

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

186,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.  
For more information visit [www.intechopen.com](http://www.intechopen.com)



## Vascular Complications in Kidney Transplantation

Alexandros Giakoustidis, Nikolaos Antoniadis and Dimitrios Giakoustidis  
*Division of Transplantation, Department of Surgery, Medical School  
 Aristotle University of Thessaloniki and Hippokration General Hospital, Thessaloniki  
 Greece*

### 1. Introduction

Kidney transplantation evolved as the treatment of choice for end-stage renal disease. Accumulated expertise and experience along with refined surgical techniques resulted in excellent patient and graft survival. Despite the improvements in surgical techniques vascular complications consists a significant and sometimes life-threatening problem that occurs in 10-20% of patients. Vascular complication can be divided in general in three main categories. Graft renal artery thrombosis and stenosis, graft renal vein thrombosis and stenosis and arterial injury.

### 2. Transplant renal artery stenosis

Transplant renal artery stenosis (TRAS) is a very common vascular complication following kidney transplantations. It is reported in a wide range of frequency occurring in 3-12% of patients (in some reports even up to 20% depending on the awareness and the available imaging means especially MDCT-angiography) (Akbar 2005, Sebastia, 2001)]. It is very difficult to have the definitive true incidence of TRAS by looking at the literature and this discrepancy is due to the different definitions, the surgeon's experience and technical skills in avoiding or preventing the TRAS and different peri-operative management. If we try to classify all arterial stenosis in kidney transplantation, we could divide it into two main categories; TRAS and proximal or pseudo-TRAS. TRAS can be categorised by the level of stenoses and this includes anastomotic stenoses, stenoses of the proper transplant renal artery and finally segmental renal artery stenoses. Proximal-TRAS refers to pre-existing or developing atherosclerotic inflow stenoses in the native iliac arteries of the transplant recipient. The incidence of proximal-TRAS has been reported to be 0%-2.4% and may become more prevalent with increasing age. Predisposing factors for transplant renal artery stenosis, are cadaveric transplant, end-to-end anastomosis, surgical clamp injury, intimal dissection, and inadequate suturing technique, long or kinking artery, prolonged cold ischemia time, acute cellular rejection, and cytomegalovirus (CMV) infection (Audard, 2006).

Patients with TRAS in the immediate post-transplant period, present oliguria or anuria and are dialysis dependent. After the first week, patients with TRAS usually present with severe renovascular hypertension. Even though severe renovascular hypertension could be

attributed to TRAS we should always keep in mind and all other causes like chronic rejection, steroid use, cyclosporine toxicity, recurrent glomerulonephritis and disease of native kidneys (Mangray, 2011, Tutone, 2005). If TRAS is not managed properly in due time, it could lead to renal dysfunction and graft deterioration. Non-invasive imaging is mandatory in the immediate post-transplant period to evaluate for possible transplant renal artery stenosis. Doppler ultrasound should be the initial diagnostic modality used because of its ability to reveal the location, length, and gross appearance of a stenosis. In addition, it is widely available, cost-effective, and does not use ionizing radiation and the nature of the transplanted renal arteries makes Doppler ultrasound an ideal screening modality (Baxter, 1995, Irshad, 2008, 2009). Whilst Doppler ultrasound is ideal in recognizing a possible lesion, categorisation and better characterisation of TRAS would be using magnetic resonance angiography with or without contrast medium (especially gadolinium-enhanced), MDCT-angiography, Radionuclide imaging that includes the administration of an angiotensin-converting enzyme inhibitor (captopril scan) shows findings similar to those of renovascular hypertension in native kidneys (Sebastia, 2001). Catheter-based angiography is the gold standard technique for diagnosing TRAS. The use of low- or iso-osmolar contrast material is recommended to reduce the risk of contrast material-induced nephropathy. When renal insufficiency is present, carbon dioxide may be substituted for an iodinated contrast agent during preliminary angiography to minimize the use of iodinated agents.

## 2.1 Definition and natural history

Unfortunately there is no consensus definition of TRAS and it would be difficult to have one in the future. Usually there is the classic presentation with refractory hypertension, deteriorating renal function, and congestive cardiac failure secondary to fluid retention. It is very impressive that marked reversal of symptoms occurs when stenosis is successfully treated (Garovic, 2005, Mangray, 2011). The timeline of TRAS usually begins at 2 months to 2 years after transplantation and hypertension is due to activation of the renin-angiotensin system (Basso, 2001, Mangray, 2011). Another important issue concerns asymptomatic normotensive patients with Doppler examination of at least 50% without evident graft dysfunction. The problem with those patients is the potential risk carrying from a procedure to treat a clinically insignificant TRAS in the short to medium term. It is unclear whether treating a non-clinically significant TRAS would have an impact in long term survival of renal grafts but since hypertension is an independent risk factor for long term renal graft survival, anything that could contribute to this direction would be beneficial. Close follow up of those patients, with significant expertise and experience of the transplant centre, along with availability of vascular interventional techniques is of paramount importance in decision making. Nevertheless, there are no reports of the long-term safety of this line of management, and the natural history of a 50% TRAS is unknown, and that conservative treatment is safe provided that there is no deterioration of kidney function (Audard, 2006, Buturovic-Ponikvar, 2003). This may not be the case for other causes, such as intimal hyperplasia, and the indication for angiography is strong when graft deterioration is revealed in the absence of other causes of graft dysfunction or chronic allograft nephropathy (CAN). Increased awareness with follow up observation should be in case of a stenosis of 80% on ultrasound examination, even in cases without suspicion of a hemodynamically significant TRAS. Due to increased susceptibility to occlusion in the presence of dehydration or cardiovascular instability, and in this case intervention should be considered.

## 2.2 Pathophysiology

Since Goldplatt et al published their study on the hypertensive role of partial reduced renal perfusion of the kidney back in 1934; there has been subsequent identification by various investigators of the role of the renin-angiotensin system, with renin being the hormone released in elevated blood levels from the ischemic kidney (Goldplatt, 1934). Angiotensin is being released enzymatically from angiotensinogen, and has various properties, including vasoconstriction, aldosterone secretion, renal sodium retention, and myocardium hypertrophy (Brewster, 2003). There is evidence that when there are two kidneys the above theory has been proven probably right, however it has been showed that this is not the case for the sole kidney or the transplanted kidney. Hypertension is also present but predominantly as a balance between volume regulation depending on salt and water retention and the renin-angiotensin dependent mechanism. It is the highly circulating volume and not the pressor effect from the renin-angiotensin system that is capable to keep a normal GFR rate with normal renin blood levels. In case that ACE inhibitors are prescribed there is a subsequent reduction in kidney perfusion and finally renal function deterioration. In patients with kidney transplantation, and in order to control hypertension, it is not uncommon to establish a diagnosis of TRAS, when an ACE inhibitor is introduced.

## 2.3 Diagnosis

Doppler ultrasonography is considered as the best screening test TRAS assessment. There are many advantages in the use of Doppler ultrasound as initial approach over other imaging methods, especially iodine contrast media. The fact that Doppler ultrasound is a noninvasive method, does not expose the recipient to the risk of iodine contrast examination, is widely and promptly available in all hospital settings makes it an excellent first choice for TRAS evaluation. The most significant limitation of the method, as in other conditions, is the fact that ultrasonography is operator dependent.

