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Congestive Nephropathy Due to Superior Vena Cava Thrombosis Following Kidney Transplantation: A Neglected Cause of Early Graft Dysfunction

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ABSTRACT

Early graft dysfunction following kidney transplantation is commonly attributed to hypoperfusion, rejection, or urological or vascular complications. Venous congestion as a primary mechanism of renal dysfunction remains under-recognized. We report a case of early post-transplant graft dysfunction caused by superior vena cava (SVC) thrombosis–induced congestive nephropathy.

A 32-year-old woman with end-stage kidney disease underwent living donor kidney transplantation after prolonged hemodialysis via a tunneled internal jugular catheter. Despite satisfactory immediate diuresis, she developed progressive oliguria within 12 hours of transplant surgery. Hemodynamics were stable, and Doppler ultrasonography showed patent graft vessels but markedly elevated intrarenal resistive indices with absent end-diastolic flow. She subsequently developed facial and neck edema suggestive of SVC syndrome. Imaging revealed chronic SVC thrombosis with extensive collateralization. Renal allograft biopsy showed features of venous congestion without evidence of rejection.

Initial conservative decongestive therapy led to partial improvement, but definitive endovascular SVC stenting resulted in rapid diuresis, normalization of intrarenal Doppler parameters, and sustained graft recovery.

This case highlights congestive nephropathy as a reversible and often overlooked cause of early graft dysfunction and underscores the importance of considering central venous obstruction in transplant recipients with prior long-term central venous catheter exposure.

Keywords

Congestive nephropathy, Superior vena cava syndrome, Kidney transplantation, Renal venous congestion, Doppler ultrasound.

Introduction

Congestive nephropathy (CN) is a hemodynamic form of renal dysfunction caused by impaired renal venous drainage leading to increased renal interstitial pressure and reduced glomerular filtration. Although traditionally overshadowed by hypoperfusion-based paradigms of kidney injury, emerging evidence suggests that venous congestion may be a dominant mechanism of renal dysfunction in several clinical settings but it is still not given due consideration in clinical practice.

Superior vena cava (SVC) obstruction, increasingly related to indwelling venous catheters, can cause marked elevations in central venous pressure and renal venous congestion. However, its role in post-transplant graft dysfunction remains poorly recognized. We report a case of early post-transplant graft dysfunction due to SVC thrombosis–induced congestive nephropathy, highlighting diagnostic challenges and pathophysiological insights.

Case Report

A 32-year-old woman with end-stage kidney disease of unknown etiology underwent living donor kidney transplantation from her mother. She had been on thrice-weekly hemodialysis for two years, including four months via a right internal jugular tunneled cuffed catheter before arteriovenous fistula maturation.

Immediate post-transplant urine output was satisfactory. However, within 12 hours, she developed progressive oliguria. Hemodynamics remained stable with no hypotensive episodes. Doppler ultrasonography demonstrated patent renal artery and vein without evidence of thrombosis, anastomotic complication, or significant perinephric collection. However, intrarenal Doppler revealed markedly elevated resistive indices with absent end-diastolic flow, a pattern suggestive of renal venous congestion.

Despite diuretic therapy, oliguria became refractory. Over the next 24 hours, the patient developed facial, neck, and upper chest edema associated with dyspnea, which was disproportionate to lower limb edema. These findings raised suspicion of superior vena cava (SVC) syndrome. Contrast-enhanced computed tomography of the chest demonstrated a chronic organized thrombus at the SVC–right atrial junction with prominent azygos and hemiazygos collaterals, consistent with chronic SVC obstruction.

A renal allograft biopsy was performed to evaluate persistent graft dysfunction. Histopathology revealed dilated peritubular capillaries and venules, mild acute tubular injury, and patchy cortical necrosis. There was no evidence of acute rejection, thrombotic microangiopathy, or calcineurin inhibitor toxicity.

An initial attempt at endovascular recanalization of the SVC thrombus was unsuccessful. The patient was managed conservatively with aggressive decongestion using high-dose diuretics and daily hemodialysis, resulting in partial improvement in urine output and symptoms. However, recurrent facial edema and worsening graft function prompted a repeat endovascular intervention. Successful balloon angioplasty and stenting of the SVC (Figure 1) resulted in rapid diuresis, resolution of craniofacial edema, normalization of intrarenal Doppler flow with restoration of end-diastolic velocities, and stabilization of serum creatinine at 1.4 mg/dL.

