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BRIEF REPORT

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Successful endoluminal thrombo-aspiration of renal graft venous thrombosis

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Abstract Renal transplant vein thrombosis is an unusual event occuring in 0.3–3 % of renal transplantations. Prognosis is uniformly poor with graft loss in nearly every case. We report here the first three cases of renal graft vein thrombosis successfully treated by percutaneous endoluminal thromboaspiration. After an initially uneventful course all recipients developed anuria and required hemodialysis. In two cases, an ultrasound examination suggested a diagnosis of venous thrombosis. Emergency arteriography and phlebography were performed, confirming the complete thrombosis of the graft veins. Thromboaspiration was carried out with full heparinization and led to renal function improvement in all cases. Grafts are still functioning 6 months after the procedure, with serum creatinine levels of 176 μmol/l, 120 μmol/l and 184 μmol/l, respectively. Thus, this procedure avoids surgical and anaesthetic risks and allows, if performed at an early stage, restoration of graft function. Great care must be taken to avoid vein wall damage, vascular suture line rupture, or pulmonary embolism.

Key words Renal graft thrombosis · Kidney transplantation · Thromboaspiration

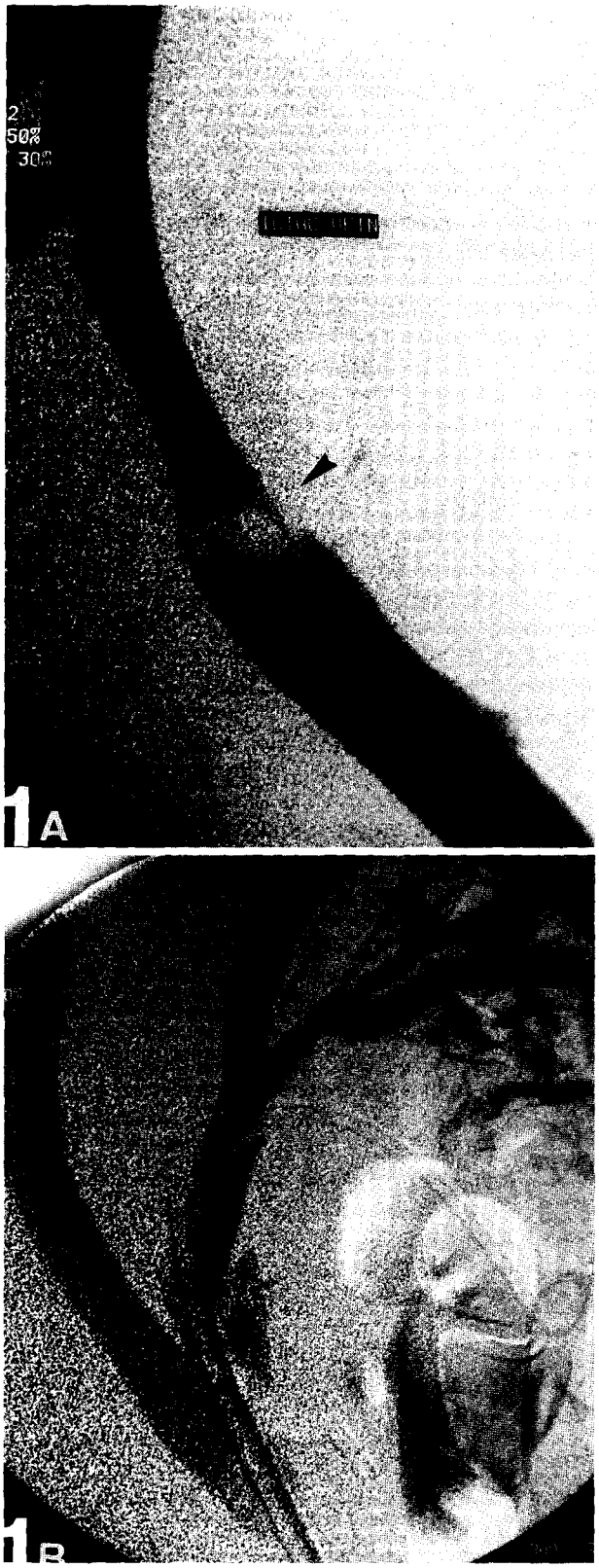
Introduction

Renal transplant vein thrombosis is an unusual event that occurs in 0.3–3 % of renal transplantations [1, 2, 3, 4]. Except for acute rejection and ilio-femoral thrombosis, several risk factors have been suggested, such as technical surgical problems, multiple donor renal veins, vessel compression, kinking or torsion, hypotension or hypoperfusion, hypercoagulable recipient state, as well as prolonged ischemia time [1, 2, 3, 4, 7]. Graft and, potentially, recipient survival may be affected by