Digital subtractive angiography (DSA), used to be the gold standard technique for establishing the diagnosis, but today tends to be replaced by MRI angiography and more recently Multi Detector CT-angiography. DSA is an invasive technique and potential complications are groin hematoma, renal artery dissection, thrombosis, perforation, and acute kidney injury caused by contrast-induced nephropathy. Doppler ultrasound can also be used to evaluate the hemodynamic changes due to TRAS. Doppler findings in TRAS include peak systolic velocity 2.0-2.5 m/s, low pulsatility index, and a parvus et tardus waveform with a systolic acceleration time of  $\geq$  to 0.1 seconds (Snider, 1989, Irshad, 2009, Baxter, 1995). Snider et al compared Doppler ultrasonography with conventional angiography and showed 94% sensitivity and 87% specificity on US (Snider, 1989). A velocity ratio of the stenotic to pre-stenotic segments of greater than 2:1 is considered supportive of the diagnosis.

Multidetectors helical CT gives accurate assessment of the site and degree of TRAS and provides accurate and valuable imaging, requires less volume of iodinated contrast medium than DSA (Sebastia, 2001). The nature of the vascular contrast medium may be of consideration, rather than the volume. Risk of contrast nephropathy is probably not related to the volume of contrast medium or the degree of renal failure (Birck, 2003, Pannu, 2004). Protection of the allograft with sufficient volume and N-acetylcysteine is recommended when intravenous contrast medium is injected, regardless of renal function and contrast volume. The alternative is to perform MRI with gadolinium, a non-iodinated contrast medium even though there have been reports of nephrogenic systemic sclerosis.

## 2.4 Treatment

Treatment options for TRAS include both surgical and endoluminal options. Primary treatment for TRAS involves Percutaneous transluminal angioplasty (PTA) with or without stent placement (Audard, 2006, Bruno, 2004). The type of arterial anastomosis that is present is the deciding factor in determining the angiographic approach utilized. The technical success rate of PTA has been reported to be as high as 94%, with a clinical success rate of 82% (Patel, 2001). Recurrent stenosis may occur in more than 10%, and allograft loss has been reported in up to 30% of cases (Fervenza, 1998). There have been reports correlating TRAS with acute cellular rejection and that long term survival is significantly higher in non-TRAS patients compared with the TRAS. Surgical revascularization is now considered rescue therapy and generally has been reserved for patients with disease unsuitable for PTA

## 2.5 Endoluminal Interventions

Since the introduction and the evolution of the endovascular interventions there has been a shift in TRAS treatment option with Percutaneous Transluminal Angioplasty (PTA) with or without the use of stent (Beecroft, 2004) being now the gold standard and the initial option of treatment. The method is considered very efficient especially in experienced hands and technical success has been reported to be greater than 90%. However when we consider the clinical impact that has on hypertension or improvement of allograft function, this is significantly lower.

As we have already pointed out, the results in clinically insignificant TRAS can be evaluated only on the degree of the radiological success. PTA with or without stenting, is carrying a significant risk for the allograft and unless a significant pressure decrease exists across the TRAS, PTA should not be undertaken. Unfortunately there is no consensus as to the appropriate value of stenosis measurement beyond which intervention is warranted, and it would be very difficult to obtain one in the future since radiological success does not always results in clinical improvement. A cut off point proposed by Schoenberg et al (Schoenberg, 2000), could be pressure decrease at least 10 mm Hg across the stenosis.

The type of arterial anastomosis that is present is the deciding factor in determining the angiographic approach utilized. If there is an end-to-end anastomosis with the internal iliac artery, commonly done in living donor allografts, then a contralateral femoral approach is utilized to make access to the downward sloping artery as easy as possible. However, if there is an end-to-side anastomosis with the external iliac artery, then an ipsilateral femoral approach is preferred by some authors to access the cephalad sloping artery (Bruno, 2004).

Results after PTA depend largely on the radiologists or vascular surgeons experience and expertise, and should have smooth cooperation with the transplant surgeons. Most of the complications relate to puncture site, but there could be also more severe complication like hemorrhage, rupture of transplant renal artery, iliac artery and loss of the allograft, in those case there could be a need for "salvage" operation. Evolution in endovascular technology, with newer pre-mounted stents, has minimised complications especially the life threatening ones and the risk for allograft loss. Rate of re-stenosis are reported to be 10% to 50% and depends on the primary cause of the stenosis, length of follow-up, and use of stents (Voiculescu A 2005). Even though there are several reports on the topic, there are limitations provided by the retrospective nature of those manuscripts and the limited number of patients. In a french study of 29 patients with TRAS treated with PTA, the technical success rate was 93.1%, and there was 27.5% re-stenosis (Audard, 2006). In other study from the US, TRAS was found in 26 (3.1%) renal allografts, and 17 were treated with PTA with a success



rate of 94%. Re-stenosis occurred in 12% of the patients [Patel NH 2001] In case of segmental branches, there is a lower success rate and the success rate is even lower for anastomotic strictures, and even though the incidence of stenosis is similar between end-to-side anastomosis to the external iliac artery and end-to-end anastomosis to the internal iliac artery, PTA in the latter situation is technically more difficult and results in a higher complication rate and more graft loss (Voiculescu, 2005).

## 2.6 Surgical correction

Surgical correction of TRAS is regarded as a difficult operation with graft loss rates exceeding 20% (Bruno, 2004). A couple of risks existing; to the recipient and to the allograft, the latter is not a contraindication to surgery, since severe TRAS could deteriorate the transplanted kidney, the patient proceeds to renal failure, and finally to end up in hemodialysis. Surgery is now considered as rescue therapy for cases unsuitable for PTA.

In general indications for surgery include: TRAS caused by kinking, anastomotic strictures and complex atherosclerotic disease. There are several options to treat TRAS; mostly excision of the stenosis with direct anastomosis to the external iliac artery and grafting with saphenous vein, recipient internal iliac artery, and preserved ABO blood group compatible deceased donor artery. Reported surgical success rates range from 63% to 92%, with recurrence in 12% of patients (Roberts, 1989). A study comparing PTA vs. surgical repair of TRAS showed an immediate and long-term success rate of 92.1% and 81.5% and 69% and 40.5% for surgical repair and PTA respectively [Benoit G 1990]. Limitations of surgical procedure are access to the artery and most importantly the subsequent warm ischemia time. A warm ischemia of 60 minutes might be tolerated by a kidney allograft that has been heparinised even though the risk for Acute Tubular Necrosis (ATN) and cortical necrosis is increased due to diminished blood flow. An alternative option even though rarely used, is back table reconstruction of a complex arterial problem and autotransplantation of the allograft.

## 3. Transplant renal artery kinks and allograft torsion

Positioning the allograft is sometimes tricky and the source of pitfalls in kidney transplantation and can result in allograft torsion. Allograft torsion can be an early or late complication. One of the main problems caused by improper positioning or torsion is the arterial kinking. Usually arterial kinks are formed due to long renal graft artery when there is a shift in the graft and/or pelvic contents that causes turn of the artery. Even though the differential diagnosis between TRAS and arterial kinking is often difficult to have, it is of paramount importance to identify an arterial narrowing due to a kink and not TRAS. Prompt diagnosis permits graft detorsion and possible salvage. The most suggestive imaging finding is a change in the axis of the transplanted kidney. CT and MR imaging can show changes in renal graft orientation and vascular pedicle kinking. Surgery remains the primary treatment for arterial kinks and only in cases where surgery is contraindicated or patients refuse surgery, we should proceed with endovascular treatment. TPA with or without stents, may increase the risk of arterial vasospasm and dissection and in addition, placing stents across kinks usually can be technically demanding.

