Myocardial infarction following sclerotherapy in a patient with a patent foramen ovale

Timothy A C Snow, Julian P McEntee, Sally C Greaves, Harvey D White

Abstract

A case is reported of myocardial infarction occurring following sclerotherapy for varicose veins in a patient with a patent foramen ovale (PFO). It is believed that microemboli crossed the PFO as a result of the procedure and caused coronary artery embolisation, resulting in the symptoms, electrocardiographic and biochemical evidence of myocardial infarction.

Case report

A 61-year-old female with a past medical history of osteopenia, varicose veins and a hysterectomy was referred to our department having developed chest pain following sclerotherapy; she had received 10 ml of microbubble sodium tetradecyl foam to her recurrent bilateral lower leg varicosities.

Following the procedure as she walked back to the changing room she developed sudden onset of central chest pain which radiated to her throat associated with light-headedness. Observations were normal and she was given oxygen and glyceryl trinitrate spray by the staff with some improvement of the chest pain.

On arrival at the hospital the pain had eased. Her blood pressure was 140/70 mmHg, pulse 66 bpm and saturations 98% on air. There was a soft systolic non-radiating murmur at the left sternal edge with normal splitting of the second heart sound, clear chest, normal JVP and no pedal oedema. Her initial ECG showed sinus rhythm with a normal axis, at a rate of 60 bpm and isolated T-wave 1 mm inversion in lead V2. However over 24 hours this progressed to widespread deep inversion (deepest 6 mm in V5) (Figure 1) in at least two coronary artery territories. Chest X-ray was normal. Her full blood count and renal function tests were normal. D-dimers were mildly elevated at 740 mcg/L (normal <500 mcg/L).

High sensitive troponin T rose from 151 ng/L (normal ≤14 ng/L) on admission to 222 ng/L after 5 hours thus she was treated as having a non-ST elevation myocardial infarction.\(^1,2\) Angiography showed normal coronary arteries with a right dominant system.

The echocardiogram, performed within 24 hours with good imaging, showed normal left ventricular size and function with no regional wall abnormalities associated with a stress cardiomyopathy (Takotsubo). All valves appeared normal. An agitated saline contrast study was negative at rest but upon valsalva manoeuvre a moderate amount of contrast was seen in the left heart consistent with a significant (PFO). This was confirmed by transoesophageal echocardiography as a moderate sized PFO with spontaneous left to right shunting with no atrial septal defect (Figure 2).
The patient was diagnosed as having a non-ST elevation myocardial infarction, thought to have been caused by micro-emboli from her sclerotherapy crossing the PFO and entering the coronary circulation. As this was felt to be related to sclerotherapy, we chose not to start anti-coagulation therapy but commenced her on aspirin and short-term clopidogrel.
She was considered for percutaneous closure of her PFO as an outpatient. On review however, it was felt that due to the clear association with sclerotherapy the aneurysmal nature of her atrial septum, which could increase the likelihood of future device erosion, and the lack of benefit shown for PFO closure by the recently published CLOSURE I trial\(^3\) that her PFO should not be closed.

**Discussion**

Sclerotherapy is a common and relatively safe procedure for the treatment of varicose veins.\(^4\) Chest tightness following foam injection is rarely associated (<0.004%) but to our knowledge there have been no cases of proven myocardial infarction or ischaemia. There are however reports of transient ischaemic attacks and strokes post-procedure. These patients have later been identified to have a patent foramen ovale\(^5\) which is prevalent in approximately 25% of the normal population.\(^6\) It is believed that micro-emboli had crossed through the defect into the left sided circulation ending in the cranial arteries.

Although to our knowledge there are no previous cases of emboli entering the coronary circulation following sclerotherapy, there are cases of thromboemboli from deep vein thrombosis causing myocardial infarction.\(^7,8\) It has also been demonstrated that in patients with stroke associated with a PFO that myocardial scars in multiple coronary territories are seen on cardiac magnetic resonance imaging.\(^9\)

Despite the lack of clinical evidence of embolisation to her other organs, had further imaging been performed areas of microinfarction may have been seen. The prevalence of asymptomatic micro-infarctions in the brain in patients with atrial fibrillation or following carotid angioplasty or endarterectomy is well documented.\(^10,11\)

**Conclusion**

Chest tightness following sclerotherapy is a rare event. However as demonstrated in this case, the possibility of myocardial infarction caused by paradoxical thromboembolism to the coronary arteries through a PFO should be considered as a potential diagnosis.

**Author information:** Timothy A C Snow, Cardiology M.O.S.S; Julian P McEntee, Registrar; Sally C Greaves, Consultant Cardiologist; Harvey A White, Professor, Green Lane Cardiovascular Service, Auckland District Health Board, Auckland

**Acknowledgement:** We thank Dr Jamie Voss for provision of transoesophageal echo images.

**Correspondence:** Professor Harvey D White, Green Lane Cardiovascular Service, Auckland City Hospital, Level 3, Building 32, Private Bag 92024, Victoria St West, Auckland 1142, New Zealand. Fax: +64 (09) 6309915; email: HarveyW@adhb.govt.nz

**References:**


