Late Onset Development Of Pericardial Effusion In A Stable Patient On CAPD And Anti-Tuberculosis Treatment
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Abstract
End stage renal disease patients are prone for pericardial diseases including pericarditis, pericardial effusion and chronic constrictive pericarditis. Pericardial effusion occurs more frequently in patients on maintenance hemodialysis than peritoneal dialysis. We present a case of asymptomatic large pericardial effusion with evidence of near tamponade on echocardiogram in a lady, 6 month after initiation of peritoneal dialysis who was on anti tuberculosis therapy. Pericardiocentesis showed a hemorrhagic exudative effusion.

Keywords: Large Pericardial effusion, CAPD

Introduction
In large pericardial effusion, more than 500ml of fluid accumulates in the pericardial space. Patients on CAPD have lower risk for pericardial effusion than patients on hemodialysis.(1). In fact patients on MHD who develop pericardial effusion can be switched over to CAPD to resolve the effusion, to avoid heparinisation.(2) Our patient developed an insidious pericardial effusion although she was clinically asymptomatic. Echocardiogram showed evidence of near tamponade. A subxiphoid pericardiocentesis was performed consecutively for 2 days as the fluid reaccumulated after the first pericardiocentesis. The causes and management of pericardial effusion in a CAPD patient are discussed.

Case Report
A 27 years old lady, diabetic and hypertensive for 2 years, ESRD on CAPD for 6 months was being evaluated for a live renal transplantation.

She was diagnosed with CKD in March 2012 and had been initiated on maintenance hemodialysis through an internal jugular line. A renal biopsy done at that time showed advanced renal damage with chronic glomerular sclerosis. In June 2012, she had unrelenting episodes of fever. All investigations including multiple blood cultures, serology, computed tomography scan, bone marrow biopsy were inconclusive. She was empirically initiated on anti-tuberculosis treatment(ATT)-isoniazid 150mg once a day(OD), pyrazinamide 750mg twice a day, rifampin 600mg OD and ciprofloxacin 500mg OD and vitamin B6. Her internal jugular line was removed and she was started on peritoneal dialysis using a Swan neck Tenckhoff double cuff catheter in July 2012. Her KT/V was 1.6 on 4 exchanges per day alternating 1.5% and 2.5% bags and an ultra filtrate of around 1500ml. She was asymptomatic and anuric.
As part of her routine pre-transplant work up an echocardiography was done in November 2012. It showed significant pericardial effusion measuring 29mm behind posterolateral wall of left ventricle and 14mm behind right atrium and right ventricle with right atrial and ventricular diastolic collapse indicating tamponade (fig-1), EF of 50%, adequate biventricular systolic function and small left pleural effusion, concentric left ventricular hypertrophy and normal sized chambers. An echocardiogram done in June had been normal. ANA, dsDNA and ANCA were negative. Chest X-ray showed cardiomegaly (fig-2). ECG was normal sinus rhythm with no ST-T changes.

She underwent pericardiocentesis and about 700ml of thick hemorrhagic fluid was aspirated (fig-3). Analysis of the fluid showed total WBC count-6930 cells/cumm, polymorphs 40%, lymphocytes 51%, eosinophils 9%, glucose-133mg/dl, total protein-5.4g/dl, LDH-912 IU/l suggestive of an exudate. Cytological examination showed numerous RBCs and mesothelial cells in a background of sheets of lymphocytes and macrophages with few neutrophils and eosinophils suggestive of chronic inflammation. No malignant or atypical cells were seen. Culture was negative, TB-PCR was negative and no AFB on smear or culture. Her haemoglobin was 11.1g/dl and packed cell volume 32.2%, urea-85mg/dl, creatinine-9.5mg/dl. There was reaccumulation of the fluid, and she underwent a second pericardiocentesis the next day. Post procedure echocardiogram after that was normal. The patient is currently asymptomatic and doing well on CAPD with no recurrence of effusion while continuing ATT.

Discussion:

The heart is encased by an outer fibrous and an inner serous pericardium. A visceral pericardium which is adherent to the epicardium and a parietal pericardium which is fused with the fibrous pericardium, constitute the serous layer. Inbetween the two layers of the serous pericardium is the pericardial space, which normally holds about 25ml of clear serous pericardial fluid which provides lubrication during the heart
motion. In a large pericardial effusion, more than 500ml of fluid accumulates. Neoplastic, idiopathic and uremia are 3 main causes of large pericardial effusion(3). Pericarditis seen in renal failure can be broadly categorised as uremic and dialysis-associated. The inflammatory state and platelet dysfunction associated with uremia are probably the cause of a hemorrhagic effusion in uremic pericarditis seen in advanced renal failure before initiation of dialysis. The typical chest pain associated with pericarditis and ST-T wave elevation on ECG are absent .On the other hand, dialysis-associated pericarditis is either due to inadequate dialysis or fluid overload which is serous,(2) which was not the case with our patient.

The incidence of pericardial effusion is less in CAPD than in maintenance hemodialysis(1). Like pleural effusion, pericardial effusion in peritoneal dialysis may occur when there is peritoneal leak through congenital asymptomatic anatomical defects in the diaphragm that become clinically significant when the intra-abdominal pressure raises during CAPD(4). But such an effusion would be serous and will manifest within first few months of initiation of CAPD. In a study by de Araujo Antunes A et al with 34 peritoneal dialysis patients, hemoglobin levels below 12.2 g/dL, Kt/V lower than 1.9 and phase angle lower than 4.5° were the best cutoffs to predict pericardial effusion in CAPD patients(5). Pericardial fluid analysis suggestive of an exudates are specific gravity > 1.015, proteins > 3mg/dl, lactate dehydrogenase >300U/dl and pericardial fluid glucose/serum glucose <1.(6)

For the treatment of pericardial effusion, if there are signs of tamponade, the next step of management is guided by local experience. Pericardiocentesis through a subxiphiod approach is safe although caution should be exercised to the formation of peritoneo-pericardial communication in a PD patient(7,8). Percutaneous drainage through an catheter with instillation of a nonabsorbable steroid provided a safe method without recurrence of effusion(9)Pericardial surgery in the form of pericardiotomy and pericardiectomy can be an option in recurrent effusion(2)

Patients on hemodialysis who develop pericarditis can be switched over to peritoneal dialysis as this heparin-free dialysis helps in faster resolution(2). Our patient developed a massive effusion 6 months after initiation of CAPD . There was rapid reaccumulation after the first pericardiocentesis. The fluid aspirated was hemorrhagic, although she was biochemically stable and there were no malignant cells. . Although she was asymptomatic, echocardiogram showed features of tamponade. This highlights the importance of periodic echocardiogram even in patients on CAPD. The absence of reaccumulation after the second pericardiocentesis rules out the possibility of mycobacterium tuberculosis or other ongoing inflammatory cause as the etiology of this pericardial effusion.

Reference:


