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CASE STUDY

PERICARDIAL EFFUSION DUE TO STENT RUPTURE AND OVERLAP SYNDROME

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ABSTRACT

A 48years old female known case of hypertension and ischemic heart disease with post percutaneous coronary angioplasty (PTCA)status to left anterior descending (LAD) and right coronary artery (RCA), was admitted to our hospital with history ofsevere breathlessness, chest pain, bilateral lower limb swelling - on and off since last 2-3 years. Initial investigation depicted moderate to severe pericardial effusion, for which she was treated with anti-tubercular drugs. Initial symptomatic relief was achieved with antikoch's treatment. But, few months later she experienced same complaints with rapid progression. Which on investigation shown to have massive pericardial effusion. This was further investigated and found to have ruptured RCA stent communicating with pericardial cavity and positive antinuclear antibody test (ANA) test. She was treated with pericardiactomy and improved.

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INTRODUCTION

The pericardium is an avascular fibrous sac that surrounds the heart. It has 2 layers visceral and parietal. Both layers are separated by pericardial space. This space contains 15 to 50 mL of fluid that act as a lubricant during cardiac movement during cardiac cycle (Harrisons 19th edition). This fluid is an ultrafiltrate of plasma, thought to originate from the visceral pericardium. The pericardium is attached to the sternum, the diaphragm, and the anterior mediastinum and is invested around the great vessels and the venae cavae, serving to anchor the heart in the central thorax. Due to its location, it also has protective functions. Pericardium has relatively inelastic physical properties, this limits acute cardiac dilatation and enhances mechanical interactions of the cardiac chambers. During stress, the pericardium dilates, and pressure increases thus resulting in pericardial effusion (Little *et al*., 2006). Accumulation of abnormal amount of and/or an abnormal character to fluid in the pericardial space is known as pericardial effusion. Pericardial effusion can be acute or chronic and patient's symptoms are greatly impacted by duration of diseases. It is caused by a variety of local and systemic disorders, or it may be idiopathic. Underlying known diseases cause includes, acute myocardial infarction (AMI), cardiac surgery, trauma, neoplasia, chest radiation autoimmune diseases, etc. The cause can also be without known underlying disease such as acute inflammatory pericarditis or previously known neoplasia (Khandaker *et al*., 2010) The diagnosis and management of pericardial diseases remain challenging because of wide manifestations and lack of clinical data. The diagnostic approach should give strong consideration to coexisting medical conditions. One of the causes is traumatic pericardial effusion secondary to post surgical or post cardiac interventional procedure. Autoimmune diseases also causes pericardial effusion, one of which is overlap syndrome (Strimel *et al*., 2017). The European Society of Cardiology (ESC) published guidelines on pericardial disease in 2004. Medical management for viral or idiopathic acute pericarditis has been centered on 3 major agents—NSAIDs, colchicine, and corticosteroids (Khandaker *et al*., 2010). Medical treatment of pericardial effusion is mainly dictated by the presence of inflammatory signs and by the underlying disease if present. Pericardial drainage is mandatory when clinical tamponade is present (Sagrista-Sauleda *et al*., 2011)

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CASE REPORT

A 48year old housewife, known case of hypertension and ischemic heart disease with post percutaneous coronary

angioplasty (PTCA) status to left anterior descending (LAD) and coronary artery (RCA), was admitted in emergency room. She also complained for severebreathlessness, chest pain, hemoptysis, bilateral lower limb swelling and disturbed sleep (orthopnea/paroxysmal nocturnal dyspnea), allincreased since 2 days before admission.

Initial physical examination

Pulse rate	104/min
Blood pressure	90/60 mmHg
Jugular venous pressure (JVP)	Raised
Pallor	+
Peripheral capillary oxygen saturation (SpO_2)	86% at room air

