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Case Report

Massive Lymphocele Causing Severe Allograft Dysfunction Following Robotic-Assisted Kidney Transplantation (RAKT): A Case Report

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ABSTRACT

Background: Lymphocele is a common postoperative complication following renal transplantation, resulting from disruption of lymphatic channels during surgery. While most lymphoceles remain asymptomatic, larger collections may lead to compressive complications, including ureteric obstruction and graft dysfunction[1]. Robotic-assisted kidney transplantation has been associated with a reduced incidence of symptomatic lymphocele, although it does not eliminate the risk.

Case Presentation: We report a 27-year-old male who developed a large symptomatic lymphocele approximately 80 days after robotic-assisted live donor kidney transplantation (RAKT). The presentation included right lower limb swelling and acute deterioration in graft function. Laboratory evaluation showed a significant increase in serum creatinine from baseline of 1.5 mg/dL to 5.8 mg/dL. Imaging revealed a large anechoic collection mimicking a distended urinary bladder causing extrinsic ureteric compression with severe hydronephrosis. A key pharmacologic contributor was the early postoperative use of Everolimus for delayed graft function. Successful restoration of graft function with initial ultrasound-guided pigtail catheter insertion and adjunctive chemical sclerotherapy.

Results: Subsequently the patient developed an acute recurrence of the lymphocele and a Secondary spike in creatinine due to an intraluminal fibrin clot occluding the catheter. Prompt percutaneous catheter clearing and mechanical flushing immediately resulted in high-volume drainage and a rapid, complete restoration of renal function to the patient's baseline of 1.5 mg/dL. Because of recurrent filling of lymphocele patient underwent laparoscopic unroofing procedure and it lead to clinical resolution of the issue

Conclusion: Lymphocele remains an important and reversible cause of graft dysfunction even in the modern era of robotic transplantation. This case underscores the necessity of prioritizing structural & mechanical aetiologies alongside immunological rejection when managing unexplained graft dysfunction, especially in patients receiving concurrent mTOR inhibitor therapy. Early diagnosis and timely intervention are essential for graft preservation.

Keywords: *Kidney transplantation, lymphocele, robotic transplant, graft dysfunction, hydronephrosis, ultrasound, Everolimus.*

INTRODUCTION

Lymphocele is defined as a lymph-filled collection without an epithelial lining that typically develops in the retroperitoneal space following renal transplantation[1]. It arises due to disruption of lymphatic vessels during donor nephrectomy or recipient iliac vessel dissection. Although most lymphoceles are small and clinically silent, larger collections can result in significant morbidity, including infection, ureteric obstruction, and allograft dysfunction[1].

The incidence of lymphocele varies widely depending on detection modality and follow-up duration, with increased detection rates attributed to routine use of ultrasonography[1]. Structural causes of graft dysfunction, such as lymphocele, are often under-recognized and may mimic immunological etiologies[3].

Robotic-assisted kidney transplantation is increasingly adopted due to its minimally invasive approach and reduced complication rates. Meta-analyses have demonstrated a lower incidence of symptomatic lymphocele with robotic techniques[2]. However, clinically significant lymphocele continues to occur, necessitating vigilance.

CASE PRESENTATION

A 27-year-old male with end stage renal disease (ESRD) underwent live-related renal transplantation, with his mother as the donor. Native kidney biopsy demonstrated chronic hypertensive changes along with features of thrombotic microangiopathy secondary to uncontrolled hypertension. Genetic evaluation for primary thrombotic microangiopathy was negative.

The patient underwent a robotic assisted kidney transplantation (RAKT) for ESRD. The early postoperative course was largely uneventful except for transient acute tubular injury on postoperative day one. Induction immunosuppression included anti-thymocyte globulin and methylprednisolone, followed by maintenance therapy with tacrolimus, mycophenolate mofetil, and prednisolone. Early allograph complications, such as acute tubular necrosis and delayed graft function, severely alter local interstitial fluid dynamics and predispose the recipient to impaired lymphatic regeneration. When the mTOR inhibitors are introduced, it acts synergistically to disrupt local tissue healing and drastically accelerate high-output retroperitoneal lymphatic leakage. [4,5,6] Everolimus was administered transiently during the first postoperative month due to delayed graft function, a factor known to increase the risk of lymphocele formation.[7,8]

The patient stabilized with a baseline serum creatinine of 1.5 mg/dL. Eighty days post-transplantation, he presented with progressive right lower limb swelling. Doppler studies of the lower limb and iliac vessels were unremarkable. Within ten days, serum creatinine increased to 5.8 mg/dL.

