DOI: 10.15825/1995-1191-2025-1-8-16

# CURRENT CONCEPTS OF VASCULAR COMPLICATIONS FOLLOWING KIDNEY TRANSPLANTATION (A LITERATURE REVIEW)

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Even with advancements in surgical techniques, vascular complications remain life-threatening conditions and can lead to graft loss and sometimes recipient death. This paper examines the causes of vascular complications following a kidney transplant (KT), as well as international experience in the application of methods for early diagnosis, treatment and prevention of these complications.

*Keywords: kidney transplantation; vascular complications; prevention of vascular complications.*

## INTRODUCTION

Vascular complications are a significant concern in kidney transplantation (KT), occurring in 3–15% of recipients, with graft loss rates ranging from 12.6% to 66.7% in affected patients [1–3].

Early vascular complications include arterial and venous thrombosis, arterial dissection, vascular damage during the retrieval procedure (cuts and lacerations), arteriovenous fistulas, pseudoaneurysms, and hematomas. Later came stenosis or kinking of the renal artery and less often of the renal vein, as well as external compression due to the presence of fluid accumulations around the graft [1, 3]. The most common complications are arterial stenoses of the graft (3–12.5 %), followed by arterial and venous thromboses (0.1–8.2%) and vascular wall dissection (0.1 %). Arteriovenous fistulas or pseudoaneurysms occur less frequently [3].

## RENAL ARTERY THROMBOSIS

Renal artery thrombosis (RAT) is the leading cause of kidney transplant loss in the early post-transplant period. It is a rare complication with a reported incidence ranging from 0.2% to 3.5% [4, 5].

RAT most often develops within minutes to hours post-transplant and can occur as a result of hyperacute rejection, anastomosis occlusion, renal artery stenosis (RAS), renal artery kinking, and blood hypercoagulable state [6, 7]. In the first hours after surgery, RAT often presents subtly, with sudden oliguria or anuria and a rapid rise in plasma creatinine being the earliest signs [5]. Other clinical signs of RAT include worsening hypertension, pain at the graft site, which may occur as a result of tissue edema [7].

Additional diagnostic methods are necessary for early detection, such as color flow mapping, which helps differentiate thrombosis from acute rejection or acute tubular necrosis [8]. Catheter angiography and magnetic resonance angiography are the primary techniques for confirming the diagnosis by revealing reduced or absent blood flow to the graft. However, in emergency situations, angiography may not always be feasible [7, 9]. Screening ultrasound with duplex scanning is considered the standard method for assessing graft perfusion [10].

In cases of complete arterial occlusion due to thrombosis, graft loss can occur. However, if thrombosis is limited to small distal branches and does not affect the main renal artery, the outcome may still be favorable [11, 12]. If the inferior pole branch is involved, it may lead to ischemia and subsequent necrosis of the ureter [13].

In cases of RAT, emergency surgery is the only viable option to salvage the transplanted kidney [5, 7]. The standard procedure typically involves thrombectomy with anastomotic repair. While the role of interventional treatment for graft artery thrombosis remains unclear, there have been reports of successful cases using catheter-directed thrombolysis, with or without percutaneous angioplasty and/or stent placement [4, 14–16].

Ayvazoglu et al. reported that among 8 RAT cases, 5 patients underwent thrombectomy with arterial anastomotic reformation, and 3 patients underwent percutaneous transluminal angioplasty, thrombolysis and stent placement [16].

Harraz et al. analyzed cases of vascular accidents – renal artery thrombosis (19) and renal vein thrombosis (4), which accounted for 23 (1%) vascular accidents among 2208 cases of live donor kidney transplant (KT) between 1976 and 2011. In 12 RAT patients (63%), the

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graft was salvaged by open revascularization. The most important stages of their surgical strategy were ensuring vascular control, revision of arterial anastomosis, thrombus extraction and perfusion of the graft with preserving solution, heparinized saline and vasodilators with removal of perfusate through venotomy followed by reanastomosis [15].

Aktas et al. reported 3 cases of salvage of renal grafts with RAT by thrombectomy and formation of a new anastomosis. Two other RAT patients underwent percutaneous transluminal angioplasty, thrombolysis and stent placement [4].

