Complete resolution of constrictive pericarditis after coronary bypass surgery

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SUMMARY

Classical constrictive pericarditis (CP) is an unusual and rare complication after coronary artery bypass grafting. It can be transient, progressive or fixed form of cardiac constriction. However recently recognized transient variant of constrictive pericarditis can be managed with medical therapy, though other progressive and irreversible forms may require pericardiectomy. We describe a 65-year-old male patient who developed a classical but a very early transient CP, just within two weeks as a result of post cardiac injury syndrome after coronary bypass surgery. The patient had a complete recovery following medical treatment.

INTRODUCTION

Constrictive pericarditis (CP) after coronary bypass surgery is a rare clinical condition which may appear early or late. The classical form of CP after cardiac surgery is usually progressive and irreversible, requiring pericardial stripping.[1] However, in some patients CP may resolve spontaneously or with medical treatment.[2,3,4]

CASE REPORT

A 65-year-old man with a long-standing history of diabetes, hypertension, and hyperlipidemia presented with unstable angina was referred for an early coronary artery bypass grafting (CABG) after diagnosing having a critical left main stem and triple vessel disease. A conventional CABG was performed under cardiopulmonary bypass. The postoperative course was uneventful and he was discharged well on post-operative day 6. However, 4 days later, the patient was re-admitted with complaints of fever, exertional dyspnoea and generalized oedema. Physical examination revealed elevated jugular venous pressure, generalized peripheral oedema, including ascites and mild hepatomegaly. The blood test findings were within normal limits except for an elevated C-reactive protein (3.1 mg/dL). Blood, urine and sputum cultures were negative. The chest radiograph showed a mild to moderate bilateral pleural effusion. (Figure 1). An echocardiography revealed a thickened pericardium and an abnormal interventricular septal movement. Pulse wave Doppler study showed significant mitral inflow velocity variation with respiration and exaggerated expiratory diastolic flow reversal in hepatic vein (Figure 1). These findings were not seen at the time of discharge earlier. These findings were highly suggestive of acute CP following CABG as part of the post-cardiac injury syndrome (PCIS). The patient was then treated with aspirin, non-steroidal anti-inflammatory drugs (NSAIDS) (celecoxib), anti-heart failure drugs including diuretics (Furosemide and Spironolactone), beta-blocker (Bisoprolol) and angiotensin converting enzyme (ACE) inhibitor (Ramipiril). As the patient could not tolerate colchicine after 2 days, we switched to steroid treatment. Prednisolone therapy (0.5 mg/kg/day) was started too, continued for 10 days, and progressively tapered over two weeks. The patient felt better and the generalized oedema disappeared. The chest radiograph and repeated echocardiography showed improvements.

The patient was discharged well on post-operative day 20. At the 3rd month of follow-up, the patient was asymptomatic. The chest radiograph showed complete resolution of pleural and pericardial effusion. The echocardiography examination revealed thinner pericardium, normalized mitral inflow velocity with respiration and expiratory diastolic flow reversal in the hepatic vein (Figure 2).

DISCUSSION

CP can be defined as a spectrum of diseases resulting from thickened, rigid and fused pericardial membranes that impair ventricular filling, leading to venous congestion and reduced cardiac output.[2,3] Common causes of CP are previous cardiac surgery, thoracic radiation, uremia, previous myocardial infarction, infection and idiopathic disease.[2,3] CP was thought to be irreversible but some have reported resolution of a transient form without surgical intervention.[3,4] Transient CP is an increasingly recognized sub-type and was first described back in 1987 by Sagrista-Sauleda et al, when a small group of subjects with CP demonstrated spontaneous and permanent resolution on serial echocardiograms.[1] Hence, it was suggested that there are several types of CP. These include classic chronic, sub-acute, transient and occult CP, all of which have their own characteristic natural history. The European Society of Cardiology has formally recognized it as a variant of CP in their updated guidelines for the management of pericardial diseases.[5]

