Physical Signs

A thrilling case of hiatus hernia

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Summary: A 65 year old woman was found to have a left parasternal heave and a systolic murmur associated with a thrill. A chest radiograph, echocardiogram and gastrograffin swallow demonstrated a massive obstructed hiatus hernia which displaced the heart anteriorly. Aspiration of the contents of the hernia led to complete resolution of the physical signs. Possible mechanisms for their production are discussed.

Introduction

Patients with massive hiatus hernias may present with postprandial epigastric discomfort, symptoms of oesophageal reflux, nausea, vomiting and breathlessness. They occasionally develop serious complications including haemorrhage from erosions or ulceration, obstruction and volvulus. We would like to describe a case presenting with some typical symptoms of massive hiatus hernia but where the physical signs suggested a cardiac rather than a gastroenterological diagnosis.

Case report

A 65 year old woman gave a one month history of anorexia and 7 kg weight loss and a one week history of vomiting after meals. A year previously a small sliding hiatus hernia had been demonstrated on barium meal when she had similar symptoms. On examination she was dehydrated and appeared cachectic but had neither cyanosis nor finger clubbing. Her pulse was regular, of normal character with a rate of 100 per minute. Her blood pressure was 100/60 mmHg and the jugular venous pressure was not elevated. She had a marked left parasternal heave and a systolic thrill palpable in the pulmonary area. There was an ejection systolic murmur loudest in the pulmonary area but easily audible at the left sternal edge and apex but it did not radiate to the axilla or carotid arteries.

Examination of the respiratory system and abdomen was normal.

Investigations showed a haemoglobin of 11.7 g/dl with a normal white cell count, urea and electrolytes. An electrocardiogram (Figure 1a) showed a sinus tachycardia, a mean frontal QRS axis of −20°, large voltage S waves in leads V1 and V2 but small QRS complexes in the limb and lateral chest leads. The appearances were not typical of right ventricular hypertrophy. The chest radiograph (Figure 2) showed a very large hiatus hernia but no other abnormality. Two dimensional echocardiography showed that the heart was displaced anteriorly by the hiatus hernia and was compressed against the anterior chest wall with considerable distortion and flattening of both ventricular cavities. No valvular abnormalities were demonstrated on echocardiography but we were unable to measure the peak flow velocities across the valves because the displacement of the heart prevented us aligning the Doppler beam with flow.

A naso-gastric tube was passed and 1000 ml of fluid aspirated. When the patient was re-examined immediately after this procedure the left parasternal heave, thrill and systolic murmur were no longer present. A gastrograffin meal confirmed the presence of a large obstructed paraoesophageal hiatus hernia. Later that day the patient passed several melaena stools with a fall in haemoglobin to 7.1 g/dl. Following a blood transfusion the patient underwent surgical reduction of the hiatus hernia and made an uneventful recovery. A repeat electrocardiogram (Figure 1b) showed that the mean frontal QRS axis of 0° and the voltages in the limb and lateral leads had increased. Post-operatively the chest radiograph and echocardiogram were normal.

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Discussion

The combination of a left parasternal heave, palpable thrill and loud systolic murmur led us to believe that the patient had significant structural heart disease, possibly pulmonary stenosis, although the presenting symptoms, chest radiograph and electrocardiogram did not support this diagnosis. Our observation that the abnormal physical signs disappeared following aspiration of fluid from the hiatus hernia supported the hypothesis that the abnormal signs resulted entirely from the compression and anterior displacement of the heart which was demonstrated echocardiographically. There have been several published reports of mediastinal tumours causing functional pulmonary stenosis and in some the cause of the murmur was demonstrated by cardiac catheterization. In these cases the pulmonary artery was compressed directly by an anterior mediastinal tumour. We have observed a parasternal heave and systolic murmur (albeit without a palpable thrill) in another patient whose heart was displaced anteriorly by a malignant sarcoma of the posterior mediastinum. In that case the signs disappeared following resection of the tumour. We have not found any previous reports of hiatus hernia causing these physical signs. The left parasternal heave presumably resulted from the displacement of the heart anteriorly against the chest wall. It is more difficult to explain the origin of the thrill and murmur since it would be unlikely that a hiatus hernia or tumour in the posterior mediastinum could directly compress any of the major arteries. It is possible that the pulmonary or aortic outflow tracts were kinked or compressed against other structures by the cardiac displacement. Alternatively there might have been distortion of the atrioventricular valve rings.
leading to functional mitral or tricuspid regurgitation but this seems a less likely explanation since the murmur was ejection rather than pan-systolic and loudest in the pulmonary area. We believe that this is the first reported case of a hiatus hernia causing a left parasternal heave, thrill and murmur.

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References


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