haemorrhagic, thrombo-embolic and septic complications or by graft rupture related to high venous pressure [1]. Prognosis is uniformly poor, with graft loss in nearly every case and no treatment of proven efficacy [1, 2, 4, 7]. We report here, to the best of our knowledge, the first three cases of renal graft vein thrombosis (RGT) successfully treated by percutaneous transluminal thrombo-aspiration with recovery of renal function.

Case reports

Case 1, a 53-year-old woman with end stage renal failure secondary to polycystic kidney disease, underwent transplantation in February 1997. She had been on hemodialysis since 1995 and had no history of arteriovenous access or deep vein thrombosis. The graft was a left kidney with two arteries and one vein, transplanted in the left iliac fossa with end to side anastomosis of the renal arteries, and vein to the external iliac artery and vein. At completion of procedure, the kidney appeared normal with normal turgor. The total cold ischemia time was 40 h. Immunosuppression included steroids, Cellcept (mycophenolate mofetil, Roche, Neuilly sur Seine, France) and initial induction using Thymoglobulin (IMTIX, Lyon, France) followed by cyclosporin-A (Neoral, Novartis, Rueil-Malmaison, France).

The initial course was complicated by delayed graft function with a daily urine output of 800 cc. On postoperative day 8, fever and pyuria broke out with neither a painfully swollen graft nor macroscopic hematuria. Urinalysis showed pyuria, and urine culture revealed *E. Coli* and *Streptococcus*. A diagnosis of pyelonephritis was made, and antibiotherapy instituted. On the same day,



◀ **Fig. 1** **a** Phlebography. Left iliac vein opacification, showing the head of the thrombus protuding into the left iliac vein. **b** Phlebography. Angiogram taken during thrombo-aspiration, showing a patent renal vein with residual thrombi

an ultrasound examination of the graft was performed, revealing graft venous thrombosis.