On examination, a tense hypogastric swelling was noted, mimicking a distended urinary bladder. Ultrasonography revealed a large anechoic collection without internal echoes, consistent with lymphocele.[3] Repeat assessment after bladder emptying confirmed the diagnosis.

Management and Outcome

Ultrasound-guided drainage was performed with insertion of a pigtail catheter. Fluid analysis demonstrated lymphocyte-rich fluid with creatinine levels similar to serum, consistent with lymphocele. There was no evidence of vascular compromise on Doppler imaging. Renal biopsy revealed acute tubular injury without features of rejection.

Serial drainage combined with sclerotherapy was performed, an approach supported by existing literature demonstrating efficacy in symptomatic lymphocele management.[5] The combined use of percutaneous drainage and embolic or chemical sclerotherapy continues to be the standard initial approach for treatment of symptomatic fluid collections following transplantation with very reproducible clinical success rates.[6,7] While the choice of agent is inconsistent, long-term cohort data underscore the fact that careful volume-targeted instillation markedly reduces local inflammation and maintains vascular integrity.[8] However, even with optimal initial tract closure, technical limitations related to catheter use and localized fluid accumulation continue to contribute to significant rates of clinical recurrence.[9,10,11] The patient initially improved; however, catheter blockage resulted in recurrence of the collection, causing hydronephrosis and severe graft dysfunction.

Following prompt percutaneous correction of the catheter obstruction, there was rapid improvement in the renal function, with serum creatinine returning to the baseline of 1.5mg/dl. The catheter and use of sclerosing agent did not completely solve the issue in this case. He was taken for laparoscopic unroofing. It solved the issue and serum creatinine stabilised.

Table 1- Demonstrates the post-transplantation clinical timeline and procedural events.

Post-transplant timeline	Clinical Manifestation/Complication	Interventions & therapeutic measure done
Day 1	Early Operative phase: acute tubular injury and signs of delayed graft function (DGF) Serum creatinine level- 3.8	Supportive management
Day 2-30	Asymptomatic; stable early postoperative course	Introduction to anti-thymocyte globulin and methylprednisolone maintenance therapy (tacrolimus, MMF, prednisolone) initiated. Everolimus administered transiently during the first month due to DGF
Day 30	Fully asymptomatic; normal recovery Baseline stabilization Serum creatinine level- 1.5 mg/dl	Regular clinical follow-up; Everolimus discontinued as allograft function stabilizes
Day 80	Onset Progressive right lower limb swelling Serum creatinine level- 1.5 mg/dl	Lower limb and iliac vessel color doppler studies performed; deep vein thrombosis (DVT) successfully ruled out.
Day 90	Onset Progressive right lower limb accompanied with tense , palpable hypogastric mass mimicking a distended urinary bladder Serum creatinine level- 5.8 mg/dl	1. USG- Revealed a massive , anechoic pelvic lymphocele causing extrinsic ureteric compression and hydronephrosis 2. Primary guided percutaneous drainage with pigtail catheter. 3. Biochemical fluid analysis (confirmation lymphocyte-rich fluid; creatinine matched serum levels) 4. Allograft core biopsy confirmed compressive ATN; negative for rejection
Day 91-93	Initial decrease in pelvic swelling; progressive relief of leg edema Serum creatinine level- declining	Serial high volume fluid drainage combined with adjunctive chemical sclerotherapy sessions
Day 94	Sudden , acute recurrence of tense hypogastric swelling and leg edema due to abrupt stoppage of catheter output Serum creatinine level- (5.2mg/dl)	Beside assessment; USG confirmed recurrence of pelvic fluid collection and secondary hydronephrosis
Day 95	Acute graft dysfunction secondary to mechanical uropathy Serum creatinine level- Elevated (3.2)	Secondary procedure- urgent percutaneous correction and mechanical flushing of the pigtail catheter, clearing an intra-luminal fibrin clot and restoring high-volume output
Day 100	Complete resolution of lower limb swelling and hypogastric mass effect Serum creatinine level- 1.5 mg/dl (Recovery)	Permanent removal of pigtail catheter done and he was taken for laparoscopic unroofing

