**Abstract:**

Introduction: Hemodialysis remains the major modality of treatment for patients with end stage renal disease. Cardiovascular events and infections are commonly encountered problems in stage V chronic kidney disease patients on long term hemodialysis. Apart from this significant proportion of patients on maintenance hemodialysis develop ascites. The cause of ascites most often due to non-compliance with salt and water, cardiac failure, hepatic vein thrombosis, chronic liver disease, spontaneous bacterial peritonitis and malignancy. Inspite of extensive workup the cause may not be identifiable in a small proportion of patients which is termed as Dialysis associated ascites. The treatment of hemodialysis associated ascites is usually not satisfactory, which consists of improvement in nutrition, search for occult infection and intensification of hemodialysis regimen.

Case report: A 33 yr old gentleman with history of hypertension and severe end stage renal failure was started on maintenance hemodialysis in June 2011. He was stable till Jan 2014, after which developed pedal edema, progressing ascites and breathing difficulty. He was found to have moderate pulmonary hypertension and left ventricular dysfunction. There was persistent ascites and breathlessness after treatment of cardiac failure and intensification of dialysis. He was evaluated for other causes of ascites and found to have tuberculosis by automated culture for AFB in peritoneal fluid. He showed a gradual reduction in the abdominal girth and ascites over 6 months after starting antituberculous therapy. Conclusion: Tuberculous peritonitis is one of the rare presentation of tuberculosis in hemodialysis patients. High index of suspicion is needed and ascites of unknown cause should be thoroughly evaluated.

**Keyword:** Chronic kidney disease, Hemodialysis, Tuberculous peritonitis, Anti Tuberculous Therapy
Introduction:
Hemodialysis remains the major modality of treatment for patients with end stage renal disease. Cardiovascular events and infections are commonly encountered problems in stage V chronic kidney disease patients on long term hemodialysis. Apart from this, significant proportion of patients on maintenance hemodialysis develop ascites. The cause of ascites most often due to non-compliance with salt and water, cardiac failure, hepatic vein thrombosis, chronic liver disease, spontaneous bacterial peritonitis and malignancy. Inspite of extensive workup the cause may not be identifiable in a small proportion of patients which is termed as Dialysis associated ascites. The treatment of hemodialysis associated ascites is usually not satisfactory, which consists of improvement in nutrition, search for occult infection and intensification of hemodialysis regimen.

Case report:
A 33 yr old gentleman with history of hypertension and severe end stage renal failure was started on maintenance hemodialysis in June 2011. He was doing weekly twice haemodialysis. He remained euvolemic requiring drugs to control hypertension and was prescribed iron and erythropoietin injections for anemia. In January 2013 patient developed bilateral pedal edema, ascites and shortness of breath for routine daily work. He had undergone cardiac evaluation which showed moderate pulmonary hypertension and left ventricular systolic dysfunction. His haemoglobin was 10 g/dL. He received treatment for cardiac failure and intensification of dialysis to weekly thrice. There was persistent ascites, inspite of resolution of pedal edema and moderate improvement in breathing difficulty. He was evaluated for other causes of ascites and found to have tuberculous peritonitis.

BLOOD INVESTIGATIONS:
Hb – 7.0g/dl, WBC: 10.1x10^3/uL, Platelet count – 210x10^3/uL , ESR (1HR) : 42 mm. Peripheral smear: NORMOCYTIC HYPOCHROMIC ANEMIA.
Blood Urea : 64mg/dl, Serum Creatinine : 6.48 mg/dl , Serum Sodium – 138 mEq/L, Serum potassium – 6.71 mEq/ L, Chloride – 105 mEq/ L, Serum bicarbonate – 15.2 mEq/ L, Serum PTH – 389 pg/ mL RBS- 72 mg/dl, Serum calcium : 10mg/dl, Serum Phosphorus : 5.9mg/dl, Serum Uric acid: 4.2 mg/dl. Serum Bilirubin total 0.4mg/dl, Direct: 0.3mg/dl, Indirect: 0.1 mg/dl.
SGPT: 12U/L, SGOT: 20U/L, ALP – 72 U/L. Serum Proteins: total: 7.6g/dl, Serum Albumin 4.0g/dl, Serum Globulin: 3.6g/dl (calculated)
Prothrombin time: Test 15.9sec, control 13.0sec, INR: 1.30.

ASCITIC FLUID ANALYSIS:
Colour: pale yellow, slightly cloudy. Volume: 2ml. Total cells: 720cells/cu mm, Neutrophils: 08%, Lymphocytes: 92%, others: background shows RBCS, macrophages (+), Reactive mesothelial cells (+)
Biochemical Analysis:
Protein 4.92 g/dl, LDH: 184U/L, Glucose: 93 mg/dl, Albumin 2.77g/dl, ADA – 7.3 U/L, Ascitic fluid aerobic culture: sterile after 48 Hrs.
Cytology:
Reactive effusion, Negative for malignancy.
Automated Culture For AFB: Mycobacterium Tuberculosis complex grown in culture.

