealand only registered medical practitioners and dentists can prescribe prescription medicines. A standing order is a written instruction issued by a medical practitioner in accordance with regulations, authorising a specified class of health professional to supply and administer specified prescription medicines in circumstances specified in the instruction, without a prescription. A standing order does not enable a person who is not a medical practitioner or dentist to prescribe medicines—only to supply and/or administer prescription medicines.\textsuperscript{18}

The overall goal of the rollout of this programme to 53 schools in CMDHB will be to improve the health of children and reduce ethnic inequalities in the rates of hospitalisation in primary school aged children for targeted health conditions (rheumatic fever and skin infections) to national average Pākeha rates or below as measured using hospital discharge data.\textsuperscript{2}

This paper describes the process of development, and the core content, of the standing orders for recognition and treatment of the targeted skin infections—impetigo, cellulitis, scabies and infected eczema. We aimed to develop simple, safe assessment protocols for RNs working in schools, including clear guidelines for when referral was required; to provide training in differentiating skin conditions; to have simple protocols for treatment using delegated authority; to maximise treatment adherence by providing medication free to the family and to have clear plans for follow-up. The plan was to pilot the clinic in one South Auckland primary school and assess feasibility with the hope that if this was successful further rollout to other high risk schools within the DHB would occur with further independent evaluation. There was the opportunity to adapt the protocols during the pilot as required.

\textbf{Methods}

A literature search for evidence-based guidelines, meta-analyses, systematic reviews, algorithms and recent randomised controlled trials (RCTs) (2000–2010) in Ovid SP Medline was undertaken assisted by a librarian who devised the search strategy (see Appendix 1). Search questions were: What is the best treatment in children aged 0–18 years for impetigo? for non surgically-acquired cellulitis? for scabies? for infected eczema?

Keywords including cellulitis, staphylococcal skin infections, furunculosis, carbuncle, impetigo; scabies, dermatitis, atopic/or eczema, skin diseases, infectious/or skin diseases, bacterial were included. Limits of English, humans and children 0–18 years were used.
The timeframe and limited resource available meant that meta-analyses and systematic reviews were assessed, with a search for RCTs published since the reviews had been completed. The quality of the evidence was evaluated using the SIGN (Scottish Intercollegiate Guidelines Network) methodology checklists.19 Evaluation was limited to medications available in New Zealand.

Guidelines for management of skin sepsis were accessed from local hospital services (KidzFirst, Starship) and the Royal Children’s Hospital Melbourne.20–22 Clinical Knowledge summaries were reviewed. Data were obtained on local patterns of antibiotic susceptibility for Staphylococcus aureus (S. aureus).

Draft protocols and algorithms were developed, which were then reviewed by all members of the writing group, and their suggestions were incorporated into subsequent drafts. External feedback was sought from experts with experience in children’s emergency care and infectious diseases. Consensus was reached on areas of disagreement. A final draft was endorsed by Middlemore Hospital’s Medicines Advisory Committee.

A training package for the nurses implementing the pilot was developed including a credentialing process for antibiotic dispensing under standing orders. Data were collected on the numbers of children seen for skin conditions, the number confirmed to have skin infections, treatments prescribed and outcomes. This study received ethical approval from the Northern Regional Ethics Committee NTX/10/09/097.

Results

Evidence base for treatment of skin infections—Cochrane reviews were found addressing treatment for impetigo, cellulitis and scabies.23–25 All had comprehensive search strategies for relevant RCTs which clearly described inclusion and exclusion criteria and covered multiple databases. Each undertook a quality assessment of trials and used appropriate rules to determine whether trials were suitable for combining in a meta-analysis. There is no Cochrane review for infected eczema but the National Institute for Clinical Excellence (NICE) in the United Kingdom had commissioned an evidence-based guideline on the management of atopic eczema in children under age 12, published in 2007.26 This included a review of the evidence relating to management of infected eczema.

The Cochrane review of RCTs of treatment for impetigo23 found 700 papers and included 57 trials with 3533 participants, (age range 0–99 years), which studied 18 topical treatments and 20 oral antibiotics. This review found evidence that for localised impetigo topical treatment is better than oral treatment. No topical treatment was superior. There were three studies of mupirocin vs placebo and one of fusidic acid vs placebo all showing greater improvement with active drug. There were four studies (440 patients) comparing fusidic acid and mupirocin with no significant difference found (RR1.03, 95% CI 0.95, 1.11). The other comparisons of topical treatments were all single studies.

For extensive lesions this review also found insufficient evidence to indicate greater efficacy of one oral antibiotic over another.23 Oral penicillin was inferior to erythromycin and cloxacillin. Many different oral regimens have been compared including differing pairs of medications and different dosing but there are no trials of flucloxacillin compared with cephalaxin, the two antibiotics most often used in local treatment regimes in New Zealand. The data were inadequate to determine ideal length of treatment or optimal dosage schedule. There was little evidence that using disinfectant solutions improves impetigo.
Another systematic review\textsuperscript{27} using a comprehensive search strategy for RCTs published in English prior to 2002 of systemically well patients of any age with impetigo identified 359 studies, with 16 meeting inclusion criteria, 12 with reasonable quality according to Jadad score. Most studies were small—9 had fewer than 100 patients, and the largest had 160 patients with impetigo. Four studies compared topical versus oral treatment and 12 compared topical treatments. This review commented on the limited high quality evidence available and recommended the use of topical antibiotic as first-line treatment in systemically well patients with limited disease. No clear definition is given of mild versus severe disease.

The Cochrane review of treatment for cellulitis\textsuperscript{24} includes 25 trials with 2488 participants. No two trials examined the same drugs, so similar drugs were grouped together. The review concludes that it is impossible to determine the best treatment. There were three studies comparing a penicillin with a cephalosporin (total 88 people) with no difference in outcome. Six trials (538 participants) compared different generations of cephalosporin and found no difference in effect. There are insufficient data on the duration of therapy. There is only one trial which had a specified group of children included, and this compared two times daily versus four times daily cephalexin with no difference found in outcomes.\textsuperscript{28}

The risk factors for cellulitis may be quite different in adult patients who commonly have underlying diabetes or venous stasis ulcers. Compliance may also vary with age as adults will usually take tablets or capsules and young children require liquid preparations. There are well recognised issues with the palatability of flucloxacillin elixir.\textsuperscript{29}

For atopic eczema the NICE review\textsuperscript{26} found evidence from case control studies and case series demonstrating that the majority of children with atopic eczema have skin colonised with \textit{S. aureus}. Where children developed overt signs of infection \textit{S. aureus} was usually involved although streptococcal species (mostly \textit{Streptococcus pyogenes}) and mixed infections were sometimes present. Two RCTs of antibiotic treatment were found. In a within person (left-right body comparison) trial including 86 patients (26 children) a topical steroid-antibiotic combination was compared with steroid alone. All patients improved within a week, with no significant difference between groups, although patients favoured the combined treatment. A RCT of 30 children with suspected \textit{S. aureus} superinfected atopic eczema randomised children to either oral cefadroxil or placebo for 2 weeks. All children on active treatment were infection free at 2 weeks compared with 9/17 in the placebo group. The guideline recommends using topical antibiotics in children with atopic eczema only in cases of localised infection, for no longer than 2 weeks.

Systemic antibiotics active against \textit{S. aureus} and \textit{Streptococcus} are recommended for widespread bacterial infections with flucloxacillin recommended as first choice, although the issue of palatability is acknowledged as a problem. Erythromycin is recommended for penicillin allergy.\textsuperscript{26}

The Cochrane review of scabies treatment included 22 trials involving 2676 people, 19 conducted in resource poor countries.\textsuperscript{25} One trial was placebo controlled, 18 compared two or more drug treatments, three compared treatment regimens, and one compared different treatment vehicles. This review concluded that topical permethrin
is the most effective treatment for scabies. The Cochrane trials using permethrin used one or two treatments. There were no trials of malathion.

We reviewed paediatric studies of cephalixin use in skin sepsis because cephalixin can be given twice daily, is funded, is palatable, an elixir is available and it was included in local guidelines in use. The regimens used for skin sepsis in studies including children that were included in the Cochrane reviews for impetigo and cellulitis used twice daily (3 trials), three times daily (two trials) and four times daily doses (3 trials).28,30–34 Cure rates with cephalixin treatment ranged from 94–100%.28,30–34

**Guidelines**—We accessed protocols and guidelines from BPACNZ (Best Practice Advocacy Centre) which provides guidance to GPs,35,36 KidzFirst (CMDHB)20, Starship,21 the Royal Children’s Hospital Melbourne,22 and the Infectious Disease Society of America (IDSA)37 which were in current use to compare antibiotic recommendations (Table 1).

| Table 1. Recommendations for oral treatment of skin sepsis or cellulitis (2010) |
|-------------------------------|-----------------------------|----------------------|-----------------|-----------------|
| **Treatment**                 | **BPAC**                    | **KidzFirst**        | **Starship**    | **Royal Children’s, Melbourne** | **IDSA** |
| First line                    | Flucloxacillin              | Cephalexin           | Flucloxacillin  | Flucloxacillin   | Dicloxacillin or cephalixin or erythromycin or clindamycin or amoxicillin-clavulanate |
| Second line                   | Amoxicillin-clavulanate or flucloxacillin | Amoxicillin-clavulanate (if unable to swallow capsules) | Cephalexin elixir | |

The Starship and BPAC guidelines have recently been updated and cephalixin is recommended for use in children as a first-line treatment now that it is fully funded by Pharmac.38,39 The Infectious Diseases Society of America Consensus recommendations recommend 5 days of treatment as adequate for uncomplicated cellulitis.37

**Antibiotic sensitivity patterns**—Environmental Science and Research (ESR) 2008 NZ data shows susceptibility of *S. aureus* to mupiricon of 91–93% % compared with 86–88% for fusidic acid.40 Auckland 2009 community data from diagnostic lab found *S. aureus* is sensitive 87–90% of the time to mupirocin and 73–79% to fusidic acid.41 CMDHB 2009 blood isolates of *S.aureus* found 91% were sensitive to mupirocin compared with 74% to fusidic acid. CMDHB wound swab sensitivities are shown in Table 2 (Personal communication, Susan Taylor,2011).
Table 2. Sensitivity of wounds swabs, Counties Manukau DHB 2010

<table>
<thead>
<tr>
<th>Antimicrobial agent</th>
<th>MSSA</th>
<th>MRSA</th>
<th>All S. aureus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penicillin/amoxicillin</td>
<td>10</td>
<td>R</td>
<td>9</td>
</tr>
<tr>
<td>Flucloxicillin</td>
<td>S</td>
<td>R*</td>
<td>82</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>93</td>
<td>76</td>
<td>90</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>93</td>
<td>83</td>
<td>91</td>
</tr>
<tr>
<td>Co-trimoxazole</td>
<td>95</td>
<td>98</td>
<td>96</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>99</td>
<td>98</td>
<td>99</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>99</td>
<td>97</td>
<td>99</td>
</tr>
<tr>
<td>Fusidic acid</td>
<td>75</td>
<td>62</td>
<td>73</td>
</tr>
<tr>
<td>Mupirocin</td>
<td>88</td>
<td>93</td>
<td>89</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Number tested</td>
<td>1609</td>
<td>351</td>
<td>1960</td>
</tr>
</tbody>
</table>

MSSA: methicillin-sensitive *Staphylococcus aureus*; MRSA: methicillin-resistant *Staphylococcus aureus*; S=predictably sensitive, R=predictably resistant; 43% of MRSA are resistant to beta lactams only.

Protocol development—A practical algorithm was developed by Steer et al to assist nurse and allied health workers in the assessment and treatment of skin conditions. This was trialled in Fiji. The diagnosis by two trained nurses was found to be highly sensitive in diagnosing any skin problem (98.7%) compared with paediatrician diagnosis, but suboptimal in differentiating between infected and non-infected scabies. We utilised this algorithm as the basis of our diagnostic recommendations, but recommended children with suspicion of fungal infections should be seen by the GP as these were not within the scope of the project as they are less likely to rapidly progress in severity (Table 3).

Table 3. Assessment tool for diagnosis of skin infections*

<table>
<thead>
<tr>
<th>Sign</th>
<th>Diagnosis</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any general danger sign</td>
<td>Very severe skin infection</td>
<td>Refer for hospital review</td>
</tr>
<tr>
<td>Extensive warm redness or swelling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swelling or redness around eyes</td>
<td>Periorbital cellulitis</td>
<td>Refer for hospital review</td>
</tr>
<tr>
<td>Localised warm tender swelling and redness</td>
<td>Cellulitis</td>
<td>Give oral antibiotic, review in 24 hours</td>
</tr>
<tr>
<td>Discrete sores/lesions with pus or crusts</td>
<td>Impetigo</td>
<td>Give topical antibiotic or oral antibiotic if extensive Follow-up in 5 days</td>
</tr>
<tr>
<td>Itchiness and papules</td>
<td>Scabies</td>
<td>Topical permethrin lotion</td>
</tr>
<tr>
<td>Round to oval flat scaly patches, often itchy</td>
<td>Fungal infection</td>
<td>Refer to GP for medical treatment</td>
</tr>
</tbody>
</table>

Antibiotic choice: The meta-analyses showed equivalent efficacy for fusidic acid and mupirocin. Although the laboratory data showed somewhat better sensitivity to mupirocin, fusidic acid is currently fully funded so this was chosen as the treatment of choice for localised impetigo. Previously mupirocin was available OTC (over the counter) but a change was instigated after S.aureus resistance increased.\textsuperscript{42}

Parents were directed to apply the fusidic acid cream three times a day until sores had healed or for up to 10 days. If the infection persisted unchanged at 5 days and compliance was confirmed then oral treatment was to be considered. If infection persisted on oral treatment then the situation was to be discussed with the delegating medical officer with consideration for referral to the general practitioner.

Where impetigo was extensive (covering >5% body surface area) then oral treatment was recommended.

Flucloxacillin elixir is especially unpalatable and tablets are not tolerated in many primary school children. For extensive impetigo and/or cellulitis we recommended the use of twice daily cephalxin, with flucloxacillin an option if the child was able to take tablets and erythromycin if allergic to cephalxin and flucloxacillin.

Cephalexin was the first choice for antibiotic because the syrup is much more palatable than flucloxacillin, and it can be taken twice daily. Cephalexin oral suspension and 500mg capsules are fully funded.

Recommended doses for cephalxin were 125mg bd <20kg, 250mg bd 20– < 30kg and 500mg bd for children >30kg for 5 days. Medsafe recommendations include twice daily dosing for children with skin and soft tissue infections, with an adult dose of 500mg twice daily. The dose was based on the Medsafe recommendations.\textsuperscript{43}

Children with cellulitis were followed up at 24 hours to assess redness and swelling (improved, worsened or same) and determine whether they were becoming systemically unwell. If they were deteriorating they were referred for medical review. Treatment was to be reviewed at 5 days. If swelling or redness persisted antibiotic treatment was to continue for a further 5 days.

There are 3 treatments for scabies available in NZ: gamma benzene hexachloride cream 1%, permethrin, and malathion. On the basis of the Cochrane review which concluded that topical permethrin is the most effective treatment for scabies, the protocol was for one treatment with permethrin lotion with review at 2 weeks.

### Implementation

Implementation in the pilot school was evaluated and results published.\textsuperscript{44} Over a 15-week period 98 children were referred for skin condition assessment. Of these 76 had one or more skin infections diagnosed. The PHN supplied medication for skin infections to 56 students.

### Discussion

Skin infections are highly prevalent among school age children and are associated with an increasing rate of potentially avoidable hospital admissions in New Zealand and internationally.\textsuperscript{4-8}

Despite the prevalence of these conditions it is not possible to develop robust evidence-based protocols and guidelines for treatment because of the lack of appropriate studies. Studies include mixed populations-few have groups limited to
children, and some required swabs positive for specific organisms as an inclusion criteria for study.

Many studies are funded by industry and are assessing new medications not those which are already available and cheap. Multiple small studies of different treatments have been undertaken but unfortunately studies directly comparing treatments that we commonly use e.g. flucloxacillin and cephalaxin are not available. Studies use variable dosing, frequency and durations of treatment. Many were conducted up to 20–30 years ago and resistance patterns are likely to have changed.

Our protocols considered the evidence available from systematic reviews, notably the Cochrane meta-analyses; local patterns of antibiotic sensitivity; palatability; simplicity of administration; local availability; and funding of medications.

We were subject to time and financial constraints which meant that we were unable to undertake a comprehensive guideline development process such as that used by the NZ Guidelines Group. Our protocols were pragmatic and were developed to be used in an environment where close monitoring of outcomes was possible so that concerns about failure to improve on antibiotics could be addressed by modifications of the protocol if necessary.

There are a number of research questions remaining—what are the best topical and oral treatments in our current environment, in what dose, how frequently and for how long? We need studies specific to children which consider the factors that will affect adherence including taste.

**Conclusion**

We were able to produce a pragmatic diagnostic algorithm which nursing staff used successfully in a pilot study to assess children with skin infections. Although robust evidence to support specific antibiotic regimens for targeted skin infections was lacking we developed practical standing orders for treatment based on literature review and taking into account other important factors, such as funding and palatability.

RN-led school health clinics targeting sore throats and skin infections are now in the process of being rolled out in 53 schools in the CMDHB region in conjunction with rheumatic fever prevention initiatives. Outcome evaluation, which will include an analysis of the effectiveness of our guidelines, is planned.

**Competing interests:** None identified.

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**References:**


Appendix 1. Search strategy

1. Cellulitis/or esp tinea/or tinea versicolor/or staphylococcal skin infections/or furunculosis/or carbuncle/or impetigo
2. Scabies
3. Dermatitis, atopic/or eczema/
4. 1 or 2
5. Skin diseases, infectious/or skin diseases, bacterial/
6. 3 and 5
7. infect$.mp.[mp=title, abstract, original title, name of substance word, subject heading word, protocol supplementary concept, rare disease supplementary concept, unique identifier]
8. 3 and 7
9. 6 or 8
10. 4 or 9
11. Limit 10 to “all child (0-18 years)"
12. Limit 11 to “therapy (specificity)"
13. Limit 11 to meta analysis
14. Limit 11 to systematic reviews
15. 13 or 14
16. Limit 11 to (consensus development conference or consensus development conference, NIH or guideline or practice guideline)
17. Limit 11 to “review articles”
18. Limit 17 to yr="2000-2010"
19. *cellulitis/or exp *tinea/or tinea versicolor/or *staphylococcal skin infections/or *furunculosis/ or carbuncle/ or *impetigo/ or *Scabies/ or (*dermatitis, atopic/ or eczema/)
20. 18 and 19
21. Limit 20 to English language
22. 12 and 19
23. Impetigo.mp. [mp=title,abstract, original title, name of substance word, subject heading word, protocol supplementary concept, rare disease supplementary concept, unique identifier]
24. Limit 23 to English language
25. Limit 24 to “all child (0–18 years)”
26. Limit 25 to yr = “2010”
Malignant hypertension: a preventable emergency
Walter van der Merwe, Veronica van der Merwe

Abstract
The Waitemata Hypertension Clinic Database 2009–2012 (Auckland, New Zealand) was searched for patients meeting the definition of Malignant Hypertension. Eighteen of 565 patients met the criteria. All patients had essential hypertension which was either undiagnosed, untreated or undertreated. Most cases responded satisfactorily to standard drug therapy, but a number were left with significant chronic kidney disease. Malignant hypertension is a life-threatening disease which should be entirely preventable with regular blood pressure checks in primary care.

Malignant hypertension is defined as very high blood pressure (usually >180/110mmHg) presenting in conjunction with evidence of rapidly progressive target organ damage, of which the most common evidence is retinal haemorrhages and exudates.\(^1\)

Pathophysiologically this represents a failure of the auroregulation seen in mild and moderate hypertension where arterial and arteriolar vasoconstriction maintains tissue perfusion at a relatively constant level and prevents the increase in pressure from being transmitted to the smaller, more distal vessels.

In malignant hypertension this autoregulation fails and rise in pressure in the arterioles and capillaries leads to damage to the vascular wall. Disruption to the endothelium then allows plasma constituents to enter the vascular wall causing narrowing or obliteration of the vascular lumen.

In the brain breakthrough vasodilatation from failure of autoregulation leads to cerebral oedema and the clinical picture of hypertensive encephalopathy. Malignant hypertension is a life-threatening emergency which can result in significant long-term sequelae, particularly chronic kidney disease, in survivors.\(^2\)

Malignant hypertension has been reported to be most common in patients with longstanding hypertension, many of whom have discontinued antihypertensive therapy.\(^3\)

Prior to the advent of effective antihypertensive therapy, malignant hypertension was both common and frequently fatal.\(^4\) Even in the modern era an in-hospital mortality rate of 2.65% is reported.\(^5\)

With universal access to medical care, and the availability of effective and well-tolerated antihypertensive drugs, malignant hypertension should be very uncommon in New Zealand.

In this article we determine the circumstances of patients presenting with malignant hypertension presenting in the modern era, and in particular whether or not the majority of cases were likely to have been preventable.
Method

The Waitemata Hypertension Clinic was established in March 2009 and patients with difficult or resistant are referred by GPs or hospital specialists. Referred patients are seen initially for full assessment by a consultant (WvdM) or (supervised) registrar.

At the initial appointment further investigations may be requested, and generally antihypertensive medication is initiated or adjusted. Follow-up visits are usually at hypertension nurse-specialist titration clinics (VvdM) where accurate electronic blood pressures are recorded and medications adjusted according to clinic algorithms.

These algorithms are based on the current guideline of the British Hypertension Society, adapted for drugs which are easily available in New Zealand. They also receive education about medical aspects of hypertension, lifestyle adjustment, and medication side-effects.

Patients are seen usually at monthly intervals until blood pressure is at target, or as close to target as can be achieved, and then are referred back to their general practitioners. On average, patients require three clinic visits prior to discharge.

All patient data is captured in an Access relational database (Microsoft Corporation, Seattle WA, USA). Patients who presented with an episode of very high blood pressure in conjunction with acute or rapidly progressive target organ damage are recorded in the database as malignant hypertension. The database was searched for that diagnosis. Renal function was calculated by the MDRD formula using serum creatinine, age, and gender.

Results

In 3.75 years (March 2009–October 2012 inclusive), 565 new patients were seen through the clinic, and of these 18 were entered as malignant hypertension (3.2%). They were either referred to the clinic following a hospital admission with malignant hypertension, or seen by the author (WvdM) as an inpatient consult, and subsequently followed at the clinic.

The demographic characteristics of the patients are listed in Table 1. Mean age at presentation was 52 years. All patients required hospital admission for their episode of malignant hypertension and were subsequently followed at the clinic until blood pressure was optimised and they were otherwise clinically stable. Those with severe chronic renal impairment receive ongoing follow-up.

Table 1. Demographic details

<table>
<thead>
<tr>
<th>Gender</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>13</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Mean (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>52 (22–86)</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Ethnicity</th>
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</thead>
<tbody>
<tr>
<td>European</td>
<td>16</td>
</tr>
<tr>
<td>Māori</td>
<td>1</td>
</tr>
<tr>
<td>Mixed race</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BMI</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30</td>
</tr>
</tbody>
</table>

The commonest modes of presentation were headaches with visual symptoms, and acute stroke. One patient presented with an aortic dissection (see Table 2). Mean blood pressure at presentation was 219/125mmHg (see Figure 1) and on further evaluation, acute kidney injury was common. Only three patients required parenteral antihypertensives in the acute phase and the rest were successfully managed with oral medication.
Table 2. “Malignant” features, comorbidities and renal function

<table>
<thead>
<tr>
<th>Pt</th>
<th>“Malignant” features</th>
<th>Comorbidities</th>
<th>Underlying cause</th>
<th>eGFR ml/min (start)</th>
<th>eGFR ml/min (end)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Retinopathy, AKI</td>
<td>Nil</td>
<td>Essential hypertension</td>
<td>21</td>
<td>33</td>
</tr>
<tr>
<td>2</td>
<td>Headache, Stroke (lacunar infarct)</td>
<td>Nil</td>
<td>Essential hypertension</td>
<td>43</td>
<td>60</td>
</tr>
<tr>
<td>3</td>
<td>Loss of consciousness, Stroke</td>
<td>Smoker, CKD 3</td>
<td>Essential hypertension</td>
<td>36</td>
<td>40</td>
</tr>
<tr>
<td>4</td>
<td>Stroke</td>
<td>Multi-infarct dementia, smoker</td>
<td>Essential hypertension</td>
<td>60</td>
<td>59</td>
</tr>
<tr>
<td>5</td>
<td>Headache, Retinopathy, LVH/LVF, AKI</td>
<td>Nil</td>
<td>Essential hypertension</td>
<td>35</td>
<td>46</td>
</tr>
<tr>
<td>6</td>
<td>Headache, AKI, Retinopathy</td>
<td>Nil</td>
<td>Essential hypertension</td>
<td>66</td>
<td>74</td>
</tr>
<tr>
<td>7</td>
<td>Headache, Retinopathy</td>
<td>Past nephrectomy for reflux nephropathy, type 2 diabetes</td>
<td>Essential hypertension</td>
<td>55</td>
<td>64</td>
</tr>
<tr>
<td>8</td>
<td>Headache, AKI</td>
<td>Depression</td>
<td>Essential hypertension</td>
<td>29</td>
<td>50</td>
</tr>
<tr>
<td>9</td>
<td>Retinopathy, AKI</td>
<td>Alcoholism, depression</td>
<td>Essential hypertension</td>
<td>21</td>
<td>34</td>
</tr>
<tr>
<td>10</td>
<td>Headache, Visual impairment, Retinopathy</td>
<td>Coronary artery disease</td>
<td>Essential hypertension</td>
<td>70</td>
<td>70</td>
</tr>
<tr>
<td>11</td>
<td>Headache, Neurological symptoms, Acute lacunar infarct</td>
<td>Previous strokes, depression, alcohol abuse</td>
<td>Essential hypertension</td>
<td>79</td>
<td>79</td>
</tr>
<tr>
<td>12</td>
<td>Severe retinopathy, AKI</td>
<td>CKD 4</td>
<td>Essential hypertension</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>13</td>
<td>Headache, Retinopathy</td>
<td>Smoker, CKD3</td>
<td>Essential hypertension</td>
<td>37</td>
<td>43</td>
</tr>
<tr>
<td>14</td>
<td>Proteinuria, Severe LVH and LV impairment</td>
<td>CKD 3, obesity</td>
<td>Essential hypertension</td>
<td>49</td>
<td>56</td>
</tr>
<tr>
<td>15</td>
<td>Cerebral haemorrhage</td>
<td>Previous stroke</td>
<td>Essential hypertension</td>
<td>41</td>
<td>106</td>
</tr>
<tr>
<td>16</td>
<td>Ischaemic optic neuropathy</td>
<td>Previous TIA, CKD 3</td>
<td>Essential hypertension</td>
<td>52</td>
<td>47</td>
</tr>
<tr>
<td>17</td>
<td>Type B thoraco-abdominal aortic dissection</td>
<td>Nil</td>
<td>Essential hypertension</td>
<td>43</td>
<td>71</td>
</tr>
<tr>
<td>18</td>
<td>Headache, AKI</td>
<td>Nil</td>
<td>Essential hypertension</td>
<td>40</td>
<td>71</td>
</tr>
</tbody>
</table>

Pt=patient.
Of the 18 patients, only 6 had been on prescribed antihypertensive therapy in the recent past, but one of these had clearly been non-adherent. An additional 4 had been commenced on therapy in the past but had stopped it for one reason or another.

