Constrictive pericarditis as a complication of coronary artery bypass surgery

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SUMMARY Although it is now recognised as a rare complication of cardiac surgery, constrictive pericarditis was diagnosed in three patients after coronary artery bypass surgery. The time interval between cardiac surgery and the development of constrictive features varied from two to six weeks. All three patients presented with severe congestive heart failure. Haemodynamic findings were characteristic of constrictive pericarditis. Pericardial thickening detected by computed tomography in one patient was useful in establishing a definite diagnosis. One of the patients had a serous constrictive effusive pericarditis, and surgical pericardial drainage was needed. The other patient underwent pericardiectomy with preservation of the grafts. The diagnosis of constrictive pericarditis should be considered in patients presenting with unexplained right sided heart failure after cardiac surgery.

Constrictive pericarditis is an uncommon disease but an important one to recognise as it is potentially curable. Bacterial infection of the pericardium after cardiac surgery is a rare but recognised cause of constrictive pericarditis, but the development of constriction without overt preceding infection has only recently been reported.2-5

Constrictive pericarditis can be induced experimentally by trauma and by injection of autologous blood in the pericardial sac.6 Traumatic haemopericardium with subsequent development of constrictive pericarditis has been reported.7 It is surprising that constrictive pericarditis does not complicate cardiac surgery more often.

We report the clinical presentation, diagnosis, and management of three patients who developed constrictive pericarditis as a complication of coronary artery bypass surgery.

Case reports

CASE 1
A 67 year old Greek man gave a three year history of typical exertional angina. He was otherwise in good health. There was a history of hypertension, and he had diabetes mellitus controlled by glibenclamide.

Fig. 1 Chest x ray film of case 1 before pericardectomy showing mild cardiomegaly and clear lung fields.
Physical examination was unremarkable, and both the electrocardiogram and chest x-ray examinations showed no abnormality (Fig. 1). Cardiac catheterisation showed good left ventricular function with inferior hypokinesia. Coronary angiography detected a normal left main stem and three vessel disease. In March 1981 he had saphenous bypass grafts placed in the left anterior descending, the marginal branch of the circumflex, and the right coronary arteries. The pericardium was left open. His postoperative course was uneventful, but in retrospect his electrocardiogram on discharge showed low voltage and poor progression of the R wave. Chest x-ray examination showed a normal heart size with a left pleural effusion. Six weeks later in Greece he started to complain of tiredness, breathlessness, and ankle swelling. He was apyrexial, and a pericardial rub was heard. Chest x-ray films showed cardiomegaly. Postcardiotomy syndrome was diagnosed. He was given diuretics and steroids and his condition improved. His heart size decreased on chest x-ray examination (Fig. 1), but his symptoms recurred.

Six months after the operation he was referred back to London. On physical examination his venous pressure was 15 cm with an M shaped contour. There was no pulsat paradoxicus, and an early third heart sound was heard. His chest x-ray film was clear, and his liver was palpable 4 cm below the costal margin. There was ascites and ankle swelling. An electrocardiogram showed sinus rhythm with low voltage. Cardiac catheterisation showed a very small vigorously contracting left ventricle and no change in the native coronary anatomy. There were three patent grafts. A right ventricular angiogram was normal. The haemodynamic measurements were typical of constriction with similar high diastolic pressures in both ventricles. An early dip and plateau were seen in the pressure tracings of both ventricles (Fig. 2). At pericardiotomy a mass of dense fibrous tissue filled the anterior mediastinum and was adherent to the pericardium. The anterior and lateral surfaces of the pericardium were excised in between the grafts, which were not disturbed. His symptoms and constrictive features resolved slowly but completely over the following six months.

CASE 2
A 55 year old man sustained an anterior myocardial infarct in April 1982. Since then he complained of typical exertional angina. He smoked 50 cigarettes a day. There was no history of hypertension. Diabetes mellitus was diagnosed in 1981 and controlled with oral hypoglycaemic agents. He was admitted for investigation in December 1982. Physical examination was unremarkable. An electrocardiogram showed sinus rhythm and signs of an old inferior myocardial infarction. Chest x-ray films were normal. Left ventricular angiography showed inferior dyskinesia with overall good function. Coronary angiogram showed a left main stem stricture and three vessel disease. Coronary bypass graft surgery was performed a month after admission; four saphenous grafts were inserted. His postoperative course was uneventful, and he was discharged 10 days later taking aspirin and dipyridamole. At the time his electrocardiogram showed minor ST-T changes and slightly lower voltage than before the operation. Chest x-ray examination showed the heart to be at the upper limit of normal size.

A month later he complained of sickness, tiredness, breathlessness, and ankle swelling over the previous two weeks. On physical examination he was apyrexial, and the venous pressure was 15 cm with pulsat paradoxicus of 15 mm Hg. His chest x-ray film was clear. Auscultation detected an early third heart sound. There was gross ascites and ankle swelling. An electrocardiogram showed very low voltage. Chest x-ray examination indicated slight cardiomegaly with bilateral pleural effusions. At cardiac catheterisation haemodynamic measurements were typical of constriction (Fig. 3). The left ventricular angiogram showed a small vigorously contracting chamber. The native coronary arteries were unchanged. There were
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Fig. 3 Haemodynamic findings in case 2. Right ventricular (RV) and left ventricular (LV) end diastolic pressures are similarly raised. The early dip does not approach the zero level (mm Hg).

three patent grafts. Echocardiography showed a pericardial effusion. Attempted pericardial tap was unsuccessful. He was referred for radical pericardectomy. At operation the pericardium was thickened, and severe adhesions to the mediastinum and sternum were found. A pericardial effusion with blood clots was drained. Because of dense adhesions between the four grafts and the pericardium an attempt at radical pericardectomy was abandoned. During the following day the gross ascites and ankle swelling subsided. Ten days later his condition was considerably improved. An electrocardiogram still showed low voltage. Two months later M mode and cross sectional echocardiography showed pericardial thickening (Fig. 4).

