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DOI: 10.4250/jcu.2009.17.3.102
pISSN 1975-4612/ eISSN 2005-9655

www.kse-jcu.org
CASE REPORT J Cardiovasc Ultrasound 2009;17(3):102-105

Recurrent Postoperative Effusive-Constrictive Pericarditis Associated with Steroid Discontinuation

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Transient effusive-constrictive pericarditis is a rare complication of open-heart surgery, but is increasingly recognized. For patients with both pericardial effusion and constrictive physiology soon after uneventful open-heart surgery, proper treatment remains to be established. We experienced a case of transient effusive-constrictive pericarditis in a 50-year-old woman who underwent aortic valve replacement due to infective endocarditis. Initially, she was treated with both prednisolone and ibuprofen, which resulted in dramatic relief of symptom. However, she suffered from a relapse of pericaridis after rapid steroid discontinuation and was stabilized by re-treatment with steroid.

KEY WORDS: Postpericardiotomy syndrome · Constrictive pericarditis · Steroid.

Introduction

Development of symptomatic pericarditis soon after uneventful open-heart surgery is rare, but is increasingly recognized. Management of the postpericardiotomy syndrome is basically symptomatic and random combinations of non-steroidal anti-inflammatory agents, colchicines and steroid have been being applied. The major adverse clinical event is recurrence of pericardits and we reported a case of recurrent pericarditis associated with discontinuation of steroid.

Case

A 50-year-old woman admitted for sudden onset facial palsy and dysarthria. Three years ago, she suffered from subacute bacterial endocarditis of the aortic valve with cultured organism of Kingella and 3-month antibiotic treatment (ceftriaxone) had been done successfully. On admission, the patient had a temperature of 36.7°C, a blood pressure of 128/54 mmHg, a pulse of 121/minute and a respiratory rate of 20/minute. An electrocardiogram showed normal sinus rhythm with a heart rate of 107/min and left ventricular hypertrophy. A chest X-ray showed moderate cardiomegaly with increased pulmonary vascularity (Fig. 1A). Both transthoracic and transesophageal echocardiography showed multiple new vegetations at the aortic valve (Fig. 2A) with severe aortic regurgitation. The right aortic sinus of Valsalva and right coronary artery was aneurysmally dilated and coronary artery-ventricular fistula drainage into right ventricle was observed. Her neurologic manifestation was considered as an embolic complication of the vegetations and emergency operation was performed. Operation finding showed moderate amount of pericardial effusion, multiple vegetations involving all aortic cusps and large sized right coronary artery communicated with right ventricular cavity, which needed aortic valve replacement, coronary artery-right ventricle fistula devision and obliteration. Identified organism by blood culture was Streptococcus mitis. Her condition was rapidly stabilized with appropriate antibiotics after operation. She was discharged uneventfully 3 weeks later and her chest X-ray showed normal heart size without any pulmonary congestion (Fig. 1B).

In 8 weeks after surgery, she had sudden onset of pleuritic chest pain with orthopnea and generalized edema. Chest X-ray revealed marked cardiomegaly (Fig. 1C). Echocardio-

• Received: June 25, 2009 • Revised: August 18, 2009 • Accepted: August 21, 2009
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graphy showed moderate amount of pericardial effusion with thickened pericardium, fibrin strands, multiple septations in pericardial space (Fig. 2B), inspiratory decrease of transmitral inflow (Fig. 2C), and markedly dilated inferior vena cava with spontaneous echo-contrast and plethora. Postoperative effusive-constrictive pericarditis was final diagnosis and ibuprofen (400 mg 3 times daily for 3 weeks) and prednisolone (1 mg/kg daily for 3 weeks) was prescribed. Her symptoms were improved very dramatically and chest X-ray showed normalized heart size within 1 week (Fig. 1D) and echocardiography revealed dramatic disappearance of pericardial effusion and constrictive physiology (Fig. 2D). The steroid was tapered over 7 weeks with improvement of symptoms and signs. Steroid was discontinued. Chest X-ray showed no cardiomegaly (Fig. 1E).

In 3 months after steroid discontinuation, she complained pleuritic chest pain and dyspnea again. Chest X-ray (Fig. 1F) and echocardiography (Fig. 2E and F) revealed cardiomegaly, recurred minimal amount of pericardial effusion encircling the entire heart, and constrictive physiology. Recurrent postoperative effusive-constrictive pericarditis potentially associated with steroid discontinuation was suspected and she had steroid medication (1 mg/kg daily) again. The tapering of steroid was more slowly over 8 months with the improvement of symptoms and signs. Chest X-ray showed normalized heart size within 1 week (Fig. 1G) and in 6 months (Fig. 1H) after re-treatment with steroid. At present, she is free of symptom with warfarin only.