USG ABDOMEN:

DISCUSSION:
Incidence of tuberculosis in Indian patients receiving maintenance hemodialysis has been reported to be 3.72 to 13.3 %. Most of the patients develop tuberculosis within a year of starting hemodialysis. In general extra pulmonary tuberculosis is more common compared to pulmonary involvement, even though some studies quoted more incidence of pulmonary tuberculosis. Increase in risk of tuberculous infection is probably due to suppressed cell mediated immunity contributed by uremia. Both the pulmonary and extra pulmonary tuberculosis in CKD patients on hemodialysis, present with or without constitutional symptoms. In the absence of sputum, diagnosis of pulmonary tuberculosis presenting with fever and parenchymal shadows is difficult as significant proportion of these patients may not show positive tuberculin test and TB quantiferon assay also may not be positive in all patients. After excluding pyogenic infection a therapeutic trial of antituberculous drugs may be the practical approach in the management of these patients. Peritoneal tuberculosis is an uncommon site of extra pulmonary infection caused by Mycobacterium tuberculosis. The risk is increased in patients with cirrhosis, HIV infection, diabetes mellitus, underlying malignancy, following treatment with anti-tumor necrosis factor (TNF) agents, and in patients undergoing continuous ambulatory peritoneal dialysis. Tuberculous peritonitis is commonly from reactivation of latent tuberculous foci in the peritoneum resulting from haematogenous spread. The original primary focus in the lung can heal and may no longer be radiographically apparent. As the disease progresses, the visceral and parietal peritoneum become increasingly studded with tubercles. Tuberculous peritonitis manifests itself in two ways. About 97 % of the patients have the exudative or moist type with ascites, 3% of the patients represent the rare fibro adhesive form, with the plastic or dry type of tuberculous peritonitis resulting in the typical ‘doughy’ abdomen. The commonest clinical feature is therefore abdominal swelling in about 82% of cases, fever is present in about 74%, weight loss in 62 %, abdominal pain in 58%, and diarrhoea in 16%. Tuberculosis of the peritoneum can mimic a variety of other abdominal disorders and unless a high index of suspicion is maintained, the diagnosis can easily be missed or delayed. DIAGNOSIS — Tuberculous peritonitis should be considered in all patients presenting with unexplained lymphocytic ascites with a serum-ascites albumin gradient of <1.1 g/dl. The diagnosis can be difficult since the onset can be insidious and it can have a variable presentation and can frequently be seen in patients with underlying liver or renal disease.Peritoneal fluid analysis — Examination of the peritoneal fluid can be helpful in raising suspicion for the diagnosis. The majority of patients have an ascitic fluid leukocyte count of 150 to 4000 mm3, with a relative lymphocytic pleocytosis. Adenosine
Adenosine deaminase — Adenosine deaminase is a purine-degrading enzyme that is necessary for the maturation and differentiation of lymphoid cells. Adenosine deaminase activity (ADA) of ascitic fluid has been proposed as a useful non-culture method of detecting tuberculous peritonitis. Examination of Acid fast stained smear of ascitic fluid has a disappointingly low yield. Direct smear for Ziehl-Neelson stain has a reported sensitivity of 0 to 6 percent. The gold standard for diagnosis is culture growth of Mycobacterium on ascitic fluid or a peritoneal biopsy. In most series, the frequency of a positive ascites culture is disappointingly less than 20 percent. The utility of cultures is even more questionable when considering the delay of four to six weeks before a result is obtained. Routine laboratory studies are nonspecific. A normal leukocyte count is present in most patients. A mild normocytic, normochromic anemia is present in approximately 50 percent of patients. Radiologic imaging — A chest x-ray may show evidence of old tuberculosis in 20 to 30 percent of patients while features of active tuberculosis are much less common. Other radiologic features such as peritoneal thickening, omental caking, and the presence of ascites with fine mobile septations on ultrasound and CT imaging may suggest the diagnosis. Tuberculin skin testing — Tuberculin testing with purified protein derivative (PPD) is positive in approximately 70 percent of patients, however a negative result is of no help in excluding the disease. Apart from TB quantiferon test, another T-cell based testing for mycobacterium tuberculosis is an FDA approved enzyme-linked immunospot assay (ELISPOT), measuring gamma interferon producing T-cell responses to early secreted antigenic targets of mycobacterium tuberculosis, has shown promising results. Polymerase chain reaction (PCR) assay, which amplifies mycobacterial 16S ribosomal RNA, show promise of rapid detection of mycobacteria. Our patient presented with worsening ascites without constitutional symptoms. He had exudative lymphocyte predominant ascites with adenosine deaminase in normal range. Patient was started on antituberculous drugs adjusted for renal function based on ascitic fluid acid-fast bacilli culture report. Patient showed a gradual reduction in the abdominal girth and ascites over 6 months. At present patient is on regular maintenance hemodialysis weekly thrice, antituberculous therapy, antihypertensives and other supportive medications. This case report is an example that ascitic fluid with unknown cause should be thoroughly evaluated, though patient may not have any symptoms of tuberculosis.

IMAGES:
Patient six months prior to treatment

After 6 months course of ATT.

REFERENCE:


