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Vascular lesions after percutaneous biopsies of renal allografts

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Abstract. On the basis of two arteriovenous fistulas, one arteriocaliceal fistula, and the literature concerning these complications, clinical symptoms, diagnostic measures, and therapeutic strategies are discussed. Decreased renal function, severe hypertension, and a bruit over the transplant site - particularly after core biopsy - are said to be indicative of an arteriovenous fistula, while persisting hematuria is seen as evidence of an arteriocaliceal fistula. In both cases, angiographic evaluation is indicated. Therapeutic possibilities include selective angiographic embolization and surgical repair. Large intraparenchymal fistulas may require wedge resection.

Key words: Vascular lesions in kidney biopsy - Arteriovenous fistulas in renal transplantation - Arteriocaliceal fistulas in renal transplantation.

Arteriovenous fistulas (AVFs) are known to occur in 0%-18% of the cases following percutaneous biopsies of native kidneys [5, 6]. No data, however, are available on arteriocaliceal fistulas (ACFs). Both types of vascular complication have reportedly been associated, on occasion, with percutaneous biopsies of renal allografts, causing significant impairment of graft function and/or severe hypertension or hematuria, respectively. We have observed two AVFs and one ACF in a series of 700 consecutive renal transplants with a total of 1170 core biopsies (0.3%). What follows is a discussion of the clinical symptoms that were observed, the diagnostic measures taken, the therapeutic strategies employed, and the results achieved, all presented in light of the existing literature.

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Case reports

Case 1

A 23-year-old man suffering from end-stage glomerulonephritis received a cadaveric renal allograft on 8 June 1984. The donor was a 35-year-old male. Cyclosporin A (CyA) and steroids were used for immunosuppression. An increase in creatinine from 2.3 mg/ml to 4.2 mg/ml led us to perform a needle biopsy (Tru-Cut Needle, Travenol, Deerfield, Illinois, USA) on day 17 after transplantation. Histological examination showed CyA damage. Two days after the biopsy, a severe macrohematuria became apparent for the first time and subsequently led to a bladder tamponade, which necessitated several endoscopic evacuations. Since hematuria persisted over a period of 19 days at varying intensities, angiography was performed and revealed an AVF in the upper portion of the kidney. In order to stop the hematuria, the artery feeding the fistula (Fig. 1) was selectively embolized with Spongostan (Spongostan M.E.D.U. D-25 Ferrosan, Soeborg, Denmark). Approximately 25%-30% of the parenchyma was lost due to this embolization. Graft function after 51 months remained stable; it was, however, reduced (creatinine 2.3 mg/ml).

Case 2

A 42-year-old woman with renal failure due to morbus Pringle-Bourneville received a kidney allograft from a 50-year-old cadaveric donor on 12 March 1984. Immunosuppressive therapy was the same as that described in case 1. After an uneventful postoperative course, the patient was discharged with largely normal graft function. In January 1987, a needle biopsy was performed because of an increase in creatinine from 1.4 mg/ml to 1.8 mg/ml and the onset of proteinuria. Histological examiantion revealed a de novo glomerulonephritis. Three weeks later, the pre-existing hypertension deteriorated even further. The response to various types of medication was negligible. During a hypertensive crisis (200/120 mm Hg), several angiolipomas ruptured spontaneously with extensive bleeding into the retroperitoneal space. In the course of an emergency laparatomy, uncontrollable bleeding required the removal of several angiolipomas, as well as bilateral nephrectomy and adrenalectomy. The latter, however, did not improve hypertension. A bruit was clearly heard over the transplant and was angiographically traced to a fistula in a large branch of the renal artery (Fig. 2). At that time, the serum creatinine level was

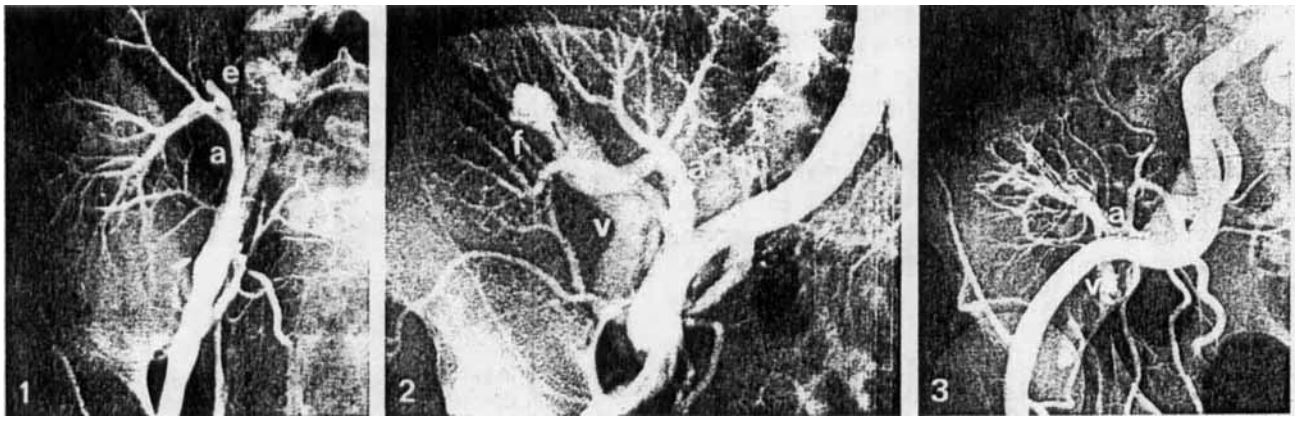

Fig. 1. Embolized arterial branch leading to the upper portion of the kidney graft. *a* Renal artery, *e* embolized arterial branch

Fig. 2. Arteriovenous fistula in a branch of the renal artery with rapid passage of the contrast medium through the fistula into the renal vein. *a* Renal artery, *v* renal vein, *f* arteriovenous fistula

Fig. 3. Arteriovenous fistula in a central branch of the renal artery with rapid passage of the contrast medium into the renal vein. The fistula cannot be precisely located. *a* Renal artery, *v* renal vein

already 4.9 mg/ml. It was decided to attempt surgical repair of the fistula. The main renal artery was followed into the hilum. Intraoperative auscultation during clamping of various branches made it possible to identify the site of the fistula. After careful dissection of both the fistula-bearing artery and vein, the fistula was transected and the artery oversewn while the vein was simply ligated. After successful surgical repair, hypertension was well controlled with prazosin and nifedipine (150/100 mm Hg). Upon discharge, the creatinine level had dropped to 3.1 mg/ml and now, 20 months later, is 2.2 mg/ml. Proteinuria remained stable with 10-30 mg/day.

Case 3

On 22 May 1987, a cadaver kidney was transplanted into a 41-year-old patient suffering from end-stage chronic glomerulonephritis. Immunosuppressive therapy consisted of CyA and low-dose steroids. A core biopsy was carried out on day 4 after transplantation in order to evaluate function. This and two additional biopsies on days 10 and 21 revealed both severe interstitial and vascular rejection. Despite pulses of steroids and repeated plasmaphereses, graft function did not improve. A perfusion scan 5 weeks after transplantation showed hardly any perfusion of the transplant, something which was confirmed by angiography. It did, however, reveal a large fistula between the main renal vein and artery (Fig. 3). Because of the severe, therapy-resistant rejection, the graft was removed without even attempting surgical repair. The patient is currently awaiting a second transplant.

Discussion

ACF is an extremely rare complication following renal allograft biopsy. Thus far, only two cases have been reported [2, 8]. In both instances bleeding was stopped by means of selective embolization. When a segmental artery is involved, as in our patient, spontaneous healing is very unlikely, whereas connection of smaller interlobular arteries with a calix may close without further treatment. Severe persistent hematuria always warrants angiographic evaluation.

In contrast, AVFs have been reported to occur in up to 18% of cases after biopsy of native kidneys when angiographically controlled [5, 6]. The degree of glomerular function impairment is thought to correlate with the shunt volume [4]. Obviously, only a small number of these lesions reach clinical relevance. It has been calculated that only 4% of AVFs persist, whereas the rest all disappear spontaneously [11].

AVFs that develop in a renal allograft are most often caused by percutaneous biopsies [1, 3, 7, 9]. In the mid-seventies, some transplant surgeons suggested performing open biopsies in order to prevent these complications from occurring [3]. However, now that transplants can be reliably located by ultrasonography, even in obese patients, this technique, never widely used, has been completely abandoned. Other causes of AVFs include accidental trauma, infection, rupture of an aneurysm, and technical failure during the procurement procedure. Yet, these occur quite seldom [7, 11]. An immunological lesion of the vessel wall during a rejection episode has also been reported to be a possible cause of AVFs [9], as has local mycosis [7]. Finally, an anecdotal case of an AVF deserves mention: a thick-walled vein was erroneously thought to be a transected pole artery and, as a result, was anastomosed ex vivo with the renal artery [9].

As for the symptomatology of AVFs, severe hypertension occurs in about 30% of the cases [10], and this may be refractory to antihypertensive medication, as was true in our second patient. Increased renin production, due to poor renal perfusion, is thought to activate the so-called Goldblatt mechanism, seriously aggravating hypertension that many

renal allograft recipients have already acquired from steroids and/or cyclosporin A therapy, from previous rejection episodes, or from their underlying renal disease. Another typical symptom of AVFs is a specific bruit that can easily be distinguished from the classic arterial stenosis type by its machine-like, drawn-out character [3]. This bruit was clearly observed in our second patient. During reoperation we found auscultation most helpful for identification of the artery deep in the hilum. Random clamping silences the bruit, thus making the feeding artery clearly identifiable.

As far as the treatment of clinically relevant AVFs is concerned, selective embolization of the feeding arterial branch should be attempted whenever possible. If this is not feasible, it might well be necessary to resect the fistula-bearing part of the kidney. If the fistula is located very centrally, involving the main artery or large segmental branches, it is worthwhile considering surgical repair. Our technique was described in the respective case report.

It is our experience that the complication rate associated with percutaneous core biopsies is clearly related to the skill of the doctor performing the biopsy. It is our policy to perform biopsies in patients whose grafts can reliably be palpated without previous sonographical evaluation. Whenever possible, the biopsy is taken from the upper part, directing the needle toward the pole rather than toward the hilum and thus minimizing the risk of traumatizing a major artery or a calix. As already mentioned, ultrasonography has proved to be very helpful in patients whose kidneys cannot be palpated.

These authors feel that there is a greater incidence of AVFs - and possibly of ACFs - in renal allografts than is commonly believed. It must be emphasized that the symptoms we have mentioned necessitate further angiographic evaluation. In both of our patients whose fistulas were able to be repaired either by surgery or embolization, bleeding in the ACF stopped immediately and renal function improved significantly after surgical repair of an AVF.

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