A 52-year-old man with a past history of alcohol dependence presented to the emergency department with vomiting and a generalized epigastric/chest pain. He had abstained from alcohol for 10 months but had relapsed a month ago and presented for detoxification and relief of his symptoms. The description of his symptoms was non-specific and it was difficult to differentiate between ischemia and gastritis. His background included several admissions to the detoxification unit and rehabilitation programmes, gastric ulcer on endoscopy with previous admissions for haematemesis and a 30-pack year smoking history.

Cardiovascular examination revealed a 1/6 systolic murmur along the left sternal edge and normal venous pressure. Auscultation of the chest was normal. Abdomen was tender in the epigastric area with active bowel sounds and no organomegaly. ECG showed sinus rhythm, normal axis, and inversion of T waves in II, III, aVF and V2-V6 (Figure 1). The patient was given sublingual nitroglycerine (GTN), an antacid and transferred to the detoxification unit. On arrival, another ECG was done which was normal with no evidence of the previously mentioned abnormalities. The patient had no symptoms when the second ECG was done an hour later. The blood work-up from the emergency department showed normal electrolytes and normal cardiac enzymes. Serial cardiac enzymes were normal. On further questioning, he reported symptoms of stable/exertional angina over the last three months. A preliminary diagnosis of Wellens variant was made and the patient was urgently referred to the cardiology department for further investigations. Under advice from the cardiology services, stress testing was done a week later. The stress test was positive and showed inferolateral ST depression associated with chest pain during the test that was relieved with GTN. He was put on the routine angiogram list. The angiogram done showed a 70% (moderate-severe) mid-left anterior descending coronary artery lesion (Figure 2) and a 50% (mild-moderate) disease in the right coronary artery. The left anterior descending coronary artery lesion was immediately stented. The patient is currently on aspirin, metoprolol and ranitidine.

Discussion

A recently published report clearly describes the syndrome, criteria for diagnosis and discussion of its implications.³ Previously described Wellens’ variants have been changes in the precordial or inferior leads only.²⁻³

In our patient biphasic T wave was seen only in lead II, deep inverted T waves extended from leads V3 to V6 and there was inversion of T waves in leads I, II, III, aVF. These coexisting changes have not specifically been reported in the literature on this syndrome.¹⁻³

Of interest was the patient being referred to the detoxification unit and not to the cardiology services. There are only...
five reviews of this syndrome in the literature, and interestingly enough, three of them are in emergency medicine journals. It is of paramount importance that the syndrome be recognized as it allows rapid and appropriate aggressive management, prevention of prolonged emergency care or inappropriate conservative therapy as these patients may be destined for a massive anterior wall myocardial infarction, and must be managed accordingly. The other issue of interest was the stress testing. The review by Tandy et al advises that stress testing be cautioned in the presence of suspected left main or left main-equivalent lesions. Instead, timely angiography should be considered in these patients. However, the review also advises that provocative testing, if done at all, should be done in conjunction with the cardiology department. Issues surrounding this uncommon syndrome are that it may not be fully recognized in the asymptomatic patient and yet it has serious implications if not appreciated and if appropriate actions are not taken.

References
1. de Zwann C, Bar FW, Wellens HJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. Am Heart J 1982;103:730-6