## 4. Thrombophilias

The thrombophilias, also referred to as hypercoagulable states, comprise hereditary or acquired conditions that predispose individuals to thrombosis. It was the third factor of

Virchow's triad that suggested that systemic alterations in the coagulability of blood, is a critical factor in thrombogenesis (Virchow, 1856). Thrombophilias are classified as congenital (inherited), acquired (secondary), or both (mixed) conditions (Schafer, 2007).

Congenital hypercoagulable states are caused by inherited thrombotic disorders due to mutations in genes encoding plasma proteins involved in coagulation mechanisms. They can be broadly classified into two categories: 1) quantitative deficiencies or qualitative defects of the physiologic anticoagulants: antithrombin, protein C and protein S deficiency, and 2) increased levels or function of the coagulation factors: factor V Leiden, prothrombin gene mutation, elevated levels of specific coagulation factors (Schafer, 2003). Congenital abnormalities of anticoagulant or procoagulant proteins result in an increased risk for venous thromboembolism (VTE) as well as arterial thrombosis with the risk to be higher in cases with decreased levels of antithrombotic proteins than in those with increased levels of prothrombotic proteins. The overall incidence of venous thromboembolism (per 100 patient-years) is found to be 1.07 for antithrombin deficiency, 0.54 for protein C deficiency, 0.50 for protein S deficiency, and 0.30 for activated protein C resistance or factor V Leiden (Bucciarelli, 1999). Half of the patients with inherited hypercoagulable state present with venous thromboembolism before the age of 45 years, particularly in the absence of well recognized risk factors, and often have a family history of thrombosis (Anderson, 2010). The secondary hypercoagulable states encompass a variety of heterogeneous disorders that have been associated with an increased risk of thrombotic complications (Schafer, 2003). Acquired hypercoagulable states include antiphospholipid antibody syndrome, cancer, heparin-induced thrombocytopenia, pregnancy and estrogen therapy, and a prior history of venous thromboembolism. Acquired coagulation defects are particularly common in patients with endstage renal disease (Wagenknecht, 1999). The prevalence of antiphospholipid antibodies in patients awaiting renal transplantation is more than 10%, but the rate of clinical events is far less than the frequency of thrombophilic states. Hyperhomocysteinemia is the typical hypercoagulable state that occurs due to a combination of inherited and acquired factors. Elevated serum levels of homocysteine have been associated with an increased risk of arterial thrombosis (myocardial infarction, stroke, and peripheral arterial disease) and venous thromboembolism (Cattaneo, 1999).

After renal transplantation the donor kidney endothelium is conditioned to exhibit a prothrombotic state as a consequence of reperfusion injury, tissue trauma, inflammation and expression of tissue factor, in addition to the recipient immune response (Key, 1992, Irish, 1999). The combination of a conditioned endothelium and a genetic or acquired predisposition to a hypercoagulable state increase the risk of thrombosis. Factors specific for the renal transplant patients that have been suggested to contribute to this thrombotic risk include the use of calcineurin-inhibiting drugs, high levels of homocysteine, diabetic nephropathy, antiphospholipid syndrome, cytomegalovirus infection, and the presence of proteinuria or nephrotic syndrome (Kujovich, 2004).

It has been proposed that inherited risk factors of venous thromboembolism, such as factor V-Leiden, prothrombin G20210A, and methylenetetrahydrofolate reductase (MTHFR) C677T, might be associated with poorer survival rates of transplanted kidneys, attributed to the context of graft perfusion defects, venous thromboembolic complications, and acute graft loss by vascular rejection, possibly reflecting immunological injury upon the vascular wall exacerbated or induced by the prothrombotic state (Heidenreich, 2003, Wuthrich, 2001). Later study with larger number of patients, did not find a statistically significant association of polymorphisms factor V-Leiden G1691A and MTHFR C677T with renal graft survival (Meyer, 2007).

Factor V-Leiden mutation or activated protein C resistance is the most common inherited thrombophilic disorder, found in 5% to 8% of the general population, in 20% of patients with a first venous thrombosis, and in up to 50% of patients with a personal or family history of recurrent thrombosis (Kujovich, 2004). In renal transplant recipients, factor V Leiden has been associated with a variety of complications after renal transplantation and a significantly higher incidence of venous thromboembolism which occurred in up to 39% of FVL carriers (Wuthrich, 2001, Irish, 1997). Also FVL carriers had 12-fold higher risk of an early graft perfusion defect (Wuthrich, 2001). A higher risk of vascular rejection was found in FVL carriers, which was linked to the presence of endotheliolitis or fibrinoid necrosis on histopathology of renal grafts lost within the first year after transplant (Ekberg, 2000).

The reported prevalence of prothrombin gene heterozygous mutation in renal transplant recipients is 3.7%, similar to that in the general population. The mutation was associated with a nearly threefold increased risk of graft failure, which was attributable to arterial, venous, or microvascular thrombosis in the majority of carriers (Fischereder, 2001, Kujovich, 2004).

A polymorphism of the methylenetetrahydrofolate reductase (MTHFR) gene coding for an enzyme that degrades the endothelium toxic product homocysteine have been associated with ESRD (Girndt, 2007). This mutation occurs in 50% to 90% of chronic dialysis patients presenting with mild hyperhomocysteinemia and have been associated with cardiovascular disease and vascular access thrombosis in this population (Mallamaci, 2005, Mallamaci, 2002). Additionally hyperhomocysteinemia can be acquired, such as in renal failure and in deficiencies of folate, vitamin B12, or vitamin B6. Even though many studies found that hyperhomocysteinemia is an independent risk factor for both first and recurrent venous thromboembolism (den Heijer, 1996, Cattaneo, 1999) and that hyperhomocysteinemia is an independent risk factor for cardiovascular disease (Ducloux D et al 2000), the effect of hyperhomocysteinemia on the risk of graft thrombosis is unknown. Antiphospholipid syndrome is the most common acquired blood protein defect associated with either venous or arterial thrombosis or both (Koniari, 2010). Antiphospholipid antibodies (APLA) are found in approximately 10% of patients awaiting renal transplantation. Since only a fraction of patients with antiphospholipid antibodies experience thrombotic complications, the description of antiphospholipid antibody syndrome (APAS), defines by the presence of antiphospholipid antibodies and a clinical history of thrombosis. Antiphospholipid antibodies include not only the lupus anticoagulant (LA) and anticardiolipin antibodies (ACLAs) but also more recently recognized subgroups of antiphospholipid antibodies (antibodies against beta-2-glycoprotein-I [B-2-GP-I]) and antibodies to phosphatidylserine, phosphatidylethanolamine, phosphatidylglycerol, phosphatidylinositol, phosphatidylcholine, and anti-annexin-V (Bick, 2003). Patients with antiphospholipid antibodies in association with other autoimmune disease, most commonly lupus, are classified as having secondary antiphospholipid syndrome. Approximately 40% of patients with SLE have an antiphospholipid (anticardiolipin) antibody (lupus anticoagulant). The presence of antiphospholipid antibodies has been recognized as an important risk factor for early allograft failure (Wagenknecht, 1999. Patients with APAS are at high risk for renovascular thrombosis and renal allograft loss was reported to be universal in the absence of anticoagulation (Vaidya, 2000). Whereas, and despite the lack of anticoagulation, no allografts were lost to thrombosis, in patients with detectable anticardiolipin antibodies but no prior history of thrombosis. In a later report all patients with antiphospholipid antibodies were successfully transplanted using postoperative anticoagulation (Morrissey, 2002).



Other prothrombotic changes that have been reported in renal transplant recipients include decreases in antithrombin, protein C and protein S levels, markedly elevated factor VIII levels and over-expression of plasminogen activator inhibitor-1 by donor epithelium (suggestive of impaired regional fibrinolysis) (Irish, 1999).

