A retrospective audit of the characteristics and treatment outcomes in patients with diabetes-related charcot neuropathic osteoarthropathy

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ABSTRACT
AIMS: To review the characteristics, management and outcomes one year after diagnosis in patients with diabetes related charcot neuropathic osteoarthropathy (CN) treated at the Diabetes Podiatry service, Waitemata District Health Board (WDHB) between 2000–2014.

METHODS: Patients with diabetes and recorded diagnosis of CN were identified from the podiatry service records. Clinical details were retrospectively obtained from WDHB databases and patient medical records.

RESULTS: Forty-one patients were included, 31 had type 2 diabetes, 10 had type 1 diabetes. At presentation, the median duration of all-type diabetes was 15 years. The median time from symptom onset to diagnosis was 17 weeks. Symptoms at presentation were: oedema (49%), warmth (73%), erythema (17%), swelling (90%) and pain (60%). Concomitant ulcers were present in 32%, deformities 83%, osteomyelitis 2% and septic arthritis 2%. Mean time to ambulation in modified shoes was 21.3 weeks (±11.5). Complication rates one year from diagnosis for ulcers, osteomyelitis, amputations and all-cause mortality were 34%, 2%, 2% and 5% respectively.

CONCLUSION: Time to diagnosis of CN was shorter than previously reported, though the high rate of deformities still suggests a significant delay in diagnosis. Increased education of healthcare professionals and people with diabetes-related neuropathy is important to ensure early diagnosis and appropriate management to reduce deformities and complications.

Charcot neuropathic osteoarthropathy (CN) is a degenerative arthropathy affecting single or multiple joints resulting from significant peripheral neuropathy. This leads to fractures or dislocations of the bones and joints of the foot. It can result in minimal structural damage or lead to longstanding deformities, ulcerations, osteomyelitis and lower-limb amputations. The most common etiology for CN is diabetes mellitus and it is universally associated with peripheral neuropathy. It tends to affect males more than females in their fifth and sixth decade of life, and the duration of diabetes prior to developing CN is at least 10 years. CN has a low prevalence of 0.08% in the general diabetic population but up to 13% in high-risk populations, however the true incidence is unknown and is partly related to the high rate of misdiagnosis. The pathogenesis of CN is unknown, however repetitive trauma is thought to cause an increase in proinflammatory cytokines, leading to persistent local osteolysis and bony destruction.

Active CN presents as warmth, swelling, erythema and pain in the foot. Patients may have no recollection of trauma to the
foot or they may recall only minor trauma such as a sprain or a twisting injury. Early active CN is a clinical diagnosis and often misdiagnosed as infection, gout or deep vein thrombosis. The average delay in diagnosis has been reported to be around 29 weeks. Al-Busaidi et al recently described two cases in Christchurch who received a late diagnosis of CN and who both experienced residual bony deformities. The diagnostic delay in these cases was four and 12 months. Their patients were initially treated as a sprain, DVT, cellulitis and arthritis prior to receiving the diagnosis of CN.

In early active CN, x-ray shows no or minimal abnormalities. However in established CN, x-ray features include capsular distention, osseous fragmentation, peri-articular debris formation, subluxations/dislocations and fractures. Delay in diagnosis and treatment leads to an increased risk of complications and long-term deformities. More recently, magnetic resonance imaging (MRI) has been recommended in early CN to confirm the diagnosis and initiate early treatment. MRI has been shown to detect stress injuries, micro fractures and soft tissue oedema in patients with normal x-rays.

The mainstay of treatment for CN is offloading and immobilisation of the foot with the goal of treatment being a stable, plantigrade foot that can be easily shod. Immobilisation can be with a total contact cast or removable cast walker and duration depends on the clinical assessment of healing. One retrospective study by Armstrong et al found that the mean duration of immobilisation was 18.5 weeks while duration to return to shoe gear was 28.3 weeks. Protective weight bearing is required after the acute episode and lifetime surveillance is required. Surgical intervention is reserved for severe deformities or concurrent osteomyelitis. Pharmacological measures such as bisphosphonates have been studied, however there is no strong evidence to support routine use.

The aim of our audit was to review the characteristics of patients with diabetes-related CN treated at the Waitemata District Health Board (WDHB) Diabetes Podiatry service between 2000 and 2014. We also reviewed time to diagnosis, management and outcomes one year from the time of diagnosis.