Discussion

Superior vena cava syndrome results from obstruction to venous return through the SVC and has classically been associated with malignancy, accounting for nearly 70% of cases [1,2]. Over the past two decades, benign and device-related causes have increased substantially and now contribute to approximately 25–30% of cases [1]. The widespread use of tunneled hemodialysis catheters is a major contributor to this shift [1,2].

In developing countries such as India, delayed arteriovenous fistula creation and prolonged catheter dependence predispose patients to central venous stenosis and thrombosis. Despite this, SVC obstruction frequently remains under-recognized, particularly when symptoms are subtle or intermittent.

The clinical manifestations of SVC syndrome depend on the level, severity, and chronicity of obstruction, as well as the extent of collateral venous development. Venous return may be redirected through the azygos–hemiazygos system, internal mammary veins, lateral thoracic veins, and vertebral venous plexus. In the present case, obstruction distal to the azygos vein allowed extensive collateralization, explaining the relatively mild and intermittent symptoms prior to transplantation.

On retrospective evaluation, the patient reported recurrent facial and neck swelling, epiphora, and dizziness during the dialysis

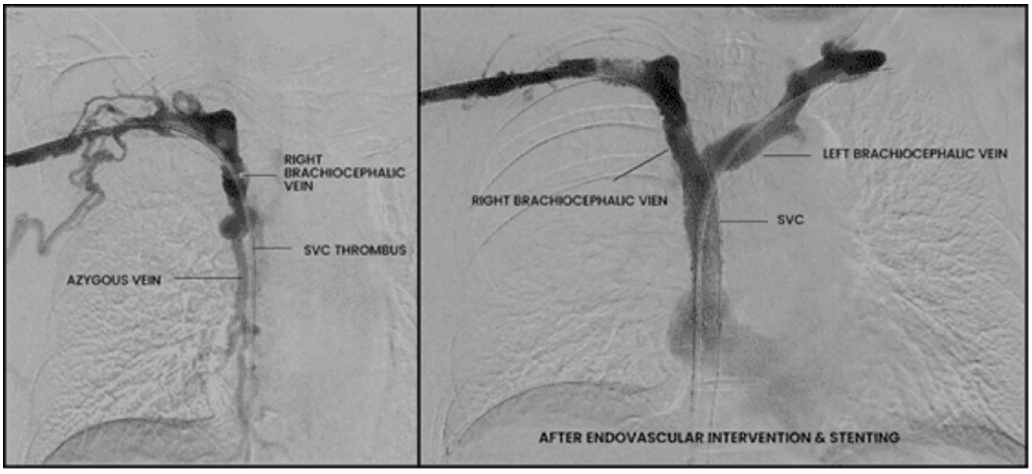

Figure 1: Digital subtraction venography demonstrating superior vena cava (SVC) thrombosis with impaired flow and collateralization (left panel), and restoration of SVC patency with improved central venous drainage following endovascular intervention and stent placement (right panel).

period. These symptoms worsened in the supine position and improved following ultrafiltration. However, they were incorrectly attributed to interdialytic weight gain, and the diagnosis of SVC syndrome was missed during the pre-transplant workup. Following kidney transplantation, aggressive intravenous fluid administration to compensate for post-transplant polyuria precipitated acute decompensation, unmasking the hemodynamic consequences of chronic venous obstruction.

Digital subtraction venography remains the gold standard for diagnosing SVC obstruction [1,3], allowing precise delineation of the site and severity of obstruction, identification of thrombus, visualization of collateral pathways, and procedural planning. Importantly, the presence of extensive collateral vessels correlates more closely with clinical severity than anatomical obstruction alone.

Endovascular therapy with balloon angioplasty and stenting has become the treatment of choice for SVC obstruction due to both malignant and benign causes. It provides rapid symptom relief, high technical success rates [3,4] and a favorable safety profile. Although post-procedural antithrombotic strategies vary, short-term anticoagulation with or without antiplatelet therapy is commonly employed.

The renal dysfunction observed in this patient is best explained by congestive nephropathy, a hemodynamic form of kidney injury caused by elevated renal venous pressure [5,6]. Traditionally, renal dysfunction in SVC syndrome was attributed to reduced renal perfusion. However, experimental and clinical evidence now suggests that venous congestion plays a dominant role [3,4,7]. A curvilinear relationship exists between central venous pressure and glomerular filtration rate, with significant decline occurring once pressures exceed approximately 6 mmHg [3,4].

