Perineal Approach to Radical Prostatectomy in Kidney Transplant Recipients with Localized Prostate Cancer

TO THE EDITOR:

In a recently published article, Yiou and associates1 suggest that the perineal approach to radical prostatectomy may decrease the risk of allograft damage in patients who have previously undergone renal transplantation. In the past year I have performed two radical retropubic prostatectomies in men with transplanted kidneys in the right iliac fossa. In both cases the surgery was unexpectedly straightforward. In obtaining exposure it was unnecessary to mobilize the peritoneum from the ipsilateral side of the pelvis in which the transplanted kidney was located, and the blade of the self-retaining Balfour retractor was simply placed above the rectus muscle on that side. The peritoneum was mobilized in the usual fashion on the contralateral side, and the blade of the Balfour retractor placed beneath the rectus muscle as usual, providing excellent exposure. It was obviously not possible to perform a pelvic lymphadenectomy on the side of the transplanted kidney, but both of these patients had T1c disease with a very low probability of pelvic lymph node metastases.

The remainder of the surgery was uncomplicated in both cases. When displacing the bladder cephalad, I was particularly careful in placing the malleable blade of the retractor so as not to injure the transplant ureter. Because the space of Retzius had not previously been entered, removal of the prostate and the subsequent vesicourethral anastomosis in both cases proceeded uneventfully. Although the perineal approach can be used in these cases, I do not believe that the retropubic approach is contraindicated in patients who have previously undergone renal transplantation.

Charles B. Brendler, M.D.
Department of Urology
University of Chicago Medical Center
5841 South Maryland Avenue
Chicago, IL 60637

REFERENCE


REPLY BY THE AUTHORS:

As reported by Kinahan et al1 radical retropubic prostatectomy can be performed safely in men with transplanted kidneys. As presented in Dr. Brendler’s letter, some precautions should be taken to avoid injury to the transplant ureter (insertion of the retractor, no ipsilateral lymphadenectomy). Thus, retropubic prostatectomy is practicable in transplanted patients. However, the surgical approach should be appropriate to each case. We have experience in the two surgical approaches (retropubic and perineal). With the use of prostate-specific antigen, prostate cancer will be detected before and after renal transplantation. In our mind, for this special population, perineal radical prostatectomy is the most appropriate surgical approach because first, in male candidates for renal transplantation, it preserves the right fossa iliaca and the bladder for future transplantation; second, in men already transplanted, it avoids any renal graft and ureteral damage. We do not contraindicate the retropubic approach, but we believe that the perineal approach is more appropriate in both these situations.

Laurent Salomon, M.D.
Hôpital Henri Mondor
Service de Chirurgie Urologique de M. le Pr. Abbou
51, Avenue du Mal de Lattre-de-Tassigny
Creteil Cedex, 94010, France

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Charles B. Brendler, M.D.
Department of Urology
University of Chicago Medical Center
5841 South Maryland Avenue
Chicago, IL 60637

REFERENCE


TO THE EDITOR:

The article by Melegos et al3 describes an interesting investigation concerning prostaglandin D synthase (also known as beta-trace protein, BTP) as a potential marker for reduced glomerular filtration rate (GFR). The authors demonstrated increased serum BTP concentrations in patients with renal failure. They also suggested that it would be worth examining whether the sensitivity for detection of renal failure could be increased using BTP rather than the quantitation of serum creatinine alone. Hoffmann et al2 came to practically the same observations.

GFR as an indicator of renal impairment is best measured by inulin or radioisotope clearance. However, these techniques are cumbersome and need specialized technical instrumentation. Thus, serum creatinine is commonly used for this purpose. Serum creatinine concentrations, however, increase only after drastic reduction of GFR (to less than 50%). Therefore an intensive search for substances that indicate reduced GFR in the creatinine-blind range has currently begun. Low-molecular-mass proteins such as beta2-microglobulin, cystatin C, and now perhaps BTP seem to be ideal candidates for this purpose.3,4 We will shortly report on our own experience with the BTP that complements the results described by Melegos et al.1

Little or no information on the relationship between BPT and GFR is available in the scientific literature. Since this

Laurent Salomon, M.D.
Hôpital Henri Mondor
Service de Chirurgie Urologique de M. le Pr. Abbou
51, Avenue du Mal de Lattre-de-Tassigny
Creteil Cedex, 94010, France

REFERENCE


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 published3 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.

Markus Giessing, M.D.
Department of Urology
University Hospital Charité
Humboldt University
Schumannstrasse 20-21
D-10098 Berlin, Germany

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REFERENCES


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

E. P. Diamandis, M.D.
Department of Pathology and Laboratory Medicine
Mount Sinai Hospital
600 University Avenue
Toronto, ON M5G 1X5, Canada

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