About 17% of patients develop constrictive physiology on

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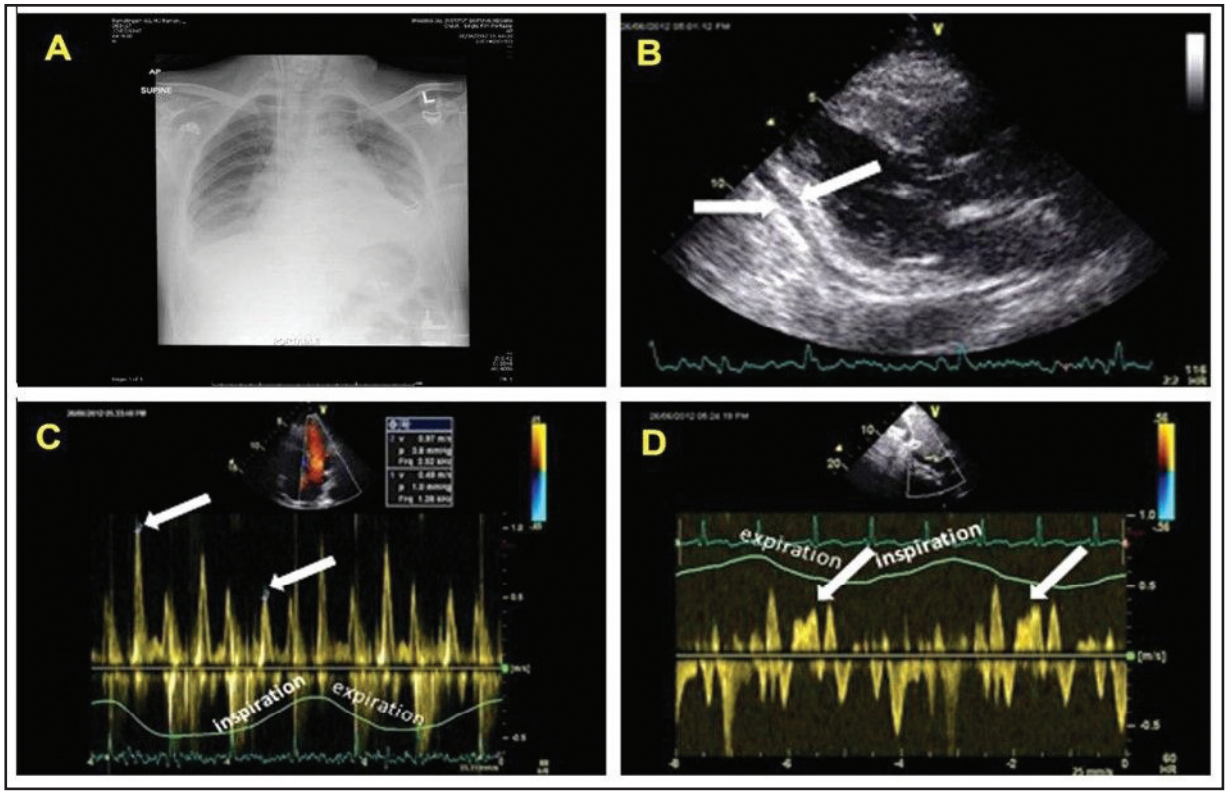

Fig. 1: Initial diagnosis: A) Chest radiograph revealed bilateral pleural effusions. B) 2D echocardiography showed thickened pericardium (white arrows). C) Pulse wave Doppler study illustrated significant mitral inflow velocity variations with respiration (white arrows). D) Pulse wave Doppler study showed exaggerated expiratory diastolic flow reversal in hepatic vein (white arrows).

