The patient reported having palpitations, heat intolerance, emotional lability and weight loss of 20 kg in the preceding six months. Laboratory testing showed: troponin-T 0.42 (normal <0.03), thyroid-stimulating hormone (TSH) <0.01, and thyroxine (T4) >100. The haemoglobin was normal and her pregnancy test was negative. She was assessed as having thyrotoxicosis with non-ST-segment elevation myocardial infarction (NSTEMI) and was treated with an infusion of unfractionated heparin, aspirin, simvastatin, atenolol, Lugol's iodine, carbimazole and cholestyramine. The pain resolved on this treatment. Several hours later the pain recurred and therefore coronary angiography was done. This demonstrated severe stenosis of the left main stem. A decision to perform emergency coronary artery bypass graft surgery was made. However, the possibility of coronary vasospasm was strongly considered and consequently surgery was cancelled. It was decided that the treatment of thyrotoxicosis be continued, with angiography repeated later. She was subsequently diagnosed with Graves’ disease.

With treatment of hyperthyroidism the symptoms resolved and her T4 levels fell. On day 9 coronary angiography was repeated; severe left main stem stenosis was still present. She was euthyroid at that stage. The beta-blocker was stopped and nitrates and dihydropryidine calcium-channel blockers were added to the treatment regimen. Angiography was done again on day 12, with a marked improvement in the calibre of the vessel. Subsequent ECGs revealed resolution of the initial changes and the patient remained pain free at discharge on day 18. She has been followed up closely in the cardiac clinic and had a normal exercise ECG. She remains euthyroid and symptom free.

**Discussion**

This report highlights a case of thyrotoxicosis due to Graves’ disease causing coronary vasospasm and NSTEMI – the first report of its kind in South Africa.

Angina has been found to be associated with thyrotoxicosis in up to 25% of patients in one series. In patients with coronary arteroma, the presence of angina reflects a mismatch between myocardial oxygen supply and demand due to the increase in cardiac workload and contractility associated with thyrotoxicosis. Al Suwaidi et al. documented 18 published reports, with a total of 34 patients, of angina pectoris associated with coronary spasm due to thyrotoxicosis, that have been confirmed angiographically since 1979. They found that the cardiac presentation varied from angina and myocardial infarction to ventricular arrhythmias, cardiogenic shock and cardiac arrest. They also noted that in these patients the manifestations of hyperthyroidism were either scarce or absent, which was also noted in the patient presented above.

Choi et al. carried out a retrospective analysis of 325 patients presenting with coronary spasm between 1994 and 2000 in Korea. They reported that of these, 8 had hyperthyroidism due to Graves’ disease. In three patients, the left main stem coronary artery was involved in the spasm. Among these patients, five were female, and all the female patients were below the age of 51. All of these patients were treated with anti-thyroid medication, calcium-channel blockers, and long-acting nitrroglycerines. They remained free of chest pain during the median follow-up period of 5 years. Resolution of chest pain with anti-thyroid treatment has also been reported by other authors.

The mechanism of coronary vasospasm in hyperthyroidism remains unknown.

**Conclusion**

Hyperthyroidism should be considered in the differential diagnosis of chest pain due to coronary spasm, particularly in young women. Thyroid function tests should be routine in patients presenting with chest pain due to coronary spasm.