

## CLINICAL STUDY

## Vascular complications after renal transplantation

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**Abstract:** *Background:* Vascular complications in renal transplant recipients are uncommon but important causes of graft dysfunction and graft loss after kidney transplantation.

*Objectives:* To document vascular complications that occurred following kidney transplantations in order to assess the incidence of these complications at our center as well as to identify possible treatment approach.

*Methods:* 103 kidney transplantations were performed in the period from 1 January 2008 to 31 December 2009. All patients after kidney transplantation underwent ultrasound examination (included colour Doppler flow and duplex Doppler ultrasound) immediately after surgery – especially in condition of anuria, then regularly according to an internal schedule and always in patients with worsened graft function.

*Results:* We detected renal vein thrombosis in 3 cases (2.9 %), artery thrombosis in 4 cases (3.9 %), one time intrarenal pseudoaneurysm (1%) and renal artery stenosis in 10 patients (9.7 %). There was no extrarenal pseudoaneurysm or arteriovenous fistula in our group of patients.

*Conclusion:* The incidence of vascular complications in our department correlates with the incidence of these complications referred in literature. We confirmed the importance of established immediate diagnosis and fast intervention once the diagnosis of vascular complication is established (*Fig. 8, Ref. 12*). Full Text in free PDF [www.bmj.sk](http://www.bmj.sk).  
Key words: vascular complication, renal transplant, kidney, ultrasound, vein, artery.

Vascular complications after renal transplantation are uncommon but important, as they may result in loss of the allograft. Vascular complications can be related to allograft as well as to the recipient's vessels. Early allograft vascular complications include thrombotic events of the renal vein or the renal artery. Vascular complications that occur later include artery stenosis, extrarenal or intrarenal pseudoaneurysms and arteriovenous fistulas (1). All of the mentioned complications have specific ultrasound characteristics so they are easily detectable with colour Doppler flow and duplex Doppler ultrasound (2).

The main risk factors for thrombosis are vascular torsion or kinking, hypercoagulation status of the recipient and hyperacute rejection (very rare complication nowadays). Renal artery thrombosis usually results from an improper surgical technique, thin arteries (especially small kidneys from paediatric donors or multiple renal arteries). Extrarenal compression by haematoma or lymphocele can lead to renal vein thrombosis (1, 3). The incidence of thrombotic complications is 0.5–6.5 %, however, renal vessel thrombosis is an important cause of graft loss in the early

posttransplant period (4). Thrombosis can be treated surgically by thrombectomy, but in majority of cases urgent graftnephrectomy is necessary. Another therapeutic possibility is catheter-directed thrombolysis with or without percutaneous angioplasty and stent placement, but this approach can be problematic in the early postoperative period (5).

Pseudoaneurysm occurs when only the artery is lacerated. The vascular injury may result from renal percutaneous kidney biopsy (intrarenal pseudoaneurysm), surgical injury in arterial anastomosis or local perivascular infection (extrarenal pseudoaneurysm). If they become symptomatic (haematuria or shunting), the treatment of choice is transcatheter embolisation, however, sometimes surgery is indicated (partial or total nephrectomy). Extrarenal pseudoaneurysms are extremely uncommon, but very dangerous because of a high risk of rupture and life-threatening bleeding. Graftnephrectomy is generally performed to prevent rupture (6, 7).

Arteriovenous fistula (AVF) is the most common biopsy-induced vascular injury. AVF occurs when an adjacent artery and vein are lacerated simultaneously. The frequency of AVF occurrence is estimated to be higher than 18% biopsy events. AVFs obviously resolve within 1–2 years, but 1/3 of them become symptomatic (haematuria) or can even lead to graft dysfunction (shunting) and need transcatheter embolisation (1, 8, 9).

Transplant renal artery stenosis (TRAS) is the most frequent posttransplant vascular complication occurring in 1–23 % (in relation to the diagnostic method used) (10). This complication is observed in the later posttransplant period (months and years).

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TRAS is a potentially curable cause of posttransplant arterial hypertension. Untreated TRAS can lead to a loss of the graft. The cause of TRAS appears to be multifactorial: suture technique, renal artery trauma during transplantation, kinking, atherosclerosis, rejection and cytomegalovirus infection. The treatment of choice is percutaneous transluminal renal angioplasty (PTRA) with or without stenting (1, 9, 10, 11, 12).

