FEATURED CASE REPORT

Reversible left ventricular dysfunction simulating a myocardial infarction after pericardiectomy

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A 39 year old man with postoperative constrictive pericarditis after pericardiectomy developed major left ventricular systolic dysfunction with an anterior wall infarct pattern on ECG but no regional wall motion abnormalities by echocardiography or serum enzymatic evidence of a myocardial infarction. The left ventricular dysfunction resolved over two weeks with supportive treatment. It is postulated that this patient's transient left ventricular dysfunction and ECG changes were caused by myocardial inflammation and oedema induced by operative trauma during pericardiectomy.

Low cardiac output states have been described after pericardiectomy. However, to the best of our knowledge, associated ECG changes simulating an acute myocardial infarction in the absence of myocardial necrosis has not been reported. The importance of recognising this condition is that it is completely reversible with supportive care and without coronary intervention.

CASE REPORT

A 39 year old man underwent urgent three vessel coronary bypass surgery for an acute myocardial infarction. Postoperatively over the next three months, the patient developed progressive congestive heart failure with dyspnoea, major lower extremity oedema, persistent right pleural effusion, and hypotension. He underwent placement of a right pleural tube and was being treated with intravenous diuretics. A viability study using delayed thallium imaging showed an apical myocardial scar but normal isotope uptake in all other regions. A technetium gated blood pool ventriculogram showed an ejection fraction of 62%. An ECG was recorded that showed sinus rhythm with non-specific ST-T abnormalities. Cardiac catheterisation showed severe native three vessel coronary artery disease with patent saphenous vein grafts to the left anterior

descending, left circumflex, and right coronary arteries. The right atrial pressure was 28 mm Hg with a prominent y descent. The right ventricular pressure was 42/18 mm Hg with a post-A wave right ventricular diastolic pressure of 30 mm Hg. The pulmonary artery pressure was 42/30 mm Hg, pulmonary capillary wedge pressure was 29 mm Hg, and left ventricular end diastolic pressure was 30 mm Hg. The cardiac index as measured by thermodilution was 1.7 l/min/m². The patient's haemodynamics were felt to be consistent with constrictive pericarditis and he underwent subtotal pericardiectomy through a median sternotomy incision. Intraoperatively, severe thickening and fibrosis of the visceral and parietal pericardium with a small posterior layer of serous fluid separating the two surfaces were present.

Postoperatively, the patient had hypotension, was inotrope dependent, and required an intra-aortic balloon pump. Figure 1 shows an ECG recorded at that time. An echocardiogram showed severe global hypokinesia of the left ventricle with no regional wall motion abnormalities and an estimated left ventricular ejection fraction of 20–25%. Despite the acute anterior infarct pattern on ECG, serial evaluation of creatine kinase and troponin I remained normal. The patient's clinical condition improved over the course of two weeks with medical management and he was successfully weaned from inotropic support. A repeat echocardiogram showed a left ventricular ejection fraction estimated at 60% without regional wall motion abnormalities. The patient's ECG reverted back to its baseline appearance (fig 2). The patient was discharged home and two years after his pericardiectomy remained without symptoms.

DISCUSSION

This is the first reported case, to the best of our knowledge, of a patient who had severe left ventricular dysfunction following pericardiectomy that masqueraded as an acute myocardial infarction, requiring mechanical and inotropic support, but that subsequently completely resolved. Symptoms attributable to constrictive pericarditis can occur as early

Figure 1 Postoperative ECG in a patient after pericardiectomy showing Q waves with ST-T elevations in the anterior leads, suggestive of an acute anterior wall myocardial infarction.

Figure 2 Follow up ECG in the same patient two weeks after pericardiectomy, showing resolution of ECG changes.

as one month to several years after open heart surgery.¹ The patient in this report is unusual in that he developed constrictive pericarditis that has rarely been described so early after coronary artery bypass surgery, most likely as a sequela of postpericardiotomy syndrome.

After pericardiectomy, a low output syndrome can occur in up to 28% of patients in the immediate postoperative period.² The ECG changes that occurred postoperatively in this patient were very suggestive of an acute myocardial infarction. However, the absence of myocardial enzyme increase and the presence of global left ventricular systolic dysfunction without a focal wall motion abnormality precluded the occurrence of an acute myocardial infarction. We postulate that this patient's low output state may have been caused by a form of "myocarditis" induced by operative trauma during visceral pericardiectomy. In fact, the presence of myocarditis is often inferred from ST segment and T wave abnormalities on the ECG³ and in some cases the clinical presentation may even simulate an acute myocardial infarction.⁴ The postoperative ECG changes in this patient then reverted to the baseline appearance that coincided with complete normalisation of ventricular function. The ECG abnormalities associated with myocarditis are also usually transient and acute myocarditis simulating an acute myocardial infarction has a good prognosis, as with this patient.

Removal of the restraining effect of the pericardium may also result in ventricular systolic dysfunction but this usually occurs in the form of regional wall motion abnormalities, especially in the basal anteroseptal region.⁵ This effect may be amplified because of the presence of severe constriction preoperatively and may have contributed, in part, to the clinical scenario in this patient. However, the ECG changes that occurred are not explained by this "unrestraining" effect.

In conclusion, this report highlights the importance of recognising that profound left ventricular failure simulating an acute myocardial infarction can occur after pericardiectomy, and that this condition can resolve rapidly with a favourable long term outcome.

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