Methods

Study design and data sources
This was a retrospective audit conducted by the Diabetes Service at WDHB whose podiatry service has maintained a list of patients treated for CN since 2000. Those who presented with an active CN between the years 2000 and 2014 were included in our study. We reviewed the electronic hospital database for demographic data and documentation of clinical encounters. If the CN diagnosis pre-dated electronic records, paper documentation was then reviewed. Clinical data such as HbA1c, diabetes management and complications were taken from a clinical encounter, usually within weeks of the date of diagnosis of CN. The presence of peripheral neuropathy was taken from documentation of this during a clinical encounter. Referral letters to the service were not archived and we therefore did not have the ability to review primary care or community records. The data were cross-referenced between different sources to ensure accuracy and completeness.

As this was a retrospective review, some clinical data could not be obtained, though it was not felt that this affected the integrity of the study. Ethics approval was not required.

Definitions
Cases of active CN were identified by the presence of warmth, erythema, acute swelling or pain with or without deformities in the affected foot in the absence of infection as the primary diagnosis. Date of diagnosis was taken as the earliest point a clinical or radiological diagnosis of CN was made. Time to diagnosis was calculated based on duration of reported symptoms at first presentation to the time of review and confirmation of diagnosis by secondary services as documented in the patient records.

Statistical analysis
Descriptive statistics, including frequencies and proportions, were used to summarise baselines variables and the findings of our study. This was done using SPSS version 23 (SPSS, Chicago, IL, USA).
Results

A total of 41 active CN cases were included and the patient characteristics are summarised in Table 1. Five patients were recorded twice as they also developed CN in the contralateral foot. No patient presented with bilateral acute CN. A disproportionate number were male and identified with Pacific Island ethnicity. Eighty-three percent required insulin therapy for diabetes management.

Table 1: Patient characteristics.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>(N=41)</th>
</tr>
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<tbody>
<tr>
<td>Mean age, yrs (range)</td>
<td>54 (34–73)</td>
</tr>
<tr>
<td>Gender, n (%)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28 (68%)</td>
</tr>
<tr>
<td>Diabetes type, n (%)</td>
<td></td>
</tr>
<tr>
<td>Type 2</td>
<td>31 (76%)</td>
</tr>
<tr>
<td>Type 1</td>
<td>10 (24%)</td>
</tr>
<tr>
<td>Diabetes treatment, n (%)</td>
<td></td>
</tr>
<tr>
<td>Insulin therapy</td>
<td>34 (83%)</td>
</tr>
<tr>
<td>Oral therapy</td>
<td>6 (15%)</td>
</tr>
<tr>
<td>Diet control</td>
<td>1 (2.5%)</td>
</tr>
<tr>
<td>Ethnicity, n (%)</td>
<td></td>
</tr>
<tr>
<td>NZ European</td>
<td>18 (44%)</td>
</tr>
<tr>
<td>Pacific Island</td>
<td>9 (22%)</td>
</tr>
<tr>
<td>Asian</td>
<td>7 (17%)</td>
</tr>
<tr>
<td>NZ Māori</td>
<td>3 (7%)</td>
</tr>
<tr>
<td>Other</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>Median duration of diabetes, yrs (range)</td>
<td>15 (1–47)</td>
</tr>
<tr>
<td>Median HBA1c, mmol/mol (range)</td>
<td>70 (36–178)</td>
</tr>
</tbody>
</table>

The majority of our referrals (46%) were from within the Diabetes Service, which included physicians, nurse specialists and podiatrists. Nineteen percent were from the orthopaedic service, 10% from community podiatrists, 5% from inpatient services, 5% from rheumatologists and the remainder were from uncertain sources. We were unable to ascertain how many were referred from primary care direct to the Diabetes Service.

The time from symptom onset to diagnosis was variable as shown in Figure 1. The median time to diagnosis was 17 weeks (1–70 weeks). Twenty (41%) patients recalled an episode of trauma preceding the development of CN. Swelling was the most common symptom at presentation in 90%; followed by warmth, pain, oedema and erythema in 73%, 60%, 49% and 17% respectively. Eighty-three percent of patients had deformities of the foot at the time of presentation. Thirty-two percent had concurrent ulcers on the ipsilateral foot. One patient presented with osteomyelitis complicating an acute CN requiring amputation and one patient required washout of a septic joint complicating a heel ulcer at presentation.

All patients underwent plain x-ray of the affected foot and 22% also had an MRI performed to confirm the diagnosis or to rule out coexisting osteomyelitis. Ninety-three percent (38/41) had abnormal x-ray findings at the time of diagnosis, with bony destruction and subluxation being the most common findings at 50% (19/38) and 37% (14/38) respectively. A number of different joints in the foot can be affected as shown in Figure 2. In our cohort, 62 joints in our 41 patients were radiologically confirmed as being affected by CN. The tarsometatarsal
joint was the most commonly affected at 35% (22/62), followed by the tarsal and metatarsophalangeal joints at 15% (9/62) each. The ankle joint showed active involvement in 13% (8/62), with the remainder involving the metatarsals and tarsal-ankle joint equally.