The most important recipient's complications are stenosis or thrombosis of iliac arteries after surgical trauma (clamp injury), atherosclerosis of iliac arteries and deep iliac vein thrombosis.

We report our experience with posttransplant complications of allograft vessels in our department.

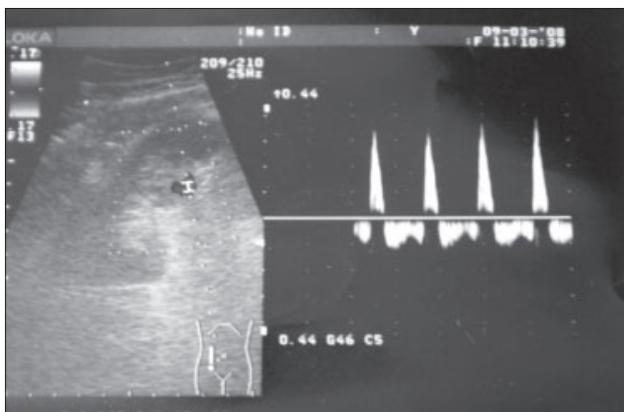
**Purpose and methods**

103 kidney transplantations were performed in the period from 1 January 2008 to 31 December 2009. All patients after kidney transplantation underwent ultrasound examination (included colour Doppler flow and duplex Doppler ultrasound) immediately after surgery – especially in condition of anuria, then regularly according to an internal schedule and always in patients with worsened graft function.

**Results**

We detected renal vein thrombosis in 3 cases (2.9 %), artery thrombosis in 4 cases (3.9 %), one time intrarenal pseudoaneurysm (1 %) and renal artery stenosis in 10 patients (9.7 %). There was no extrarenal pseudoaneurysm or arteriovenous fistula in our group of patients. In our series, no deaths occurred among patients with allograft vascular complications.

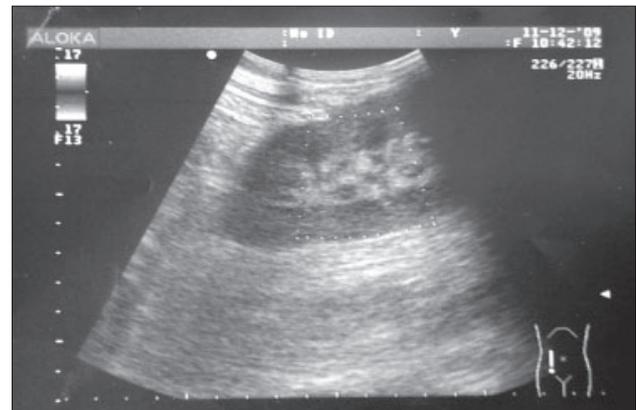
Renal vein thrombosis was clinically manifested by primary anuria. In 2 cases we diagnosed thrombosis by ultrasound examination, in 1 case this complication was recognized during surgical revision because of bleeding. Blood perfusion on the kidney was recovered only in 1 case – after successful surgical thrombectomy (Figs 1, 2). 2 years after transplantation, renal function of salvaged kidney is stable. In two cases vascular complica-



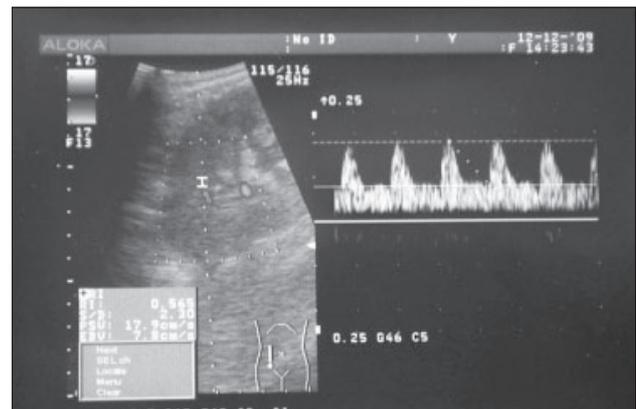
**Fig. 1. Thrombosis of the renal vein of allograft – before recanalisation (AF, 59 yr).**



