A 12-year-old girl was admitted to the hospital with a history of fatigue, abdominal pain, vomiting and dark urine. On admission, she had arthralgia, oedema and a palpable purpuric rash on the lower extremities. Past medical history revealed that a tonsillectomy had been performed at the age of 9 years, and Graves’ disease was diagnosed at the age of 10 years. She was commenced on propylthiouracil and subsequently remained euthyroid state on a stable dose of propylthiouracil. She was hypertensive, and laboratory investigations were: ESR 59 mm/h, BUN 74 mg/dl, serum creatinine 1.8, serum albumin 3.2 g/dl, cholesterol 177 mg/dl, serum IgA 291 mg/dl, creatinine clearance 13 ml/min/1.73 m², antinuclear antibody negative and anti-ds DNA antibody negative. Urinalysis showed many RBCs/HPF and proteinuria 30 mg/dl. She was diagnosed as having acute nephritic type of HSP nephritis, and treated with methylprednisolone pulse therapy followed by prednisolone, calcium-channel blocker (Madinine) and propylthiouracil. She achieved complete remission of HSP nephritis including resolution of haematuria and proteinuria at 1.4 years of follow-up. About 3 years after the development of HSP, the patient is still in complete remission without recurrence of vasculitis.

Our cases suggest that HSP may develop in a patient with Graves’ disease receiving the usual dose of propylthiouracil or no treatment. Therefore, it is likely that not only propylthiouracil use but also Graves’ disease itself may be a triggering factor for the development of HSP in susceptible individuals. However, a large prospective study may further elucidate the clear relationship between HSP and Graves’ disease or propylthiouracil use.

Conflict of interest statement. None declared.

The Institute of Kidney disease, Department of Pediatrics, Yonsei University College of Medicine, Seoul, Korea. Email: jsyonse@yumc.yonsei.ac.kr

Ji Hong Kim
Duk Hee Kim
Ho Sung Kim
Pyung Kil Kim


doi:10.1093/ndt/gfl287
leucocyte counts. In our patients, three out of 23 (13.04%) CAPD patients given lercanidine developed non-infective turbid peritoneal dialysis.

Differential diagnosis of non-infectious cloudy peritoneal dialysate includes cellular causes (i.e. leucocyte, eosinophils, red cells and malign cells) and non-cellular causes (i.e. triglyceride and drugs) [2]. Yoshimoto et al. [1,3] reported that benidipine, manidipine, nisoldipine and nifedipine caused cloudy dialysate on CAPD patients. In their study, it was revealed that the fluid contained an elevated triglyceride concentration. As far as we know this is the first report in the literature that lercanidine causes cloudy dialysate in the CAPD patients.

Conflict of interest statement. None declared.

1 Trabzon Training and Research Hospital Cevat Topal
2 Sutcu Imam University, Nephrology Ekrem Dogan
Kahramanmaras, Turkey Reha Erkoc
3 Yuzuncu Yil University Yasemin Soyoral
Nephrology, Van Turkey
Email: hayriyesayarlioglu@yahoo.com; hayriyesayarlioglu@gmail.com


doi:10.1093/ndt/gfl269
Advance Access publication 30 May 2006

Effect of creatinine assay standardization on the performance of Cockcroft–Gault and MDRD formula in predicting GFR

Sir,

The recent article of van Biesen et al. [1] illustrates the importance of standardization of serum creatinine assays when using formulas to estimate glomerular filtration rate (GFR). We provide further proof of the importance of standardization of creatinine. We previously compared the performance of the modification of diet in renal disease (MDRD) formula and the Cockcroft–Gault (CG) formula in healthy persons and normo-albuminuric diabetic patients [2]. As reported in this Journal, we concluded that the MDRD equation was less accurate than CG formula in predicting GFR (measured by inulin clearance). Our data were based upon a creatinine assay, based on the Jaffé kinetic reaction performed on a Hitachi 747 auto-analysers.

Meanwhile it has been shown that correct use of the MDRD equation necessitates calibration of the creatinine assay against the assay used by the Cleveland Clinic Laboratory [3].

The Cleveland Clinic Laboratory originally used a Beckman modified kinetic rate Jaffé reaction. Recently, samples of the MDRD study were re-analysed and compared with a creatinine assay using isotope dilution mass spectrometry (IDMS) as the gold standard [4]. IDMS results were highly correlated to enzymatic creatinine, measured by Roche technology. Therefore, this enzymatic assay should preferably be used in the MDRD equations.

We recently introduced the Roche enzymatic method in our Hospital. Comparison of the enzymatic method with our former Jaffé method according to an approved evaluation protocol [National Committee for Clinical Laboratory Standards, Evaluation Protocol number 9 (EP-9)] revealed: y (enzymatic creatinine) = 1.266 × (Jaffé creatinine) − 29. Based on this formula, we recalculated our creatinine data and applied the new formula [4]:

\[
170 \times \left[ \frac{[\text{enzymatic creatinine (mg/dl)}]}{0.95} \right]^{0.999} \\
\times (\text{age})^{-0.176} \times \left[ \frac{\text{Serum albumin (mg/dl)}}{\text{Nitrogen (mg/dl)}} \right]^{-0.170} \\
\times \left[ \frac{\text{albumin (g/dl)}}{0.762} \right]^{10.318}
\]

Results are given in Table 1. It is apparent that the use of standardized creatinine assay has a major influence on the reported differences between the formula-derived GFR and the true GFR. In fact, in contrast to our earlier finding, our recalculated data indicate that the MDRD formula is probably more accurate than the CG formula, also in persons with normal GFR. Reporting of MDRD-GFR using creatinine assays that are not calibrated using a gold standard should be discouraged.

Conflict of interest statement. None declared.

Table 1. Measured and predicted GFR in healthy subjects and diabetes patients without [2] and with standardization of creatinine assay

<table>
<thead>
<tr>
<th>GFR (inulin clearance)</th>
<th>Former Jaffé method</th>
<th>New enzymatic method</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CG</td>
<td>ΔCG - GFR</td>
</tr>
<tr>
<td>Healthy subjects (n = 46)</td>
<td>107 ± 11</td>
<td>112 ± 17</td>
</tr>
<tr>
<td>Diabetic patients (n = 46)</td>
<td>122 ± 18</td>
<td>119 ± 16</td>
</tr>
</tbody>
</table>

Values are expressed as means ± SD; GFR, glomerular filtration rate (ml/min/1.73 m²); CG, prediction of GFR with Cockcroft–Gault formula (ml/min/1.73 m²); MDRD, prediction of GFR with the MDRD formula (ml/min/1.73 m²).