

information is necessary to judge the usefulness of this parameter, we quantified beta₂-microglobulin, serum creatinine, and BTP in patients with diabetic nephropathy. Forty-one patients with a diminished GFR (less than 80 mL/min) were compared with 74 patients with a normal GFR (greater than 80 mL/min). Inulin clearance was measured for all patients as the reference standard. The results were compared with those obtained for BTP, which was quantified by a different but equally sensitive method (latex particle-enhanced immunonephelometry using rabbit polyclonal antibodies) as already described by Melegos *et al.*¹ Our results that were recently published² indicated the following. (1) Similar to serum creatinine, serum BTP shows a curvilinear behavior in relation to GFR. (2) There is a significant correlation between GFR and the reciprocal concentrations of serum BTP. This is comparable to the results obtained for serum creatinine ($r = 0.672$ versus $r = 0.666$). (3) Serum-BTP is a better discriminator between patients with normal and reduced GFR than beta₂-microglobulin or creatinine as indicated by the receiver operating characteristics (ROC) curve analysis (area under the curves 0.853, SE 0.039 versus 0.771, SE 0.049 or 0.746, SE 0.053).

Our data support the view that BTP may be a suitable indicator of reduced GFR even in the creatinine-blind range and, of great interest for urologists, to characterize the kidney function using sensitive parameters other than creatinine.

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REPLY BY THE AUTHORS:

Earlier this year, we reported our findings on serum levels of prostaglandin D synthase (PGDS) in patients with renal failure.¹ Using an immunofluorometric procedure for measuring PGDS in serum, we found highly elevated levels of this enzyme in patients with various forms of renal failure and treatments. PGDS is produced by the brain, is present at high levels in cerebrospinal fluid (CSF), and diffuses into the general circulation, ending up in the urine. Minimal reduction in GFR causes marked increases of PGDS concentration in serum. Thus, this analyte may be useful in assessing patients with early renal failure.

Subsequent to our publication, Priem *et al.*² independently reported similar findings. In the current letter, they summarize their data which essentially conclude that PGDS concentration in serum (PGDS is also known as beta-trace protein, BTP) correlates negatively with GFR and positively with creatinine. They also found that this new marker is superior to both creatinine and beta₂-microglobulin as a marker of GFR. These authors analyzed BTP with a new immunonephelometric procedure.

The concordance of our study¹ and the data of Priem *et al.*² confirms that PGDS may be a new, valuable marker of early renal failure. We need more extensive clinical studies to verify and extend these original observations. The commercial availability of methods for quantifying PGDS in serum should facilitate these new studies. An enzyme-linked immunosorbent assay for PGDS measurement in serum has recently become available from Diagnostic Systems Laboratories, Inc., Webster, Texas. PGDS has further utility as a marker of obstruction of the epididymis/vas deferens when measured in seminal plasma.³

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