Postpericardiotomy syndrome: What a pulmonologist must know

Sir,

Postcardiac injury syndrome consists of both postmyocardial infarction syndrome and postcardiotomy syndrome (PCS). This entity is well known to cardiologist and cardiac surgeons, but pulmonologists are often confused when they deal with these patients. We present here, a patient with recurrent bilateral pleural effusions consequent to pericardiectomy done for constrictive pericarditis.

A 28-year-old male, nonsmoker, presented with fever, right-sided pleuritic chest pain, and increasing shortness of breath for the last 10 days. He had been diagnosed with constrictive pericarditis 6 months ago and was operated for the same 2 weeks before these symptoms started. The pericardiectomy was uneventful, and the thickened pericardium could be removed only anteriorly. There was past history of Tubercular intestinal obstruction 10 years ago for which laparotomy and bowel resection was done. On evaluation, he had features suggestive of right-sided moderate pleural effusion. Thoracocentesis was done, and 1500 ml of straw colored fluid was aspirated. The fluid was lymphocytic, exudative with 90% lymphocytes, sugar 78 mg/dl, protein 4.8 g/dl, lactate dehydrogenase 765 U, and adenosine deaminase 12 U/L. Post-thoracocentesis, the patient was asymptomatic for around 7 days when he again developed shortness of breath. On evaluation, he had massive left-sided effusion, with biochemical characteristics same as the previous right-sided effusion. A left-sided intercostal drainage (ICD) tube was inserted and 3 L of hemorrhagic pleural fluid was removed. The fluid had a hematocrit of 4% as compared to the blood hematocrit of 37% establishing the diagnosis of a hemorrhagic pleural effusion. The patient was started on antituberculous therapy and referred to our center with the ICD tube *in situ*. At presentation, he had blood pressure of 100/60 mmHg, pulse of 132/min with baseline atrial flutter, room air saturation 94%; respiratory rate of 20/min with axillary temperature of 100.5 F. His investigations showed hemoglobin of 12.6 g/dL, total leukocyte count 12,200/mm^3 and platelet count 227,000/mm^3, serum bilirubin was 0.8 mg/dl, serum urea was 34 mg/dl, and creatinine was 0.9 mg/dl. There was no abdominal swelling or pedal edema. His chest X-ray revealed cardiomegaly with right-sided moderate pleural effusion and left-sided ICD tube. Two-dimensional - ECHO revealed thickened pericardium posterior and inferior to left ventricle, posterior to left atrium, and behind right atrium, grossly dilated atria with an ejection fraction of 45%. Computed tomography chest was done which showed right-sided loculated pleural effusion with pleural thickening, ICD tube in place on the left side with minimal left-sided pneumothorax. To diagnose the cause of pleural effusion the patient underwent medical thoracoscopy and pleural biopsy on the right pleural space which revealed extensive thickening of parietal pleura along with multiple adhesions. Pleural biopsies were taken, and 200 ml of hemorrhagic fluid was drained. Pleural biopsy was noncontributory to diagnosis and revealed nonspecific inflammation. Postthoracoscopy multiple doses of intrapleural urokinase were given, but the residual pleural thickening and loculation were significant enough to prevent the right-sided lung from expanding.

To ascertain the cause of his bilateral pleural effusions, literature search was done, and a diagnosis of postpericardiotomy syndrome (PPS) was made on the basis of having 3 out of 5 cardinal symptoms of PCS,[1,2] consisting of fever, pleuritic chest pain, and new and worsening pleural effusion. He was started on high dose Aspirin (1 g/day) to which he showed good response, in terms of disappearance of fever and pleuritic chest pain. In view of the failure of fibrinolytic therapy and nonexpansion of lung he underwent thoracoscpic decortication of the right-sided pleura. Postoperative period was uneventful, and the patient subsequently underwent radiofrequency ablation of the aberrant cardiac conduction pathways to treat the baseline atrial fibrillation. He was discharged on the 5th postoperative day. The patient was reviewed 2 months postsurgery. He was asymptomatic, and a repeat chest X-ray chest showed minimal right-sided pleural thickening. In conjugation with the cardiologist, the dose of Aspirin was gradually tapered and he is on 150 mg of Aspirin every day.

The first description of PPS was given in 1953 in patients undergoing mitral valve surgery who developed fever and pleuritic chest pain following surgery.[3] Time interval for symptoms to develop is variable and can be from several days to weeks to months after surgery. The exact etiology of this condition is unknown but is linked to an aberrant increased immune response in the postoperative period.[4,5] Patients with PPS generally presents with fever without an obvious cause after the 1st postoperative week, pericardial friction rub, new or worsening pericardial effusion, pleuritic chest pain, and new or worsening pleural effusion. A consensual diagnostic criterion has been given based on two recent clinical trials (COPPS and COPPS-2), and diagnosis of PPS can be made if patients present with at least two of the above-mentioned five clinical findings.[1,2] The incidence of PPS varies from 9% to as high as 50% in various studies.[6] Patient with younger age and blood transfusion have an increased risk, whereas diabetics have a lower risk of developing this entity.[6] For most

patients PPS is a self-limiting condition, but its duration can be highly variable. In general, PPS only lasts for a few weeks and persistence beyond 6 months is exceptional.[7] Failure to identify the relationship between the antecedent operation and the occurrence of the pleural effusion often results in misdiagnosis, as was the case in our patient. Most patients show relief in pain and effusion with bed rest, Aspirin and other similar agents. Corticosteroid and Colchicine have been tried in patients with persistent symptoms, despite being on Aspirin.[8]

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Conflicts of interest
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Ankit Kumar Sinha, Amit Jose, Arjun Khanna, Deepak Talwar

Metro Centre for Respiratory Diseases, Metro Multispeciality Hospital, Noida, Uttar Pradesh, India
E-mail: doc.khanna@gmail.com

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