

Letters to the Editor

Kidney Size and Glomerular Filtration Rate in Type 1 (Insulin-Dependent) Diabetes Mellitus Revisited

Dear Sir,

The question of what determines the elevation of glomerular filtration rate in some Type 1 (insulin-dependent) diabetic patients remains vexed. Almost two schools of thought have evolved, one favouring the role of increased kidney volume, and by implication glomerular filtration surface area, as the major determinant of glomerular filtration rate (GFR) [1], and the other supporting the view that alterations in the hormonal and metabolic milieu account for the major part of this elevation [2].

Gundersen and Mogensen [3], using multiple regression analysis on the combined data from three studies, came to the conclusion that the increased kidney size in Type 1 diabetic patients is the major and perhaps the sole determinant of the increased GFR. However, as these authors recognise, there is no way that a statistical analysis can open up the ring of interdependent variables kidney volume → GFR → kidney volume. Moreover, analyses of data in previous studies are hampered by small numbers of patients and use of different techniques for the estimation of renal volume [4–6].

We have made paired measurements of kidney volume using an ultrasound technique [7], and GFR, as ⁵¹Cr EDTA clearance, using the single bolus method [8] in 35 Type 1 diabetic patients and 13 age-and sex-matched control subjects. The normal range for GFR was 88–135 ml/min per 1.73 m² (mean 113) and for kidney volume 210–298 ml/1.73 m² (mean 255). The Type 1 diabetic patients as a group had increased GFR (96–178 ml/min per 1.73 m² (mean 129) and kidney volume 211–412 ml/1.73 m² (mean 297).

In the control group we found no significant correlation between kidney volume and GFR (r=0.06; NS). This is in agreement with Christiansen et al. [5], who used similar techniques for kidney volume estimation, but in contrast to Mogensen and Andersen [4], who de-

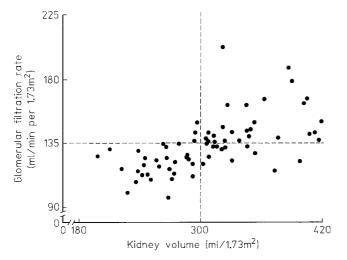


Fig. 1. A plot of 69 paired measurements of kidney volume and glomerular filtration rate in 35 Type 1 diabetic patients. The broken lines designate the upper limits of the normal ranges. Only three measurements with kidney volume at the upper limit of the normal range out of 31 with normal kidney volume (9.7%) had slightly elevated GFR, while 12 of 38 measurements with high kidney volume (31.6%) had normal GFR, even with grossly elevated kidney volume

duced kidney volume from IV pyleograms, and Puig et al. [6], who deduced it from plain X-rays and used creatinine clearance as an estimate of GFR. Conversely, we found as others before [4–6], a strong correlation between kidney volume and GFR in the diabetic group as a whole (r=0.75, p<0.001). However, no significant correlation was found in those Type 1 diabetic patients with normal GFR (r=0.26; NS), while a strong correlation was maintained in those diabetic patients with supranormal GFR (r=0.69, p<0.01).

Figure 1 shows paired measurements of kidney volume and GFR taken in 35 diabetic subjects. In some patients, determinations were repeated several times under different metabolic conditions as part of a treatment trial. It appears that Type 1 diabetic patients with normal kidney volume are highly unlikely to develop a high GFR, whereas it seems perfectly possible to have a normal GFR despite an elevated kidney volume. To have a high GFR a large kidney seems to be required ($Chi^2 = 18.56$, p < 0.0005). Thus kidney volume undoubtedly plays an important role in the genesis of the high GFR in Type 1 diabetes, but this can only be a permissive, not a primary causative one. In other words, elevated kidney volume is a necessary but not sufficient pre-requisite for the development of high GFR in Type 1 diabetes, and other non-morphological factors must therefore also be important.

Yours faithfully, M. Wiseman, and G.C. Viberti

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