A longitudinal study of kidney structure and function in adults

Dear Editor,

I read the article by Kariyanna et al. [1] with great interest. The authors discussed a relatively neglected issue in clinical nephrology, namely interaction between kidney size and function. The authors drew some important conclusions which, in my opinion, are difficult to accept due to limitations of the study. I will briefly discuss these limitations.

Firstly, the authors used ultrasound to determine kidney size in an obese population, in which the sensitivity reduces dramatically (BMI 30.2 ± 4.9). Ultrasound was also performed by various technicians with different machines. Intra- and inter-observer difference was not controlled in the study which claims that kidneys shrink at a rate of 0.072 cm per year. Ultrasound is not the gold standard as it was suggested by the authors to determine kidney size. In a comparative study, Ninan et al. [2] only compared ultrasound with abdominal plain X-ray, intravenous pyelogram, and renal angiogram but not with renal MRI or CT. To detect such subtle changes, standard MRI or CT scans should have been used or at least more measurements should have been performed with ultrasound (median follow-up is 3.7 years in the study).

Secondly, the MDRD equation is not the perfect way to calculate the actual glomerular filtration rate. Twenty-four-hour urine collections, or insulin clearance ideally, would be better. Equations are especially prone to error in elderly and obese patients as applied in this study.

Thirdly, it is not evident from the paper what the authors used as kidney size: the maximal longitudinal length of the bigger kidney or the mean of the two? We know size differences between the two kidneys as a variant of normal size can exist [3]. Some diseases such as stone disease, ischaemic nephropathy and obstructive nephropathy may further disproportionately affect the kidneys. Thus, a more exact analysis of kidney size should be offered.

Lastly, a standard rate of atrophy for all aetiological subclasses of chronic kidney disease seems unreasonable. Severity of the disease changes from patient to patient and various aetiologic subclasses progress at different rates. Thus, a constant atrophy irrespective of the aetiology may be a result of the retrospective study design and relatively small sample size. Al-Said et al. showed that even simple renal cysts may affect kidney size and function inversely [4,5]. However, in the current study, the authors did not mention the status of simple renal cysts in their patients.

In conclusion, it is very difficult to ascertain from this that kidney atrophy occurs independently of the underlying aetiology of chronic kidney disease by such a data set.

Conflict of interest statement. None declared.

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References


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The diffusion gradient between ionized calcium in dialysate and plasma water—corrected for Gibbs–Donnan factor—is the main driving force of net calcium balance during haemodialysis

Sir,

I read with interest the debate on calcium mass balance [1–3]. We then compared in the same patient two consecutive sessions respectively with 1.5 and 1.25 label calcium, sampling from the dialyser in/out at both dialysate and blood ports, after the start and before the end of the session, for total calcium (Roche colorimetric-cresolphthalein) and ionized calcium (Nova Biomedical Stat Profile®). CMB has been determined at the dialysate side (pCMB) and blood (pCMB) side, by an original formula that allows the calculation of solute integral concentration (Cint), avoiding dialysate collection. The solute Cint is calculated by the following equations [4]:

\[
\varepsilon = t / \ln \left( \frac{\text{CaD}}{\text{CaP}} \right)
\]

Eq.1

\[
\text{Cint} = t \times \text{Ca}^{++} \times \exp \left( \frac{\varepsilon}{t} \right)
\]

Eq.2

Dialysate-side CMB:

\[
\text{diffD} = \text{intCa}^{++} - \text{intCa}^{++} \times Q_d \times t
\]

Eq.3

\[
\text{convD} = \text{intCa}^{++} \times Q_f \times t
\]

Eq.4

\[
\text{globalD} = \text{diffD} + \text{convD}
\]

Eq.5

Blood-side CMB is then obtained as follows:

\[
\text{diffB} = \text{intCa}^{++} - \text{intCa}^{++} \times Q_pw \times t
\]

Eq.6

\[
\text{convB} = \text{intCa}^{++} / 1.12 \times Q_f \times t
\]

Eq.7

\[
\text{globalB} = \text{diffB} + \text{convB}
\]