## RENAL VEIN THROMBOSIS

Renal vein thrombosis (RVT) is one of the major vascular complications after KT and one of the major causes of immediate graft loss [27]. According to various reports, the incidence of early post-transplant RVT ranges from 0.1% to 5.5% [17], 0.3% to 4.2% [27], and 0.14% [5]. RVT usually develops within the first 5 days after surgery, with peak incidence within the first 48 hours, although there are cases of delayed RVT occurring after the first postoperative week [7].

Common causes of RVT are anatomical anomalies or technical problems during surgery [27], including graft vein kinking, vascular endothelial injury during surgical manipulation or during graft pretreatment [18, 19]. External compression caused by hematoma or lymphatic fluid accumulation is also referred to as a direct cause of RVT [6].

The short and thin-walled nature of the right renal vein (RRV) often presents technical challenges during right KT [17, 18]. However, Natour et al. reported that the side of KT is not directly associated with RVT risk [20]. To overcome these technical difficulties, lengthening the RRV by incorporating a segment of the inferior vena cava or gonadal vein in deceased donor right KT has proven effective. This approach facilitates venous anastomosis and is likely a key factor in the currently low incidence of RVT [21].

Higher doses of cyclosporine are also associated with a higher incidence of venous thrombosis. Another important reason is hypercoagulable states such as antithrombin III, protein C or protein S deficiency [22].

Clinically, RVT usually presents with a significant increase in the size and density of the graft, pain or discomfort in the graft area, increasing hematuria with rapidly decreasing urine output, proteinuria, graft dysfunction, oliguria, and/or complete anuria [23]. Indirect signs include ipsilateral lower extremity edema, subfebrile temperature, and, in severe cases, massive retroperitoneal hemorrhage.

Doppler ultrasound with color flow mapping is the primary and first-line diagnostic method for detecting RVT. They can detect absence of blood flow in the renal vein, swollen graft, and abnormal arterial signal with a plateau-like reverse diastolic flow [7, 8, 17]. Loss of corticomedullary differentiation and paranephral fluid can also be detected [7]. Magnetic resonance or computed tomography venography is a more accurate but less frequently used procedure as a routine assessment method [17].

Surgical intervention is almost always required when RVT is detected in a kidney transplant. According to Cambou et al., venous thrombosis was the cause of graft loss in 86.4% of cases, with only intraoperative thrombosis being associated with better graft survival (63.5%), which is probably due to the possibility of immediate intervention. In the case of surgical intervention, the graft is subjected to new ischemia-reperfusion injury with consequences in the form of delayed recovery of function or eventually graft function may not recover at all [17].

In a study by Fathi et al., three out of seven grafts with RVT were salvaged after open thrombectomy [24]. A study conducted by Haberal et al. reported that two out of four grafts were salvaged after emergency revision venous thrombectomy with restoration of blood flow. In two patients, treatment was unsuccessful and graftectomy was performed [25], which was necessary to prevent progressive congestive edema and graft rupture, which can lead to life-threatening bleeding and patient death [26]. According to Harraz et al., two RVT cases were resolved by percutaneous catheter-directed thrombolytic therapy and one case by thrombectomy and revascularization [15].

According to Lerman et al., immediate intervention within 1 hour of the onset of thrombosis can salvage the graft. Lerman et al. report a case in which the graft was salvaged after open thrombectomy and reimplantation of the kidney [27].

Early RVT has a poor prognosis. Evidence suggests that intraoperative monitoring for vascular complications is critical to the success of transplantation if intraoperative RVT occurs. In addition, screening for prothrombotic conditions in potential kidney recipients with risk factors for thrombosis and the justified use of perioperative anticoagulant therapy are important strategies to prevent thrombus formation [28].

## RENAL ARTERY STENOSIS

The incidence of transplant RAS is reported to range between 1% and 23% [33]. RAS is the most common vascular complication following a KT [4, 6, 16]. The wide range of incidence has been attributed to the lack of uniformity in the definition of the condition and the different imaging techniques used to make the diagnosis [29]. Other reports suggest that RAS affects about 3% of all renal transplants [7].