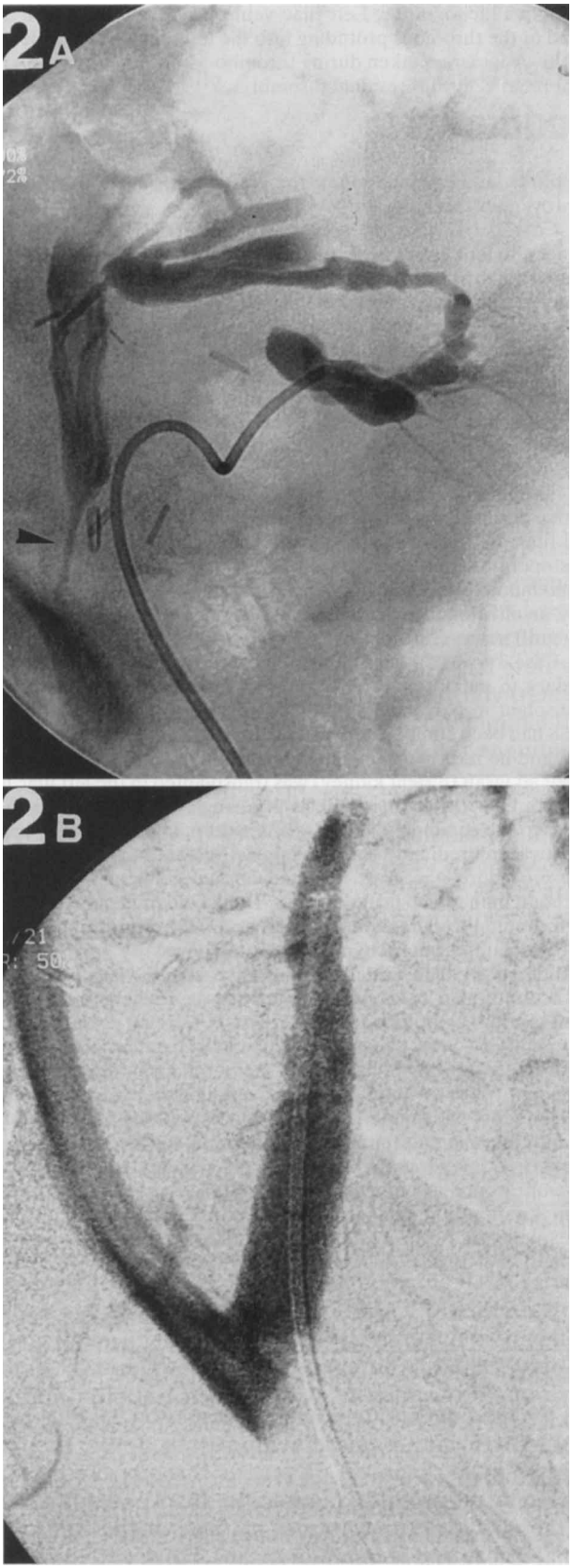
Case 2, a 43-year-old woman with end stage renal failure secondary to unknown nephropathy, underwent transplantation in March 1998. She had been on hemodialysis since 1995 and had no history of arteriovenous access or deep vein thrombosis. The graft was a right kidney with two arteries and one vein transplanted in the left iliac fossa with end to side anastomosis of the renal arteries, and vein to the external iliac artery and vein. At completion of the procedure, the kidney appeared normal with normal turgor. Total cold ischemia time was 30 h 30 min. Immunosuppression included steroids, Cellcept and initial induction using Thymoglobulin followed by cyclosporin-A (Neoral).

The initial course was complicated by delayed graft function with a normal daily urine output, followed by slow but effective renal function improvement, allowing hemodialysis withdrawal. On postoperative day 12, oliguria, macroscopic hematuria and acute renal failure broke out with a painfully swollen graft. On the same day, an ultrasound examination of the graft was performed, revealing graft venous thrombosis.

Case 3 was a 50-year-old man with end-stage renal failure secondary to membranoproliferative who received his second renal transplant in November, 1991. His first renal transplant in April 1988 had been complicated by a recurrence of the initial nephropathy, and he returned to hemodialysis in January 1991. The second renal allograft (a right kidney) was transplanted in the left iliac fossa with end-to-side anastomosis of a single artery, and two short veins to the common iliac artery and external iliac vein. The upper vein was injured and required surgical restoration. At the end of the procedure, the graft regained normal color and turgor, but without immediate urine output. Total cold ischemia time was 50 h. Immunosuppression included steroïds, azathioprine and initial induction using Thymoglobulin followed by cyclosporin-A (Sandimmun). The initial postoperative course was complicated by acute tubular necrosis. The patient was discharged on day 30 with a serum creatinine level of 177 μmol/l. Renal dysfunction occurred after 1 year, (creatinine = 200 μmol/l) and renal biopsy was performed, showing recurrence of membranoproliferative glomerulonephritis, but without rejection or drug toxicity. In September, 1995, 4 years after undergoing transplantation, the patient was readmitted to the unit for acute oliguric renal failure with fever, hypertension, microscopic hematuria and a painfully swollen graft. The ultrasound examination of the graft seemed to indicate renal vein thrombosis, leading to graft biopsy. In the absence of histologic explanation of the acute renal failure, a graft CT scan was performed after 10 days, revealing venous thrombosis.