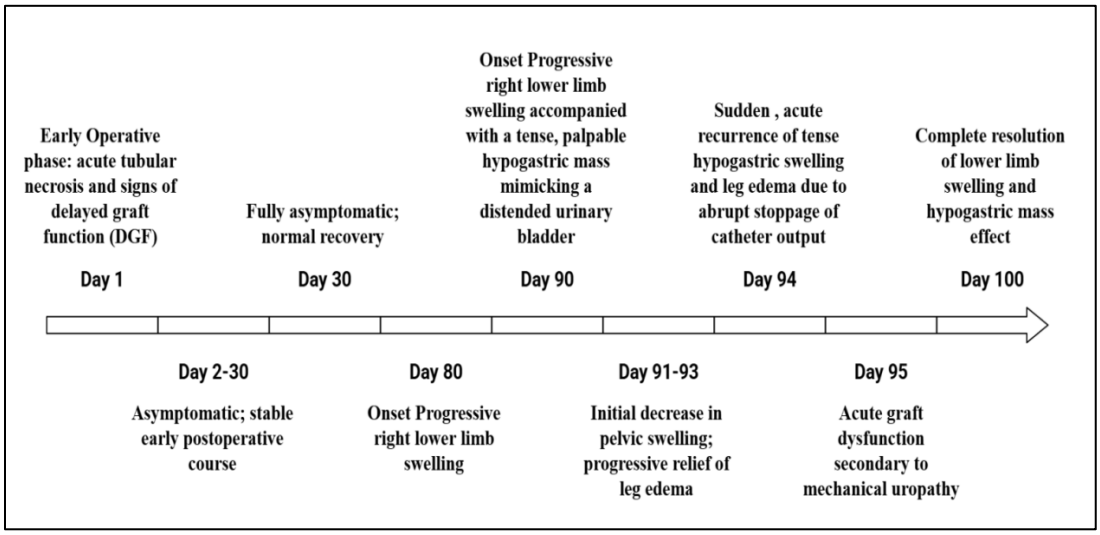

Figure 1- Demonstrates the post-transplantation clinical timeline

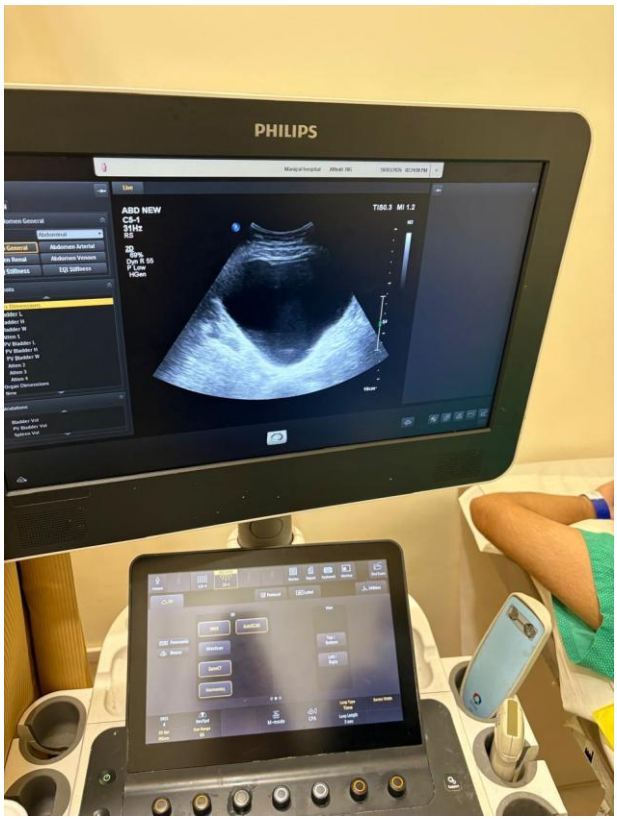

Figure 2. Ultrasound image showing a well-defined anechoic fluid collection in the pelvis adjacent to the renal allograft, with posterior acoustic enhancement and no internal echoes, consistent with a lymphocele.

