SHORT REPORT

The flat electrocardiogram—systole or asystole?

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Abstract

A case of stone heart syndrome is reported in a pregnant 27 year old West African patient, who suffered syncopal symptoms shortly before cardiac arrest. The electrocardiographic features were those of asystole, but direct examination of the heart at emergency thoracotomy in the A&E department revealed tetanic contracture of the organ. At necropsy, the heart was of normal weight but showed areas of fibrosis surrounding the bundle of His. The discrepancy between the ECG features and the physiological state of the heart raises the possibility that other cases of apparent asystole may not be what they seem.

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Keywords: cardiac arrest; stone heart; asystole; myocardial fibrosis

Case history

A 27 year old West African patient was admitted to the accident and emergency department in cardiac arrest. One hour before her final col-

lapse, she had suffered a syncopal episode and had fallen on the stairs at home. She quickly recovered from this initial episode and had not apparently suffered serious injury from the fall according to her husband.

On examination she had no cardiac output and no spontaneous respirations. There were no external signs of injury. The cardiac arrest was managed according to contemporary Resuscitation Council (UK) guidelines.¹ Following endotracheal intubation and the insertion of a right external jugular vein cannula, the initial electrocardiogram rhythm was noted to be asystole. This was treated with adrenaline and atropine. Following a second dose of adrenaline 1 mg, fine ventricular fibrillation could not be ruled out, and two 200 J shocks were delivered, after which the ECG trace was again isoelectric and certainly characteristic of asystole.

After 15 minutes of resuscitation, there was still no spontaneous return of circulation, and external cardiac massage was not producing an impulse in either the carotid or the femoral arteries. Left anterolateral thoracotomy was performed for this indication,² and to investigate the possibility of cardiac tamponade: a hard, cricket ball sized mass was found in the mediastinum, which appeared to represent the entire heart. Internal cardiac massage was also ineffective, and resuscitation was discontinued after a further five minutes.

NECROPSY EXAMINATION

Necropsy findings were unremarkable apart for the cardiovascular system. The heart weight was 305 g. Histology of the myocardium showed patchy myocardial fibrosis involving the bundle of His (fig 1). Both carotid arteries had intact 0.5 cm aneurysmal swellings at their bifurcation.

The uterus contained a gestational sac of around 12 week size.

Discussion

Examination findings in this case were those of stone heart syndrome, first described by Wukasch *et al* in 1972.³ The original paper described 13 patients who developed a tetanic contracture or myocardial rigidity during cardiopulmonary bypass. Attempts to resuscitate these hearts using pharmacological and physical means were uniformly unsuccessful. At necropsy, all of the patients were found to have left ventricular hypertrophy, and 12 of 13 had

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Figure 1 High power photomicrograph of the heart, showing cell degeneration and fibrosis in the bundle of His (large arrow). Compare with the underlying myocardium (small arrow) which is normal. Haematoxylin and eosin stain, $\times 215$.

myocardial fibrosis. Heart weights were available for 10 of the patients, and all were increased.

The mechanism of the development of stone heart syndrome is thought to be one of irreversible contracture of ischaemic heart muscle, with exhaustion of adenosine triphosphate (ATP) supplies. There is some evidence that the use of calcium salts in resuscitation may precipitate this condition,⁴ and catecholamines are known to aggravate cardiac damage in ischaemia.⁵ Both endogenous and exogenous levels of adrenaline are raised to very high levels in myocardial infarction and cardiac arrest, because of stress and treatment.⁶ A recent paper from Japan reported the phenomenon of firm myocardium during open chest resuscitation, but the significance of the presenting rhythm was not explored.⁷

It is not clear whether pregnancy might have compromised heart function in this patient, but at 12 weeks gestation cardiac output is increased by 1–1.5 litres/min, requiring extra cardiac work. Additionally, a digoxin-like immunoreactive substance (endoxin) is produced during normal pregnancy,⁸ which would tend to interfere with normal ion flux in the cardiac muscle cells and produce an increase in intracellular sodium.

The heart was of normal size and weight, with no evidence of left ventricular hyper-

trophy. However, in common with the patients originally described, this woman had myocardial fibrosis. No past medical history was available to suggest an aetiology, but it is probable that the fibrosis, which involved the bundle of His, precipitated a cardiac arrhythmia and cardiac arrest, resulting in irreversible ischaemic change in the heart. While the flat ECG trace would normally be diagnosed as asystole, the heart was in fact in the directly opposite state, that of tetanic contracture.

It is possible that many other cases of apparent asystole are not what they seem.

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