Unusual Cause Of Sudden Onset Ultra Filtration Failure
In Long Standing Diabetic CAPD Patient

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Abstract: In this report we describe a 65 year old female who presented with sudden onset of ultrafiltration failure and was subsequently diagnosed to have sclerosing peritonitis on biopsy. She was managed with removal of PD catheter and switching over to HD. This case report highlights the importance of peritoneal biopsy with ultra structural study in CAPD patients.

Key Words: Sclerosing peritonitis, ultra filtration failure, CAPD

Introduction:
Peritoneal membrane is exposed continuously to unphysiologic dialysate solution containing high percentage of glucose, glucose degradation products, low pH, and neovascularisation. Diabetic changes in the blood vessels can produce peritoneal damage leading to ultrafiltration problem. Recurrent episodes of peritonitis can also produce peritoneal membrane failure due to adhesion and or due to changes in the transport status as a result of injury to mesothelial cells, interstitium or peritoneal capillaries. Unnoticed leak into the pleural cavity, retroperitonium, genital region leads to ultrafiltration failure.

Case report:
A 65 year old female, known case of Diabetes mellitus for 15 years, Hypertension for past 15 years, Chronic Kidney Disease – Diabetic nephropathy (5 years) on CAPD since 2 years (February 2005). Patient was on 4 exchanges of 2.5 x 2 exchanges alternate with 1.5% x 2 exchanges each of 4 hrs dwell time. She was a low average transporter. There was one episode of Staph. aureus peritonitis within one month of initiating PD.

In February 2007, patient presented to us with sudden onset of decreased outflow subsequently leading to nil outflow of 3 days duration with features of volume overload. She was initiated on hemodialysis. X Ray KUB showed catheter tip in the bony pelvis in appropriate position (Fig 1). Leak was ruled out by appropriate clinical examination. There was no history of constipation and she had good compliance. Repositioning was done by mini laparotomy to left iliac fossa, extensive adhesions were present intraperitoneally with very less space in pelvic cavity. Lysis of adhesions was done. Patency of catheter was ensured. After 3 days while attempting flushing of catheter there was no outflow, despite the catheter being in appropriate position. Hence mini laparotomy was done which showed recurrence of adhesions. Catheter was removed and peritoneal biopsy was taken and sample sent for light microscopy and electronmicroscopy. Light microscopy findings were fibrocollagenous connective tissue containing focal dense infiltrations of plasma cells, focal necrosis with infiltration of plasmacells, lymphocytes and neutrophils. There was no granuloma and Acid fast stain for Mycobacteria was also negative. Adipose tissue showed many blood vessels and foci of hemorrhage. Lightmicroscopy diagnosis was focal necrosis with subacute to chronic inflammation (Fig 2, 3). Electronmicroscopy findings were interstitial fibrosis,
thickened and laminated capillary basement membrane, thickened peritoneal lining membrane and increased fibroblasts and macrophagic cells in interstitial space (fig 4, 5). All these findings were consistent with chronic fibrosing peritoneal disease.

Patient was discharged and she was doing well on hemodialysis since February 2007 but she died in May 2009 during dialysis due to cardiac cause. She did not have abdominal pain or obstruction suggestive of sclerosing encapsulating peritonitis after removal of catheter till her death.

Discussion:

Ultrafiltration failure is defined as fluid over load in association with ultrafiltration volume of less than 400ml in a modified peritoneal equilibration test (I). Sclerosing encapsulating peritonitis (SEP) is an uncommon (1-2% of PD patients) but serious complication of chronic PD. Clinically patient may present with abdominal pain, nausea, vomiting, malnutrition and bowel obstruction, blood stained dialysate or loss of ultrafiltration. It is insidious in onset and it can even present after transfer to hemodialysis or transplantation (2). The small intestine is bound or encapsulated by a thick fibrous layer, rendering the peritoneal surface opaque. The fibrous layer resembles a thick shaggy membrane, marble, cocoon or fruit rind. The bowel so exposed may appear normal (3).

Etiology is multifactorial. It may be idiopathic (4), acetate use, recurrent or subclinical peritonitis (5,6), use of bacterial filters (7), multiple abdominal surgeries (8), intra peritoneal contamination with chlorhexidine, hypertonic acidic dialysate (6) beta blockers (6), high intradialytic peritoneal content of fibrinogen (9), intraperitoneal antibiotics like tetracycline and presence of plastic particles intraperitoneally are some of the causes.

Diagnosis was done by clinical, imaging and by biopsy. On X-Ray imaging when looking for catheter position if tip is in pelvis with outflow block - fibrin block, omental trapping or rarely an adhesion are differential diagnosis. USG shows increased small bowel peristalsis, tethering of bowel to posterior abdominal wall, echogenic strands and new membrane formation (10). The characteristic trilaminar
appearance of the bowel wall may also suggest this diagnosis (11). CT findings also vary – loculated ascites, adherent bowel loop, narrowing of bowel lumen, thickening of peritoneal membrane may be marker of subsequent development of SEP (12 & 13).

**Microscopic findings:**

Peritoneal alterations associated with PD begin with mesothelial modification. Simple sclerosis is a part of morphological alterations associated with PD. In normal conditions, a layer of microvilli covers normal, flat mesothelial cells, the intercellular junctions between mesothelial cells are tight and cells are flat in section with bulging nucleus (14-16). After start of PD microvilli reduce in number and subsequently disappears, intercellular junction tend to loosen and mesothelial cell becomes cuboidal. After prolonged period of dialysis histological alterations occur in the submesothelial layers. Normally mesothelial basement membrane is thin and single but after prolonged dialysis it thickens and duplicates. Basement membrane of blood vessels is also thin and single before dialysis but with prolonged dialysis it also thickens and duplicates. Normally sub mesothelial tissue consist of areolar tissue 2-3 mm thick with reticulate elastic lamina, occasional collagen and low density of mast cells, fibroblasts and macrophages. During dialysis submesothelial edema with increase in cellular element is seen. After prolonged dialysis microscopic particles of plastic material from bags, tubes and catheter can be found in the peritoneum. In sclerosing peritonitis progression of sclerosis occurs – inflammatory infiltrates, calcifications and typical vascular alterations which are not found in simple sclerosis are seen. The peritoneal surface is reduced to rough thickened membrane. There is aggressive sclerosis beginning in the sub mesothelial layers causing rigidity and thickening of intestinal loops progressively impeding their motility. In extreme cases the sclerotic process completely fixes intestinal loops completely preventing their movement. The mesentery, stomach, liver, spleen, gall bladder, abdominal wall may also be effected by sclerosis. The sclerosis is non homogenous and one area of abdomen may be more affected than the other forming a mass and this is described as “abdominal cocoon”. Microscopically mesothelium is almost absent, sclerotic tissue consist of dozens of irregular layers and this main constant characteristic of sclerosing peritonitis. Fibroblasts and mesoblasts are main cells in matrix and occur throughout the sclerotic tissues, more in deeper layers. In sclerosing peritonitis unlike simple sclerosis the muscle layers is compressed. Thickening of sclerotic tissue reaches very high values between 1000 and 4000μm as compared to simple sclerosis where 40-50 μm is the proposed upper limit. The thickening of sclerotic tissue is the best pathological criteria for differentiation for the two. The cellular infiltrate like mesoblasts, fibroblasts, leucocytes, erythrocytes, isolated macrophages and giant cells are found in sclerotic tissue of many cases of sclerosing peritonitis but never in simple sclerosis. Peritoneal calcifications may also be seen in sclerosing peritonitis (17-25).

Therapy of sclerosing peritonitis include both medical and surgical measures. Medical measures includes corticosteroids, immunosuppressents, total parenteral nutrition, phosphatidylcholine, progesterone and tamoxifen have been tried. Surgical measures include surgery for intestinal occlusion, suspension of PD and removal of catheter as done in our case. We do not know whether in future she would have progressed to SEP or not.

**Conclusion:**

An elderly diabetic lady on CAPD for 2 years who presented with sudden onset of outflow block with ultrafiltration failure was found to have extensive adhesions on mini laparotomy with ultrastructural findings consistent with chronic fibrosing peritoneal disease. This shows the importance of peritoneal biopsy in diagnosing the sclerosing peritonitis and thus suspension of peritoneal dialysis and early removal of catheter with switch over to hemodialysis may help, but we do not know whether in future these patients progress to sclerosing encapsulating peritonitis or not.

**References:**

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