Clinical Report

A rare cause of chylous ascites

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Abstract

We report a patient with end-stage renal disease status after two renal transplantations. Milky-like ascites was noted since the immunosuppressant agent was switched to sirolimus (1 mg/day). Chylous ascites was diagnosed owing to the triglyceride of dialysate to serum being 15.98:15.99. Series studies were all negative. It is highly suspected that the cause of chylous ascites is sirolimus related because surgically related lymph vessel injury happens most often 6 months after transplantation. Sirolimus-related chylous ascites is a rare cause of chylous ascites but the incidence rate increases after transplantation. Side effects of sirolimus include hyperlipidemia, anemia, thrombocytopenia, hepatotoxicity, delayed wound healing and a high rate of lymphoceles, lymph edema, and pulmonary alveolar proteinosis. Chylous ascitis has improved since the switch from sirolimus to other immunosuppressant agents.

Keywords: chylous ascites; peritoneal dialysis; renal transplantation; sirolimus

This case report describes the case of a 55-year-old woman with end-stage renal disease (ESRD) due to IgA nephropathy who had received two renal transplantations, in 1988 and 1999. In the following 10 years, her renal function gradually deteriorated owing to chronic rejection, and uraemic signs such as anasarca and oliguria inevitably developed. Stepwise initiation of peritoneal dialysis using peritoneal dialysis catheter implantation by the Moncrief and Popovich technique was conducted in August 2009, and the immunosuppressant was then switched to sirolimus (Rapamune 1 mg/day) for better rejection control. During the procedure, the flushed peritoneal drain from the implanted Tenckhoff catheter was clear. However, on extraction of the buried catheter in November for intolerable uremia, milky-like ascites was noted (Figure 1). Biochemical study of the turbid peritoneal dialysate showed an elevated white cell counts (WBC 200/mm3) with 100% lymphocytes, and dialysate cultures did not yield any pathogens. The triglyceride level in the dialysate was 15.98 mmol/L, and the serum triglyceride level was 11.99 mmol/L. Chylous ascites was diagnosed. Subsequent studies including malignancy work-ups and anatomic obstructive causes of lymph vessels were all negative. On reviewing the patient’s medications, no calcium-channel blocker was prescribed.

After excluding the main causes of chylous ascites, sirolimus (1 mg/day) was considered to be the major offending agent. After tapering of the sirolimus, the peritoneal dialysate became clear and the dialysate triglyceride level declined to 5.16 mmol/L.

Chylous ascites are uncommon, and the milky appearance is due to a high triglyceride content, >11.1 mmol/L. In general, the main causes of chylous ascites involve disruption of the lymphatic system from trauma, obstruction due to abdominal malignancy or cirrhosis, infection such as tuberculosis, and medication, for example a calcium-channel blocker (Table 1) [1, 2]. However, additional differential diagnoses of chylous ascites in renal trans-
plantation patients include surgical lymph vessel injury and the immunosuppressant agent sirolimus [3]. Regarding surgical related lymph vessel injury, lymphoceles usually occur in the first 6-months post-transplantation.

Sirolimus has been increasingly used in transplantation medicine [4]. The side effects of sirolimus treatment are dose dependent, and include hyperlipidemia, anemia, thrombocytopenia, hepatotoxicity, delayed wound healing and a high rate of lymphoceles, lymph edema and pulmonary alveolar proteinosis [5]. The risk of sirolimus-related lymphoceles is 12–15% [3]. The mechanism of sirolimus-induced lymphoceles is unclear, but may be related to disruption of proliferative signals necessary to seal perivascular lymphatics and to promote wound healing [4]. After the main causes of chylous ascites are excluded, sirolimus toxicity should be considered. Switching sirolimus to other immunosuppressive agents led to complete resolution.

**Conflict of interest statement.** None declared.

**REFERENCES**


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