All patients were thoroughly evaluated and investigated for secondary causes of hypertension, including primary renal disease, phaeochromocytoma, primary aldosteronism and sleep apnoea. Three (patients 3, 12 and 18) had renal biopsies to exclude a primary glomerulonephritis.

In no patient was a secondary cause of hypertension identified, and the three renal biopsies showed hypertensive changes only. In all the additional patients with renal impairment, it was judged to be secondary to hypertension (Table 3).

### Table 3. Antihypertensive medications

<table>
<thead>
<tr>
<th>Pt</th>
<th>On BP drugs at presentation</th>
<th>IV drugs used</th>
<th>Number of drugs</th>
<th>Discharge drug details</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No (previous)</td>
<td>No</td>
<td>4</td>
<td>Frusemide, Metoprolol, Cilazapril, Felodipine</td>
</tr>
<tr>
<td>2</td>
<td>No</td>
<td>No</td>
<td>2</td>
<td>Cilazapril, Amlodipine</td>
</tr>
<tr>
<td>3</td>
<td>No</td>
<td>No</td>
<td>3</td>
<td>HCTZ, Cilazapril, Amlodipine</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>No</td>
<td>4</td>
<td>(Had RSDN) Cilazapril, Chlorthaldione, Doxazosin, Felodipine</td>
</tr>
<tr>
<td>5</td>
<td>No (previous)</td>
<td>Yes – Labetolol</td>
<td>5</td>
<td>Lisinopril, Amlodipine, Frusemide, Atenolol, Doxazosin</td>
</tr>
<tr>
<td>6</td>
<td>No</td>
<td>No</td>
<td>1</td>
<td>Candesartan</td>
</tr>
<tr>
<td>7</td>
<td>Prescribed but non-adherent</td>
<td>No</td>
<td>7</td>
<td>Felodipine, Cilazapril, Chlorthaldione, Metoprolol, Spironolactone, Clonidine, Candesartan</td>
</tr>
<tr>
<td>8</td>
<td>No</td>
<td>No</td>
<td>4</td>
<td>Amlodipine, Cilazapril, Spironolactone, Chlorthaldione</td>
</tr>
<tr>
<td>9</td>
<td>No</td>
<td>No</td>
<td>3</td>
<td>Labetolol, Amlodipine, Cilazapril</td>
</tr>
<tr>
<td>10</td>
<td>No</td>
<td>No</td>
<td>2</td>
<td>Cilazapril, Metoprolol, (?Spironolactone)</td>
</tr>
<tr>
<td>11</td>
<td>Yes</td>
<td>No</td>
<td>4</td>
<td>Felodipine, Cilazapril, Metoprolol, Chlorthaldione</td>
</tr>
<tr>
<td>12</td>
<td>No</td>
<td>No</td>
<td>4</td>
<td>Amlodipine, Labetolol, Frusemide, Lisinopril</td>
</tr>
<tr>
<td>13</td>
<td>Yes</td>
<td>No</td>
<td>6</td>
<td>Amlodipine, Candesartan, Spironolactone, Bendrofluazide, Atenolol, Doxazosin</td>
</tr>
<tr>
<td>14</td>
<td>No (previous)</td>
<td>No</td>
<td>4</td>
<td>Lisinopril, Amlodipine, Carvedilol, Chlorthaldione</td>
</tr>
<tr>
<td>15</td>
<td>Yes</td>
<td>Yes – Hydralazine and Labetolol</td>
<td>3</td>
<td>Lisinopril, Amlodipine, Indapamide</td>
</tr>
<tr>
<td>16</td>
<td>Yes</td>
<td>No</td>
<td>4</td>
<td>Cilazapril, Metoprolol, Felodipine, Doxazosin</td>
</tr>
<tr>
<td>17</td>
<td>No</td>
<td>Yes – GTN and Labetolol</td>
<td>4</td>
<td>Candesartan, Felodipine, Metoprolol, Doxazosin</td>
</tr>
<tr>
<td>18</td>
<td>No (previous)</td>
<td>No</td>
<td>4</td>
<td>Cilazapril, Felodipine, Carvedilol, Spironolactone</td>
</tr>
</tbody>
</table>

Mean 3.78

Pt=patient.
In 17 of the 18 patients blood pressure responded fairly simply to standard antihypertensive therapy and of these 17, 15 achieved blood pressures <140/90mmHg. Patient 10 had a BP 164/70mmHg on two drugs, and had a third drug added, but moved out of area so we have no follow-up data on her.
Patient 16 was an octogenarian and a final blood pressure of 148/70mmHg was judged satisfactory. Patient 4 had truly refractory hypertension with blood pressures persistently around 180/110mmHg on 7 drugs. She was eventually referred for renal sympathetic denervation (RSDN) and has ended up with a BP averaging 140/80mmHg on four drugs.

Mean number of drugs at discharge was 3.78 (1–7). All patients received an ACE-inhibitor or angiotensin receptor blocker, 16/18 a DHP calcium channel blocker, 12/18 a beta blocker or combined alpha-beta blocker, 8/18 a thiazide diuretic, 5/18 an alpha blocker, 4/18 spironolactone, and 3/18 frusemide. Average eGFR at presentation was 44ml/min and in 14/18 patients it was <60ml/min. Average final GFR was 57ml/min and in 10 patients it was <60ml/min.

**Discussion**

Malignant hypertension is an uncommon but life-threatening complication of hypertension. Among our 18 cases, all were based on essential hypertension in otherwise healthy individuals, which was either undetected, untreated, or inadequately treated. The majority responded satisfactorily to standard antihypertensive drug therapy.

Most had a degree of acute kidney injury at presentation, which improved significantly with treatment. Nevertheless the majority were left with significant chronic kidney disease which will certainly affect their long-term cardiovascular risk and life expectancy. Two or three are likely to eventually require renal replacement therapy.

The malignant phase of hypertension could likely have been prevented in all 18 patients had their hypertension been detected and/or adequately treated. These cases are a reminder that an annual blood pressure check should be mandatory in all individuals, including young and middle-aged men who may otherwise present to a doctor very infrequently.

It is also a reminder the in treated hypertensive patients, achievement of target blood pressure is important, and that referral to a hypertension specialist should occur in individuals who are resistant to, or intolerant of, standard therapies.

**Competing interests:** None identified.

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**References:**


Can we improve the prevention and detection of congenital abnormalities? An audit of early pregnancy care in New Zealand

Nicola Arroll, Lynn Sadler, Peter Stone, Vicki Masson, Cindy Farquhar

Abstract

Aim To determine whether there were “quality gaps” in the provision of care during pregnancies that resulted in a perinatal death due to congenital abnormality.

Method Perinatal deaths from congenital cardiovascular, central nervous system or chromosomal abnormality in 2010 were identified retrospectively. Data were extracted by retrospective clinical note review and obtained by independent review of ultrasound scans.

Results There were 137 perinatal deaths due to a congenital cardiovascular (35), central nervous system (29) or chromosomal abnormality (73). First contact with a health professional during pregnancy was predominantly with a general practitioner. First contact occurred within 14 weeks in 85% of pregnancies and there was often a significant delay before booking. Folate supplements were taken by 7% pre-conceptually and 54% of women in the antenatal period. There were 20 perinatal deaths from neural tube defects that could potentially have been prevented through the use of pre-conceptual folate. Antenatal screening was offered to 75% of the women who presented prior to 20 weeks and 84% of these undertook at least one of the available antenatal screening tests. Review of ultrasound images found five abnormalities could have been detected earlier.

Conclusion Delay in booking or failure to offer screening early were the most common reasons for delay in diagnosis of screen detectable abnormalities. The preventative value and timing of (pre-conceptual) folate needs emphasis.

There were 704 perinatal deaths in New Zealand in 2010, and 30% of these perinatal deaths were due to congenital abnormalities. Congenital abnormality is the most common cause of perinatal death in New Zealand, and therefore a review of the care received during pregnancy was designed to identify areas for improvement.

It was hypothesised that a number of congenital abnormalities could be prevented if folate was taken prenatally. It was also hypothesised that a number of chromosomal abnormalities and neural tube defects could be detected earlier through current regimens of antenatal screening in the first or second trimester.

Antenatal screening has been an accepted part of antenatal care in New Zealand since 1968. In February 2010 the National Screening Unit introduced a new guideline for routine antenatal screening, to be offered to all pregnant women, which includes a nuchal translucency scan and blood test (levels of plasma protein-A and beta human chorionic gonadotrophin) between 11 and 13 weeks gestation.
If women are unable to access the first trimester screening, a second trimester blood test (levels of beta human chorionic gonadotrophin, alpha fetoprotein, unconjugated oestriol and inhibin A) between 14 and 20 weeks is also available.

The first and second trimester screening combination of tests is calibrated to identify an increased risk of Trisomy 21, with a lower sensitivity to identify an increased risk of other trisomies and chromosomal abnormalities (trisomy 13, 18, triploidy, Turner’s and Klinefelter’s syndromes).

Second trimester serum screening will also identify an increased risk of open neural tube defects (anencephaly, acrania, spina bifida and encephalocele). All women are offered a fetal anatomy scan at around 20 weeks gestation to check for fetal abnormalities.

Antenatal screening and ultrasound are key tools for identifying congenital abnormalities early in pregnancy. Early identification gives parents a greater number of options for treatment, and may reduce the number of late terminations of pregnancy (after 20 weeks gestation).

Late terminations are associated with increased risks to the mother, greater maternal distress and additional requirements for statutory registration of death.²

Method

Perinatal deaths resulting from cardiovascular system, central nervous system or chromosomal congenital abnormality during 2010 were identified from the Perinatal and Maternal Mortality Review Committee (PMMRC) dataset.

The PMMRC dataset of perinatal deaths is a compilation of data submitted by Lead Maternity Carers (LMCs), clinicians, PMMRC District Health Board (DHB) local coordinators, death notifications and some additional data from births deaths and marriages (BDM).

The perinatal deaths included in the audit occurred between 1 January and 31 December 2010. For fetal deaths, the date of birth is used as ‘date of death’. Only fetuses and babies who died from 20 weeks gestation up to 27 days after birth are included in this audit. This means that a significant number of fetuses are not included in this audit as the pregnancy would have ended prior to 20 weeks and were therefore not within the scope of the PMMRC.

The classification system of cause of death that has been adopted by the PMMRC is the Perinatal Society of Australia and New Zealand (PSANZ) system of classification (PDC).³

Perinatal deaths included in this audit were classified as central nervous system (PDC 1.1), cardiovascular (PDC 1.2) or chromosomal abnormalities (PDC 1.5). These sub-classifications of congenital abnormalities are the most likely to be detected by first and second trimester antenatal screening. The hospital notes, Lead Maternity Carer (LMC) notes and General Practitioner (GP) notes were requested where applicable for each of the pregnancies included in the audit.

An audit tool was developed to gather key demographic data and information on pregnancy care. The notes for each of the women were reviewed and the data points for each woman entered into an Excel spread sheet.

A review of ultrasound images was undertaken. Using the ultrasound reports included in the women’s notes we were able to identify women who had scans between 10 weeks and the gestation at which the abnormality was detected.

Ten weeks was used as the cut off as it is the lower limit at which most congenital abnormalities can be identified on ultrasound. Women who had a scan after 10 weeks which was reported as normal were included in the ultrasound audit [n=82/137 (60%)].

Static images for the ultrasounds were reviewed by one specialist with expertise in ultrasound and maternal fetal medicine using the Picture Archiving and Communication System (PACS) or in a DVD format. The reviewer was aware that the pregnancy had ended in a perinatal death due to congenital
abnormality; however the type of abnormality was not known. None of the ultrasound scans that were reviewed had been reported by the reviewer.

**Results**

137 of the 211 perinatal deaths from congenital abnormality reported to the PMMRC were identified and confirmed as cardiovascular system (26%), central nervous system (21%) or chromosomal abnormalities (53%).

Of the 137 women included in the audit, six sets of LMC clinical notes were not available as they had either been given to the woman or the midwife had left the country. In three cases there was no specific reason for the midwife not to have retained a copy of the notes.

Table 1 shows the maternal demographic data by perinatal death classification. Median BMI was higher for women with a baby with a central nervous system abnormality compared to women with other congenital abnormalities but this difference was not statistically significant (p=0.13).