Twenty-three (56%) patients were treated in a total contact cast while removable casts were used in the majority of the remaining patients. The mean time till patients were ambulatory in modified shoes was 21.3 weeks (± 11.5 weeks). Within one year from diagnosis of CN, 17 (34%) patients had developed a foot ulcer, two patients suffered a further fracture, one developed osteomyelitis and one required an amputation. There was an all-cause mortality of 5%.

Discussion

This audit of CN presentation and management at WDHB is the largest series published in New Zealand. While acknowledging that it does not reflect the true incidence, partly due to patient relocation, misdiagnosis and that some patients may have been treated privately, we feel that we have captured most, if not all, new cases in our DHB especially over recent years. All patients with CN and diabetes should be referred to a secondary podiatry service without delay and our service works closely with the orthopaedic and vascular service to optimise outcomes.

Our audit found that the characteristics and demographics of our patients presenting with active CN generally align with international studies. The time to diagnosis of 17 weeks is shorter than described in previous studies. The early symptoms of CN are often attributed to minor trauma or possible infection and disregarded by patients, which can delay true diagnosis. We were unable to ascertain all the direct causes of delay in our retrospective audit.

The early stages of active CN requires a high index of suspicion and if treated early, long-term deformities and complications may be prevented. Figure 3 shows the joint destruction that can occur with CN particularly when there is a delay in diagnosis. Figure 3a shows the normal appearances of the foot with soft tissue swelling only at the time of symptom onset in one of the patients in our study cohort. When the CN
was diagnosed four months later, the x-ray showed the typical destructive CN changes with collapse of the midfoot, destruction of the tarsometatarsal and ankle joints, and periosteal new bone at the bases of the first to fourth metatarsal. Once joint destruction has occurred, it is irreversible; therefore it is critical that offloading of the foot occurs early. Most of our patients presented with established deformities suggesting a significant delay to diagnosis and appropriate treatment.

MRI was underutilised as a diagnostic tool in our cohort. WDHB had limited access to MRI imaging from 2003 up until 2007. This likely impacted on the low numbers of MRIs performed in our patients in the early years. When performed, this was to rule out coexisting infection in cases when the diagnosis of CN was evident on x-ray.5

There was an underrepresentation of general practitioner (GP) referrals in our patient group suggesting GPs may refer these patients to a number of secondary services such as emergency departments, general physicians or orthopaedics, particularly since infection is a common misdiagnosis. If there is a high suspicion of CN without coexisting infection, we recommend a direct referral to the Diabetes Podiatry service.

While patients had a low risk of developing amputations and were ambulatory in modified footwear within five months, a significant number nevertheless developed foot ulcers. CN leads to collapse of the midfoot and results in bony prominences that lead to overlying ulceration. Ulcers predispose patients to infection and potentially amputation. Treatment of CN and ulcers requires casting and intensive, often weekly, review and input from a specialist diabetes podiatry service and long-term follow-up.

Conclusion

While CN has a low prevalence among patients with diabetes even in those with peripheral neuropathy, it is a very significant complication and has important implications for the individual. By diagnosing CN in the early stages, deformities and subsequent complications may be prevented. Our study and the cases reported by Al-Busaidi et al4 highlight that there are still significant delays in diagnosis in these patients.

It is important that not only health professionals but also patients are aware of the presenting symptoms of CN and the need to present for early treatment. An opportune time for this education could be during their annual diabetic foot check with their GPs or diabetes teams. A high level of clinical suspicion is needed to suspect the diagnosis in patients with long-standing diabetes and peripheral neuropathy.

As stated in the BPAC New Zealand guidelines, a suspected acute presentation of CN is considered an emergency and warrants prompt referral directly to a specialist diabetes podiatry service. An MRI scan should now be considered an essential part of the work up of a swollen, erythematous foot in a patient with diabetes-related peripheral neuropathy.5 Timely offloading of the foot is crucial to preserve the architecture of the foot and prevent complications such as deformities, ulcers and amputation. Once joint destruction occurs, this is irreversible, therefore early diagnosis is critical.

Any medical professional presented with a patient with diabetic peripheral neuropathy and an acutely red, swollen and warm foot needs to consider Charcot neuropathic osteoarthropathy as a diagnosis and refer urgently to a specialist diabetes foot service.
Competing interests: Nil.

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REFERENCES: