Pathogenesis of hemodialysis ascites remains unexplained. Extracellular fluid overload, peritoneal membrane changes, disturbed lymphatic drainage, hypoalbuminemia, congestive heart failure, constrictive pericarditis, hyperparathyroidism, pancreatitis, hepatitis and uremic toxins might contribute to the formation of ascites [1]. We would like to report our experience on hemodialysis patients with intractable ascites, their histopathology of peritoneum and the therapeutic approach.

Five patients with nephrogenic ascites were identified. The cause of the renal failure was chronic glomerulonephritis in 2 patients, chronic pyelonephritis and diabetes mellitus in 1 patient and there was 1 case of unknown etiology. None of the patients had received peritoneal dialysis before. All patients developed ascites after starting hemodialysis. Patient compliance to water and salt restriction was poor. All had ascites with protein concentrations > 30 g/l (table 1).

After obtaining informed consent, the patients had a double-cuffed Tenckhoff catheter inserted and ‘Baxter Healthcare System’ was used. Continuous ambulatory peritoneal dialysis (CAPD) was applied 4 times/day with 2 liters of dialysis solution. The catheters were inserted surgically and during insertion peritoneal biopsies were taken. Microscopic examination of peritoneum biopsies showed increased fibrosis and inflammatory cellular infiltration. When peritoneal equilibration test [2] was performed on day 7, according to dialysate/plasma (D/P) creatinine ratio, 3 patients were included in ‘high average transport’, 1 patient in ‘high transport’ and 1 patient ‘low average transport group’. CAPD was tolerated well and by the disappearance of the ascites, the patients were free of symptoms like abdominal distension and fullness sensation.

CAPD was discontinued in 2 patients at months 3 and 6 due to recurrent peritonitis attacks. In the remaining 3 patients, CAPD are at months 11 and 17 and still continue without any
problem. Those patients whose CAPD had to be discontinued were free of ascites clinically at 11 months.

The prognosis of patients with nephrogenic ascites is rather dismal [3]. Intensive hemodialysis, rigid fluid control, albumin infusions, abdominal paracentesis, reinfusion of ascites via a peritoneovenous catheter and intraperitoneal instillation of steroids, tetracyclines, or other drugs were reportedly not useful [3, 4].

Anatomical or functional changes of the peritoneum may play a role in ascites formation. Extracellular fluid overload is an important factor in the pathogenesis. This type of ascites has exudative forms and the peritoneum undergoes some histopathological changes. In conclusion, for the treatment of nephrogenic ascites, CAPD may be used with success and the dialysis is effective.

References


328
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Duranay/Erbilen/Bali/Sahin/Ate§/Hasanoglu/Yakinci