**Table 1. Maternal demographic data**

<table>
<thead>
<tr>
<th>Perinatal death classification (PSANZ PDG)</th>
<th>Central nervous system 1.1</th>
<th>Cardiovascular system 1.2</th>
<th>Chromosomal abnormalities 1.5</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=35</td>
<td>n=29</td>
<td>n=73</td>
<td>n=137</td>
</tr>
<tr>
<td>Priotised ethnicity</td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Maori</td>
<td>8</td>
<td>22.9</td>
<td>5</td>
<td>17.2</td>
</tr>
<tr>
<td>Pacific Peoples</td>
<td>9</td>
<td>25.7</td>
<td>1</td>
<td>3.4</td>
</tr>
<tr>
<td>Indian</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Other Asian</td>
<td>1</td>
<td>2.9</td>
<td>7</td>
<td>24.1</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>2.9</td>
<td>5</td>
<td>17.2</td>
</tr>
<tr>
<td>NZ European</td>
<td>16</td>
<td>45.7</td>
<td>11</td>
<td>37.9</td>
</tr>
<tr>
<td>Mother's age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>4</td>
<td>11.4</td>
<td>2</td>
<td>6.9</td>
</tr>
<tr>
<td>20-24</td>
<td>12</td>
<td>34.3</td>
<td>10</td>
<td>34.5</td>
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<tr>
<td>25-29</td>
<td>8</td>
<td>22.9</td>
<td>5</td>
<td>17.2</td>
</tr>
<tr>
<td>30-34</td>
<td>5</td>
<td>14.3</td>
<td>9</td>
<td>31.0</td>
</tr>
<tr>
<td>35-39</td>
<td>5</td>
<td>14.3</td>
<td>3</td>
<td>10.3</td>
</tr>
<tr>
<td>40+</td>
<td>1</td>
<td>2.9</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>BMI (median (IQR))</td>
<td>29.4 (21.7-34.6)</td>
<td>23.1 (20.5-28.7)</td>
<td>24.3 (21.9-27.4)</td>
<td>24.9 (21.6-31.2)</td>
</tr>
</tbody>
</table>

Folate supplements were reported to have been taken by only 7% of women pre-conceptually and 54% of women antenatally. Of the 21 whose babies were diagnosed with neural tube defects (congenital abnormalities amenable to folate use) only one out of 21 was recorded as taking pre-conceptual folate and 13 out of 21 (62%) antenatal folate.

A majority of women [93 (68%)] were first seen by their GP during pregnancy, while 30 women (22%) had their first contact with a self-employed LMC, eight were first
seen in hospital or by a school nurse and first contact was unknown for seven of the women.

Figure 1 shows the association between gestation in weeks at which the mother was first seen by a health professional on the x axis compared to the gestation in weeks when she booked with a LMC. Dashed lines are shown at 10 weeks when booking is advised and solid lines at 14 weeks which is the final gestation for first trimester screening.

Overall 114 women (83%) were seen by a health care provider before 14 weeks, while only 90 women (66%) booked with a LMC before 14 weeks.

Figure 1. Scatter plot of gestation at first health professional visit and gestation at booking with LMC among women whose babies died of PDC 1.1, PDC 1.2 and PDC 1.5 in New Zealand 2010

Figure 2 shows an overview of the gestation at first contact and screening history of the study population. First and second trimester screening are reported together as only 17 women had second trimester serum screening and some of these women also had a nuchal translucency in first trimester.

Of the 82 who took up the offer of first/second trimester screening 27 had a nuchal translucency scan alone, 38 had combined first trimester screening, 13 had a nuchal translucency scan and second trimester bloods, and four had second trimester bloods alone.
Figure 2. Outcomes of first and second trimester nuchal translucency and serum screening among perinatal related deaths from central nervous system, (PDC 1.1), cardiovascular system (PDC 1.2) and chromosomal (PDC 1.5) congenital abnormalities in New Zealand 2010

* Includes 6 cases of spina bifida who did NOT have second trimester serum testing so would not have been detected.

There were eight women who had a nuchal translucency scan but did not have a first trimester blood test done so no risk was reported (2010 guidelines prevent ultrasound providers from reporting the risk based on the nuchal translucency measurement alone).

Of these eight women, two had second trimester blood tests; low risk in one case and an increased risk result in the other case. The increased risk pregnancy was found to be Trisomy 21 and the only abnormality in this group of eight that was amenable to screening.

Table 2 shows a breakdown of the screening results by abnormality and gives false negative rates. A false negative was defined when a woman was deemed by appropriate screening to be low risk for the identified abnormality.

Trisomy 21 had a low false negative rate in this sample with only one out of the nine Trisomy 21 pregnancies who underwent screening receiving a low risk result. While the false negative rate for all potentially screen detectable abnormalities is shown it should be noted that the only true false negatives are those for Trisomy 21 which is the abnormality the screening test is currently calibrated for.
Table 2. First and second trimester screening results by congenital abnormality group

<table>
<thead>
<tr>
<th>Congenital abnormality perinatal related deaths 2010</th>
<th>Total</th>
<th>First or second trimester screening</th>
<th>Increased risk result</th>
<th>False negative screen</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>n %</td>
<td>n %</td>
<td>n %</td>
</tr>
<tr>
<td>CNS abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly/acrania**</td>
<td>10</td>
<td>40 %</td>
<td>375 %</td>
<td>20 %</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>9</td>
<td>89 %</td>
<td>0 %</td>
<td>100 %</td>
</tr>
<tr>
<td>Encephalocele</td>
<td>2</td>
<td>0 %</td>
<td>- %</td>
<td>- %</td>
</tr>
<tr>
<td>Other CNS*</td>
<td>14</td>
<td>50 %</td>
<td>- %</td>
<td>- %</td>
</tr>
<tr>
<td>Cardiac abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cardiac*</td>
<td>29</td>
<td>62 %</td>
<td>14 %</td>
<td>- %</td>
</tr>
<tr>
<td>Chromosomal abnormalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trisomy 21**</td>
<td>18</td>
<td>50 %</td>
<td>8 %</td>
<td>1 %</td>
</tr>
<tr>
<td>Trisomy 20**</td>
<td>1</td>
<td>100 %</td>
<td>0 %</td>
<td>100 %</td>
</tr>
<tr>
<td>Trisomy 18**</td>
<td>19</td>
<td>63 %</td>
<td>5 %</td>
<td>42 %</td>
</tr>
<tr>
<td>Trisomy 13 [includes 13/18]**</td>
<td>6</td>
<td>67 %</td>
<td>25 %</td>
<td>75 %</td>
</tr>
<tr>
<td>Turner syndrome**</td>
<td>3</td>
<td>67 %</td>
<td>0 %</td>
<td>0 %</td>
</tr>
<tr>
<td>Klinefelter’s syndrome**</td>
<td>2</td>
<td>50 %</td>
<td>0 %</td>
<td>0 %</td>
</tr>
<tr>
<td>Other chromosomal*</td>
<td>18</td>
<td>67 %</td>
<td>1 %</td>
<td>17 %</td>
</tr>
</tbody>
</table>

*Other CNS, all cardiac or other chromosomal are not screen detectable. However in some cases the first trimester screening will show an increased result due to a large nuchal translucency measurement indicating illness in the fetus.

** First trimester nuchal translucency with or without first or second trimester serum screening

† False negative rate has been calculated for potentially screen detectable abnormalities only other CNS, all cardiac, and other chromosomal are not screen detectable so no false negatives are reported.

† Only two of eight cases of spina bifida had second trimester serum screening (Spina bifida is only detectable by second trimester serum screening or ultrasound in the second trimester and ultrasound is the primary method for detecting spina bifida).

The final diagnosis was made significantly earlier in screen detectable abnormalities where antenatal screening occurred in either the first or second trimester [median gestation 19.5 weeks (IQR 19-21)] compared to when no screening was done [21 weeks (IQR 19-25)], p=0.02.

Termination was offered earlier in pregnancy when the pregnancy resulted in a screen detectable congenital abnormality and the mother had undergone screening in the first or second trimester [median gestation of 20 weeks (IQR 19-21) compared to 21 weeks (IQR 20-24) among unscreened women], although this difference was not statistically significant.

126 women (92%) had an anatomy scan. The median gestation at the anatomy scan was 19 weeks, ranging from 19 to 26 weeks. Of the 126 women who had an anatomy scan 96 (76%) were reported as abnormal, 16 (13%) as unclear and 14 (11%) were reported as low risk. The unclear group was made up predominantly of scans where
part of the fetal anatomy was not adequately seen which could have been due to imaging issues or to abnormality.

Eighty-two cases where a valid scan was available (131 ultrasound scans) were reviewed. Twenty five scans met the criteria for review but were unable to be obtained. The review of available scans identified five cases where the reviewer was able to identify the abnormality earlier. This included one trisomy 18, one triploidy, one central nervous system abnormality and two cardiovascular system abnormalities. There was an additional case where the images were not available where it was thought that there was potential for the abnormality to have been detected earlier due the severe nature of the abnormality.

There were four sets of anatomy scan images that were not available for review. Of the 20 sets of anatomy scan images that were available, seven were missing key views which meant that the scan should not have been reported as complete.

Discussion

There were 137 perinatal deaths where the primary antecedent cause of death was cardiovascular, central nervous system or chromosomal congenital abnormality in 2010. These constitute a significant portion (19%) of the 704 perinatal deaths during 2010 in New Zealand.

The purpose of this audit was to identify areas where there is potential to improve care for women with babies with congenital abnormalities and to reduce the number of perinatal deaths due to these types of congenital abnormalities firstly through prevention and secondly through earlier detection.

Only one of the 21 mothers who had a pregnancy that resulted in a neural tube defect was recorded as having taken folic acid pre-conceptually and 13 were recorded as having taken it antenatally.

The Ministry of Health recommends that all women who are planning a pregnancy or who are pregnant take 0.8mg of folic acid (5mg if in a higher risk group). If taken for at least one month prior to conception and during the first three months of pregnancy, folate can reduce the incidence of neural tube defects (risk ratio for folate use for reducing NTDs 0.28, 95% confidence interval 0.15 to 0.52).

Pre-pregnancy counselling including optimising treatment of medical conditions, identifying personal and family history of congenital defects, advising on folate prophylaxis, smoking cessation and educating about the potential value of booking early with an LMC are all important ways to identify at risk pregnancies and to reduce risk of congenital abnormalities.

A delay between first contact with a health care professional and booking with a lead maternity carer (LMC) was identified. In a majority of cases the first contact during the pregnancy was with a general practitioner (or general practice nurse). This first contact occurred before the cut off for first trimester screening (13 weeks and six days) in 83% of pregnancies examined, however only 66% of women went on to book with an LMC before 14 weeks.

This highlights a need to facilitate booking with a LMC or for GPs to take the responsibility for providing antenatal screening. If GPs continue to provide first line
antenatal care, they should be targeted for first trimester antenatal screening education.

In February 2010 the antenatal screening guidelines in New Zealand changed. Prior to 2010 the main antenatal screening tests for congenital abnormalities were the nuchal translucency and anatomy scans. In February 2010 new guidelines were implemented that required all eligible pregnant women to be offered a nuchal translucency scan combined with a blood test prior to 14 weeks gestation.

Women who presented after 14 weeks could be offered the 2nd trimester blood test up to 20 weeks gestation. There was a range of screening test combinations undertaken by the women included in this audit, which may reflect the changes that were made to the screening programme during this period, although it may also reflect women’s choices. It would be useful to evaluate whether there continues to be variation in antenatal screening options performed.

First and second trimester antenatal screening is calibrated for maximal sensitivity for Trisomy 21, although it does identify risk for other chromosomal abnormalities. The calibration of the screening optimising diagnosis of Trisomy 21 is evidenced by the higher false negative rates in pregnancies which resulted in Trisomy 18 or Trisomy 13 in this review.

Screening could be calibrated for greater detection of Trisomy 18 and Trisomy 13, but the costs and benefits of these additional investigations would need to be evaluated. The true false negative rates for any chromosomal abnormality can only be assessed by a review of all cases of congenital abnormality. To do this would require an improved register of congenital abnormalities in New Zealand.

The efficacy of the anatomy scan as a tool for identifying abnormalities is partly due to high uptake of this test (92% in this review), and partly due to a high rate of detection (76% of the anatomy scans in this data set were reported as abnormal).

To be most effective, the anatomy scan should be done at 20 weeks which is a recent change from 18 weeks and reflects in part the changing maternal habitus as increasing obesity has made it more difficult to obtain the required images.

Having the anatomy scan later increases the chance of an abnormality being detected in all women, although this needs to be weighed against the effects of a later diagnosis.

The anatomy scans that on review were determined to be incomplete but were reported as completed are an area of concern. With usual practice a sonographer scans the patient and captures the required images and then a radiologist reviews and reports on the static images. When the required images are not captured the anatomy of the baby is not able to be adequately assessed.

Ensuring that all required images are obtained and getting women to return for a further scan should the required images not be obtained are important steps to improving detection of abnormalities. Failure to obtain a standard view may be an indicator that there is an abnormality.
There were five cases where the ultrasound reviewer was able to detect the abnormality earlier. These cases suggest ultrasound is an area where the detection of congenital abnormalities could be improved.

The failure to retain static images by ultrasound operators was identified as an issue. Given continued improvements in electronic storage, the reduction in cost of this service and importance to audit and review, ultrasound providers should be retaining copies of ultrasound scans. Further, this practice aligns with retention of clinical notes by clinicians in other specialties.

Of the 137 women included in the audit there were six sets of midwifery notes that were not available for review. All lead maternity carers are legally required to retain a copy of pregnancy notes for 10 years.6

**Conclusion**

This audit aimed to determine whether there were quality gaps in the care of women whose babies died from congenital abnormality. This included the hypotheses that a number of congenital abnormalities could be prevented if folate was taken prenatally and that a number of chromosomal abnormalities and neural tube defects could have been detected earlier.

Preconceptual folate supplements were not widely reported as used across all types of abnormalities, levels of folate supplement use reported increased antenatally. It is likely that given the low levels of preconceptual folate use reported that a number of the neural tube defects could have been prevented had folate supplements been taken preconceptually.

A number of the abnormalities could potentially have been detected earlier this includes 23% of women who saw a health care practitioner before the cut off for either first or second trimester screening and who were not offered screening.

There were also three neural tube defects that could potentially have been detected earlier including an anencephaly that was not detected at the nuchal translucency scan and two spina bifidas that were not detected during the second trimester screening. This may reflect the fact that second trimester screening is no longer optimised for detecting spina bifida as not all spina bifidas are screen detectable.

Similarly there were ten chromosomal abnormalities that could potentially be detected by screening but the screening algorithms are not optimised to detect them. There was one case of Trisomy 21 that was a false negative.