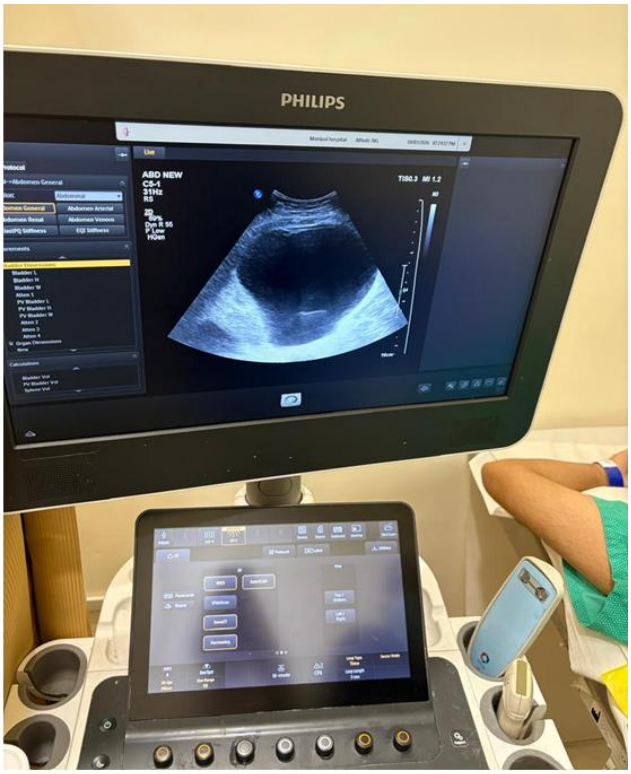

Figure 3. Repeat ultrasonography demonstrating persistence of a large anechoic pelvic collection occupying the hypogastric region, mimicking a distended urinary bladder.

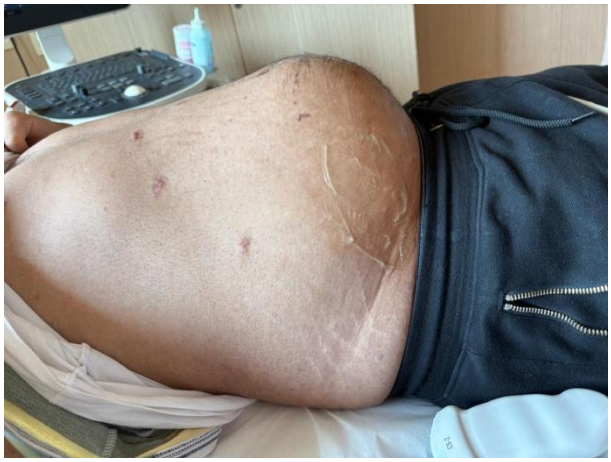

Figure 4. Clinical photograph showing tense hypogastric swelling corresponding to underlying fluid collection in a post–renal transplant recipient.

DISCUSSION

Lymphocele remains one of the most common surgical complications following renal transplantation, with incidence ranging widely depending on diagnostic criteria.[1] It typically develops within weeks to months after transplantation and may present with nonspecific symptoms, often mimicking rejection or drug toxicity.[1,6]

The underlying pathophysiology involves lymphatic leakage from disrupted vessels during surgical dissection.[1] Multiple risk factors have been implicated, including obesity, delayed graft function, use of mTOR inhibitors such as everolimus,[4,5,10] high-dose steroids, anticoagulation therapy, and acute rejection episodes[7,8],[9]. The use of everolimus in our patient likely contributed to lymphocele formation by delaying endothelial healing and tissue repair.

While most lymphoceles are asymptomatic, larger collections may exert mass effect on adjacent structures. Ureteric compression can lead to hydroureteronephrosis and obstructive uropathy, as demonstrated in previous reports.[4] In severe cases, compression of the renal parenchyma may mimic the Page kidney phenomenon[1].

Ultrasonography remains the cornerstone of diagnosis, typically demonstrating an anechoic perinephric collection[3]. Differentiation from other fluid collections, particularly the urinary bladder, is critical and can be achieved through dynamic assessment before and after voiding.

Management strategies depend on symptom severity and size of the collection. Small lymphoceles often resolve spontaneously, whereas symptomatic cases require intervention.[5] Percutaneous drainage with or without sclerotherapy is widely accepted as first-line therapy, although recurrence rates remain significant[5,10,11,12]. Various sclerosing agents, including ethanol, povidone-iodine, and tetracycline derivatives, have been used with variable success[11],[12].

For recurrent or large lymphoceles, laparoscopic fenestration into the peritoneal cavity is considered an effective and definitive treatment option[9],[13]. Adjunctive therapies such as octreotide have also been explored in refractory cases[13].

Despite advancements in surgical techniques, including robotic-assisted transplantation, lymphocele formation cannot be entirely prevented[2]. This case underscores the importance of considering structural causes in the differential diagnosis of graft dysfunction. Early recognition and timely intervention are crucial, as prompt drainage can reverse renal impairment and prevent unnecessary renal replacement therapy[3].

CONCLUSION

Lymphocele remains a significant and potentially reversible cause of graft dysfunction following renal transplantation, even in the era of robotic-assisted surgery. A high index of suspicion, early imaging, and prompt intervention are essential to preserve graft function and improve patient outcomes.

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