**Fig. 2. Thrombosis of the renal vein of allograft – after recanalisation (AF, 59 yr).**



**Fig. 3. Thrombosis of the renal artery of allograft – before recanalisation (MP, 28 yr).**



**Fig. 4. Thrombosis of the renal artery of allograft – after recanalisation (MP, 28 yr).**

tions led to urgent graftnephrectomy. Renal artery thrombosis appeared 3 times in the early posttransplant period (primary anuria) and led to graftnephrectomy. The fourth case was observed 2.5 months after transplantation as a consequence of antirejection treatment with high doses of corticosteroids, high doses of erythropoetin for pure red cell anaemia and presence of

inherent hypercoagulation status of the recipient. In cooperation with NUSCH Hospital immediate rheolytic pharmacomechanical thrombectomy and catheter-directed thrombolysis with subsequent percutaneous renal stent implantation were performed. Three months after renal artery recanalisation the patient is doing well, with no necessity of dialysis, renal function is stable with serum creatinine 351  $\mu\text{mol/l}$  and diuresis 3600 ml per day (Figs 3, 4). Intrarenal pseudoaneurysm was recognised in 1 patient 1 month after percutaneous renal biopsy (Fig. 5). No bleeding was detected. Transplant renal artery stenosis was discovered in 10 cases. Suspicion based on clinical symptoms and ultrasound signs was confirmed by contrast-enhanced computed tomographic (CT) angiography (in 2 patients) or gadolinium-enhanced magnetic resonance (MR) angiography (in 6 patients). In 2 cases there was no graft perfusion found in early ultrasound examination, immediate surgery (without CT or MR) discovered kinking of the renal artery. Correction of the kidney position led to graft function recovery. The third case of kinking was detected 3 months after transplantation, but it was not considered as hemodynamically important so we preferred a conservative approach. In 7 patients renal artery stenosis was confirmed. In 6 cases TRAS was hemodynamically significant, so percutaneous transluminal angioplasty with or without stenting (5 vs. 1) was performed (Figs 7, 8). In 4 cases the intervention led to signifi-

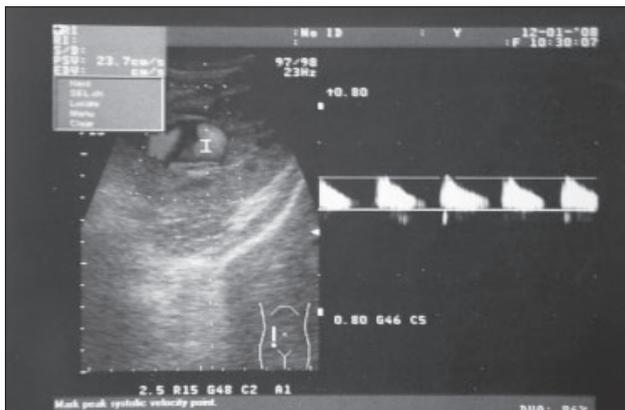


Fig. 5. Intrarenal pseudoaneurysm after renal graft biopsy (ZG, 29 yr).

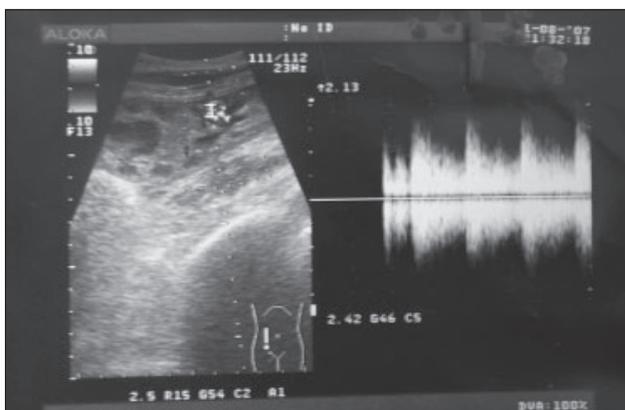


Fig. 6. Arteriovenous fistula after renal graft biopsy (AS, 16 yr).