**Recommendations where improvements could be made based on the findings include:**

- All women should receive pre-conceptual counselling to optimise maternal health, identify obstetric or familial risk factors, discuss current medical conditions and refer to specialists as required.

- A media campaign for pre-conceptual folate is required along with further investigation of the fortification of bread with folate.
• Education of all women is required about the importance of booking with a LMC before 10 weeks.

• Education and support should be offered so that primary care providers are able to effectively offer first trimester screening, interpret screening results and facilitate expeditious booking with an LMC.

• If screening has not already been arranged then LMCs should offer all women first or second trimester screening, as required by the Ministry of Health.

• There should be a review of the current algorithms used in New Zealand’s first and second trimester screening programme and consideration of the cost benefit of using algorithms calibrated for maximal sensitivity for all chromosomal abnormalities.

• There should be a review of the efficiency and adequacy of the antenatal screening program’s guidelines for reporting results for nuchal translucency in a patient who has not had a serum sample taken to avoid delays in reporting risk from the nuchal scan.

• False negative screening tests should be reviewed by the screening unit.

• All LMCs should document pre-conceptual folate and antenatal folate use including when the woman commenced taking folate and the dose.

• Ultrasound services should retain copies of all ultrasounds and audit their images to ensure accurate measurements are obtained during scanning, in particular during the nuchal translucency scan.

• Enhancement of the current birth defects register to include congenital abnormalities where a perinatal death occurred.

Competing interests: None identified.

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References:

IgE-mediated food allergy—diagnosis and management in New Zealand children

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Abstract

**Aim** To summarise the diagnosis and management of IgE-mediated food allergy (FA) in New Zealand children.

**Method** A review of the scientific literature and subsequent consensus development.

**Results** FA is a common problem in New Zealand children with management necessitating accurate diagnosis, appropriate risk management, and reassessment over time.

**Conclusion** This paper highlights the importance of a structured approach to diagnosis and management of FA in New Zealand children, guided by appropriately skilled health professionals.

Key points

- Food allergy is a common paediatric condition.
- The history of an immediate allergic reaction is critical in interpretation of skin-prick test (SPT) or serum specific IgE (ssIgE, also referred to as RAST or EAST).
- Specialist paediatric referral should occur in any child with anaphylaxis, allergy to more than one food allergen, or where the primary care practitioner is not confident about diagnosis, test interpretation, or management.
- Children and young people with food allergy should have advice about allergen avoidance and a written management plan detailing the signs, symptoms and management of allergic reactions.
- Children with IgE-mediated food allergy require regular follow-up. Many food allergies are not persistent and need reassessment over time.

Introduction

IgE-mediated food allergy (FA) is common, affecting up to 10% of children under the age of 5 years. Parental perception is that up to 30% of pre-schoolers may be affected. Management of FA involves accurate diagnosis of the specific allergen(s), advice on allergen avoidance, risk assessment with provision of an appropriate action plan, and follow-up.

Many children grow out of FA. Reassessment, often including cautious reintroduction of the offending allergen, is an important part of ongoing management.
Recent evidence based reviews have reinforced the paucity of quality evidence on which to base decisions about diagnosis and management of FA. However FA is a common paediatric condition and regardless of poor evidence clinical care needs to be offered to these patients.

This consensus document has been developed by the Allergy Special Interest Group of the Paediatric Society of New Zealand, providing a current guide to managing children with IgE-mediated FA in New Zealand (NZ).

**Definitions**

FA is defined as an adverse immunologic reaction to a food protein. Many FA are IgE-mediated immediate hypersensitivity reactions, while immunological mechanisms other than IgE also occur. Food intolerance does not have an immunological mechanism. Some food intolerance is clearly defined (e.g. lactose intolerance) but much is not; non IgE-mediated food allergy and food intolerance will not be considered further in this document.

Sensitisation is defined as the presence of specific IgE detected on SPT or ssIgE.

**Epidemiology**

There are no data on rates of FA in NZ children. In Australia up to 10% of 1 year olds have proven food allergy. Milk, egg and peanut allergy account for about 75% of early food allergies. Other common allergens include fish, shellfish, tree nuts, kiwifruit, sesame, and also wheat and soy.

Atopy is a risk factor for FA, with most children with FA having eczema. There is often a family history of atopy and sometimes of FA. While atopy is inherited, allergy to a specific allergen is not and importantly IgE sensitisation to specific allergens does not necessarily imply causation of eczema.

**Prevention**

It is poorly understood why some children develop FA while most develop tolerance. Maternal allergen avoidance during pregnancy or breast feeding does not prevent FA in the infant. Later introduction of peanut, egg, and cow’s milk to the infant’s diet is associated with an increased rate of allergy to that food.

Prospective studies are currently evaluating early introduction of common food proteins as a strategy to prevent FA. While general infant feeding guidelines often suggest introduction of solids at about 6 months of age, allergy prevention advice is that solids can be introduced to the infant’s diet from 4 months onwards, with no role for avoidance of commonly allergenic foods.

**Diagnosis**

Clinical Features of IgE-mediated allergy—The history of an allergic reaction is important in assessing possible FA. Factors to consider include:

- Signs and symptoms of IgE-mediated allergic reactions are varied with no single feature always present (Table 1).
• Onset of symptoms in IgE-mediated FA is often within minutes of exposure to an allergen. Delay of symptom onset more than two hours after ingestion is uncommon.\(^{12}\)

• Most allergic reactions occur after ingestion of an allergen, with patients having different thresholds to trigger reaction. Skin contact with an allergen may result in local reactions but seldom causes severe reactions. Inhalation in the vicinity of peanut butter is unlikely to cause a reaction.\(^{13}\) Reactions following inhalation in other situations (e.g. cooking fish) can occur.

• Most IgE-mediated reactions resolve quickly. Anaphylaxis can be biphasic, with recurrence of symptoms after initial apparent resolution.\(^{14}\) Persistence of urticaria beyond 6-8 hours makes FA a less likely cause unless there is ongoing allergen exposure.

• Many reactions occur with the first known ingestion of an allergen. If a food allergen is regularly consumed and tolerated then development of allergy to that food is uncommon.

Table 1. Signs and symptoms of an IgE-mediated allergic reaction

<table>
<thead>
<tr>
<th>Cutaneous</th>
<th>Urticaria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Angioedema</td>
</tr>
<tr>
<td></td>
<td>Flushing / erythema</td>
</tr>
<tr>
<td></td>
<td>Itch</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Watery rhinorrhoea</td>
</tr>
<tr>
<td></td>
<td>Sneezing</td>
</tr>
<tr>
<td></td>
<td>Tongue swelling *</td>
</tr>
<tr>
<td></td>
<td>Hoarseness / laryngeal oedema *</td>
</tr>
<tr>
<td></td>
<td>Cough *</td>
</tr>
<tr>
<td></td>
<td>Wheeze *</td>
</tr>
<tr>
<td></td>
<td>Stridor *</td>
</tr>
<tr>
<td>Gastrointestinal tract</td>
<td>Vomiting</td>
</tr>
<tr>
<td></td>
<td>Abdominal pain</td>
</tr>
<tr>
<td></td>
<td>Diarrhoea</td>
</tr>
<tr>
<td>Cardiovascular / general</td>
<td>Pallor *</td>
</tr>
<tr>
<td></td>
<td>Dizziness *</td>
</tr>
<tr>
<td></td>
<td>Collapse *</td>
</tr>
</tbody>
</table>

* Features of anaphylaxis, defined as a severe allergic reaction with involvement of cardiovascular and/or respiratory systems.

Anaphylaxis—Anaphylaxis is a severe, systemic allergic reaction with circulatory or respiratory compromise.\(^{15}\) Anaphylaxis due to food allergic reactions most often involves respiratory features rather than cardiovascular, and a history suggestive of respiratory involvement as part of a reaction should be sought.

Eczema—Most young children with FA have a history of eczema. Young infants with severe eczema have an increased likelihood of also having FA.\(^{16}\) In breast fed infants transfer of food allergens via breast milk may contribute to eczema.

Most children with eczema are atopic and thus often sensitised to multiple foods on testing, but in the absence of a suspicious history this may not translate into specific
foods being triggers for eczema flares. Screening with large panels of allergens is not recommended in eczema. There has been little benefit from food exclusions for treatment of eczema in trials.

Table 2. Food allergy testing

<table>
<thead>
<tr>
<th>Test</th>
<th>Pro</th>
<th>Con</th>
<th>Caveat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin prick test</td>
<td>Immediate result Inexpensive Can test using food in question if no commercial allergen test available Strongly positive results likely to indicate clinical allergy</td>
<td>Operator and reagent dependent Extremely rarely associated with severe allergic reaction Unreliable with concomitant antihistamine use or dermographism Weakly positive results may or may not indicate clinical allergy</td>
<td>For both SPT and food ssIgE it is possible to have weakly positive tests associated with clinical allergy and strongly positive tests to foods that are clinically tolerated. Neither SPT nor ssIgE predict the severity of allergic reaction</td>
</tr>
<tr>
<td>ssIgE</td>
<td>Change in level over time may predict development of tolerance Strongly positive results likely to indicate clinical allergy</td>
<td>Results not immediately available Relatively expensive Not available for all potential allergens Requires venipuncture Weakly positive results may or may not indicate clinical allergy</td>
<td></td>
</tr>
</tbody>
</table>

In the absence of an immediate IgE-mediated reaction, the potential benefits of food exclusion for management of eczema should be weighed carefully against the potential risks (e.g. failure to thrive, cost). Any food exclusion for eczema should be considered a trial, with the intent being to reintroduce the food after a period of weeks.

Good skin care is the basis of eczema treatment, regardless of potential food triggers. Education needs to be provided including advice on avoidance of irritants, use of moisturisers, and use of topical steroids as appropriate.

Investigations—The first purpose of allergy testing is to confirm the cause of an allergic reaction. Even a convincing history suggesting IgE-mediated FA will not always be confirmed on investigation. Testing for specific IgE can help avoid unnecessary or prolonged periods of dietary restriction. Confirming IgE-mediated FA aids ongoing management and helps predict natural history.

In addition there may be a role for testing where there may be other potentially significant allergens, acknowledging that this may identify clinically unimportant sensitisation in some children. However children presenting after one food allergic reaction may have other food allergies; in one study 40% of infants presenting with cow’s milk allergy had egg allergy.

Screening for large groups of allergens is not recommended, but testing a small range of common allergens may be considered (e.g. testing peanut in a one year old...
presenting with egg allergy who is already ingesting milk and wheat). Importantly foods that are already tolerated should not be tested.

Investigation will be either by SPT or ssIgE (Table 2). SPT are reported as millimetre of wheal, with 3mm taken to indicate presence of specific IgE. Historically ssIgE have been reported as a grade (e.g. 1+ to 6+), but more commonly now the result is reported as KIU(A)/L. As described below the interpretation of test results is highly dependent on the patient’s history.

Other tests are not valid for investigation of IgE-mediated FA. Which test is used will depend on availability of the test and the food in question. Where one test is negative despite a suggestive history, undertaking the other test is appropriate. Routine testing with both SPT and ssIgE is not necessary.

For some foods predictive SPT and ssIgE results have been published; strongly positive results indicate a higher likelihood of allergic reaction on supervised food challenge (Table 3).11

Table 3. Allergy tests and food challenge outcomes

<table>
<thead>
<tr>
<th>ssIgE results predicting chance of reaction at challenge &gt;95%26,30</th>
<th>Egg in infants under 2 years</th>
<th>&gt; 7kU/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Egg</td>
<td>&gt; 2kU/l</td>
<td></td>
</tr>
<tr>
<td>Milk</td>
<td>15kU/l</td>
<td></td>
</tr>
<tr>
<td>Peanut</td>
<td>14kU/l</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Allergy skin prick test results predicting chance of reaction at challenge &gt;95%31</th>
<th>Egg in infants under 2 years</th>
<th>8mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Egg</td>
<td>6mm</td>
<td></td>
</tr>
<tr>
<td>Milk</td>
<td>7mm</td>
<td></td>
</tr>
<tr>
<td>In infants under 2 years</td>
<td>5mm</td>
<td></td>
</tr>
<tr>
<td>Peanut</td>
<td>8mm</td>
<td></td>
</tr>
<tr>
<td>In infants under 2 years</td>
<td>4mm</td>
<td></td>
</tr>
</tbody>
</table>

The key to interpretation of investigations is the patient’s history. The pre-test probability influences interpretation of test results.20 Where a child has had symptoms highly suggestive of an IgE-mediated reaction then any positive ssIgE/SPT test may be taken as confirmation of diagnosis. Where the history is less clear or where there is no history of exposure (i.e. a lower pre-test probability) then a more strongly positive test result will be needed to confirm a diagnosis, and may need to be combined with a supervised food challenge for confirmation. The threshold results in Table 3 apply to a selected population with high pre-test probability, and may not have the same applicability in a more general population.

Molecular allergy tests (Component Resolved Diagnostics, CRD) are becoming more readily available.21 For example in some populations ssIgE to Ara h 2 helps predict outcome of peanut challenge.22 The range of CRD will evolve with time, and their use in other situations (e.g. to determine tolerance of heat treated forms of cow’s milk and egg) will also expand with further data.
Management

Referral guidelines—Specialist paediatric referral is recommended for children with FA with
- Definite or possible anaphylaxis.
- Allergy to cow’s milk or multiple food allergies, where expert advice is needed.
- Where there is uncertainty about the diagnosis or interpretation of results.
- Food sensitisation on ssIgE / SPT, where supervised challenge may be necessary to clarify whether there is clinical allergy.
- Allergy to foods such as peanut and nut where the risk of severe allergic reactions is higher.
- Children with asthma and FA, with asthma a risk factor for severe food allergic reaction on accidental exposure.
- Children whose FA persists past 5 years of age.