DISCUSSION

Transient effusive-constrictive pericarditis is a rare complication of open-heart surgery but important disease entity, since these patients are not indicated for pericardiectomy. Transient effusive-constrictive pericarditis was originally described in the English literature by Sagristá-Sauleda et al. [1] in 1987. Transient inflammation or fibrosis of the pericardium associated with viral or bacterial infection or immunologic mechanism after acute effusive pericarditis has been proposed as a mechanism of this transient effusive-constrictive pericarditis.[2] In 2004, Haley et al.[3] described 36 patients who met the criteria for the diagnosis of transient constrictive pericarditis. At that reports, they described that the causes for the transient constrictive pericarditis were diverse and most common cause was prior cardiovascular

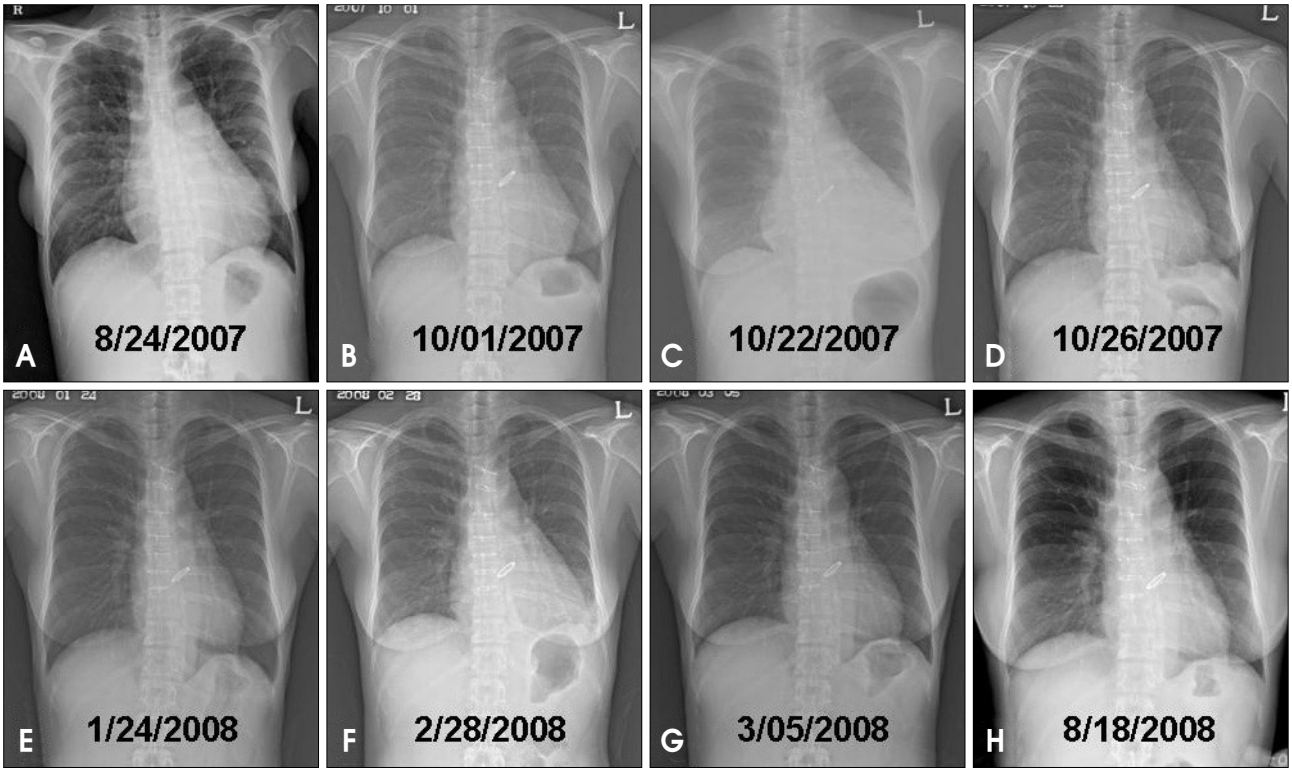

Fig. 1. A series of chest X-ray. Preoperative chest X-ray showed moderate cardiomegaly with increased pulmonary vascularity (A). Cardiomegaly and pulmonary congestion after operation was resolved (B). In 8 weeks after surgery, chest X-ray showed aggravated cardiomegaly compared with previous one (C). Under the diagnosis of postoperative effusive-constrictive pericarditis, patient was treated with prednisolone and ibuprofen for 7 weeks. Chest X-ray showed normalized heart size within 1 week (D) and after treatment (E). In 3 months after steroid discontinuation, chest X-ray revealed cardiomegaly again (F). The patient was suspected as recurrent effusive-constrictive pericarditis potentially associated with steroid discontinuation. Chest X-ray showed normalized heart size within 1 week (G) and in 6 months (H) after re-treatment with steroid.

