Case Report

Cloudy dialysate as a presenting feature of superior vena cava syndrome

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Introduction

The onset of cloudy dialysate fluid in a peritoneal dialysis (PD) patient usually heralds infectious peritonitis [1]. However, infection does not explain all cases of increased dialysate turbidity. When culture-negative cloudy dialysate is encountered, other aetiologies must be considered. These include the presence of: increased polymorphonuclear cells due to intra- or juxta-peritoneal inflammation; eosinophils, seen in association with intraperitoneal free air; red blood cells; malignant cells; and an elevated triglyceride content. The association of chylous ascites with lymphoma, pancreatitis, or catheter-related trauma in PD patients is well-recognized [2–4]. Drug-induced elevations of dialysate triglyceride have been reported also [5,6]. To our knowledge, there have been no reports of chylous peritoneal dialysate associated with progressive superior vena cava (SVC) syndrome.

Case

The patient is a 59-year-old woman with end-stage renal disease (ESRD) secondary to diabetes mellitus. She had been receiving haemodialysis via a right internal jugular Hickman catheter for approximately 2 months when the catheter clotted with associated thrombosis of the SVC. The patient had undergone placement of a subcutaneously tunnelled Tenckhoff catheter 4 weeks earlier in anticipation of a switch to PD. The haemodialysis catheter was removed, anticoagulation medication was started, and PD was initiated. Nevertheless, she developed SVC syndrome, manifested initially by mild facial swelling. During the next 2 weeks, the patient complained of progressively worsening periorbital oedema and bilateral upper extremity swelling. Concomitant with her worsening upper body oedema, she reported intermittent cloudy dialysate. She denied abdominal pain, nausea, vomiting, diarrhea, fever or chills, and exam revealed no abdominal tenderness. The patient was not taking a calcium channel blocker. Cell counts of the white dialysis effluent revealed fewer than $5 \times 10^6$ leukocytes/l and rare red blood cells. Routine, fungal and AFB cultures revealed no growth. Cytology of the fluid was elevated at 25 mg/dl.

Eight weeks after starting PD, the patient presented with mid-epigastric abdominal pain, nausea, and vomiting of 2 days duration. Serum lipase was significantly elevated at 6430 U/l (normal range 20–209) and a diagnosis of acute pancreatitis was made. The cell count of the PD fluid revealed only $5 \times 10^6$ leukocytes/l and the culture was again sterile. The triglyceride level in the effluent was again elevated at 33 mg/dl. Over the ensuing 72 h the patient experienced symptomatic improvement concomitant with a fall in her serum lipase to 313 U/l. Yet, her dialysate effluent remained intermittently cloudy.

During the next 4 weeks, the patient’s facial and upper extremity oedema worsened causing her new difficulty with speech, swallowing and breathing. Her dialysate effluent continued to be intermittently cloudy, particularly after her long nocturnal dwells. Echocardiography revealed a widely patent foramen ovale and proven right-to-left atrial shunt. The risk of paradoxical embolization was felt to be sufficiently high as to preclude thrombolysis and/or stenting of the SVC. An innominate vein to right atrial bypass was eventually performed utilizing a pericardial tube conduit. In the immediate postoperative period, the patient experienced some waxing and waning upper body oedema and she reported 1 day of cloudy white dialysate. However, in the 18 months since surgery, her symptoms have remained significantly improved and her PD effluent has remained clear. Several repeat triglyceride levels have been less than 10 mg/dl. A postoperative magnetic resonance imaging study with gadolinium demonstrated patency of the graft based on residual internal flow signal and decreased distension of the azygous vein, which had previously served as the major collateral around the obstructed SVC.

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Discussion

This case exemplifies the challenge of establishing a definitive diagnosis when culture-negative turbid dialysate is encountered. Our patient’s dialysate was characterized by normal leukocyte counts, normal red blood cell counts, no organisms, and no malignant cells. Significant fibrin strands were not visible on inspection. The triglyceride level was elevated each time the patient was noted to have cloudy dialysate and it was normal when the dialysate cleared. Therefore, we believe the most likely cause of our patient’s cloudy dialysate was the elevated triglyceride content. Certainly, the triglyceride levels we observed do not meet the often used definition of chylous ascites, i.e. a grossly milky fluid with a lipid content greater than that of plasma, a protein content more than half that of plasma, and/or the presence of microscopic fat [7]. However, triglyceride levels comparable to those we observed have been previously reported in association with turbid peritoneal dialysate [5].

Impaired lymphatic drainage in association with SVC syndrome is well recognized. Animal studies have demonstrated the development of chylous effusions in thoraces of dogs following experimental obstruction of the SVC [8]. In man, nontraumatic chylorrhea has been reported as a consequence of SVC thrombosis complicating either central venous line or LeVeen shunt placement [9,10]. Similarly, the association of chyloperitoneme with the SVC syndrome (in a non-dialysis patient) was reported by Abadoglu et al. who described a patient with Behcet’s disease that presented with, among other manifestations, SVC syndrome and chylous ascites [11].

A basic review of the anatomy and physiology of intestinal lymphatic drainage suggests a plausible mechanism whereby SVC obstruction might result in elevated dialysate triglyceride content. The source of triglyceride in chylous ascites has been shown to be nascent chylomicrons. These are the largest and lowest density lipoproteins produced by intestinal mucosal cells and are characterized by a primarily triglyceride core surrounded by a monolayer of apolipoprotein B [12]. These particles are released into a vast network syndrome and we have observed have been previously reported in association with turbid peritoneal dialysate [5].

Our patient’s cloudy dialysate did not meet the often used definition of chylous ascites, i.e. a grossly milky fluid with a lipid content greater than that of plasma, a protein content more than half that of plasma, and/or the presence of microscopic fat [7]. However, triglyceride levels comparable to those we observed have been previously reported in association with turbid peritoneal dialysate [5].

We have described the case of a woman who presented with chylous dialysate concomitant with SVC syndrome and we have offered a plausible mechanism whereby SVC syndrome might result in elevated peritoneal dialysate triglyceride content. To our knowledge this is the first report of chylous dialysate associated with SVC syndrome in a patient performing peritoneal dialysis.


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