In vitro data suggest that immunosuppressive drugs like cyclosporine and OKT3 may increase the risk of thrombosis but an independent clinical association with allograft thrombosis is unproven (Gruber, 1989, Abramowicz, 1992). Specifically, the prothrombotic effects of cyclosporine include activation of monocytes to express tissue factor, increased platelet aggregation, endothelial dysfunction and activation of the intrinsic coagulation pathway, impaired fibrinolysis and impaired activation of protein C (Carlsen, 1988, Fishman, 1991, Bombeli, 1996, Evans, 1997, Levi, 1992).

Thrombotic complications after renal transplantation are usually catastrophic. Inherited and acquired hypercoagulable states have to be considered prior to kidney transplantation and proper prophylactic treatment initiated for the purpose to improve transplant outcome. Andrassy et al. provided specified screening recommendations for thrombophilia prior to kidney transplantation (Andrassy, 2004). A general screening for thrombophilia, to include factor V Leiden, prothrombin gene G20210A polymorphism, antithrombin III, protein C and S activity, antiphospholipid antibodies and lupus anticoagulant, should be performed in all children and adolescents because they have the highest risk for thrombotic complications. Adults only with history of thrombotic events should be thoroughly screened (recurrent AVC fistula thrombosis should be taken into account). When risk factors are absent, screening only for antiphospholipid antibodies and lupus anticoagulant is recommended.

There is also no consensus on the optimal management of renal transplant patients with thrombophilic disorders. Treatment strategies to reduce thrombotic risk including heparin, warfarin, and aspirin have been evaluated in several reports. While inadequate anticoagulation may place the patient at risk for thrombosis, preemptive or intense perioperative anticoagulation can result in postoperative bleeding (Morrissey, 2002, Murashima, 2010, Friedman, 2001). Morrissey et al. (Morrissey, 2002) recommended management approach for renal transplant patients with thrombophilia. Patients with diagnosis or suspected to have thrombophilia is suspected, the risk for thrombotic complications should be stratified as low, intermediate or high. In high risk are patients with inherited thrombotic disorder and history of at least two thrombotic episodes. For the high risk patients indefinite oral anticoagulation has been advocated. In intermediate risk are patients with a known inherited thrombotic disorder who are asymptomatic or have experienced a single thrombotic event. They should receive adequate prophylaxis in high-risk situations such as surgery for a minimum of 6 months. For patients with no suspicion of thrombophilia, no anticoagulation or a short term postoperative anticoagulation may be given.

## 5. Renal vein and artery thrombosis

Renal transplantation is established as the preferred treatment for most cases of end-stage renal disease. Postoperative vascular complications include thrombosis of renal vein and artery, with a delay in the diagnosis and management of these complications leading to significant morbidity for the recipient, with a high risk of graft loss and mortality (Akbar, 2005). It consists of a rare complication that often results in graft loss, with reported incidence ranging from 0.4% to 6% (Rouviere, 2002, Giustacchini, 2002). Bakir et al reported

that thrombosis represented 45% and 37% of renal allograft loss at 3 and 12 months (Bakir, 1996).

### 5.1 Renal vein thrombosis

Renal vein thrombosis (RVT), although an unusual event, most often has graft loss as a result (Figure 1). The causes that may lead to this serious complication include compression due to hematomas or lymphoceles, angulation or kinking of the vein, anastomotic strictures, or an underlying state of deep venous thrombosis or hypercoagulability (Penny, 1994). RVT usually occurs suddenly and towards the end of the first week of an otherwise uncomplicated kidney transplantation. Specifically it occurs in the first 2 weeks post transplant, with 80% occurring in the first month and 93% within the first year (Kobayashi, 2007). Clinical presentation is initiated by oliguria and hematuria with a tender swollen graft, which if ruptured, is accompanied by life-threatening bleeding (Kobayashi, 2007).

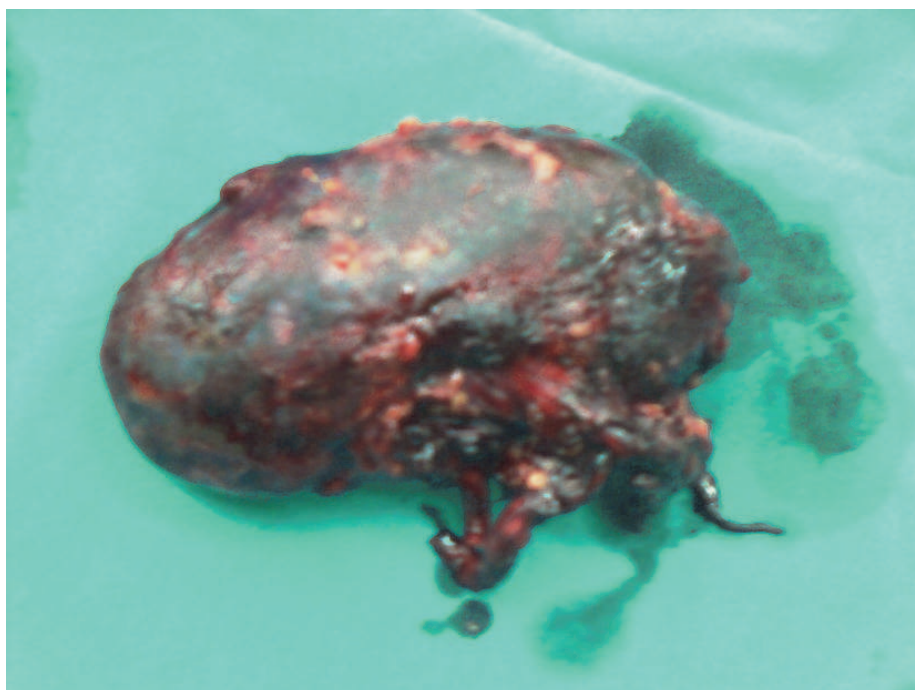


Fig. 1. Specimen of renal allograft nephrectomy due to renal vein thrombosis

### 5.2 Renal artery thrombosis

This uncommon complication which may occur most often as an early but also as a late event after kidney transplantation consists a devastating clinical condition leading frequently to graft loss. Renal artery thrombosis (RAT) onset most often follows a technical problem such as intimal dissection, kinking or torsion of the vessels. Risk factors include poor cardiac output, hyperacute rejection, unresponsive acute rejection, and a hypercoagulable state. It presents with a rapid onset of oliguria. In cases of segmental infarct, there can be lack of symptomatology or a presentation of renal dysfunction and increased blood pressure. When RAT occurs as a late event, it could be attributed to renal artery stricture or its manipulation post-operatively e.g. during angiography, or usually due to graft rejection.

### 5.3 Diagnosis

An early clinical diagnosis is very important for both RVT and RAT, even during the morning ward round. Diagnosis of these complications is established by colour flow Doppler studies, demonstrating in RVT a swollen graft with a crescent of clot along the convex margin of the kidney. In this case it is essential that the patient is taken immediately to theatre. Under normal clinical conditions, the spectral Doppler renal arterial waveform shows high resistive index with reversal of diastolic flow. On the contrary, in RAT a lack of flow in the renal artery is demonstrated, with the presence of intraluminal filling defects. In RAT, diagnosis is set by Doppler studies or at time of surgical exploration, however by that time it is not possible for the graft to be saved due to the kidney's low tolerance to warm ischemia (Rouviere, 2002).