RAS is a potentially reversible cause of refractory post-transplant hypertension, where narrowing of the renal artery of the graft obstructs blood flow and leads to renal hypoperfusion [30]. RAS is typically diagnosed

between 3 months and 2 years after transplantation, though earlier or later manifestations can occur [31]. This complication should be suspected in patients with treatment-refractory arterial hypertension, worsening condition, elevated creatinine levels, decreased graft function, reduced urine output, or unexplained fluid retention [7]. Occasionally, clinically asymptomatic stenoses are incidentally detected during routine ultrasound surveillance of the recipient and graft [31].

Most RAS cases are prone to progression leading to graft loss. Therefore, early and accurate diagnosis of this condition is important [31]. Digital subtraction angiography is widely considered the gold standard for imaging RAS, but it is an invasive technique requiring the use of nephrotoxic contrast agents. Computed tomographic angiography and magnetic resonance angiography with contrast are also commonly used imaging techniques to diagnose RAS [31]. However, Doppler ultrasound angiography is preferred as the initial imaging choice because of its accessibility and non-invasiveness [9]. Signs of RAS are seen at the narrowing site and include an elevated peak systolic velocity (PSV), an abnormal ratio of PSV in the main renal artery compared to the superior iliac artery, and an aliasing effect due to turbulence. A PSV of 340–400 cm/s at the anastomosis site is considered a reliable criterion for RAS, which, however, should be considered in the context of other parameters [7].

Once the diagnosis is established, various surgical interventions are performed for hemodynamically significant stenosis [32]. Endovascular techniques, including percutaneous transluminal angioplasty and stent placement, are the first-line therapy. In refractory cases or with complex anatomical features, open surgical intervention follows [7].

Earlier studies have shown that following an endovascular procedure, a significant majority of patients experienced improvement in their kidney function (85–93%) and blood pressure (63–83%) to a more normal level. The risk of recurrence ranged from 10% to 33%, but was lower when angioplasty was combined with stenting [33].

This is supported by a meta-analysis by Ngo et al., which noted higher patency rates with stent placement for RAS compared to angioplasty alone (90.4% *vs.* 73%) [34]. Drug-eluting balloons and stents have also been successfully used to treat RAS [35]. A recent systematic review further demonstrated that endovascular treatment of RAS preserves graft function and hemodynamic parameters [35].

## RISK FACTORS FOR THROMBOTIC COMPLICATIONS

Risk factors for thrombotic complications can be broadly categorized into three groups: donor-related, recipient-related and those caused by to the peculiarities of the surgical intervention itself. Thrombosis may also develop as a result of a technical error during anastomosis, intima damage, decreased blood flow due to constriction or twisting of vessels, stasis, hypercoagulation, or exceeding the target levels of immunosuppressive drugs in the blood [3]. Other causes include acute rejection and external compression by hematoma or lymphocele [5].

### Donor risk factors for thrombotic complications

Donor risk factors for thrombotic complications include advanced donor age, vascular anomalies in the graft (such as multiple vessels), use of the right kidney as a graft, deceased donor kidneys versus living related donor kidneys, and prolonged warm and cold ischemia times. A donor age older than 60 years [36], or in some reports older than 50 years [6], is considered a significant risk factor for renal graft thrombosis.

As the demand for kidney transplants surpasses donor organ availability, medical professionals are increasingly utilizing kidneys with multiple renal arteries (MRAs) to expand the donor pool. While MRAs often require vascular reconstruction during transplantation, which was historically associated with a higher risk of early post-transplant complications such as graft thrombosis [37], recent studies suggest that this is no longer a significant concern. Modern surgical techniques and improved perioperative management have contributed to comparable outcomes between grafts with MRAs and those with single renal arteries, with no significant increase in postoperative complications or adverse events [37–42].

Only one retrospective study from Iran reported significantly higher surgical complication rates after transplantation of renal allografts with multiple arteries compared to cases that did not require vascular reconstruction. The authors found vascular complications in 25.4% of patients with multiple arteries, compared to 8.2% in recipients of single-artery grafts [43]. The presence of multiple arteries has previously been identified as an independent risk factor for bleeding [44], with a nephrectomy rate of 22% in severe cases. This may explain the findings of Salehipour et al. [43], where the reported bleeding rate was 6.1%, compared to 1.9% in the study by Osman et al.

There is no consensus on whether right renal grafts increase the risk of thrombosis compared to left renal grafts. One report suggests that the use of a right donor kidney increases the risk of thrombosis [36]. The main explanation proposed is that right KT may present technical difficulties because of the short and more ‘fragile’ right renal vein [18, 19].