Some children with FA may be looked after in primary care. Management needs to include advice on allergen avoidance, provision of an action plan and follow-up for possible resolution of FA. Eventual referral for specialist supervised food challenge may be necessary.

Allergen avoidance—Management of FA involves avoidance of known allergens to minimise reactions on accidental exposure. Commercially packaged food from NZ declares the presence of common allergens. Caution is needed with food packaged in other countries.

For some allergens (e.g. egg and milk) total avoidance is not always necessary. Up to 75% of egg or milk allergic children may tolerate these as an ingredient in well cooked foods (e.g. baked foods). This is sometimes apparent on the initial history (e.g. a child who reacted to scrambled egg but tolerates cake containing egg). At other times this tolerance may develop as the allergy is resolving. There are published guidelines for liberalising egg as an ingredient in baking at home in selected patients.

Many families and doctors are confused by labels that warn about potential traces of an allergen. While avoiding all of these products will be the least risky option, this is difficult to achieve. Risk management decisions about these products should be made by families in discussion with their doctor and dietitian. It is our experience that most children tolerate some products with “may contain traces” warnings without signs or symptoms of allergic reaction.

Risk management—Anaphylaxis may be the initial presentation of children with IgE-mediated FA. Children with less severe initial reaction can have anaphylaxis on further accidental allergen ingestion.
Factors that increase the risk of severe allergic reaction include:

- Age, with life-threatening and fatal food allergic reactions more commonly reported in older children, adolescents and young adults.
- Asthma, as almost all patients who die of food allergy also have asthma.
- Peanut/Nut allergy as these are responsible for a significant proportion of fatal food allergic reactions. Cashew nut anaphylaxis is often severe.\(^{56}\)

Other factors to consider as part of risk management include:

- Access to emergency care.
- Ability to comply with allergen avoidance.
- Comorbidities and other medications.

**Action plan**—All children and young people with FA should have a written plan detailing the signs and symptoms of allergic reactions and what action should be taken. The plan needs to be available to all caregivers.

- A variety of plans are available for download on [www.allergy.org.au](http://www.allergy.org.au) (the website for ASCIA—Australasian Society of Clinical Immunology and Allergy). Use of standardised plans is recommended to minimise confusion amongst caregivers.
- The history and risk assessment will determine whether the plan includes an adrenaline autoinjector (EpiPen®, Anapen®). Guidelines on who should have an adrenaline autoinjector are available on [www.allergy.org.au](http://www.allergy.org.au). Use of adrenaline ampoules with a needle and syringe is not a safe alternative.\(^{27}\)
- Antihistamines may be part of the plan, used for symptom relief. Antihistamine use does not prevent or treat anaphylaxis. Use of a non-sedating antihistamine is preferred.\(^{28}\)
- Referral for education of carers at schools and preschools should be made to local providers, often local Public Health Nurses.
- For children with asthma the action plan may include instructions on the use of bronchodilators, to be used after intramuscular adrenaline for any FA reaction with respiratory symptoms.
- MedicAlert should be considered particularly for older children and adolescents.

**Dietetic support**—Dietetic input is important for children with cow’s milk allergy, with multiple food allergies, or with allergy to foods that are hard to avoid such as wheat and soy. If maternal allergen avoidance is advised, maternal dietetic advice will be needed.

**Cow’s milk alternatives in infancy**—Infants with cow’s milk allergy need an alternative for weaning or for supplementation of breast feeding. Goat’s milk is not an option as there is extensive cross-reactivity. Special authority funding is available for extensively hydrolysed formula (eHF) and amino acid formula (AAF) for infants meeting PHARMAC criteria, with requirements for regular re-evaluation, and a “step
down” from the more expensive AAF to alternatives as possible. Many infants with cow’s milk allergy can tolerate soy.8

Follow up—Children with IgE-mediated FA need follow up to determine whether the allergy is persistent, to consider retesting and food challenge if appropriate, and to ensure that action plans remain up to date, with review of adrenaline autoinjector use as needed.

The natural history of FA is often of resolution. Overall most milk (80% by age 5 years), egg (66% by age 7 years), wheat and soy allergies will resolve. For most children, peanut (80%), nut (90%) and fish allergy will be persistent.3 Follow-up testing frequency depends on the food in question and the interval history of reactions; intervals of less than 12 months are generally unnecessary.19

During follow-up there needs to be age-appropriate transition of responsibility for managing the FA. Some older children may develop troublesome food aversion and fear of trying new foods; anxiety about FA should be actively sought and managed.

Food challenge—Double-blind, placebo-controlled food challenge (DBPCFC) is the gold standard for diagnosis of FA in clinical studies, but it is rarely part of clinical practice. Supervised open food challenge may be used to clarify diagnosis, determine whether sensitisation is clinically relevant, or to determine resolution.

The decision to undertake a food challenge will depend on:

- Reactions – recent reactions indicate persistent allergy.
- Investigations—strongly positive SPT or ssIgE make tolerance unlikely.
- Family preference and chance of resolution – for some families a 50% chance that an allergy has resolved may mean they are keen to pursue challenge, while others may prefer to wait until the chances of resolution are higher.

Supervised food challenges should be carried out using established protocols with access to immediate medical back-up and resuscitation, in case a severe reaction occurs.

In NZ most food challenges will be done in hospital settings with paediatric specialist supervision. Food challenge does not necessarily inform as to on-going risk—a mild reaction at a small dose indicates persistent FA and a challenge is stopped, with the result not precluding a more severe reaction on subsequent exposure.

Resources

www.allergy.org.au (ASCIA website) with allergic reaction plans, adrenaline autoinjector guidelines, eczema management plans and patient information sheets. ASCIA proves open access online e learning courses for education on food allergy and anaphylaxis.

www.allergy.org.nz is an organisation providing support, information and advocacy, plus education kits for schools and preschools.
Conclusion

Food allergy is a common problem for NZ children. Management necessitates:

- Accurate diagnosis with appropriate investigation
- Education about allergen avoidance, with dietetic assistance as appropriate
- Risk assessment and provision of an appropriate allergic reaction action plan
- Supervised challenges where appropriate
- Follow up for possible resolution and ongoing risk assessment.

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Questions about New Zealand’s health system in 2013, its 75th anniversary year

Robin Gauld

Abstract

New Zealand’s health system turns 75 in September, 2013. This article suggests that it is a time for celebration but also reflection on whether we have achieved the aims of the 1938 Social Security Act which laid out a set of principles for health care delivery. The article looks at questions of access, equity and service integration. It outlines why the health system we have today is shaped the way it is, and asks whether we should recommit to the original 1938 aims or develop a new set of principles for our health system.

This year, 2013, is a significant occasion for the New Zealand health system for it marks the 75th anniversary of the laying of its contemporary foundations. Such an anniversary is a time for celebration but also for reflection on whether we have achieved the goals sought in the passage, on 14 September 1938, of the Social Security Act under the government of Michael Joseph Savage.

It is also a time for debate around how well our present health system functions and performs in the light of the 1938 Act’s aims. This article, therefore, reflects on the goals of the Social Security Act for health care, whether we have achieved them and what the key barriers to this have been. It also asks whether we should recommit to the original 1938 aims for health care, or develop a new set of principles that reflect how the New Zealand health system is presently structured and performs.

What the Savage Government aimed for

When the Savage Government presided over the 1938 Social Security Act (SSA), for health care, they did so with an intention of creating universal access to a comprehensive national health service. New Zealand was, thus, the first democratic capitalist country in the world to attempt this. Indeed, the National Health Service in the United Kingdom was not enabled until a decade later.

The intent of the Savage Government was laudable, given the pre-existing circumstances: health care was not a right, its delivery was highly variable, access depended to a great degree on ability to pay and whether services were even available, and the provision of public services was limited.1–3

The goals of the SSA were visionary, including that:

- Health care should be universally available and a fundamental right without barriers to access;
- all New Zealanders should have equal access to the same standard of treatment;
• the health system should have a preventive rather than curative focus; and
• services should be integrated not fragmented between primary and hospital-based care.³

These goals, in many ways, align closely with international thinking today – from agencies such as the World Health Organization – around what health systems should aim for in terms of overall design.⁴,⁵ This makes it all the more important that we ponder whether, 75 years on, we have achieved the SSA aims. The next section takes up this question.

**Have we achieved the aims of the Social Security Act?**

Over time, we have built a New Zealand health care system that performs anywhere from poorly to superbly depending on which of the many indicators one looks at. Health system performance measurement is complex and it is often difficult to get a clear picture of performance across an entire system.⁶

In 2011, in an attempt to provide an overall performance rating for the New Zealand health system, a group of medical students and I developed a scorecard which rated New Zealand against 64 international and national benchmark indicators using routinely-collected data. This allowed us to compare New Zealand’s performance with the best across a range of different dimensions. The method for this and findings are presented in more detail elsewhere.⁷

In brief, we selected indicators across a series of performance categories (healthy lives, efficiency, quality, access and equity). Where international data were used, benchmarks were set by averaging performances for the three highest-performing systems with the New Zealand score rated against the benchmark in a simple numerator-denominator calculation. For local data, the same process was employed with the three top-performing DHBs used for establishing the benchmark.

The result was a reasonable 71%, but a notably poor 57% for equity and only 64% for access raising serious questions about achievement of at least two of the four SSA goals listed above, discussed in further detail below.

**Universality, access and equity**—In terms of universality, New Zealand has performed reasonably well in the broadest sense. All permanent residents, regardless of socio-economic status or tax-contributions, are entitled to a range of health and disability services that are largely government-funded through general taxes. This places New Zealand alongside other developed nations that consider health care to be a fundamental right.

Yet the 64% for access given in our scorecard highlights a range of problem areas. While no-one will be turned away from a hospital emergency department when in need of health care, and which is free, access to services across the spectrum of care in New Zealand remains problematic. As ours and many other studies consistently highlight, it is patchy, far from universal, and varies by socioeconomic status, service, ethnicity and region.⁸

Perhaps most problematic are the fees charged to see a general practitioner (GP) or allied primary care provider such as a practice nurse. Data variously show a proportion of New Zealanders avoid seeing the doctor or filling a prescription due to
cost barriers. For example, some 26% of patients in poor health surveyed in 2011 by the Commonwealth Fund had cost-related barriers to health care.\textsuperscript{9}

Jatrana and Crampton’s analyses of Statistics New Zealand data collected in 2004–05 show 15.5% of the population avoided seeing a doctor due to cost.\textsuperscript{10} The subsequent 2006–07 New Zealand Health Survey suggested only 1.7% of respondents (but 4.1% of Māori) fell into this category.\textsuperscript{11}

While the last two of these studies have different findings, one interpretation may be that increased subsidies introduced through the 2000s aimed at removing financial barriers have been effective and demonstrate progress toward removing cost barriers. On other measures, such as physical access to a GP, New Zealand performs comparatively well with Commonwealth Fund data showing three-quarters of patients in poor health able to get a same or next day appointment.\textsuperscript{9}

When it comes to hospital and specialist care, questions also abound around universality, equity and access. Waiting lists for non-urgent services, as we know, have long been a feature of our health system which successive governments have worked to improve the management of.\textsuperscript{12}

Certainly, there is more transparency now with the use of scoring and booking systems than two decades ago. The electives target in place since 2009 has also driven a renewed government and DHB commitment to improving public service access and waiting times.

Yet access continues to vary by DHB and specialty,\textsuperscript{13,14} while periodic media reports suggest it could be becoming increasingly difficult to access public hospital specialist services despite the influence of a national target for improving elective services access.\textsuperscript{15} Certainly, access remains problematic from the perspective of doctors as shown in a 2012 Commonwealth Fund survey in which 75% expressed concerns about the length of time their patients were waiting to see a specialist.\textsuperscript{16}

The implication is that those who can pay privately or have health insurance will receive the care they need, while those unable often suffer until they become unwell enough to reach the threshold over which they may be seen and treated. It is difficult to believe this scenario was an intent of the original SSA, nor what most New Zealanders want today.

**Preventive not curative focus**—To be fair, successive governments have worked to build a preventive focus for the New Zealand health system.\textsuperscript{12} Indeed, the guiding legislation for the present DHB system requires a focus on inter-sectoral planning, health needs assessments and health promotion. However, over the years the best of policy intentions have often failed due to the powers of particular interest groups, an over-riding focus in the health system on personal health and institutional arrangements discussed in more detail below that work against good cross-sectoral planning which prevention requires.

**Integration**—Integration is an area of considerable concern, given the potential we have for this in New Zealand and a history of attempting to better integrate services.\textsuperscript{17,18} In practical terms, integration means that patients perceive the health professionals they see—whether primary care or hospital based—as all working for the same system as you would expect at different branches of your bank. In this sense, most
patients with multiple health care encounters would probably suggest there is limited integration, although we lack good data about this based on patient experiences.\textsuperscript{18} Again, Commonwealth Fund surveys indicate that some 30\% of patients with chronic care needs experience problems with care coordination.\textsuperscript{9} Other studies into care coordination and integration have revealed similar challenges.\textsuperscript{18}

In New Zealand, we have the essential ingredients for integration. We have a single-payer health system in that the government pays for most health care and most social services are centrally-funded. Health funding incorporates disability support services funding, unlike many other countries where this is by a separate agency. We also have CEOs in each DHB, with ultimate responsibility for the organisation of care in their region.

The money and, very importantly, lines of authority are therefore linear. In contrast, many countries have multi-payer systems meaning health care providers receive their income from many different sources, complicating attempts to run a single system.\textsuperscript{19} The most prominent example is the USA. Why then have we failed after 75 years to achieve an integrated health care system; and how do we make sense of the various other shortcomings touched upon above? We need to go back to the late-1930s.