### 5.4 Treatment

Following establishment of diagnosis for RVT, the treatment of choice is urgent thrombectomy. However graft salvage may not be possible, in which case graft nephrectomy is usually required. In case thrombectomy is applied early, within 1 hour following the event, graft salvage can be achieved. The increased risk of swelling, edema and also a possible rupture of the kidney graft in such a condition, makes urgent exploration essential. Systemic anticoagulants can be applied as treatment only in cases of partial vein thrombosis.

The surgical treatment for renal graft thrombosis includes laparotomy, thrombectomy and ultimately a possible graft nephrectomy. Several authors describe endoluminal therapy for renal graft thrombosis; however the exact role of interventional radiologic treatment is not yet well-defined (Obed, 2008). The technique for percutaneous treatment involves placing the tip of a catheter within the thrombus, 1 cm distal to the surgical anastomosis, with infusion of a thrombolytic agent (Rouviere, 2002). Because transcatheter thrombolysis revascularizes arteries at a slower rate than surgical thrombectomy, patients with a heavy clot burden should be primarily offered surgical treatment (Hedegard, 2009). Transcatheter thrombolysis should be limited to low clot burden, segmental artery thrombosis, or high-risk surgical candidates. Additionally catheter-directed thrombolytics should be avoided in the first 2 weeks following kidney transplant due to the immature anastomotic suture line. On the other hand RAT can be determined as a terminal event, which can be averted only if poor graft function can be attributed to arterial inflow and in this case intervention should be immediate. By the time the diagnosis is set, the transplanted kidney is lost.

### 5.5 Prevention

Although in many cases of renal allograft vascular thrombosis, no cause can be identified, epidemiological studies have attempted to categorise risk factors as modifiable, including drugs and the surgical procedure among others, and nonmodifiable, including age, diabetes mellitus and vascular anomalies. Additionally studies have identified changes in coagulation or fibrinolysis promoting a more thrombotic state, as risk factors as well.

Prevention may hold an important role in avoiding the formation of vascular thrombosis. This requires of course a combination of different measures such as avoiding prolonged cold and warm ischemia. Attention to precise surgical technique, use of preservation solution such as University of Wisconsin solution and an immediate and effective management of rejection, should all be outlined as important in the prevention strategy.

Additionally identification and management of thrombophilic states could act as a preventive measure against renal vascular thrombosis, with a possible need for routine screening and directed therapy to reduce the risk of thrombosis and graft loss, however no consensus for either strategy have been introduced. Previous reports indicate a possible laboratory investigation to potential recipients with a previous history or family history of thrombotic events, such as deep and superficial vein thromboses, pulmonary emboli, fistulas having been thrombosed or incidents of multiple occlusions of central venous dialysis catheters, as well as patients undergoing preemptive transplantation with a living donor kidney (Andrassy, 2004). The risk of thrombosis must be balanced against that of bleeding. For known thrombophilia and a history of clinical events, perioperative heparinization followed by long-term anticoagulation with warfarin has shown good results, including successful retransplantation. However since results of the few available, prospective randomized studies on heparin use in renal transplant patients, show conflicting conclusions, one understands that there is a great need for a preoperative classification of thrombotic and hemorrhagic risk among renal transplant candidates and for establishment of consensus guidelines.

6. Extrarenal pseudoaneurysm

Extrarenal arterial pseudoaneurysms in renal transplantation are rare, and their prevalence is less than 1% (Bracale, 2009). Extrarenal pseudoaneurysms are directly related to arterial anastomosis, percutaneous nephrostomy placement and infectious causes. It is usually asymptomatic and rarely can cause renal dysfunction or compression of adjacent structures (Bracale, 2009). When extrarenal pseudoaneurysms become large, there is a strong indication to be surgically removed to avoid spontaneous rupture and loss of the allograft (Figure 2).

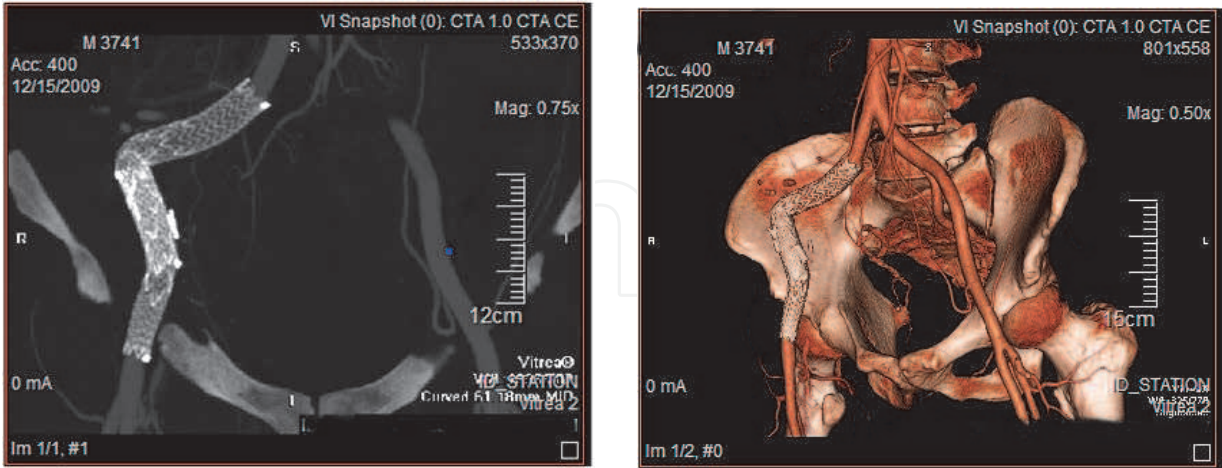


Fig. 2. External iliac artery pseudoaneurysm presented as a complication of renal vein thrombosis and allograft nephrectomy. The pseudoaneurysm was formed at the stump of the arterial anastomosis due to inflammation. Preemptive treatment with double stent placement was successfully performed.



## 7. Arteriovenous fistula

Arteriovenous fistula (AVF) is a well-recognized vascular complication of percutaneous biopsy. The reported incidence of AVF ranges between 0.5 and 16% (Martinez, 1998). An arteriovenous fistula (AVF) may be formed when both arterial and venous walls are punched by the biopsy needle. Mostly they are asymptomatic, rarely may cause persistent hematuria or recurrence of hematuria, hypertension and deterioration of renal function. Rarely, renal graft ischemia may be the result of steal phenomenon from a large AVF (Harrison, 1994, Matsell, 1992, Cruzado, 1999). Factors that may predispose to the development of arteriovenous fistula include early posttransplant period, the presence of hypertension, sclerosis and interstitial fibrosis, the formation of intrarenal hematoma (Schwarz, 2008). About 70% of AVF cases resolve spontaneously within weeks or months (Matsell, 1992).

Doppler sonography allowing noninvasive diagnosis of AVFs is the diagnostic examination of choice (Ozbek, 1995). Angiography is the reference standard as it confirms the presence of the AVF, accurately assesses its size and location, and permits endovascular treatment (Loffroy, 2008). On Doppler, the AVF shows a focal area of turbulent flow and a localized region of disorganized color that extends outside the normal vessels on color Doppler. In the area of AVF, the duplex US shows a very high velocity with low resistive index in the feeding artery with arterialization of the flow in the draining vein (Irshad, 2009).