These difficulties were partially addressed by Fallani et al., who utilized cryopreserved vascular grafts to lengthen renal vessels. Their study analyzed KT outcomes between 2012 and 2020, focusing on right kidney

grafts, including those with abnormal vascularization and cases requiring vessel lengthening. For grafts with multiple vessels, vein lengthening resulted in shorter warm ischemia and overall surgery times, making them comparable to standard grafts. For right kidney grafts using vessel lengthening, surgical times were similar to those of left kidney grafts. The authors concluded that the functional outcomes of these surgical approaches were comparable [46].

In another study, researchers from Spain found no significant difference in the use of right or left donor kidneys with respect to early posttransplant thrombosis. To control for donor-related factors, they compared the outcomes of contralateral kidney transplants from the same donor (24 pairs of transplants). However, they observed a striking discrepancy in the thrombosis rates based on the side of transplantation: 21 thrombosed grafts on the right (87.5%) compared to only three on the left [46].

An increased risk of thrombosis in deceased donor kidneys compared with grafts from living related donors has not been consistently observed in either earlier studies [43, 47] or more recent research. A recent study analyzing a cohort of 446 patients who received grafts from both living and deceased donors found no significant difference in renal artery thrombosis rates between the two groups [48].

While early studies did not identify warm ischemia time as a risk factor for thrombosis [44], a more recent study from Tunisia highlighted prolonged warm ischemia as a potential risk factor for vascular thrombosis [49]. An increased incidence of thrombosis has been reported in cases where cold ischemia time exceeded 24 hours [36].

### Recipient-related risk factors for thrombotic complications

Recipient-related factors are considered more critical for KT outcomes than donor-related factors [50]. Recipient-related risk factors include age, the underlying disease leading to end-stage renal disease (ESRD), comorbidities, and the choice, order, and duration of renal replacement therapy (RRT).

It is well established that recipients younger than 5 years and older than 50 years are more susceptible to graft thrombosis [35]. However, KT is increasingly being performed in older patients [51–54]. In the United States, the proportion of patients aged 65–74 years on the transplant waiting list has risen from 2% in the 1990s to over 10% in 2012. Similarly, in Europe, the average recipient age has increased by 10 years over the past two decades. For example, in the Netherlands, the proportion of kidney transplant recipients over 65 years of age grew from 22.8% in 2005 to 39.8% in 2017 [53, 55].

The underlying disease that led to ESRD significantly influences transplant outcomes. Individuals with diabetes mellitus and diabetic nephropathy are at a significantly increased risk of thrombosis due to hyperactive platelets, elevated prothrombotic clotting factors, and impaired fibrinolysis [56]. Diabetes accelerates the development of atherosclerosis, further increasing the risk of thrombotic complications. Cardiovascular disease and angiopathy associated with atherosclerosis also contribute to a higher likelihood of thrombosis in these patients [6].

Obesity (BMI >30 kg/m) has been associated with an increased risk of thrombosis, as well as a paradoxical protective effect against postoperative bleeding [41, 64]. Given the global rise in obesity, more high-BMI patients are undergoing KT [57]. While BMI likely interacts with other patient-specific factors, it remains an important consideration for transplant candidacy and post-transplant outcomes [58].

The choice, order, and duration of RRT can influence transplant outcomes significantly. A systematic review analyzing 76 studies (1968–2019) found that preemptive transplantation offers better long-term survival and a lower risk of graft loss [59].

Preemptive transplantation – where a kidney transplant is performed before the initiation of dialysis – has been shown to significantly improve long-term survival and reduce the risk of graft loss. A systematic review that analyzed 76 studies from 1968 to 2019 confirmed these benefits, highlighting the importance of early transplantation in optimizing patient outcomes [59].

The selection, sequence, and duration of RRT play a crucial role in transplant outcomes. A systematic review of 76 studies (1968–2019) found that preemptive transplantation significantly improves long-term survival and reduces the risk of graft loss [59].