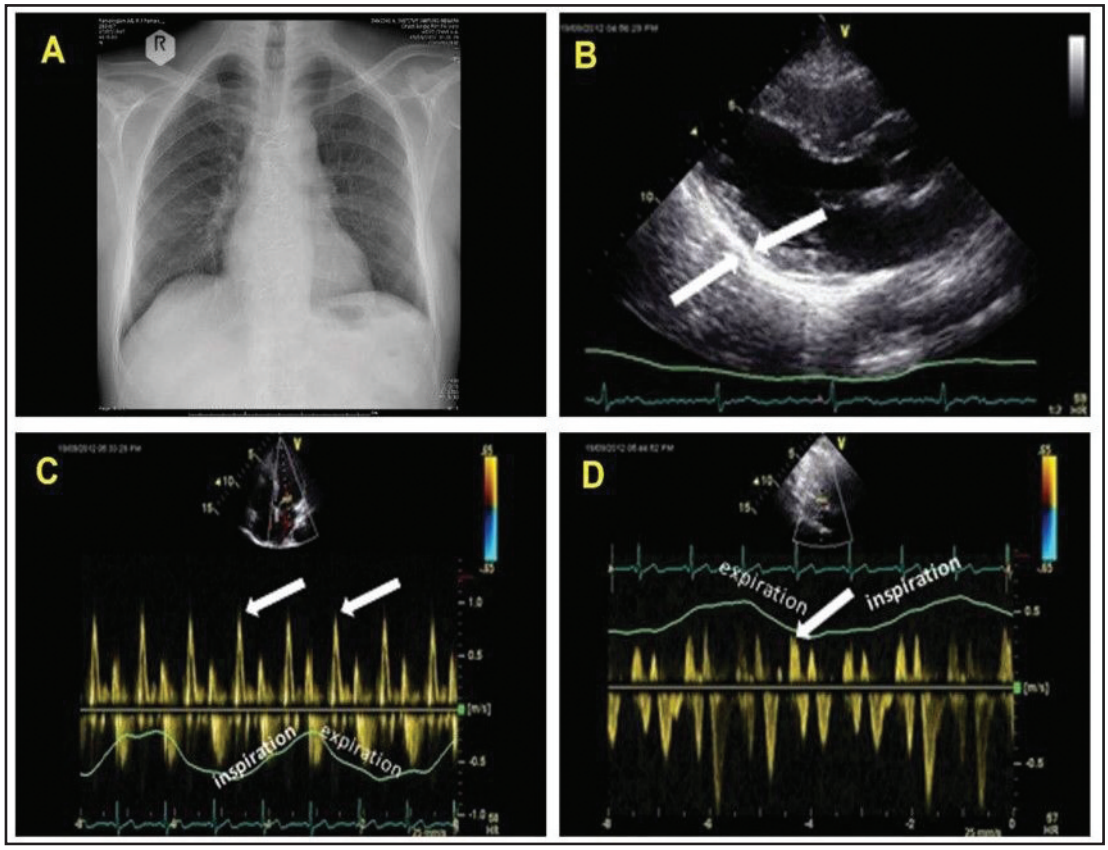

Fig. 2: Ten weeks after completion of treatment: A) Chest radiograph revealed complete resolution of the pleural effusions. B) 2D echocardiography showed normalised pericardium (white arrows). C) Pulse wave Doppler study illustrated normalised mitral inflow velocity variations with respiration (white arrows). D) Pulse wave Doppler study showed significantly reduced expiratory diastolic flow reversal in hepatic vein (white arrow).

postoperative echocardiography after open heart surgery but typical CP following coronary bypass surgery is a rare phenomenon with occurrence rate of 0.2 ~ 0.3%.[2,3] The interval between surgery and development of symptoms varies from 1 to 204 months. The common features are dyspnoea (81%), chest pain (34%), and fatigue (29%); peripheral edema (90%) and elevated jugular venous pressure (86%).

The precise mechanism of CP after cardiac surgery remains unknown. Many reported that CABG is a traumatic procedure with intraoperative irritation to the pericardium by the physical manipulation of surgeons and later continuous violent friction between the pericardium and the beating heart leading to PCIS.[2,3,4] Eui Im et al. suggested that post-operative pericardial effusion and normal left ventricular ejection fraction were predictors of constrictive after CABG.[3]

Our patient showed acute form of CP which appeared very early, just less than 2 weeks after the CABG, which has not reported before. We decided to treat this symptomatic patient medically. Unfortunately, our patient could not tolerate colchicine which is the first line of treatment in acute pericarditis with effusion.[5] Corticosteroids has been effective in reversing pericardial constriction only in some cases when it is given within the first 6 weeks of the surgery.[5]

CONCLUSION

Acute transient constrictive pericarditis after CABG can be treated successfully with medical therapy resulting in complete resolution.

CONFLICT OF INTERESTS

None to declare.

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