The Case | Milky urine

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A 36-year-old woman, born and raised in India but living in Italy for the past 10 years, presented with complaints of backache and passage of milky urine for the past 6 years. This was accompanied by occasional passage of white or pinkish clots. There was no dysuria, graveluria, or fever. She used to visit India every year; and did not give any history of lymphangitis or elephantiasis. Her physical examination was normal. Urinalysis showed uniformly milky urine (Figure 1). Dipstick examination showed 3+ protein, trace sugar and microscopy revealed 10-12 erythrocytes per h.p.f. On standing, it showed layering with fat on top, followed by a layer of fibrin clots and cellular and other particulate debris at the bottom. The bacterial culture was sterile. Ultrasound examination showed a normal urinary tract and kidneys. This milkiness cleared after addition of equal volume of ether. She underwent a cystoscopy that showed efflux of milky urine from the left ureteric opening. Retrograde pyelogram was performed in Trendelenberg position and dye was instilled without any pressure.

What is the diagnosis? What would the retrograde pyelogram show?
Retrograde pyelogram (Figure 2) shows leakage of the radiocontrast agent from the pelvicalyceal system into the lacteals on the left side.

Chyluria is characterized by the passage of intestinal lymph (chyle) in urine, and develops as a result of communication between obstructed and dilated lymphatic vessels and the urinary tract. The commonest cause in endemic regions is lymphatic filariasis due to *Wuchereria bancrofti* or *Brugia malayi*. A number of other parasites have been implicated but the etiological relationship is unclear. Nonparasitic etiologies include tuberculosis, retroperitoneal abscess and malignancies, trauma, and pregnancy.

This patient had received multiple courses of diethylcarbamazine in the past for treatment of presumed filarial infection. Repeated examinations failed to reveal microfilariae in blood or urine. As she was troubled, a decision was made to treat the chyluria. A ureteric catheter was placed in the left renal pelvis, and nine instillations of 0.5% povidone-iodine solution were carried out at eight hourly intervals. The urine cleared, but milkiness returned after 2 days after which 10 instillations of 1% silver nitrate were performed. This led to complete clearing of the urine. A repeat retrograde pyelogram showed complete disappearance of the pyelolymphatic connections (Figure 3). The patient has been followed up for 9 months and is symptom free.

Renal lymphatics follow the renal vein, and through lateral aortic glands end up into the lumbar trunks. These drain into the cisterna chyli along with the intestinal trunks. Parasitic infestation leads to obliteratorive lymphangitis and impairment of valvular mechanism. Chyle from either cisterna chyli or the intestinal trunk regurgitates into the lumbar trunk and causes chyluria by rupture into pelvicalyceal system. Biochemical analysis and demonstration of lymphocytes and chylomicrons in urine confirm the diagnosis of chyluria. A combination of cystoscopy, lymphangiography, lymphoscintigraphy, and retrograde pyelography helps in localizing the site of leak. Filaricidal drugs are usually not helpful as the infection is burnt out by the time chyluria develops. Consumption of a diet that excludes fat, supplemented by medium chain triglycerides, helps in some cases. Definitive management requires renal pelvic sclerotherapy. Sclerosants induce an inflammatory reaction in the lymphatic vessels leading to closure of the communicating lymphatics by fibrosis. Recurrence is noted in 15–20% cases; either due to inadequate sclerotherapy or opening up of fresh communication; late recurrence is secondary to recanalization of obstructed lymphatics. Cases that fail sclerotherapy may need open or laparoscopic chylolymphatic or retroperitoneoscopic pyelolymphatic disconnection, microsurgical lymphovenous anastomosis or autotransplantation.

REFERENCES