CASE 3
A 47 year old man had a history of myocardial infarction complicated by ventricular fibrillation in 1980. Since then he had exertional angina. He smoked 12 cigarettes a day. He was admitted for investigation in June 1982. Physical examination showed no abnormalities. Electrocardiogram showed signs of old inferior and anterior myocardial infarction. A heart size at the upper limit of normal and clear lung fields were evident on the chest x ray films. Cardiac catheterisation showed overall good left ventricular function, but there was anteroapical dyskinesia. Coronary angiography indicated two vessel disease. He was referred for coronary bypass surgery and had two saphenous grafts three days after admission. His postoperative recovery was uneventful. At discharge, his electrocardiogram showed minor ST-T changes and lower voltage than before the operation. The heart shadow was unchanged on x ray examination.

A month later when seen as an outpatient he complained of feeling that his head and neck were congested and of tiredness, breathlessness, and ankle swelling during the last few days. On physical examination his venous pressure was 8 cm with an M shaped pulse contour. His chest was clear, and heart sounds were normal. There was pitting oedema over both ankles. An electrocardiogram showed low voltage. On chest x ray examination the heart size was unchanged, but small bilateral pleural effusions were seen. Ventilation perfusion scan, superior vena cavaogram, and left leg venogram were normal. Echocardiogram showed no pericardial effusion. Computed tomography of the thorax indicated pericardial calcification and thickening (Fig. 5). At cardiac catheterisation the right ventricular angiogram showed a small, but well contracting chamber. Right sided haemodynamic measurements were characteristic of constriction. Constrictive pericarditis after cardiac surgery was diagnosed. As the patient was unwilling to undergo further surgery it was decided to defer pericardiectomy. He was seen three months later when his symptoms and clinical features were unchanged.

Fig. 4 M mode echocardiogram of the left ventricle in case 2 showing parallel motion of the visceral and parietal pericardial layers, implying that they are adherent (arrows). PW, posterior wall; IVS, interventricular septum; RV, right ventricle.
Discussion

Constrictive pericarditis as a consequence of cardiac surgery was first recognised at necropsy and seems to be a surprisingly rare complication of cardiac surgery. A few cases have been reported after coronary artery bypass surgery, prosthetic valve replacement, and atrial septal repair. The diagnosis should be suspected in patients presenting with right sided heart failure after operation, even if there is associated left ventricular failure.

One of the reasons for the late emergence of constriction as a complication of cardiac surgery is the difficulty in recognising it in patients who have had surgery for congenital or valvular heart disease and who often have other reasons for raised venous filling pressures. The symptoms and clinical features in our patients, who had only coronary disease, were typical of constrictive pericarditis. The pathological findings varied from constrictive effusive pericarditis in the patient (case 1) with pericardial thickening, adhesions, and a pericardial effusion containing blood clots to those of established chronic constrictive pericarditis as described by Paul Wood.

The main differential diagnosis in patients presenting with signs of constriction early after cardiac surgery is between constrictive effusive pericarditis and late cardiac tamponade. Haemodynamic studies in serous effusive constrictive pericarditis show a prominent dip plateau ventricular pressure. The early dip does not approach the zero level (mm Hg) as in chronic constrictive pericarditis as a result of the effusive element. In cardiac tamponade the early diastolic dip is absent. This is explained by the...
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Constrictive pericarditis is a condition where the pericardium, a sac that surrounds the heart, is thickened and adherent, causing constriction of the heart's chambers. This results in impaired filling of the ventricles during diastole, leading to symptoms such as dyspnea, jugular vein distension, and peripheral edema. The diagnosis is often made with echocardiography, which shows a characteristic pattern of equalization of the left and right ventricles during systole.

The pathogenesis of constrictive pericarditis can be multifactorial, involving a combination of factors such as trauma, infection, and previous cardiac surgery. Residual blood and clots within the pericardial space can contribute to the formation of adhesions between the pericardium and the heart muscle, leading to constrictive physiology.

Pericardial irrigation with povidone-iodine solution has been suggested as a method to treat constrictive pericarditis, particularly in cases where reoperation is necessary. Irrigation can help to remove residual blood and clots, potentially improving diastolic filling and reducing symptoms.

Medical treatment with diuretics and steroids has been successful in resolving constrictive signs in some cases. However, surgical removal of the pericardium may be necessary in cases where the condition is not responsive to medical treatment. Radical pericardiectomy, where the entire pericardium is removed, is a more aggressive approach and can provide long-term relief in some cases.

It is important to recognize that constrictive pericarditis can be a complication of coronary artery bypass surgery, and proper management of this condition is crucial to prevent adverse outcomes. Early recognition and appropriate intervention can significantly improve patient outcomes.

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pericardium as can be excised.

CONCLUSIONS

There is evidence that blood accumulation within the pericardial space plays an important part in the pathophysiology of constrictive pericarditis secondary to cardiac surgery. Possibly with secondary failure of the mechanisms of dissolving blood clots by the pericardial mesothelial cells.

The diagnosis should be considered in patients presenting with right sided heart failure after cardiac surgery. Computed tomography is the best available method of screening postoperatively patients in whom there is a suspicion of developing constriction. Haemodynamic studies will confirm the diagnosis.

Radical pericardiectomy is the treatment of choice. Medical treatment or surgical drainage may, however, partially resolve the constrictive signs when an effusive element is also present in constrictive pericarditis so avoiding the potential risk of graft damage during decortication.

References

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