In most cases AVFs close spontaneously within a few months, but they warrant observation to exclude the need for therapeutic intervention. The likelihood of and time to spontaneous closure in renal allografts, and the optimal time for therapeutic intervention are not predictable (Loffroy, 2008). Treatment has been recommended when bleeding persists for more than 72 h, renal function deteriorates markedly, lesion enlarges and there is suspicion of steal phenomenon. Endovascular superselective embolization is the therapeutic procedure of choice, as loss of normal parenchyma is minimal, with success rate of approximately 88% with no significant loss of allograft function (Loffroy, 2008, Tarif, 2002). In the majority of cases, successful embolization can be achieved using coils or microcoils. Schwarz et al (Schwarz, 2008) proposed a hemodynamic prognostic test to predict which AVF would probably profit from AVF coiling, by comparing Doppler sonographic resistive indices of the main renal artery and the non-AVF associated segmental arteries (Schwarz, 2008). The resistive index of the main renal artery should be at least 0.05 less than that of the non-AVF-associated segmental renal arteries indicating under-perfusion of the rest renal parenchyma.

## 8. Conclusions

Renal transplantation is regarded as an optimal treatment for End-stage renal disease. Improvements in surgical techniques and advanced immunosuppressive drugs have resulted in remarkable survival of patients and renal grafts. However complications occur in both the immediate postoperative period and later. Awareness for early post-operative complications, like renal vein and artery thrombosis could save allografts and patients. Cardiovascular disease remains the most frequent cause of death and transplant loss after kidney transplantation, with hypertension present in vast majority of kidney transplant recipients and a risk factor for cardiovascular disease. Improvements in imaging



modalities and interventional techniques resulted in earlier identification and management of TRAS. It is of paramount importance for the transplant surgeon to keep in mind that early and late vascular complications after renal transplantation could be very challenging and potentially allograft and/or life threatening

## 9. References

- Akbar SA, Jafri SZ, Amendola MA, Madrazo BL, Salem R, Bis KG. (2005). Complications of renal transplantation. *Radiographics*, Vol. 25, No. 5, (Sep-Oct 2005), p:1335-1356.
- Anderson JA, Weitz JI (2010). Hypercoagulable states. *Clinics in Chest Medicine*, Vol. 31, No. 4 (December 2010), p:31:659-73.
- Andrassy J, Zeier M, Andrassy K. (2004). Do we need screening for thrombophilia prior to kidney transplantation? *Nephrology Dialysis Transplantation*, Vol. 19, Suppl 4, p:iv-64-iv-68.
- Audard V, Matignon M, Hemery F, Snanoudj R, Desgranges P, Anglade MC, Kobeiter H, Durrbach A, Charpentier B, Lang P, Grimbert P. (2006). Risk factors and long-term outcome of transplant renal artery stenosis in adult recipients after treatment by percutaneous transluminal angioplasty. *American Journal of Transplantation* Vol. 6, No. 1, (January 2006), p:95-99.
- Bakir N, Sluiter WJ, Ploeg RJ, van Son WJ, Tegzess AM. (1996). Primary renal graft thrombosis. *Nephrology Dialysis Transplantation*, Vol. 11, No. 1, (January 1996), p:140-147, 1996
- Basso N, Terragno NA (2001). History about the discovery of the rennin angiotensin system. *Hypertension*, Vol. 38, No 6, (December 2001) p: 1246-1249.
- Baxter GM, Ireland H, Moss JG, Harden PN, Junor BJ, Rodger RS, Briggs JD. (1995). Colour Doppler ultrasound in renal transplant artery stenosis: which Doppler index? *Clinical Radiology*, Vol. 50, No. 9, (September 1995), p: 618-622.
- Beecroft JR, Rajan DK, Clark TW, Robinette M, Stavropoulos SW. (2004): Transplant renal artery stenosis: outcome after percutaneous intervention. *Journal of Vascular Interventional Radiology*, Vol. 15, No 12, (December 2004), p: 1407-1413, 2004.
- Benoit G, Moukarzel M, Hiesse C, Verdelli G, Charpentier B, Fries D. (1990): Transplant renal artery stenosis: Experience and comparative results between surgery and angioplasty. *Transplant International*, Vol. 3, No. 3, (October 1990), p:137-140.
- Birck R, Krzossok S, Markowetz F, Schnülle P, van der Woude FJ, Braun C. (2003). Acetylcysteine for prevention of contrast nephropathy: meta-analysis. *Lancet*, Vol. 362, No. 9384, (August 2003), p:598-603, 2003.
- Bick RL (2003). Antiphospholipid thrombosis syndromes. *Hematology Oncology Clinics of North America*, Vol. 17, No. 1, (February 2003), p:115-47.
- Bracale UM, Carbone F, del Guercio L, Viola D, D'Armiento FP, Maurea S, Porcellini M, Bracale G. (2009). External iliac artery pseudoaneurysm complicating renal transplantation. *Interactive Cardiovascular Thoracic Surgery*, Vol. 8, No. 6, (June 2009), p:654-60.
- Brewster UC, Setaro JF, Perazella MA. (2003). The renin-angiotensin-aldosterone system: cardiorenal effects and implications for renal and cardiovascular disease states. *The American Journal of Medical Sciences*, Vol. 326, No. 1 (July 2003):15-24.

- Bruno S, Remuzzi G, Ruggenenti P. (2004). Transplant renal artery stenosis. *Journal of the American Society of Nephrology*, Vol. 15, No. 1, (January 2004), p:134-141.
- Bucciarelli P, Rosendaal FR, Tripodi A, Mannucci PM, De Stefano V, Palareti G, Finazzi G, Baudo F, Quintavalla R (1999). Risk of venous thromboembolism and clinical manifestations in carriers of antithrombin, protein C, protein S deficiency, or activated protein C resistance: a multicenter collaborative family study. *Arteriosclerosis Thrombosis and Vascular Biology*, Vol. 19, No. 4, (April 1999), p:1026-33.
- Buturovic-Ponikvar J. (2003). Renal transplant artery stenosis. *Nephrology Dialysis Transplantation*, Vol. 18, No. S5, (July 2003), p:v-74-v-77.
- Cattaneo M (1999). Hyperhomocysteinemia, atherosclerosis and thrombosis. *Thrombosis and Haemostasis*, Vol. 81, No. 2, (February 1999), p:165-76.
- den Heijer M, Koster T, Blom HJ, Bos GM, Briet E, Reitsma PH, Vandenbroucke JP, Rosendaal FR (1996). Hyperhomocysteinemia as a risk factor for deep-vein thrombosis. *The New England Journal of Medicine*, Vol. 21, No. 334, (March 1996), p:759-62.
- Cruzado JM, Torras J, Domínguez J, Sancho C, Alsina J, Grinyó JM. (1999). An unusual cause of post-biopsy oliguria in an allograft. *Nephrology Dialysis Transplantation*, Vol. 14, No. 8, (August 1999), p:2022-4.
- Ducloux D, Motte G, Challier B, Gibey R, Chalopin JM (2000). Serum total homocysteine and cardiovascular disease occurrence in chronic, stable renal transplant recipients: a prospective study. *Journal of the American Society of Nephrology*, Vol. 11, No. 1, (January 2000), p:134-7.
- Ekberg H, Svensson PJ, Simanaitis M, Dahlbäck B (2000). Factor V R506Q mutation (activated protein C resistance) is an additional risk factor for early renal graft loss associated with acute vascular rejection. *Transplantation*, Vol. 27, No. 69, (April 2000), p:1577-81.
- Fervenza FC, Lafayette RA, Alfrey EJ, Petersen J. (1998). Renal artery stenosis in kidney transplants. *American Journal of Kidney Disease*, Vol. 31, No. 1, (January 1998), p:142-8.
- Fischereder M, Schneeberger H, Lohse P, Krämer BK, Schlöndorff D, Land W (2001). Increased rate of renal transplant failure in patients with the G20210A mutation of the prothrombin gene. *American Journal of Kidney Diseases*, Vol. 38, No. 5, (November 2001), p:1061-4.
- Friedman GS, Meier-Kriesche HU, Kaplan B, Mathis AS, Bonomini L, Shah N, DeFranco P, Jacobs M, Mulgaonkar S, Geffner S, Lyman N, Paraan C, Walsh C, Belizaire W, Tshibaka M.(2001). Hypercoagulable states in renal transplant candidates: impact of anticoagulation upon incidence of renal allograft thrombosis. *Transplantation*, Vol. 27, No. 72, (September 2001), p:1073-8
- Garovic VD, Textor SC. (2005): Renovascular hypertension and ischemic nephropathy. *Circulation*, Vol. 112, No. 9, (August 2005), p:1362-1374.
- Girndt M, Heine GH, Ulrich C, Köhler H. DialGene Consortium. (2007). Gene polymorphism association studies in dialysis: vascular access. *Seminars in Dialysis*, Vol. 20, No. 1, (Jan-Feb 2007), p:63-67.