Several large-cohort studies have reported a significantly higher incidence of thrombotic graft loss in patients who underwent peritoneal dialysis (PD) before transplantation compared to those on hemodialysis (HD) [60]. This increased risk is attributed to elevated levels of procoagulant factors, such as apolipoprotein A and coagulation factors II, VII, VIII, IX, X, XI, and XII, in PD patients, likely due to a moderate nonspecific inflammatory response of the peritoneum to dialysate exposure. However, other studies suggest that the type of dialysis does not significantly influence the risk of graft thrombosis [61].

## PATHOPHYSIOLOGY OF HEMOSTATIC CHANGES IN RENAL TRANSPLANT RECIPIENTS

In KT, all three components of Virchow's triad – endothelial damage, circulatory stasis, and a hypercoagulable state – are present [62, 63]. These factors can contribute to thrombosis due to issues arising during vascular anastomosis and reconstruction, vascular intima damage, reduced blood flow from vessel constriction, compression, or twisting, as well as graft congestion. Additionally, the hypercoagulable state may result from

the pathophysiology of chronic kidney disease (CKD) itself or associated comorbidities [3].

Historically, patients with end-stage CKD were considered to be in a "hypocoagulant" state, meaning they had an increased tendency to bleed rather than clot. In the early 1980s, studies demonstrated that patients with uremia often suffer from anemia, and their bleeding time is strongly dependent on anemia. Correcting anemia to a hematocrit level above 30% was found to significantly reduce bleeding time and the severity of hemorrhagic manifestations [64].

Recent studies have identified CKD as a risk factor for thromboembolic complications. CKD is associated with a prothrombotic state, marked by elevated tissue factor activity, increased fibrinogen, and higher D-dimer levels [65]. The disease also involves systemic inflammation, oxidative stress, activation of the renin-angiotensin-aldosterone system, anemia, microalbuminuria, hyperhomocysteinemia, hyperparathyroidism, and bone and mineral metabolism disorders. Specific apolipoprotein isoforms and platelet hyperreactivity contribute to a hypercoagulable state, increasing the risk of arterial and venous thromboembolic events and accelerating the progression of kidney failure [63, 66].

Paradoxically, while ESRD patients are at increased risk for thrombosis, they also have a higher propensity for bleeding, with a 4% incidence of postoperative bleeding after transplantation compared to 1% in general surgery [67]. This may be attributed to profound imbalances between procoagulant and anticoagulant mechanisms in CKD patients.

## PREVENTION OF THROMBOTIC COMPLICATIONS IN KIDNEY TRANSPLANTATION

Rapid identification of thrombotic complications is crucial for successful prophylaxis in KT, as treatment options are largely limited to emergency surgery and thrombectomy. While perioperative prophylactic antithrombotic therapy appears justified, no standardized protocols currently exist [68]. Existing thrombosis prevention guidelines primarily focus on venous thromboembolism and do not specifically address thrombosis at surgical sites [69]. Additionally, the European Association of Urology guidelines do not recommend routine postoperative prophylaxis with unfractionated or low-molecular-weight heparin for low-risk living-donor transplant recipients [70].

Patients with advanced CKD face a delicate balance between thromboembolic risk and bleeding complications, making anticoagulant therapy particularly challenging. Due to hemostatic alterations and multiple comorbidities, CKD and ESRD patients are often excluded from clinical trials evaluating anticoagulation therapy [71, 72]. Consequently, there is limited evidence on the efficacy and safety of anticoagulants in this population, and no standardized risk assessment scale exists to accurately determine individual thromboembolic and hemorrhagic risk. Additionally, pharmacokinetic and pharmacodynamic alterations related to impaired renal function further complicate anticoagulation management [63].

As a result, reliable data for establishing evidence-based guidelines on perioperative antithrombotic therapy remain limited [73]. Consequently, different medical centers implement their own protocols for perioperative anticoagulation and postoperative thromboprophylaxis [68].

## CONCLUSION

Advancements in surgical techniques, perioperative management, and immunosuppressive therapy have significantly improved KT outcomes and patient survival. However, vascular complications remain a major challenge, posing risks to both the graft and the recipient. A thorough analysis of these complications highlights three key areas requiring special attention: the intrinsic characteristics of the donor organ, recipient-associated risk factors, and the complexity of the surgical procedure – all of which underscore the importance of preventive strategies.

*The authors declare no conflict of interest.*

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*The article was submitted to the journal on 13.08.2024*