Three cases of elevations in serum creatinine secondary to a monoclonal IgM have been previously described in patients known to have Waldenström’s macroglobulinemia. 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.

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Ethanol lock solution as an adjunct treatment for preventing recurrent catheter-related sepsis—first case report in dialysis setting

Sir,

Preliminary studies have reported the successful use of ethanol lock or flush techniques in preventing or controlling catheter-related infections in oncology patients [1] and in those on total parenteral nutrition [2–4]. Ethanol irrigation has also been used for valve disinfection of the Lifesite/C213 catheter [5]. We describe here the first documented case of use of an ethanol lock technique as adjunctive treatment for preventing dialysis catheter infection in a patient with recurrent bacteraemia.

A 70-year-old woman with a central venous catheter (CVC) at the right internal jugular site developed sepsis. The CVC was removed and replaced by another inserted at the left internal jugular site, and treatment with vancomycin plus gentamicin was initiated (day 1). Both peripheral blood culture and CVC tip quantitative culture (TQC) yielded methicillin-resistant Staphylococcus aureus (MRSA). On day 18, persistent MRSA bacteremia prompted removal of the CVC, which, when cultured, also grew MRSA. Another CVC was placed at the left subclavian site. On day 22, because of severe sepsis and CVC dysfunction, the CVC was changed, with a new CVC being placed at the left internal jugular site. Cultures from the blood and CVC tip were sterile. Venous thrombosis was excluded by ultrasound examination and endocarditis by transoesophageal echocardiography. On day 24, intermittent haemodialysis was initiated with two silicone uncuffed tunnelled dialysis catheters replacing the last CVC over guidewire exchange. No further CVC was inserted. Ethanol lock technique (each catheter was filled with 3 ml of a 60% ethanol solution containing 500 IU of unfractionated heparin per ml) was started on day 24 and repeated after each dialysis session. The locks were removed at the beginning of each dialysis session. On day 26, the patient’s fever disappeared. Repeated dialysis catheter-drawn peripheral blood cultures were sterile. On day 32, because of adverse cutaneous reactions, vancomycin and gentamicin were replaced by rifampin and trimethoprim-sulfamethoxazole. Both antibiotics and lock technique were stopped on day 53. No catheter malfunction was observed during dialysis sessions. On day 60, the dialysis catheters were systematically removed and replaced over guidewire. Contrast phase inverted microscope (DMIL, Leica) examination at magnifications 400×, 1000×, and 4000× showed no microcracking of the distal tip of the removed catheters. Six months later, the patient was persistently dialysed using the catheters inserted on day 60, without relapse of catheter infection.

Locking ethanol to the catheter lumen may induce systemic spill-out of the ethanol. We did not measure blood ethanol concentration after ethanol lock, but our patient experienced no adverse events. In a study performed in children, flushing a 74% ethanol solution through catheters into children with catheter-associated bacteremia induced only mild adverse effects [1].

Ethanol is an effective disinfectant with a broad range of antimicrobial activity; it is safe, easy to use, inexpensive and has never been implicated in acquired resistance. However, an ethanol concentration above 40% is required to inhibit bacterial growth in established biofilms [6]. For our patient, we used a 60% ethanol solution. Catheter occlusion was observed after 100% ethanol lock in a recent report, which, however, did not specify the type of catheter used [7]. In our case we used silicone catheters, whose mechanical properties are not altered even after prolonged ethanol exposure [8].

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