- Giustacchini P, Pisanti F, Citterio F, De Gaetano AM, Castagneto M, Nanni G. (2002). Renal vein thrombosis after renal transplantation: An important cause of graft loss. *Transplantation Proceedings*, Vol. 34, No. 6, (September 2002), p:2126-2127
- Goldblatt H, Lynch J, Hanzal RE, Summerville WW. (1934). Studies on experimental hypertension, I: the production of persistent elevation of systolic blood pressure by means of renal ischaemia. *The Journal of Experimental Medicine*, Vol. 59, No. 3, (February 1934), p:347-379.
- Harrison KL, Nghiem HV, Coldwell DM, Davis CL. (1994). Renal dysfunction due to an arteriovenous fistula in a transplant recipient. *Journal of the American Society of Nephrology*, Vol. 5, No. 6, (December 1994), p:1300-6.
- Hedegard W, Saad WE, Davies MG. (2009). Management of vascular and nonvascular complications after renal transplantation. *Techniques in Vascular Interventional Radiology*, Vol. 12, No. 4, (December 2009), p:240-62
- Heidenreich S, Junker R, Wolters H, Lang D, Hessing S, Nitsche G, Nowak-Göttl U. (2003). Outcome of kidney transplantation in patients with inherited thrombophilia: data of a prospective study. *Journal of the American Society of Nephrology*, Vol. 14, No. 1, (January 2003), p:234-9.
- Irish AB, Green FR, Gray DW, Morris PJ. (1997). The factor V Leiden (R506Q) mutation and risk of thrombosis in renal transplant recipients. *Transplantation*, Vol. 64, No. 4, (August 1997), p:604-7.
- Irish A. (1999). Renal allograft thrombosis: can thrombophilia explain the inexplicable? *Nephrology Dialysis Transplantation*, Vol. 14, No. 10, (October 1999), p:2297-303.
- Irshad A, Ackerman S, Sosnouski D, Anis M, Chavin K, and Baliga P. (2008). A Review of Sonographic Evaluation of Renal Transplant Complications. *Current Problems in Diagnostic Radiology*, Vol. 37, No. 2 (Mar-Apr 2008), p:67-79
- Irshad A, Ackerman SJ, Campbell AS, Anis M. (2009). An overview of renal transplantation: current practice and use of ultrasound. *Seminars in Ultrasound CT and MR*, Vol. 30, No. 4, (August 2009), p:298-314.
- Key NS (1992). Scratching the surface: endothelium as a regulator of thrombosis, fibrinolysis, and inflammation. *The Journal of Laboratory Clinical Medicine*, Vol. 120, No. 2, (August 1992), p:184-6.
- Kobayashi K, Censullo ML, Rossman LL, Kyriakides PN, Kahan BD, Cohen AM. (2007). Interventional radiologic management of renal transplant dysfunction: Indications, limitations, and technical considerations. *Radiographics*, Vol. 27, No. 4, (Jul-Aug 2007), p:1109-30
- Koniari I, Siminelakis SN, Baikoussis NG, Papadopoulos G, Goudevenos J, Apostolakis E. (2010). Antiphospholipid syndrome; its implication in cardiovascular diseases: a review. *Journal of Cardiothoracic Surgery*, Vol. 3, No. 5, (November 2010), p:101-10.
- Kujovich JL. (2004). Thrombophilia and thrombotic problems in renal transplant patients. *Transplantation*, Vol. 15, No. 77, (April 2004), p:959-64.
- Loffroy R, Guiu B, Lambert A, Mousson C, Tanter Y, Martin L, Cercueil JP, Krausé D. (2009). Management of post-biopsy renal allograft arteriovenous fistulas with selective arterial embolization: immediate and long-term outcomes. *Clinical Radiology*, Vol. 63, No. 6, (June 2008), p:657-65.

- Mallamaci F, Zoccali C, Tripepi G, Fermo I, Benedetto FA, Cataliotti A, Bellanuova I, Malatino LS, Soldarini A. CREED Investigators. (2002). Hyperhomocysteinemia predicts cardiovascular outcomes in hemodialysis patients. *Kidney International*, Vol. 61, No. 2, (February 2002), p:609-14.
- Mallamaci F, Bonanno G, Seminara G, Rapisarda F, Fatuzzo P, Candela V, Scudo P, Spoto B, Testa A, Tripepi G, Tech S, Zoccali C. (2005). Hyperhomocysteinemia and arteriovenous fistula thrombosis in hemodialysis patients. *American Journal of Kidney Diseases*, Vol. 45, No. 4, (April 2005), p:702-7.
- Mangray M, and Vella JP. (2011). Hypertension After Kidney Transplant. *American Journal of Kidney Diseases*, Vol. 57, No. 2, (February 2011), p:331-341
- Martinez T, Palomares M, Bravo JA, Alvarez G, Galindo P, Entrena AG, Osuma A, Asensio C. (1998). Biopsy-induced arteriovenous fistula and venous aneurysm in a renal transplant. *Nephrology Dialysis Transplantation*, Vol. 13, No. 11, (November 1998), p:2937-9.
- Matsell DG, Jones DP, Boulden TF, Burton EM, Baum SL, Tonkin IL. (1992). Arteriovenous fistula after biopsy of renal transplant kidney: diagnosis and treatment. *Pediatric Nephrology*, Vol. 6, No. 6, (November 1992), p:562-4.
- Meyer M, Laux G, Scherer S, Tran TH, Opelz G, Mytilineos J. (2007). No association of factor V Leiden, prothrombin G20210A, and MTHFR C677T gene polymorphisms with kidney allograft survival: a multicenter study. *Transplantation*, Vol 27, No. 83, (April 2007), p:1055-8.
- Morrissey PE, Ramirez PJ, Gohh RY, Yango AY, Kestin A, Madras PN, Monaco AP. (2002). Management of thrombophilia in renal transplant patients. *American Journal of Transplantation*, Vol 2, No. 9, (October 2002), p:872-6.
- Murashima M, Konkle BA, Bloom RD, Sood SL, Grossman RA, Brunelli SM, Stein SH. (2010). A single-center experience of preemptive anticoagulation for patients with risk factors for allograft thrombosis in renal transplantation. *Clinical Nephrology*, Vol. 74, No. 5, (November 2010), p:351-7
- Obed A, Uihlein DC, Zorger N, Farkas S, Scherer MN, Krüger B, Banas B, Krämer BK. (2008). Severe renal vein stenosis of a kidney transplant with beneficial clinical course after successful percutaneous stenting. *American Journal Transplantation*, Vol. 8, No. 10, (October 2008), p:2173-2176
- Pannu N, Manns B, Lee H, Tonelli M. (2004). Systematic review of the impact of N-acetylcysteine on contrast nephropathy. *Kidney International*, Vol. 65, No. 4, April 2004), p:1366-1374.
- Patel NH, Jindal RM, Wilkin T, Rose S, Johnson MS, Shah H, Namyslowski J, Moresco KP, Trerotola SO. (2001). Renal arterial stenosis in renal allografts: retrospective study of predisposing factors and outcome after percutaneous transluminal angioplasty. *Radiology*, Vol. 219, No. 3, (June 2001), p:663-667.
- Penny MJ, Nankivell BJ, Disney AP, Byth K, Chapman JR. (1994). Renal graft thrombosis: a survey of 134 consecutive cases. *Transplantation*, Vol. 58, No. 5, (September 1994), p:565-569.