Patient history revealed that she had dyspnoea on exertion (NYHA class 2-3) and intermittent episodes of chest pain from last 2-3 yrs. On further investigation and coronary angiography (CAG), she was found to have double vessel disease with block in LAD (100%) and block in RCA (90%). Immediately she underwent PTCA to LAD and RCA and symptomatic relief was achieved. Later 6-7 months, she again started experiencing chest pain and dyspnoeaon exertion. After investigation, she was found to have pericardial effusion, for which pericardiocentesiswas done. On microscopic examination, lymphocyte predominant leukocytosis and exudative nature of pericardial fluid was noted. Empirical anti-tubercular treatment was initiated, as her Ziehl-Neelsen (ZN) stain and TB PCR on pericardial fluid was negative. She continued the treatment for almost a year until she again reported same complaints as above. Dyspnea was progressive in nature (NYHA class 4), diffuse, continuous type of chest pain, aggravated by exertion. Same time started intermittent type of hemoptysis, initially occurring for 3-4 times weekly, which progressed to 2-3 episodes/day, more in early morning and after rising from bed. Immediate 2D ECHO depicted massive pericardial effusion and cardiac tamponade with 60%ejection fraction (EF). No regional wall motion abnormality (RWMA). Transesophageal echocardiogram (TEE) - EF 60%, large ECCENTRIC mass on tricuspid valve, ruptured sinus of Valsalva. High resolution CT ofthorax suggested right lower lobe collapse and consolidation, gross pericardial effusion. Multinodular calcific RT UL OPASCITIES, multiplemediastinal lymphadenopathy of significant size. Sputum R/M-Budding yeast cells, C/S-MDR Klebsiella pneumonie.

CAG was planned and work for other causes of pericardial effusion was done. CAG showed patent LAD stent and aneurismal mass on proximal RCA. Distal fills via collaterals. RCA stent in pericardial space.ST depression in inferior leads with T wave inversion with electrical alternance was noted in Electrocardiogram (ECG).

Hematological investigation reports

Hemoglobin	10.5 (Iron deficiency picture on ps)
Total leukocyte/Platelet count	Normal
Renal Function Test (RFT)	Normal
Liver Function Test (LFT)	Normal
HIV/ HBs Ag/HCV	Negative
PT/INR/BT/CT	Normal
Arterial blood gas (ABG)	Normal
Creatine Kinase-MB	Negative
Troponin test	Negative
Antinuclear antibody (ANA) test by immunofluroscense	Positive (homogenous)
PM-SCL Antibodies	Borderline positive

For the repeated episodes of pericardial effusion she underwent pericardiectomy. And same treated with antibiotics as per culture and sensitivity report for consolidation. Patient improved symptomatically and hemodynamically.

DISCUSSION

Pericardial effusion is the presence of an abnormal amount of and/or an abnormal character to fluid in the pericardial space (Harrison's 19th edition). The spectrum of pericardial effusions ranges from mild asymptomatic effusions to cardiac tamponade (Imazio *et al.,* 2012). Most of the time clinical picture of the patient leads directly to the diagnosis of pericardial effusion. As in patients with chest pain of pericarditic characteristics or in patients with underlying disease, such as acute myocardial infarction, cardiac surgery, end-stage renal disease or widespread metastatic neoplasm. Patients without previous known diseases seek medical attention due todyspnea or chest discomfort (Sagrista-Sauleda *et al*., 2011) Lady presented at our hospital had undergone PTCA to LAD and RCA. She also had dyspnea or chest pain. Classical symptoms include dyspnoea on exertion progressing to orthopnoea, chest pain, and/or fullness. Additional occasional symptoms like nausea, dysphagia (oesophagus) and hiccups (phrenic nerve) may also be observed. Physical examination generally shows signs like neck vein distention with elevated jugular venous pressure at bedside examination, pulsusparadoxus, and diminished heart sounds on cardiac auscultation. Pericardial friction rubs are rarely reported (Little, 2006). Severity of pericardial effusion can be acute (> 6 weeks) or subacute (from 6 weeks to 6 months) or chronic (More than 6 months). It can be fibrinous, effusive, adhesive or constrictive. Number of etiological agents may be responsible for pericardial effusion, most common being infections (viral, bacterial, especially tuberculosis). Other non-infectious agents include Neoplasia, connective tissue diseases, pericardial injury syndromes (post-myocardial infarction effusions, post-pericardiotomy syndromes, post-traumatic pericarditis either iatrogenic or not), rheumatic fever, metabolic causes (especially hypothyroidism, renal failure), myopericardial diseases (especially pericarditis, but also myocarditis, heart failure), and selected drugs (i.e. minoxidil, hydralazine) (Harrison's 19th edition).

Form the literature, we found five major surveys that has been published on the characteristics of moderate to large pericardial effusions (Corey *et al*., 1992; Sagrista-Sauleda *et al*., 2000; Levy *et al*., 2003; Reuter *et al*., 2005). Report for the surveys suggest, overallprevalence of malignant or infectious aetiologies ranges from 15 to 50%.Most of the pericardial effusion cases still remain idiopathic in developed countries (up to 50%). Other causes includes, cancer (10–25%), pericarditis and infectious causes (15–30%), iatrogenic causes (15–20%) and connective tissue disease (5–15%). Among all tuberculosis was the dominant cause in developing countries (>60%). Tuberculosis is diagnosed, if patient has diagnosis of tuberculosis elsewhere in the body (i.e. pulmonary), or with a lymphocytic pericardialexudate with elevated adenosine deaminase (ADA) levels. Antituberculosis therapy is expected in countries (i.e. Africa and India), where tuberculosis is endemic (Imazio *et al.,* 2012). In our case, patient had lymphocyte predominant leukocytosis and exudative nature of pericardial fluid, but her Ziehl-Neelsen (ZN) stain and TB PCR on pericardial fluid was negative. Hence, she was initiated with empirical anti-tubercular treatment. She continued the

treatment until she experienced same complaints after a year. Pervious history along with NYHA class 4 dyspnoea, continuous type of chest pain, aggravated by exertion and intermittent type of hemoptysis suggested injury to the pericardium.

Pericardial effusion resulting from injury of the pericardium constitutes the post-cardiac injury syndrome (PCIS). This was first described after myocardial infarction by Dressler in 1956. It can develop after cardiac surgery, after blunt trauma, penetrating cardiac trauma or cardiac perforation due to catheter or rarely acute myocardial infarction. Clinical picture mimics acute viral/idiopathic pericarditis (Harrisons 19th edition; Khan *et al*., 1992) PCIS is regarded as an immunopathic disease entity. When laboratory investigation suggested normal haematological parameter with normal RFT and LFT, we suspected involvement of immunopathic disease. We found ANA test and PM-SCL Antibodies test for the patient to be positive. Anti PM-SCL antibodies are generally associated with calcinosis cutis, myositis, overlap syndrome, cardiac manifestations (Associated with positive anti PM-SCL antibodies), Pericarditis (pericardial pain and either a pericardial friction rub or pericardial effusion), arrhythmia requiring treatment, complete heart block, or Death due to Systemic sclerosis related heart disease (Koschik *et al*., 2012)

Echocardiography and ECG are also useful tool for investigation. Early in acute pericarditis ST elevation in association with PR depression. Classically, the ECG changes of acute pericarditis evolve through 4 progressive stages: stage I, diffuse ST-segment elevation and PR-segment depression; stage II, normalization of the ST and PR segments; stage III, widespread T-wave inversions; and stage IV, normalization of the T waves (Little, 2006). Although ECG is standard and widely used method, CT and magnetic resonance imaging (MRI) can allow assessment of the entire chest and detection of associated abnormalities in the mediastinum, lungs and adjacent structures. Both of them also offer the advantage of possibility of identifying hemorrhagic effusions or clots within the pericardium (Sagrista-Sauleda *et al*., 2011). 2D ECHO for our patient depicted massive pericardial effusion and cardiac tamponade with 60% EF. Whereas, ST depression in inferior leads with T wave inversion with electrical alternancewas noted in ECG. Even CT was useful, High resolution CT of thorax suggested right lower lobe collapse and consolidation, gross pericardial effusion. Most of the acute idiopathic pericarditis respond to Nonsteroidal anti-inflammatory drug (NSAID) treatment. However, autoimmune pericardial effusions may need anti-inflammatory medications. Other pharmacological agents used in treatment for pericardial effusion includes, corticosteroids (eg, prednisone, methylprednisolone, prednisolone), Antibiotics (eg, vancomycin, ceftriaxone, ciprofloxacin, isoniazid, rifampin, pyrazinamide, ethambutol), Sclerosing agents (eg, tetracycline, doxycycline, cisplatin, 5-fluorouracil) and antineoplastic therapy (eg, systemic chemotherapy, radiation) may be used depending on the etiological factors. Hemodynamic support may also be required. Surgical treatments includes; pericardiostomy, pericardotomy, thoracotomy, sternotomy, pericardiocentesis (Harrioson's 19th edition; Khandaker *et al*., 2010; Sagrista-Sauleda *et al*., 2011) For our patient pharmacotherapy was initiated with antibiotics. Pericardiectomy was done for repeated episodes of pericardial effusion. Further, patient improved symptomatically and hemodynamically.

Conclusion

A 48 year old lady with pericardial effusion due to stent rupture and overlap syndrome treated with total pericardiectomy, significantly improved symptomatically and clinically.

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