haemodialysis therapy for end-stage renal failure secondary to diabetes mellitus and hypertension. The operation was uneventful and the kidney functioned well. The serum creatinine declined from 14.5 mg/dl pre-operatively to 1.36 mg/dl on day 22 post-transplantation. The patient received immunosuppressive therapy with prednisolone, tacrolimus and mycophenolate mofetil. Twenty-seven days after transplantation, the patient presented to our emergency department with general oedema and abdominal distension. The serum creatinine concentration was 1.4 mg/dl, and the ultrasound scan of the abdomen showed a large amount of ascites. Diagnostic paracentesis was performed and 21 of fluid were drained. The ascitic fluid was milky in gross appearance with the triglycerides content at 188.2 mg/dl, which was compatible with chyle [5]. The other biochemical analysis revealed cell count at 90 WBCs/mm² with all lymphocytes and was sterile for aerobic, anaerobic and tuberculous cultures. The creatinine concentration of ascites was 1.61 mg/dl, vs a urinary creatinine concentration of 20.6 mg/dl, which excludes the possibility of the fluid being of urinary origin. The lymphoscintigraphy revealed transient flush of lymphatic flow in the right lower quadrant area, abnormality of lymphatic circulation and disturbance in the lower abdomen. An abdominal magnetic resonance imaging (MRI) on day 9 after the diagnosis of chyloperitoneum showed neither enlarged lymph node nor abnormal peri-toneal fluid collection, although there was localized fluid over the anterior aspect of the transplanted kidney, which was suspected to be a lymphocele. Dietary intervention with high protein, low fat and medium-chain triglyceride was implemented and maintained for 2 months, and the patient’s symptoms did not worsen. A repeat abdominal ultrasonographic examination revealed the disappearance of lymphocele on day 65 post-transplantation. During the 15 months follow-up, the patient was free from ascites and the transplanted kidney worked well.

Post-operative chylous ascites is a rare complication that is caused either by surgical interruption of lymphatic channels or by the fenestration of lymphoceles to the peritoneum. The diagnostic tools are limited to prevent further graft damage by nephrotoxic contrast. Lymphoscintigraphy and 3D MRI may be helpful in demonstrating abnormality of lymph vasculature, especially in renal transplantation. Dietary intervention, during and after chyloperitoneum, is recommended according to our experience. The rapid recovery of chylous ascites may mean that the injury to the lymphatic system is less severe in transplanted procedure.

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Table 1. Creatinine values using different methods

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>Initial Sample</th>
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<th>Sample 3</th>
<th>Sample 4</th>
<th>Sample 5</th>
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IgM monoclonal protein presenting as pseudohypercreatininaemia

Sir,

A 63-year-old male was referred for a serum creatinine that was noted to be elevated and increasing. Routine blood tests at a community-based laboratory had revealed a urea of 7.1 mmol/l and creatinine 238 μmol/l (normal 71–133) performed on dry slide technology (DT 60, Johnson & Johnson Clinical Diagnostics). Two days later, his creatinine was 255 μmol/l on the same instrument. Renal sonogram and urinalysis were normal. A 24 h urine sample showed 0.08 g/day protein, and a creatinine clearance of 45 ml/min, based on a serum creatinine of 187 μmol/l (Vitrous 250, Johnson & Johnson Clinical Diagnostics). A repeat creatinine 3 months later on the same technology showed his creatinine to be 305 μmol/l.

He was referred to us for further evaluation. Past medical history and review of systems was unremarkable; he was on no regular medications, and denied the use of tobacco or alcohol. Repeat creatinine was 99 μmol/l (using the Hitachi 717 (Roche Diagnostics) employing a Jaffe reaction). Other labs included a normal haemoglobin, platelet count, electrolytes, calcium, phosphorus and urea. We subsequently measured creatinine in repeated and duplicate samples on the DT 60, the Vitrous 250, the Jaffe reaction and another enzymatic method (Table 1). His total protein was slightly elevated at 86 g/l, and so a serum protein electrophoresis was ordered, revealing a monoclonal IgM κ at a concentration of 28.6 g/l. A presumed diagnosis of pseudohypercreatininaemia secondary to a monoclonal protein was made. A radiopharmaceutical glomerular filtration rate was measured to be 93 ml/min, using Tc-99m DTPA. A computed tomography scan and bone marrow biopsy revealed no evidence of Waldenstrom’s macroglobulinaemia or multiple myeloma.

