Histopathologically, peritoneal membrane showed diffuse hyalinization, without mesothelial cell lining, and bioincompatible of peritoneal dialysis solution are a key event during the pathogenesis. Peritonitis remains a major problem in Peritoneal Dialysis. Excessive peritoneal inflammatory responses lead to mesothelial cell exfoliation and thickening of the submesothelial, resulting in peritoneal fibrosis and sclerosis.

Methods: We report two cases of the patient in Continuous Ambulatory Peritoneal Dialysis (CAPD), focus on PM histopathologic changes.

Results: Case 1. A male, 47 years old, suffered from repeated peritonitis. He was transferred to hemodialysis. After fourth episode of infection, the catheter was malfunctioned and removed. He was done , however CAPD catheter was malfunctioned and removed. He was done on CAPD for 6 years with glucose base solution. The peritoneal membrane histopathology showed diffuse fibrosis and hyalinization, without mesothelial cell lining nor inflammatory cell infiltrate. He was transferred to hemodialysis.

Case 2. A 28 years old male, came with chief complaint of fever, abdominal pain, and cloudy effluent in CAPD fluid. On third episode of peritonitis, Empirical treatment with cefotaxime intraperitoneal was done, however CAPD catheter was malfunctioned and removed. He was done on CAPD for 6 years with glucose base solution. The peritoneal membrane histopathology showed diffuse fibrosis and hyalinization, involving vessels, foci of lymphoplasmacytic cells infiltration, without mesothelial cell. He was transferred to hemodialysis.

The success of PD was dependent on the structural and functional integrity of the PM. The mesothelium lines the PM and is the first line of defense against chemical / bacterial insult. Peritonitis remains a major complication of PD and cause of ultrafiltration/technique failure, morbidity and mortality. The mortality risk of PD-related peritonitis was 16-18%. Bioincompatible PD solution is associated with production of proinflammatory and profibrotic cytokines. High peritoneal glucose exposure is associated with increased incidence of relapsing/recurrent peritonitis and conduct proinflammatory and profibrotic reactions in the peritoneal cavity leading to fibrosis and malfunction after 6-7 years. Peritoneal fibrosis was detected in 50% and 80% of PD patients within one and two years on PD. Pathological alterations in the vasculature of peritoneal include hyalinization of the blood vessels, vasculopathy, and submesothelial thickening.

Conclusions: Peritonitis episode altered peritoneal structure and functional. Neglected infection and repeated infection led to PD catheter removal. Glucose based solution may a contributing factor for membrane failure due to inflammation and fibrosis after 6-7 years. No conflict of interest