Biochemical risk assessment and invasive strategies for acute coronary syndromes without ST-segment elevation
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Chapter 9

A bridge to Brugada

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ABSTRACT

In acute cardiac care, prompt diagnosis and management is mandatory. The electrocardiogram (ECG) remains a crucial investigation in the management of ischemic heart disease and arrhythmias. A case is discussed, where the ECG changes caused by the Brugada syndrome and those caused by ischemia, aggravated by myocardial bridging, intertwine.
Case report
A 35-year-old man presented to the Emergency Department with complaints of ongoing chest pain for 2 h after an episode of syncope. He had collapsed on the way home from a social event. The duration of loss of consciousness was not known, as there had been no witnesses. After regaining consciousness, he had continued to have chest pain that radiated to his left arm and was accompanied by nausea and perspiring. The patient denied the use of any drugs.

The patient described similar episodes of collapse throughout his life. He had not sought cardiac evaluation earlier. There was no family history of sudden cardiac death (SCD) or premature atherosclerosis. Physical examination showed a patient who was pale and still in pain. There were no further abnormalities on examination, and he was haemodynamically stable. The electrocardiogram (ECG) showed sinus rhythm with distinct concave ST-segment elevations in leads V1 and V2 (Figure 1). Echocardiography showed hypokinesia of the anterolateral wall with no other abnormalities.

FIGURE 1
Electrocardiogram showing marked bowl-shaped ST-segment elevation leads V1 and V2.
In view of the ongoing chest pain and ST elevation, emergency coronary angiography was performed. This showed the patient’s coronary arteries with marked bridging of the mid-left anterior descending artery (mid-LAD) (Figure 2). Specifically, there was no evidence of vasospasm or coronary stenosis. Left ventricular angiography confirmed hypokinesia of the anterior wall. The patient was treated with β-blockade.

FIGURE 2
Coronary angiogram of the LAD during systole, showing bridging of the mid-LAD (arrow). The left lower panel shows the same segment of the LAD during diastole.
On further ECG monitoring, the ST-segment abnormalities were seen to appear and disappear repetitively without any relation to chest pain. Furthermore, laboratory results for markers of myocardial necrosis, including Troponin T, remained negative. Repeat echocardiography after 3 days showed that the regional wall motion abnormality had resolved.

As there was a strong suspicion of the Brugada syndrome, a drug challenge was performed. After infusion of 100 mg flecainide, a clear type 1 (coved type) Brugada ECG became visible (Figure 3). In Figure 3, the leads V5 and V6 have been replaced by leads V1 and V2 positioned in the third intercostal space (ICS).

**FIGURE 3**
ECG after flecainide infusion showing a coved (type 1) ST segment elevation in the right precordial ST segments with a conduction delay.

![ECG after flecainide infusion](image)

V5 and V6 are replaced with V1 and V2 at the third ICS.

The Brugada syndrome was diagnosed, and the patient was referred for DNA screening and implantable cardiac defibrillator implantation. During his hospital admission, no episodes of fast polymorphic ventricular tachycardia (VT) were seen.
DISCUSSION

There are numerous conditions that can mimic myocardial infarction (MI) on the surface ECG. Several electrocardiographic clues can be used to differentiate these conditions from true MI. Nonetheless, as this case illustrates, multiple mechanisms can interweave, requiring accurate, swift diagnosis and subsequent management.

As in ST-elevation MI (STEMI), time from onset of symptoms to reperfusion is an important factor in the MI size and patient outcome; all suspected STEMI patients should undergo rapid evaluation for reperfusion therapy. Current guidelines advocate prompt intervention with primary PCI within 90 min of first medical contact. In case of logistic difficulties, fibrinolysis can be considered alternatively.

At presentation, the patient showed a ST elevation pattern that was not ‘classic’ for acute MI. The ST-segment elevation in the precordial leads was not convex as is mostly seen in STEMI, but concave. Furthermore, distinct reciprocal depressions lack. However, the suspicion of STEMI remained high due to the accompanying clinical symptoms and hypokinesia of the anterior wall as seen with echocardiography. Because of the important therapeutic implications, in case of doubt, an acute coronary angiography can be performed, followed by PCI, when suitable.

Myocardial bridging is a relatively common finding. Anatomically, the ‘bridge’ is overlying myocardium covering the coronary artery. Angiographically, this anatomic finding correlates to systolic compression of the affected segment of the coronary artery as the myocardium contracts. Most commonly, the mid-portion of the LAD is involved. Although generally considered to be a benign condition, multiple cases of ischemia, infarction, and arrhythmias have been reported.

The Brugada syndrome is a well-characterized clinical and electrocardiographic syndrome with typical ECG alterations, the absence of structural cardiac abnormalities, and a predisposition for life-threatening ventricular tachyarrhythmias. In the Brugada syndrome, the prominent repolarization abnormalities usually occur after acute episodes of electrical instability and often show periodic normalization. Several stimuli, such as autonomic influences and anti-arrhythmic drugs, are known to modulate the occurrence of the typical ECG abnormalities. When the suspicion is high, sodium channel blockers are used as a pharmacological test to expose the syndrome.
Type 1 ST-segment elevation in the right precordial leads is diagnostic of Brugada syndrome and is characterized by a coved ST-segment elevation ≥2 mm (0.2 mV) followed by a negative T wave. Type 2 (saddle back) and type 3 (saddleback or coved <1 mm elevation) should not be considered diagnostic. The placement of the right precordial leads in a superior position (up to the second ICSs above normal) can increase the sensitivity of the ECG for detecting the Brugada phenotype, both in the presence or absence of a drug challenge.

Brugada syndrome is definitively diagnosed when a Type 1 ST-segment elevation (Brugada ECG) is observed in the presence or absence of sodium channel blocking agent in combination with one or more of the following: documented VF, polymorphic VT; a family history of SCD (<45 years old); coved-type ECGs in family members; inducibility of VT with programmed electrical stimulation; syncope; or nocturnal agonal respiration.

Although associations between the Brugada syndrome, coronary myocardial bridging, and vasospastic angina have been suggested in the literature, a causal relationship has not been shown. In this particular case, anterior wall ischemia and subsequent stunning of the left ventricle could have been aggravated by bridging of the mid-LAD during polymorphic VT caused by the Brugada syndrome. The mechanism by which bridging could aggravate ischemia during VT might be by the reduction of the diastolic interval resulting in diminished coronary perfusion of the myocardium distal to the bridge.

In addition, it has also been suggested that patients with Brugada syndrome might be at higher risk for ischemia-related SCD. This phenomenon may be caused by changes in the calcium and potassium channel currents, increasing the susceptibility for malignant arrhythmias.

Conclusion
Although probably not causally interrelated, the coexistence of myocardial bridging and Brugada syndrome may be relevant in affected individuals. Interaction between the provoked ischemia and the specific repolarization abnormalities that are caused by the Brugada syndrome could provide an electrophysiological substrate that may increase individual susceptibility to life-threatening ventricular tachyarrhythmias.
REFERENCES