Elevated venous pressure is transmitted to the renal veins, leading to increased interstitial pressure within the non-compliant renal capsule. This results in compression of post-glomerular capillaries and tubules, impaired lymphatic drainage, neurohormonal activation, sodium retention, inflammation, oxidative stress, and progressive reduction in glomerular filtration [3,6,8] [Figure 2].

Congestive nephropathy is most commonly described in heart failure but has also been reported in pulmonary hypertension, isolated tricuspid regurgitation, elevated intra-abdominal pressure, and venous outflow obstruction [6,10]. Diagnosis remains challenging due to the absence of definitive diagnostic criteria. Intrarenal Doppler ultrasonography can provide useful clues; elevated resistive indices with absent or reduced end-diastolic flow should raise suspicion of venous congestion [9].

Histopathological features of congestive nephropathy are not well established. Available reports describe dilated peritubular capillaries and veins, mild acute tubular injury, limited interstitial fibrosis, and relatively preserved glomeruli [10]. Improvement in renal function following decongestion remains the most practical diagnostic clue. In the present case, biopsy findings and dramatic improvement following SVC stenting strongly support venous congestion as the dominant mechanism of graft dysfunction.

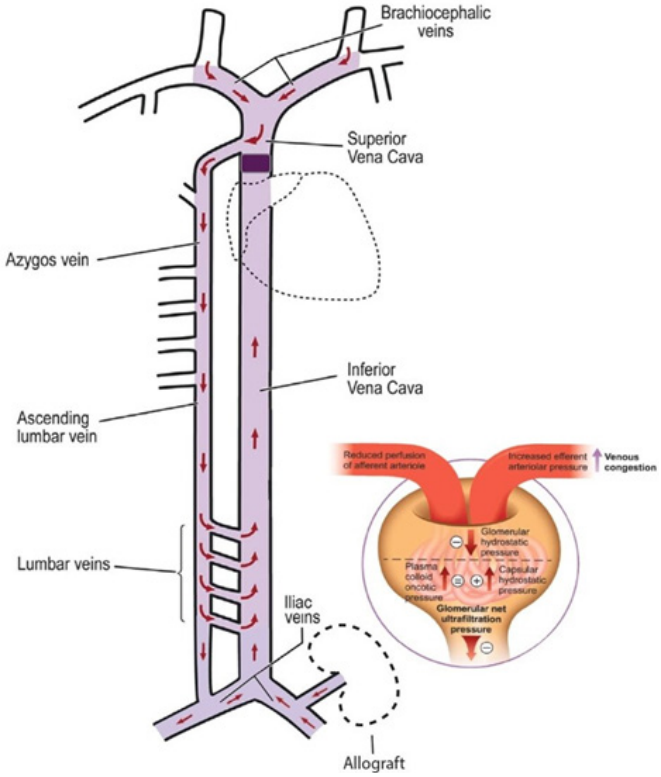

Figure 2: Pathophysiology of congestive nephropathy in venous outflow obstruction.
Schematic representation showing impaired venous drainage from a renal allograft due to obstruction of the venous system, with diversion of blood through collateral pathways including the lumbar, ascending lumbar, azygos, and brachiocephalic veins. Elevated renal venous pressure leads to venous congestion, increased efferent arteriolar pressure, and reduced effective renal perfusion. The inset illustrates the glomerular hemodynamic consequences of venous congestion, including increased capsular and glomerular hydrostatic pressures, reduced transglomerular ultrafiltration pressure, and consequent decline in glomerular filtration rate, culminating in congestive nephropathy.

Take-home Messages

- Superior vena cava obstruction is an under-recognized complication of long-term tunneled hemodialysis catheter use and may remain clinically silent before transplantation.
- Venous congestion can be a dominant mechanism of early post-transplant graft dysfunction, even in the presence of stable hemodynamics and patent graft vessels.
- Elevated intrarenal resistive indices with absent end-diastolic flow on Doppler ultrasonography should prompt evaluation for renal venous congestion.
- Congestive nephropathy lacks definitive diagnostic criteria; improvement in renal function following decongestion remains a key diagnostic clue.
- Pre-transplant assessment for central venous obstruction

should be considered in transplant candidates with prior central venous catheter exposure to prevent potentially reversible graft dysfunction.

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