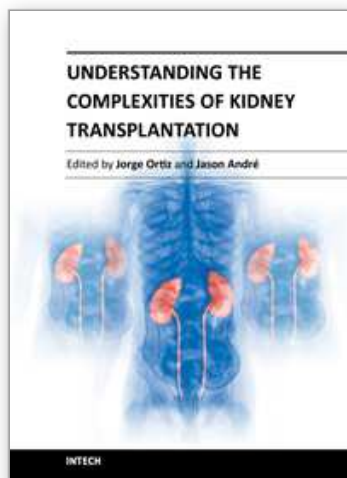
- Roberts JP, Ascher NL, Fryd DS, Hunter DW, Dunn DL, Payne WD, Sutherland DE, Castaneda-Zuniga W, Najarian JS. (1989). Transplant renal artery stenosis. *Transplantation*, Vol. 48, No. 4, (October 1989), p:580-583.
- Rouvière O, Berger P, Béziat C, Garnier JL, Lefrançois N, Martin X, Lyonnet D. (2002). Acute thrombosis of renal transplant artery: Graft salvage by means of intra-arterial fibrinolysis. *Transplantation*, Vol. 73, No. 3, (February 2002), p:403-409
- Sebastià C, Quiroga S, Boyé R, Cantarell C, Fernandez-Planas M, Alvarez A. (2001). Helical CT in Renal Transplantation: Normal Findings and Early and Late Complications. *RadioGraphics*, Vol. 21, No. 5, (Sep-Oct 2001), p:1103-1117
- Schafer AI, Levine MN, Konkle BA, Kearon C. (2003). Thrombotic disorders: diagnosis and treatment. *Hematology American Society of Hematology Educational Program*, p:520-539
- Schafer, AI. (2007). Hypercoagulable State. In *Cardiovascular Medicine*, Willerson JT, Wellens HJ, Cohn JN, Holmes DR, pp:2423-2438, Springer-Verlag, London.
- Schwarz A, Hiss M, Gwinner W, Becker T, Haller H, Keberle M. (2008). Course and relevance of arteriovenous fistulas after renal transplant biopsies. *American Journal of Transplantation*, Vol. 8, No. 4, (April. 2008), p:826-31.
- Snider JF, Hunter DW, Moradian GP, Castaneda-Zuniga WR, Letourneau JG. (1989). Transplant renal artery stenosis: Evaluation with duplex sonography. *Radiology*, Vol. 172, (September 1989), p:1027-30.
- Schoenberg SO, Bock M, Kallinowski F, Just A. (2000). Correlation of hemodynamic impact and morphologic degree of renal artery stenosis in a canine model. *Journal American Society Nephrology*, Vol. 11, No 12, (December 2000), p:2190-2198
- Tarif N, Mitwalli AH, Al Samayer SA, Abu-Aisha H, Memon NA, Sulaimani F, Alam A, Al Wakeel JS. (2002). Congenital renal arteriovenous malformation presenting as severe hypertension. *Nephrology Dialysis Transplantation*, Vol. 17, No. 2, (February 2002), p:291-4.
- Tutone VK, Mark PB, Stewart GA, Tan CC, Rodger RS, Geddes CC, Jardine AG. (2005). Hypertension, antihypertensive agents and outcomes following renal transplantation *Clinical Transplantation*, Vol 19, No. 2, (April 2005), p: 181-192
- Virchow R. *Gesammelte Abhandlungen zur Wissenschaftlichen Medtzin*. Frankfurt, Germany: *Medinger Sohn*, 1856:219- 732.
- Vaidya S, Sellers R, Kimball P, Shanahan T, Gitomer J, Gugliuzza K, Fish JC. (2000). Frequency, potential risk and therapeutic intervention in end-stage renal disease patients with antiphospholipid antibody syndrome: a multicenter study. *Transplantation*, Vol. 69, No. 7, (April 2000), p:1348-52.
- Voiculescu A, Schmitz M, Hollenbeck M, Braasch S, Luther B, Sandmann W, Jung G, Mödder U, Grabensee B. (2005). Management of arterial stenosis affecting kidney graft perfusion: a single-centre study in 53 patients. *American Journal of Transplantation*, Vol. 5, No. 7, (July 2005), p:1731-1738.
- Wagenknecht DR, Becker DG, LeFor WM, McIntyre JA. (1999). Antiphospholipid antibodies are a risk factor for early renal allograft failure. *Transplantation*, Vol. 68, No. 2, (July 1999), p: 241-246.



Wuthrich RP, Cicvara-Muzar S, Booy C, Maly FE. (2001). Heterozygosity for the factor V Leiden (G1691A) mutation predisposes renal transplant recipients to thrombotic complications and graft loss. *Transplantation*, Vol. 72, No. 3, (August 2001), p: 549-50.

IntechOpen

IntechOpen



## **Understanding the Complexities of Kidney Transplantation**

Edited by Prof. Jorge Ortiz

ISBN 978-953-307-819-9

Hard cover, 564 pages

**Publisher** InTech

**Published online** 06, September, 2011

**Published in print edition** September, 2011

Kidney transplantation is a complex field that incorporates several different specialties to manage the transplant patient. This book was created because of the importance of kidney transplantation. This volume focuses on the complexities of the transplant patient. In particular, there is a focus on the comorbidities and special considerations for a transplant patient and how they affect kidney transplant outcomes. Contributors to this book are from all over the world and are experts in their individual fields. They were all individually approached to add a chapter to this book and with their efforts this book was formed. Understanding the Complexities of Kidney Transplantation gives the reader an excellent foundation to build upon to truly understand kidney transplantation.

### **How to reference**

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Alexandros Giakoustidis, Nikolaos Antoniadis and Dimitrios Giakoustidis (2011). Vascular Complications in Kidney Transplantation, Understanding the Complexities of Kidney Transplantation, Prof. Jorge Ortiz (Ed.), ISBN: 978-953-307-819-9, InTech, Available from: <http://www.intechopen.com/books/understanding-the-complexities-of-kidney-transplantation/vascular-complications-in-kidney-transplantation>

**INTECH**  
open science | open minds

### **InTech Europe**

University Campus STeP Ri  
Slavka Krautzeka 83/A  
51000 Rijeka, Croatia  
Phone: +385 (51) 770 447  
Fax: +385 (51) 686 166  
[www.intechopen.com](http://www.intechopen.com)

### **InTech China**

Unit 405, Office Block, Hotel Equatorial Shanghai  
No.65, Yan An Road (West), Shanghai, 200040, China  
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元  
Phone: +86-21-62489820  
Fax: +86-21-62489821

© 2011 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the [Creative Commons Attribution-NonCommercial-ShareAlike-3.0 License](https://creativecommons.org/licenses/by-nc-sa/3.0/), which permits use, distribution and reproduction for non-commercial purposes, provided the original is properly cited and derivative works building on this content are distributed under the same license.

IntechOpen

IntechOpen