Table 1. Creatinine values using different methods
Three cases of elevations in serum creatinine secondary to a monoclonal IgM have been previously described in patients known to have Waldenström's macroglobulinaemia. These falsely elevated results occurred with an enzymatic assay (Roche Diagnostics) and were confirmed to be normal using high performance liquid chromatography (HPLC) [1]. Fortunately, this appears to be an uncommon problem, although possibly under-recognized. The mechanism remains unknown, although its occurrence with a number of different assays implies that there may be more than one. The previous report in the literature referred to an aqueous test method [1]. Our patients’ serum was tested with similar methods without any resulting interference.

As clinicians who see patients frequently for elevated serum creatinine values, nephrologists must be aware of the limitations of this test. Pseudohypercreatininemia secondary to a monoclonal protein should be considered in patients with an isolated creatinine elevation, particularly when associated with a normal urea, or an elevated total protein. Measuring the serum creatinine using a completely different method may prevent other unnecessary tests or procedures.

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**Creatinine and GFR: an imperfect marriage of convenience**

Sir,

While I would like to congratulate Fontsere [1] for his recent work that further elucidates the relationship between glomerular filtration rate (GFR) and serum creatinine, [1] it is not without some consternation that I watch the evolution of the use of creatinine as a marker for GFR. Since it has always been a marriage of convenience at the expense of accuracy, I think that no one should be shocked that Fontsere, as have others before him [2], found that such equations are not an accurate measure of GFR. Researchers in the early 20th century considered inulin to be the best marker for GFR, but it was inconvenient because it required a timed urine collection and the administration of an exogenous substance [3]. Oddly enough, when Rehberg [4] first proposed the use of creatinine, it was that as an exogenous administration; because there were many substances in the serum that were not really creatinine but gave Jaffe’s reaction, one could not rely on endogenous creatinine. However, since tubular secretion of creatinine often counterbalanced the overestimation in the serum by non-creatinine chromogens, a marriage of convenience was born, because the measurement of an endogenous creatinine clearance did not require the inconvenience of the administration of an exogenous substance. By the time micropuncture studies confirmed that the advantage of inulin over creatinine, we knew that creatinine clearance was indeed an inadequate measure of GFR [5], yet we continued to use creatinine because it was convenient, and by the late 20th century we had had so much experience with creatinine clearance that we really knew more about human diseases and symptoms at any given creatinine clearance than at the true GFR. In reality, we do not use the creatinine clearance to estimate GFR since we have so little experience with real inulin clearances, but we use it because we have become comfortable with creatinine and have such a vast experience with creatinine clearances that we can better predict symptoms at any known creatinine clearance than we could if we had an inulin clearance or an actual GFR. Most equations are designed to fit well with mean measured GFRs [6], but practicing physicians, unlike administrators and epidemiologists, do not deal with means but individual values. Similarly, as Fontsere correctly notes, anything that upsets that tenuous balance and interferes with the production, (including diurnal variations) [7–9] measurement [10–13] or secretion [14–16] of endogenous creatinine will further alter those estimations of GFR from equations using a solitary serum creatinine.

Therefore, the problem is not limited to one particular equation. The problem is and has always been with creatinine itself and our own search for convenience. Creatinine clearance is not and has never been synonymous with GFR, and all of the regression analysis will not make it so because the serum creatinine depends upon many factors other than filtration. We should not be surprised that the more approximations that we make, the less accurate our data becomes. The problems come when we actually delude ourselves (and others) into thinking that these equations actually represent an actual GFR.

Conflict of interest statement. None declared.