\textbf{Compromise and consequent institutional foundations}—Others have documented the political bargain struck between the government and the then very powerful medical profession, represented through the New Zealand Branch of the British Medical Association (NZBMA) as it was called, required as it would have been almost impossible to implement the SSA otherwise.\textsuperscript{1,20}

The NZBMA opposed the government’s proposals for funding doctors, which was likely to be some form of government-funded ‘national insurance’ that could come via a capitation model that would pay a fixed sum per annum per patient enrolled with a doctor and mean no direct patient charges to see a doctor. The NZBMA was of the view that such a model would undermine the doctor-patient relationship as it would interrupt the ‘personal arrangements’ between the two parties if a third payer – the government—became involved.\textsuperscript{1}

Yet this view flew in the face of advice received from Sir Henry Brackenbury, vice-president of the British Medical Association (UK), who visited New Zealand in 1937. Brackenbury was a firm believer that national health insurance was the best method for funding. Fee for service medicine, he said, effectively reduced medical practice to the status of selling goods over the counter instead of fostering the principle of the doctor being the professional health advisor to the individual. Brackenbury believed doctors should be able to give full attention to patients without having to worry about presenting them with a bill.\textsuperscript{1} In sum, there was something of a conflict of views between BMA leadership and the New Zealand Branch.

The bargain eventually struck between the government and NZBMA in order to get the 1938 reforms implemented was as follows:

- GPs would maintain their independence and private business ownership model, and their ability to directly charge each patient for services provided. They would also receive a subsidy per visit from the government, meaning patients directly paid around a third of the cost.\textsuperscript{20}
Doctors would be permitted to work part-time in public hospitals for which they would be paid a salary, while maintaining their capacity to also work in the private sector. Hospitals would have no patient fees.¹

In this bargain, we had the establishment of institutional arrangements that remain in place today. These foundations have led to a health system with quite separate service delivery compartments and methods of funding: GPs and specialists in private practice serve largely their own and their patients’ interests, not those of the whole health system or public; public hospitals, similarly, function independently, despite employing the same specialists working privately.

In primary care, various attempts over the years to alter fee structures or integrate with hospital services have been troubled by a mix of resistance and substandard policy.² However, recent trends suggest a closer alignment of GPs and primary care within the broader health system, while raising questions around how they might be further advanced. These include the increasing involvement of GPs and Primary Health Organisations (PHOs) in DHB activities and the gradual building of integrated local health systems, which should be propelled by the 2013 ‘New PHO Services Agreement’ and Alliancing arrangements being implemented in each DHB region.

There has also been a gradual shift in recent years in some areas toward salaried general practice with capitation funding under PHOs, and GPs do play a crucial ‘gatekeeping’ role in the health system which is absent in many countries. This role could be given increasing prominence with the post-2008 policy emphasis on ‘better, sooner, more convenient’ and its implications for moving more services, and enhancing their coordination, under the umbrella of primary care.

When it comes to hospital care, debating the dual system goes to the heart of how some 40% of our medical specialists practice. That is, with both a public and a private hat on. Some say the dual system gives patients choice, but it also drives inequities in access through charges that are prohibitive to many. New Zealand is unusual in having no method for setting private fees which, in many EU and Asian countries, are often exactly the same as in public and, in this regard, a driver of equitable access which is an explicit aim of their health systems.²²³

We need debate around private fees in which there is little transparency, especially when New Zealand’s private specialists rely on publicly-subsidised systems – the GP gatekeepers—for patient referrals and public hospitals for crucial backup and support when patients have complications. And if private practice is quickly accessible what is hindering similarly timely access in public hospitals?

The embedded nature of our health system described above has meant an almost inevitable path dependency that seems difficult to shift from.²⁴ This path is largely due to resistance of the NZBMA and policy compromises made in the short period after 1938.

We need to ask, 75 years on, whether it was appropriate for one group to dominate so strongly in the policy process over 70 years ago, whose interests have best been served, and what public value and good the resulting system has brought long-term to the New Zealand public and to the development of our health care delivery arrangements?
We need to ask, also, whether we should accept for our health system what has become status quo for its foundations? We know why the health system we have today is structured the way it is, but we need more debate and research into how well it performs.

We need to involve the public in this debate, and our health professionals. We need to ask whether we should recommit to the 1938 principles, as outlined above, and aim to achieve these; or whether we need a new set of principles that more accurately reflects the status quo.

If the latter, should we expect some patients to face access barriers, especially fees for primary care services; should we be resigned to compartmentalisation in our health system and the complexity this creates, particularly for developing strategies aimed at integration and at preventive approaches to health care delivery; and should we accept that those who can pay get swifter access to non-urgent services. This is the year for debating such questions and thinking about the design of our health system for the years to come.

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**References:**

Scalp involvement by *Sarcoptes scabiei var hominis* resembling seborrhoeic dermatitis in two immunocompromised patients with systemic lupus erythematosus

Antonia Birry, Paul Jarrett

**Abstract**

Scabies is a common condition in New Zealand but scalp infestation by the mite is not often considered. Topical treatments traditionally do not involve the scalp. We report two cases of immunocompromised patients with systemic lupus erythematosus (SLE) who had scalp infestation clinically mimicking seborrhoeic dermatitis.

**Case report**

**Case 1**—A 37-year-old Cook Island Māori female with established SLE and cutaneous involvement, was referred to the Dermatology Clinic for the assessment of a further rash that she had developed approximately 4 weeks after returning from the Cook Islands. The itchy rash had persisted despite treatment with 5% permethrin cream. Her medications were prednisone 40 mg once daily and hydroxychloroquine 400 mg once daily.

She had an eczematous rash affecting the trunk and limbs, however no burrows were seen. In the hair bearing areas of the scalp there was an inflammatory dermatosis consisting of scale and erythema resembling seborrhoeic dermatitis (Figure 1). A skin scrape of the scalp confirmed the presence of *Sarcoptes scabiei var hominis*.

**Figure 1. Case 1—scale and erythema in the scalp resembling seborrhoeic dermatitis**
She was treated with two doses of ivermectin (200 µg/kg) 7 days apart. The rash on her scalp and body resolved. Subsequent skin scrapes of the scalp revealed no further mites.

**Case 2**—An 18-year-old Tongan female admitted with arm cellulitis was referred by the surgical services to the Dermatology Inpatient service. She presented with several weeks of itching and a rash over the scalp, trunk and limbs. She had an established diagnosis of SLE. Her medications were prednisone 60 mg once daily and azathioprine 100 mg once daily which she had been taking for 7 months.

Examination revealed multiple typical scabetic burrows. There was a scalp dermatosis confined to the hair bearing areas consisting of scale and erythema resembling seborrheic dermatitis.

A scalp scraping confirmed the presence of *Sarcoptes scabiei var hominis* (Figure 2).

*Figure 2. Case 2—*Sarcoptes scabiei var hominis* from scalp scraping*

She was successfully treated with a combination of topical 5% permethrin cream applied to all of the skin (including the face and scalp) and 1% malathion shampoo.
Discussion

There are few reports of scalp involvement by *Sarcoptes scabiei var hominis*.1–8 The hair bearing areas of the scalp are often not considered to be a site of active infestation by the scabies mite and traditionally topical treatments are not applied to this area.

Immunocompromised patients (due to drugs, disease or both) may present with crusted or “Norwegian” scabies. Crusting refers to the clinical appearance of the skin often around the hands due to a very high mite burden. In these circumstances the scalp should be considered as a potential reservoir of infection however neither of these two reported cases had such crusting. Immunosuppression is commonly but not always needed for scalp involvement.1

General advice from the Centre for Disease Control and Prevention (CDC) is to apply the cream from the neck down but in infants and young children to also treat the entire head and neck including the scalp. The CDC recommends that ivermectin should be considered in patients who have failed treatment with, or who cannot tolerate, Food and Drug Administration (FDA)-approved topical medications for the treatment of scabies but notes that it is not FDA approved.9 In 2012, Pharmac subsidised the use of ivermectin, subject to preconditions, for crusted scabies and institutional outbreaks.10

There are previous reports of scalp scabies mimicking seborrhoeic dermatitis3,4 and scabies involving the scalp in the setting of immunosuppression and connective tissue disease.1–4,6,8 Ivermectin has been used previously to treat three patients with dermatomyositis who had scalp involvement.3

In these two additional reported cases the mite burden was large enough to have been able to identify them by scalp scrapings and therefore the scalp would have constituted a significant source of re-infection for the patients and their families if left untreated.

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A rare complication of cytarabine therapy

Praveen Ramakrishnan Geethakumari, Sreejith G Nair

Clinical presentation—A 50-year-old woman developed pain, swelling and redness of palms and feet, 1 week after induction chemotherapy for acute myeloid leukaemia with cytosine arabinoside (cytarabine/Ara-C) and daunorubicin (7+3 regimen).

The lesions evolved with exfoliation; resolution after management with petrolatum emollients and pain control (Figures 1 and 2). What is the diagnosis?

Figure 1. Palmar exfoliation

Figure 2. Plantar erythema and desquamation seen secondary to exposure to cytosine arabinoside
Answer and Discussion—Palmar-plantar erythrodysesthesia, hand-foot syndrome (HFS), Burgdorf’s reaction or chemotherapy-induced acral erythema, manifests as bilateral extremity paraesthesias, pain, swelling, erythema, desquamation and rarely bullae or ulceration, 1 week to 3 months after chemotherapy with healing over weeks.

It has been associated with cytotoxic agents like pegylated liposomal doxorubicin, cytosine arabinoside, docetaxel, capecitabine and multi-kinase inhibitors like sorafenib and sunitinib.

Pathogenic mechanisms postulated include microvascular injury, free radical damage, and differences in temperature and eccrine gland distribution in the acral regions. Differential diagnoses include chemotherapy-induced Raynaud syndrome, drug reactions, paraneoplastic manifestations and graft-vs-host disease.¹

Prevention involves avoidance of friction, moisturisers and regional cooling. Manifest HFS entails dose interruption or reduction based on severity. Drugs evaluated for the prevention and management of HFS include pyridoxine, celecoxib, topical uridine, corticosteroids and dimethyl-sulfoxide.¹,²

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Short metacarpal and metatarsals

Gopal Chandra Ghosh, Bhimarey Kategary, Arnab Sarkar, Krishnarpan Chatterjee, Brijesh Sharma

Clinical—A 42-year-old female admitted to us with complaints of fever and cough (with purulent expectoration for 5 days) was diagnosed as a case of pneumonia. Her past history was uneventful. On general physical examination her height was 148 cm and she had a round face. Her left hand had short fourth fingers, and both feet had short fourth digits (Figures 1a & 1b). Her mental status and skin examination were normal. X-ray of the hands and feet revealed short left fourth metacarpal and both fourth metatarsals (Figure 2). Serum calcium, phosphate, parathormone concentrations were normal and NCCT of head was normal.

What is the diagnosis?
**Answer**—As the patient had clinical features of short height, round face, short fourth metacarpal and metatarsals—suggestive of Albright hereditary osteodystrophy (AHO) with normal biochemical parameters like serum calcium, phosphate and parathormone—we diagnosed her as a case of *Albright hereditary osteodystrophy (AHO) associated with pseudopseudohypoparathyroidism.*

**Discussion**—Albright hereditary osteodystrophy (AHO) is a rare metabolic disorder first described by Fuller Albright in 1942.¹ AHO is observed in pseudohypoparathyroidism (PHP) type Ia and Ic, which are two of the four subtypes of pseudohypoparathyroidism (PHP).

When AHO is present with normal levels of serum calcium, phosphorus and parathyroid hormone, then it is termed pseudopseudohypoparathyroidism (PPHP).² Short fourth and fifth metacarpals and metatarsals are characteristic features of Albright hereditary osteodystrophy (AHO). It is the phenotypic manifestation of the alpha subunit of Gs protein of PTH receptor deficiency.²

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**References:**
Systemic retinoids for recurrent keratoacanthomas

Faisal R Ali, John T Lear

A 64-year-old female presented with a 14 year history of recurrent keratoacanthomas occurring on sun-exposed sites. There was no family history of skin disease.

One particularly problematic lesion on the dorsal aspect of the right hand was excised and recurred five times. Given these multiple, failed excisions, acitretin (35 mg o.d.) was instigated. The size and infiltration of the keratoacanthomas improved greatly within 8 weeks and all lesions clinically resolved and no new lesions appeared after 6 months.

Retinoids are vitamin A derivatives, whose use is well established for hyperkeratotic disorders such as psoriasis and acne. Examples include isotretinoin, acitretin and etretinate. They purportedly interfere with expression of epidermal growth factor receptor genes.¹

Systemic retinoids can be of benefit in reducing the frequency of keratinising tumours, including keratoacanthomas, actinic keratoses and squamous cell carcinoma¹,² and their use should be recalled as prophylaxis in patients with recurrent such tumours, notably organ transplant recipients.

Common side-effects include teratogenicity, dryness of skin and mucosa and hepatic and cholesterol dysfunction.

Figure 1. Representative keratoacanthoma on right dorsal hand
Figure 2. Following 8 weeks acitretin (35 mg o.d.), size and induration of keratoacanthomas had much improved

Figure 3. Following 6 months acitretin, keratoacanthomas had clinically resolved

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References:


The Legacy of Percy Pease

Percy Pease was New Zealand’s first full-time paediatric surgeon and he firmly established paediatric surgery as a specialty in this country.

Born into humble beginnings in small-town rural South Africa in 1937, he faced great adversity before entering medical school.

Percy initially trained as a paediatrician but his qualifications were not recognised in South Africa. As he was in imminent danger of arrest there he crossed the border to Swaziland in 1968, leaving all his possessions behind.