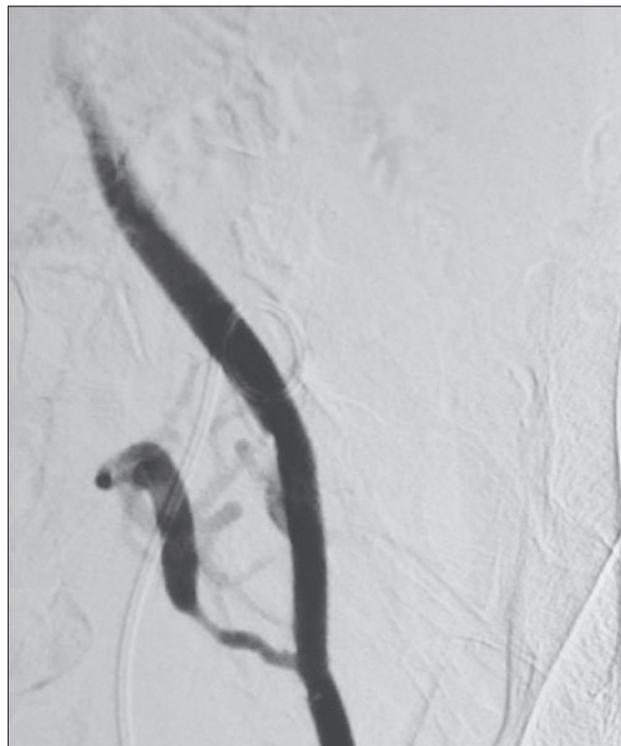


Fig. 7. Renal angiography before PTR (EM, 51 yr).

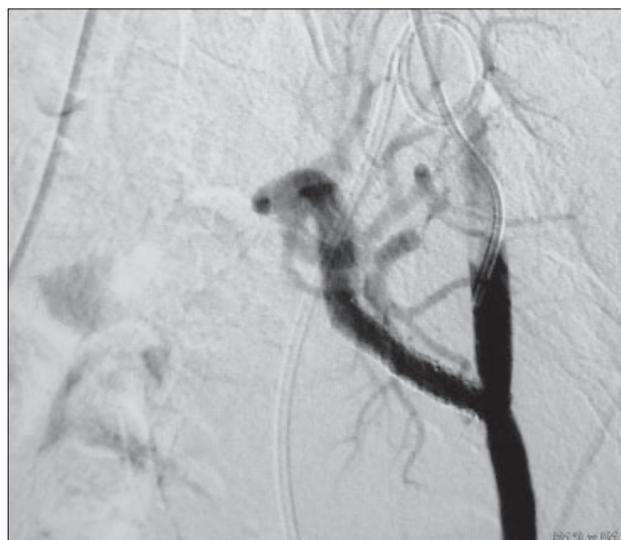


Fig. 8. Renal angiography immediately after PTR with stenting (2 stents) (EM, 51 yr).

cant improvement of renal function and blood pressure, while in the remaining 2 cases there was no change of renal parameters or blood pressure. In one case TRAS was not hemodynamically significant, the patient had a stable renal function and well controlled hypertension and is on regular observation. During a period under consideration we didn't find arteriovenous fistula in our group of patients. In Figure 6 we show one from previous period of time.

## Discussion

Vascular complications in renal grafts, including thrombosis and arterial stenosis, range from 0.5 to 23 % (4, 10). In comparison with this data, the incidence of various vascular complications in our casuistics is slightly lower, it ranges from 1 % to 9.7 %.

Not surprisingly the most frequent complication in our group of patients was stenosis of the renal artery. It's incidence was 9.7 % which is in accordance with literature data. In the mentioned period of time we didn't lose any graft because of this complication. We are convinced in fact that protection of graft function is in direct relation to regular preclusive ultrasound examinations and fast intervention once the diagnosis of the renal artery stenosis is established.

In accordance with other's experiences (1, 3, 4) the most common vascular complication which led to the graft loss in our series was thrombotic event in an early posttransplant period. We lost 5 allografts all together – 3 because of the renal artery thrombosis, in 2 cases the reason was thrombosis of the renal vein. Although in 3 cases the diagnosis was done in short period after transplantation, the life-threatening bleeding was observed and an urgent surgical intervention led to graftnephrectomy. In two patients the appropriate diagnosis was done to late.

We recognise several risk mechanisms causing vascular complications in our cases – wide disparity in vessel size, multiple renal arteries, hypercoagulable state, atherosclerosis of the donor or recipient vessels, kinking of artery and angulation of the vein. We suppose that the main reason of renal artery stenosis is faulty suture technique, excessive reparation changes in surround of vessels after surgical revisions and acute rejection. Complications as pseudoaneurysm or arteriovenous fistula were very rare so we consider renal biopsy as a safe diagnostic performance after renal transplantation.

## Conclusion

The incidence of vascular complications in our department correlates with the incidence of these complications referred in literature. We confirmed the importance of established immediate diagnosis and fast intervention once the diagnosis of vascular complication is established.

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