Results

At the time of the ultrasound examination, all recipients were anuric and required hemodialysis. In the first two cases only, a diagnosis of venous thrombosis was suggested by ultrasound examination. In all patients, arteriography showed the normal patency of the transplant renal artery and its main branches. However, there was



◀ **Fig. 2** **a** Arteriography. Selective catheterisation of an arterio-venous fistula clearly showing a tight stenosis of the transplant vein. **b** Phlebography. Normal vein patency after percutaneous transluminal angioplasty plus stent implantation

no flow within it, the runoff phase did not opacify, and the nephrogram phase was not visible. Selective transplant vein phlebography showed complete occlusion of the main renal veins and their branches. In one patient, the thrombus had protruded into the external iliac vein. Thromboaspiration was achieved by an ipsilateral femoral venous route. Three thousand units of heparin were given to the patients at the begining of the procedure. The patients did not receive any fibrinolytics. The main graft vein was carefully catheterized and an 8 French long sheath was placed into it, close to the anastomosis. Declotting was performed with 8 French straight and curved aspiration catheters (Cordis, Miami), which were placed into the main transplant vein and into its branches and gently moved to and fro, aspiration being made manually with a 50 cc syringe. In all cases, the procedure restored satisfactory graft vein patency within 20 min, reestablishing normal flow into graft arteries and veins.

For the first case, underlying venous stenoses were successfully dilatated 4 months later, with placement of an endovascular stent, and, for the third patient, immediately. In the second case, a bulky perirenal haematoma secondary to graft rupture induced severe graft compression and led to decompression surgery. Control arteriography confirmed the restoration of satisfactory venous flow, and no further complications occured in any of the cases. All patients underwent full heparinization, initiated during the procedure in order to prevent allograft thombosis recurrence, this was followed by long-term anticoagulation therapy. After a few days, renal function improved in all cases, allowing hemodialysis withdrawal and hospital discharge. Grafts are still functioning 6 months later with a serum creatinine of 176 μmol/l in case 1 (6 months after the venous angioplasty), 120 μmol/l in case 2, and 184 μmol/l in the last case.

Discussion

The overall reported incidence of primary renal graft vascular thrombosis (without acute rejection or extension from iliofemoral venous thrombosis) varies from 0.5 to 6.2%. Thrombosis usually occurs within 2 weeks of renal transplantation (cumulative occurrence of 90% at day 14) [1, 7]. Among these vascular thromboses, arterial thrombosis represents 0.2–3.5% and venous thrombosis 0.3–3% [1, 2, 3, 7]. Indeed, 30–40% of early (90 days) graft failures and one third of the graft losses

in the first year post transplant are related to vascular thrombosis [1, 2, 7].

In the case of the first two patients, graft thrombosis occured early, as usually described at 8 and 12 days after renal transplantation, respectively, while both recipients presented a delayed graft function. In contrast, for the last patient, thrombosis occurred 4 years after renal transplantation, while he was suffering from chronic renal failure because of chronic rejection and recurrence of the initial nephropathy.

Graft vein thrombosis usually induces a rapid onset of oliguria or anuria, hematuria and a painfully swollen graft, which may progress to rupture, hemorrhage and shock. Many of these signs are not specific for allograft renal vein thrombosis, especially when graft function is delayed. Knowing the fate of patients with venous graft thrombosis, early diagnosis is mandatory. A noninvasive tool, renal duplex scanning, can be helpful as it shows suggestive signs such as arterial reverse flow, disappearence of the venous signal on Doppler studies or evidence of the thrombus itself. However, in our third case, renal duplex scanning failed to show thrombosis because of the presence of two veins. Diagnosis was made by CT scan and confirmed by arteriography. If the clinical suspicion of a venous thrombosis is strong enough, vascular contrast studies must immediately be performed.

Several risk factors are said to underlie this complication, but studies are limited by the small number of cases. Well recognized risk factors for RGT include technical surgical problems [1, 3, 4], vessel torsion or compression, perioperative hemodynamic status [1], past history of venous thrombosis [1, 7], diabetic nephropathy of recipient [1, 2], donor's right kidney [1], discrepancy in the size between donor and recipient vessels, immunosuppressive therapy (cyclosporin, OKT3) [1, 2, 7], and prolonged cold ischemia time [1, 7]. Other risk factors include peritoneal dialysis [5], membranous glomerulopathy and multiple graft vessels [1, 3].

