Country cardiograms case 45: Answer

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Interpretation of the electrocardiogram (ECG) shown in Figure 1 (on page 144) reveals normal sinus rhythm at a rate of about 75 beats/min. The QRS complex is normal in both duration (0.06 s) and axis (+30°). The corrected QT interval is prolonged (0.532 s). There is diffuse T-wave inversion in limb leads I, II, aVL and aVF, and precordial leads V2 through V6. There is no ST-segment elevation, nor are there any pathologic Q waves. The clinical history and T-wave inversion are suggestive of an ischemic etiology. Although in the absence of ST elevation one cannot comment on the coronary arterial distribution and precise myocardial territory involved, the diffuse nature of the ECG changes implies the involvement of a large portion of the myocardium. This would be unusual in occlusive atherosclerotic ischemic heart disease. However, it can occur in patients with a dominant left anterior descending artery that wraps around the apex of the heart, supplying the inferior wall as well as the anterolateral walls.

Based on the clinical history, ECG changes and laboratory investigations, the initial diagnosis was non–ST elevation myocardial infarction, and the patient was transferred to a tertiary care centre. She underwent urgent cardiac catheterization, which revealed patent coronary arteries with minimal atherosclerosis. The left ventriculogram showed severe hypokinesis with apical ballooning. Stress-induced cardiomyopathy was diagnosed.

Stress-induced cardiomyopathy is an increasingly recognized clinical entity that most often presents with signs and symptoms resembling acute coronary syndrome or frank myocardial infarction. Patients may also present with dyspnea, as well as arrhythmia. Stress-induced cardiomyopathy was first described in Japan about 20 years ago, where it was given the name takotsubo (meaning "octopus trap" in Japanese) cardiomyopathy.[1] It is also commonly referred to as apical ballooning or broken heart syndrome.

Patients often present with ECG changes and modest elevations in troponin levels in the absence of demonstrable coronary arterial occlusion, stenosis or spasm. Common presenting ECG changes vary and can include ST-segment elevation, typically in the anterior precordial leads, T-wave inversion and other nonspecific abnormalities.[2] Stress-induced cardiomyopathy has been reported to account for about 1%–2% of patients presenting with suspected myocardial infarction.[3]

Patients with stress-induced cardiomyopathy are most commonly postmenopausal women experiencing an acute — often emotional — stressor. This cohort accounts for at least 80% of cases.[4] Other reported acute triggers include intracranial trauma, hemorrhagic or ischemic strokes, medical illness, surgery and exogenous catecholamine administration.[5] No trigger is identified in about one-third of patients.[6] The underlying pathophysiology is not clear; however, the association with stressful events supports neurologic and catecholamine-mediated mechanisms. Although attempts have been made at developing diagnostic criteria,[7] none have been formally adopted.

Because of the lack of reliable diagnostic criteria, patients who present to the emergency department with clinical

presentations suggestive of stress-induced cardiomyopathy should receive the usual treatment for acute coronary syndrome. This may involve fibrinolytic therapy, if clinically indicated. Diagnosis is often confirmed on cardiac catheterization, which reveals either normal coronary arteries or only mild atherosclerotic disease in the context of left ventricular apical ballooning with reduced systolic function.

The prognosis for stress-induced cardiomyopathy is good, with the average recovery time for left ventricular dysfunction being 2–3 weeks.[5] The risk of recurrence is up to 10%.[5] Although there are no specific treatments, complications such as heart failure and arrhythmia should be managed in the usual manner.

For the question, see page 144.

Competing interests: None declared.

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Country Cardiograms

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