He next went to England and gained his surgical qualifications before working at the Birmingham Children’s Hospital. During his time there the opportunity to travel to New Zealand arose.

When Percy arrived in 1974 the specialty of paediatric surgery was still in its infancy in New Zealand.1 For many years surgery on children was viewed as part of general surgery, and surgeons would operate on children relying on their general surgical training. These early pioneers made a remarkable contribution given their general scope and limited resources. A notable example was Henry Barrett, a provincial surgeon in New Plymouth who treated the first surviving child with oesophageal atresia in the southern hemisphere.2

Princess Mary Hospital in Auckland was Percy's first place of practise, purpose-built in 1918 for wounded American servicemen in the Pacific (Figure 1).3 It had an expected lifespan of 5–10 years but would become the home of the first Paediatric Thoracic Surgical Unit, under the bastion of Laurie Smith and Percy Pease. Percy was determined to deliver a paediatric patient-centred service, and together with Paul White, campaigned in the face of considerable opposition before the eventual opening of Starship Children's Hospital in 1991.

Percy’s reputation as a clinician and surgeon drew children from throughout New Zealand and the South Pacific. He undertook regular pro-bono medical missions to Samoa from 1979 and Tonga from 1981. He realised that many of the conditions he would encounter could be looked after in the first instance in the islands if the local surgeons were supported and upskilled.

A prime example of Percy’s immense contribution was his lifetime work in the sub-specialty of paediatric thoracic surgery, summarised in Table 1. He was quoted as saying, “Seeing a child smile up at you from their hospital bed after life-changing surgery makes it all worthwhile”.

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Table 1. Summary of the lifetime contribution to paediatric thoracic surgery by Percy Pease

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of cases</th>
<th>Clinical details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchoscopy</td>
<td>526</td>
<td>78 for foreign body.</td>
</tr>
<tr>
<td>Oesophagoscopy</td>
<td>957</td>
<td>58 ingestions, 33 foreign body, 10 caustic stricture.</td>
</tr>
<tr>
<td>Congenital cystic adenomatous malformation</td>
<td>40</td>
<td>5 pleuropulmonary blastoma, 1 bilateral, 1 demise with cerebral metastasis.</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>32</td>
<td>Lobectomies</td>
</tr>
<tr>
<td>Bronchogenic cyst</td>
<td>5</td>
<td>Resections</td>
</tr>
<tr>
<td>Sequestration</td>
<td>11</td>
<td>4 Extra-lobar sequestration, 7 intra-lobar sequestration</td>
</tr>
<tr>
<td>Congenital lobar emphysema</td>
<td>6</td>
<td>1 subsequent lipoblastoma</td>
</tr>
<tr>
<td>Congenital diaphragmatic hernia</td>
<td>25</td>
<td>20 congenital diaphragmatic hernia, 2 eventrations, 1 bilateral, 2 Morgagni, 1 traumatic rupture.</td>
</tr>
<tr>
<td>Mediastinal tumour</td>
<td>18</td>
<td>9 median sternotomy – 14 anterior 3 posterior 1 anterior &amp; posterior</td>
</tr>
<tr>
<td>Congenital oesophageal pathology</td>
<td>65</td>
<td>57 OA / TOF, 1 oesophageal web, 2 oesophageal lungs, 5 oesophageal duplication cysts.</td>
</tr>
<tr>
<td>Thoracic tumours</td>
<td>15</td>
<td>9 neuroblastoma, 2 ganglioneuroma, 1 plasma cell granuloma, 2 Asken tumour, 1 bronchial carcinoid tumour</td>
</tr>
</tbody>
</table>

Percy had a gregarious personality and a philosophy that encouraged co-operation and respect in the treatment of patients. Throughout his 46 years of paediatric surgical
service he maintained strong and supportive working relationships with clinicians and colleagues within his hospital and around the country. He inspired and trained many of the current Paediatric Surgeons around the country.

With his recent passing an annual symposium bearing his name is dedicated to maintaining the ideals of co-operation, support and respect amongst colleagues, qualities he very much encouraged.  

He is fondly remembered and missed by colleagues and his legacy will continue. As Percy himself aptly surmised, “There have been lots of laughs and lots of tears. I’ve enjoyed every minute”.

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References:
RMO patient safety forums in New Zealand: agents for change

The value of Resident Medical Officers (RMOs) input in improving the quality and safety of clinical care is increasingly recognised but remains under-utilised. Junior medical staff are often poorly prepared to contribute to quality and safety improvement efforts despite often having a strong interest in developing the skillset to contribute to change and frequently being “in the best position to identify how things could work better on the ground”.

RMOs regularly change clinical runs, hospitals, District Health Boards (DHBs) and sometimes even countries. It is well recognised that patient safety suffers when medical staff change runs particularly at the beginning of a new academic year. This regular rotation not only means that RMOs are at increased risk of being involved in adverse events but are well placed to recognise patient safety issues as the ‘fresh eyes’ in the organisation and are able to compare things that work well (or don’t) with other places they have worked. A survey of junior doctors in the UK discovered that two-thirds recognised system problems at least weekly or daily. Not capitalising on these observations is a “disservice to doctors in training and the patients they care for.”

A key method for improving quality of care is being able to voice concerns (to the appropriate staff), learn from avoidable adverse events, and be involved in the process of finding ways to prevent future errors. This can be achieved through interactive workshops, courses and forums.

Facilitated forums amongst peers provide a chance to raise patient safety issues and experiences from real life events in a confidential and professional environment. They support reflection, highlight how systems, processes and human factors combine to result in adverse events and provide an opportunity to discuss potential solutions and ways of reducing patient harm in the future. The overriding aim of RMO patient safety forums is to empower junior doctors to talk freely about the issues they experience and to lead to actions that address safety issues. Regular forums allow for review of progress against issues raised previously, recording new concerns, and evaluating the value of the forums.

Such RMO patient safety forums have been initiated at Capital and Coast and Hutt Valley DHBs in slightly different formats. At Capital and Coast DHB the RMO meetings are joined by the Patient Safety Officer and Deputy Chief Medical Officer, whereas the sessions at Hutt Valley DHB are facilitated by a Medical Registrar with an interest in patient safety. This collaboration ensures that senior staff are made aware of RMO patient safety concerns and can take into consideration their suggestions for improvement in the hospital. Involving senior staff and managers gives the group greater ability to make changes and a different perspective on the hospital’s safety and quality activity, policies and procedures.

An advantage of the Capital and Coast forums is having senior medical officers involved with specific authority to take action and follow up on issues. However
having more senior staff present could prevent RMOs from disclosing some problems. A benefit from the Hutt meetings is protected six-weekly time slots; whilst it has been a challenge at Capital and Coast to find a time RMOs can regularly attend.

Examples of improvements in the quality of clinical care achieved through the RMO patient safety forums at Capital and Coast and Hutt Valley DHBs include:

- Follow up of concerns regarding unrealistic expectations being placed on junior medical staff
- Improved hospital orientation for RMOs
- Improved communication with community pharmacists to prevent patients from being able to use a controlled drug prescription twice
- Better understanding of the roles and requirements of phlebotomists and RMOs
- More efficient processes for blood transfusions
- Weekly drop in sessions run by Cardiology Registrars for RMOs to discuss interesting or complex ECGs

The RMOs have suggested topics for guest speakers to present at interactive sessions. Topics covered have included medico-legal aspects of documentation, basics of quality and clinical governance, use of Early Warning Score and pre-assessment clinics.

There is commonly a sense of disconnection between clinical staff and hospital management with junior doctors tending to believe managers prioritise performance measures over patient safety. Our experience has demonstrated that involving senior management staff in RMO forums leads to improved communication, understanding and collegiality between both groups.

Clinical staff involved in adverse events are often referred to as the second victim, and can suffer emotional distress which can have a negative impact on their performance at work and general wellbeing. Evidence suggests junior doctors benefit from assistance with reflection rather than formal reporting systems alone, and that learning from errors is maximised when constructive feedback is offered. Doctors report that discussing errors and adverse events they have been involved in with colleagues is the most valuable and useful resource in learning from and coping with the experience.

The RMO patient safety forums provide a unique accessible safe place to raise and address concerns and have led to four key improvements in addressing patient safety and RMO training:

- Better communication and support around medical errors and disclosure of errors between RMOs, senior clinical and managerial staff;
- Tailored teaching addressing specific needs requested by RMOs;
- Addressing gaps in induction and orientation; and
- A better understanding of patient safety hospital procedures (such as using incident forms).
We hope that those working in other DHBs will be encouraged to involve RMOs more in quality improvement and consider introducing their own patient safety forums or further evaluate those already in place.

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Coroner’s inquest

*From Dominion Notes. Published in NZMJ 1912;9(40).*

AUCKLAND, April 5.

An extraordinary state of affairs was disclosed at an inquest yesterday touching the death of Wm. Cunningham, an elderly man who died early on Monday morning in a place off Cook Street, behind the Market Hotel.

James Dunn said he had lived with deceased for the past two and a half years in a loose-box at the rear of the Market Hotel. He thought this loose-box belonged to the owner of the Market Hotel.

The police report stated that the body of deceased was in a disgusting state. The stable hut where he had lived in company with Dunn was filthy. It smelt badly and appeared to require the immediate attention of the Health Department.

“This is an amazing state of affairs,” remarked the coroner, “with health officers and sanitary arrangements. I want the jury to realise the manner in which these officers perform their duty.”

A juryman: How can these people live in such places? (To the last witness) Did you pay rent?

Dunn: No, but I worked for the hotel people now and again.

The coroner: It is a shocking state of affairs. It will be for the Health Department to say whether a man was ever told off to visit this place. It is quite evident that it has not been visited for years by a sanitary inspector.

A juryman: I would class this loose-box as a dog-box.

In accordance with the medical testimony, a verdict of death from fatty degeneration of the heart was returned. The jury added the following rider:--

"We desire to call the attention of the Hon. the Minister in charge of the Health Department, and also the attention of the "Mayor and Councillors of the City of Auckland to the loathsome and insanitary condition of the loose-box or stable situated off Cleave's Avenue, at the back of the Market Hotel, Auckland."

When questioned by a reporter last evening in connection with the above rider, the District Health Officer said: “The inspection of all the premises in the city, which such a rider implies, requires inspectors, and inspectors require money. If the citizens of Auckland wish to have such inspection made, let them elect on their local bodies men who are prepared to spend the necessary cash and the inspection will be carried out.”
Prevalence of depression in patients referred with snoring and obstructive sleep apnoea (OSA)

Sleepiness, the main symptom of OSA, can often overlap or be confused with fatigue; fatigue indicating tiredness and lack of energy because of mental, physical, and emotional disorders. This study aims to review the relationship, if any, between OSA and depression. Ninety-seven percent of 240 patients referred because of snoring to a sleep clinic participated.

The subjects responded to two depression questionnaires. Thirty-two percent had a positive response to the 14-question Hospital Anxiety and Depression Scale (HADS) test. This result correlated significantly with the Mini-International Neuropsychiatric Interview (MINI) test. Fifty-three percent had either doctor-diagnosed depression (28%) and/or a positive HADS or MINI (25%). The researchers conclude “depending on classification, 32–53% of patients with snoring had depressive symptoms or were on treatment, which is significantly greater than the Australian average of 21%.”

Internal Medicine Journal 2013;43:630–634.

Age-adjusted D-dimer cut-off values in older patients with suspected venous thromboembolism

When venous thrombosis is suspected, the D-dimer assay is valuable. If it is not raised, venous thrombosis is unlikely. The conventional cut-off value is 500µg/L. If this level is not exceeded, most clinicians would not proceed to imaging. However, D-dimer levels increase with age and the use of an age-adjusted cut-off value (age×10µg/L in patients aged 50 years or more) may be a safer and more efficient strategy.

This meta-analysis reviews 13 studies involving more than 12,000 patients with suspected thromboembolism in whom D-dimer testing [using both conventional (500µg/L) and age-adjusted (age×10µg/L in patients aged >50 years) cut-off values] and reference testing were performed.

The conclusion was that the specificity of D-dimer testing increased substantially when the age adjusted cut-off value was applied and was more than doubled in the eldest patients (>80 years). The researchers estimate that imaging could be correctly avoided in 30 to 55% of elderly patients with suspected thromboembolism. However, if the clinical probability is high, they recommend imaging irrespective of the assay results.

BMJ 2013;346:f2492.
Nurse practitioners to the rescue?

Apparently the U.S. health care system is at a critical juncture in health care workforce planning. The nation has an acknowledged shortage of primary care physicians.

It has been suggested that expanding the supply and scope of practice of nurse practitioners will address this problem. Unsurprisingly these proposals are controversial. This paper reports on a national postal-mail survey of 505 physicians and 467 nurse practitioners. The questionnaire included scope of work, practice characteristics, and attitudes about the effect of expanding the role of nurse practitioners in primary care. The response rate was 61.2%.

Physicians reported longer working hours, seeing more patients, and earning higher incomes than did nurse practitioners. A total of 80.9% of nurse practitioners reported working in a practice with a physician, as compared with 41.4% of physicians who reported working with a nurse practitioner.

The nurses were more likely than physicians to believe that they should be allowed hospital admitting privileges and be paid equally for the same clinical services. The conclusions were that the physicians and nurse practitioners do not agree about their respective roles in the delivery of primary care.

Controversial, as expected.

Erratum

**Letter:** Lessons from the February 2011 Christchurch Earthquake for the training and preparation of Post Graduate Year 1 Doctors

*Dale C Sheehan, John Thwaites, Blair York, Jaejin Lee*

19-Apr-2013 - Vol 126 No 1373

Please be advised that the authors in bold above were missing from the original publication.

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