In our cases, the only objective risk factor was a long cold ischemia time in all cases, and multiple and injured veins in the third patient. In no case did we find a hypercoagulable state of the recipient such as antithrombine III, protein C or protein S deficiencies, resistance to activated protein C, or presence of antiphospholipid antibodies.

Prognosis of RGT is generally poor, with graft loss in nearly all cases, except when urgent surgical thrombectomy is carried out [1, 2, 4, 7]. Percutaneous transluminal thrombo-aspiration is a very simple concept. Initially used for arterial embolectomy [9], indications have been extended to venous thrombosis [8]. This technique allowed, in our three cases, very fast – less than 20 min – and complete transplant – vein declotting with preservation of renal function and satisfactory creatinine level at month 6. This procedure avoids surgical and anaesthetical risks. Its own risks appear very low, they are mainly:

1 Vein wall damage, which should be prevented by a very gentle motion of the aspiration catheters;
2 Vascular suture line rupture, which one should be aware of when attempting renal vein declotting soon after transplantation. Placing the sheath tip into the renal vein carefully avoids repeated catheterisation of the suture line and decreases such risks. Great care must be taken during this procedure when the time interval between transplantation and transluminal thromboaspiration is short.
3 Pulmonary embolism. Following removal of the bulk of thrombi from the graft vein, the flow restored within it may dislodge some small thrombi not adherent to the vein wall and not yet aspirated. These may cause tiny pulmonary emboli which will be treated in any event by the heparin injected during and after the procedure. However, we believe that large pulmonary emboli are not possible: the total amount of thrombus contained in the kidney is limited, aspiration removes most it. Large pieces of clot would be blocked, if necessary, by the introducer sheath still in place in the transplant vein, or by a stenosis of the transplant vein. In our opinion simultaneous fibrinolysis has to be avoided because of the haemorrhagic risk.

We observed graft function improvement in all cases, but recovery of renal function was slow, with obtained nadir serum creatinine levels at 35, 25, and 92 days post procedure, respectively. Creatinin reached the baseline value only for the second patient. So prompt diagnostic and immediate intervention are essential in order to save the grafts.

Closing, we can state that renal graft thrombosis is a serious complication of kidney transplantation and an important cause of early graft loss. Early percutaneous endoluminal thrombo-aspiration seems to be an effective and safe procedure.

References

1. Bakir N, Sluiter WJ, Ploeg RJ, van son WJ, Tegzess AM (1996) Primary renal graft thrombosis. Nephrol Dial Transplant 11: 140–147
2. Gruber SA, Pescovitz MD, Simmons RL et al (1987) Thromboembolic complications in renal allograft recipients. A report from the prospective randomized study of Cyclosporine versus Azathioprine-Antilymphocyte Globulin. Transplantation 44: 775–778
3. Hilfiker ML, Feddersen RM, Gibel LJ, Smith AY, Harford AM, Sterling WA (1992) Allograft renal vein thrombosis in a kidney with two veins. Transplantation 54: 738–739
4. Merion RM, Calne RY (1985) Allograft Renal Vein Thrombosis. Transplant Proc 17: 1746–1750
5. Murphy BG, Hill CM, Middleton D et al (1994) Increased renal allograft thrombosis in CAPD patients. Nephrol Dial Transplant 9: 1166–1169
6. Peek D, Kootstra G, Christiaans MHL, van Hoof JP (1995) Renal graft thrombosis. Transplantation 60: 311–311
7. Penny MJ, Nankivell BJ, Disney APS, Byth K, Chapman JR (1994) Renal Graft Thrombosis. A Survey of 134 consecutive cases. Transplantation 58: 565–569
8. Poulain F, Raynaud A, Bourquelot P, Knight C, Rovani X, Gaux JC (1991) Local thrombolysis and thromboaspiration in the treatment of acutely thrombosed arteriovenous hemodialysis fistulas. Cardiovasc Intervent Radiol 14: 98–101
9. Starck E, Dermott J, Crummy A, Turnipseed W, Acher C, Burgess J (1995) Percutaneous aspiration thrombo-embolectomy. Radiology 156: 61–66