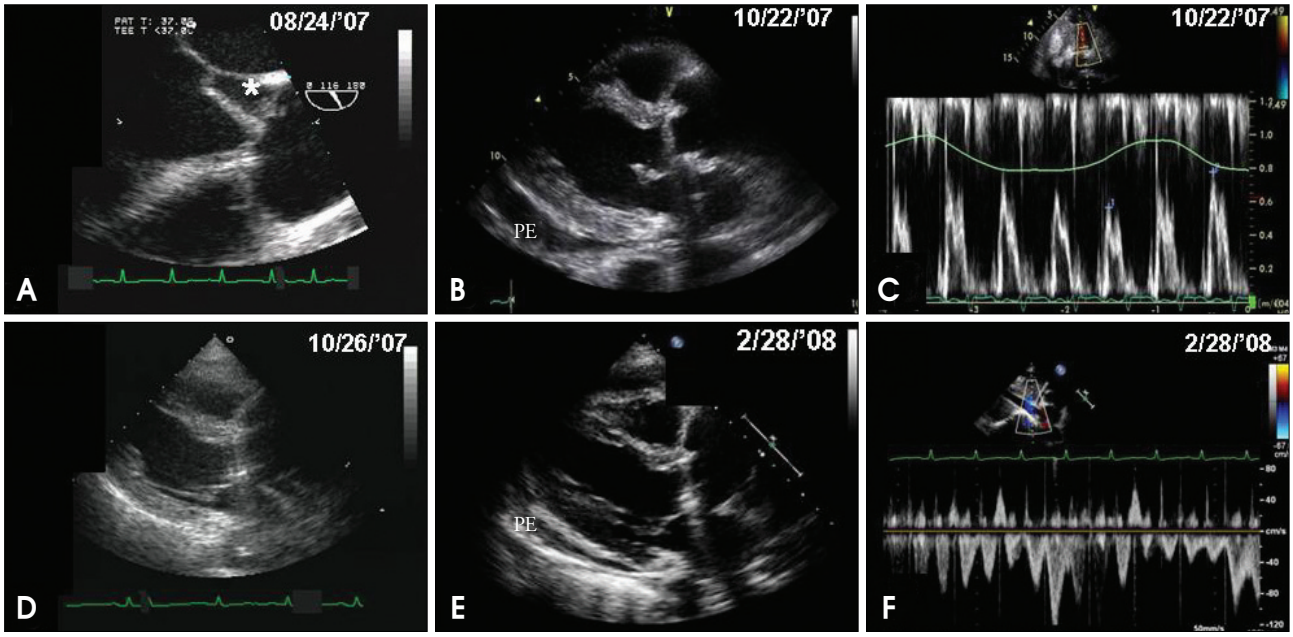

Fig. 2. Representative echocardiograms. Preoperative transesophageal echocardiography showed multiple vegetations attached to aortic valve (A). After uneventful aortic valve replacement, moderate pericardial effusion (PE) with typical constrictive physiology developed (B and C), which resolved dramatically with medical treatment (D). After steroid discontinuation, pericardial effusion (E) and constrictive physiology redeveloped (F). Asterisk: vegetations.

surgery (25%). In Korea, Yang et al.[4] reported 11 patients with transient constrictive pericarditis in 2001. They showed that tuberculosis (10/11 patients) was the most important etiology of transient constrictive pericarditis in Korea.

Postpericardiotomy syndrome develops days to months after cardiac and pericardial injury.[5][6] Management of the postpericardiotomy syndrome is basically symptomatic and random combinations of non-steroidal anti-inflammatory agents, colchicines and steroid have been being applied. Recently, Imazio et al.[7] showed that most of recurrent pericarditis might be an autoimmune disease and colchicine plus conventional therapy led to a clinically important and statistically significant benefit over conventional treatment, decreasing the recurrence rate in patients with a first episode of acute pericarditis.[8] But their study included acute pericarditis of diverse causes (idiopathic, viral, and autoimmune causes, including postpericardiotomy syndromes and connective tissue diseases). Thus, it is not certain if their results could be applied to postpericardiotomy syndrome patients. The major adverse clinical event of postpericardiotomy syndrome is recurrence of pericarditis and optimal management of recurrent postpericardiotomy syndrome has not been also established.

Our case is postpericardiotomy syndrome with pericardial effusion and constrictive physiology. After administration of steroid and ibuprofen, the constrictive physiology was dramatically resolved. However, there was a recurrence of constrictive physiology after rapid steroid discontinuation. The mechanism of pericarditis recurrence is not clear and the role of steroid discontinuation remains elusive. As colchicine is reported to be a better option to prevent recurrence of pericardits,[8] it would be worthy to have a randomized comparison study for patients with pericardiotomy syndrome. However, before getting those data, clinicians should be aware that recurrence is possible with rapid steroid discontinuation after dramatic improvement of pericarditis and physicians' vigilance is needed during care of patients with postpericardiotomy syndrome, which is expected to increase along with increased numbers of open heart surgery.

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