Eq.8

where \(Q_d\) = dialysate flow (L/min), \(Q_pw\) = plasma water flow (L/min), \(Q_f\) = ultrafiltration rate (L/min), \(t = \) dialysis time (min) and 1.12 = Donnan coefficient [5]. The average ionized/total calcium was 53.9 ± 0.02% in blood and 86.7 ± 0.02% in dialysate, similar to Basile’s data. From the changes in ionized calcium concentrations (Table 1), we may observe that using CaD = 1.5 mmol/L after the start of dialysis, an evident increase at the blood side (from 1.21 to 1.34) does correspond to a consensual opposite decrease at the dialysate side (from 1.31 to 1.20), suggesting a net calcium transfer from dialysate to blood, and a similar trend is maintained at the end of dialysis. Using CaD = 1.25 mmol/L, these variations are less evident; notwithstanding, despite an apparent positive gradient from blood to dialysate (1.17 vs 1.10), the variations in concentration do suggest a transfer of calcium from dialysate to blood: this may be explained by the Donnan effect of plasma proteins. The CMB data (Table 2) allow us to examine calcium transfer: it must be pointed out that if we compare \(\mu\)CMB and \(\nu\)CMB mass, balances do not close, due to rapid diffusion equilibrium with the calcium buffer pool in periosteum and exchangeable bone surface calcium [1]; therefore, \(\mu\)CMB has to be considered underestimated, while \(\nu\)CMB is most reliable. Using CaD = 1.5, the diffusive \(\mu\)CMB is strongly negative (−282.5 mg), while \(\nu\)CMB is positive (+182.29 mg), though underestimated for the reason above considered; the convective \(\nu\)CMB is slightly negative, and we must note that it closes almost perfectly in the comparison of blood and dialysate side if corrected taking account of the Donnan factor (see Eq. 7 above), as ultrafiltrate calcium is lower than plasma water concentration. The resulting global CMB shows, therefore, a strongly positive calcium transfer from dialysate to blood. On the contrary,

### Table 1. Results of samples performed in/out at blood and dialysate side of the dialyser, measuring both total and ionized calcium

<table>
<thead>
<tr>
<th>CaD</th>
<th>Unit</th>
<th>Initial</th>
<th>Final</th>
<th>Integral</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.5</td>
<td>mEq/L</td>
<td>4.6</td>
<td>4.8</td>
<td>4.699</td>
</tr>
<tr>
<td>1.5</td>
<td>mEq/L</td>
<td>4.7</td>
<td>5.0</td>
<td>4.849</td>
</tr>
<tr>
<td>1.25</td>
<td>mEq/L</td>
<td>3.0</td>
<td>3.0</td>
<td>3.000</td>
</tr>
<tr>
<td>1.25</td>
<td>mEq/L</td>
<td>2.8</td>
<td>2.8</td>
<td>2.800</td>
</tr>
<tr>
<td>1.5</td>
<td>mmol/L</td>
<td>1.21</td>
<td>1.22</td>
<td>1.215</td>
</tr>
<tr>
<td>1.25</td>
<td>mmol/L</td>
<td>1.34</td>
<td>1.37</td>
<td>1.355</td>
</tr>
<tr>
<td>1.5</td>
<td>mmol/L</td>
<td>1.31</td>
<td>1.27</td>
<td>1.290</td>
</tr>
<tr>
<td>1.25</td>
<td>mmol/L</td>
<td>1.20</td>
<td>1.22</td>
<td>1.210</td>
</tr>
</tbody>
</table>

The integral value is obtained by an original formula (see text).

### Table 2. CMB was measured from integral Ca²⁺ (see text)

<table>
<thead>
<tr>
<th>Blood side</th>
<th>Dialysate side</th>
</tr>
</thead>
<tbody>
<tr>
<td>CaD = 1.5 mmol/L</td>
<td>CaD = 1.25 mmol/L</td>
</tr>
<tr>
<td>Diffusive</td>
<td>Convective</td>
</tr>
<tr>
<td>182.29</td>
<td>−67.75</td>
</tr>
<tr>
<td>67.76</td>
<td>4.6</td>
</tr>
<tr>
<td>114.55</td>
<td>4.6</td>
</tr>
</tbody>
</table>

The values are converted from millimole to milligram.