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Case Report

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An Unusual Case of Allograft Nephromegaly Secondary to Renal Lymphangiectasia Presenting Several Years Following Deceased Donor Kidney Transplantation

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Abstract

Introduction: Renal lymphangiectasia is a rare condition characterized by nephromegaly secondary to benign intrarenal or extrarenal lymphatic dilatation with perinephric leakage. Only a few isolated case reports exist following kidney transplantation (KT).

Methods: We report herein a case of allograft nephromegaly occurring several years following deceased donor kidney transplantation.

Results: The patient underwent KT in 2009 and initially did well. In 2017, he noticed fluid retention with lower extremity and abdominal swelling that improved with diuretic therapy and was not associated with proteinuria. He returned in 2023 at which time he had significant weight gain and intermittent leakage of clear fluid from his transplant incision. Computerized tomographic imaging of the abdomen and pelvis revealed ascites, fluid-filled left inguinal and supraumbilical hernias, and allograft enlargement with perinephric fluid. He was again managed medically with diuretic therapy. He eventually presented in 2026 with recurrent allograft nephromegaly, a large perinephric fluid collection with extension into the psoas muscle, and fluid-filled hernias. Multiple imaging modalities and repeated percutaneous drainage procedures as well as attempted lymphatic ablation failed to reveal a specific etiology or completely resolve the above findings. Due to the infiltrative nature of the imaging findings, a kidney biopsy was recommended but the patient refused. Serum creatinine has remained stable in the 1.6 mg/dl range with no significant proteinuria. Symptoms have improved with diuresis and intermittent drainage.

Conclusion: Renal lymphangiectasia is an extremely rare condition that is largely a diagnosis of exclusion. Pathophysiology is unclear although the few cases reported following KT have occurred many years later. Management is primarily medical and expectant although symptomatic patients can be treated with repeated aspiration, drainage, or rarely surgical peritoneal fenestration. Nephrectomy is rarely indicated in the absence of suspected malignancy.

Keywords: Ascites; Allograft Nephromegaly; Deceased Donor Kidney Transplant; Lymphatic Obstruction; Perinephric Fluid

Abbreviations: BMI: Body Mass Index; CT: Computerized Tomography; FSGS: Focal Segmental Glomerulosclerosis; KT: Kidney Transplantation; MRI: Magnetic Resonance Imaging; PET: Positron Emission Tomography.

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INTRODUCTION

Renal lymphangiectasia is an extremely unusual benign condition characterized by distended peripelvic, intrarenal, and perirenal lymphatics leading to extracapsular lymphatic leakage, nephromegaly, perinephric fluid collections, and ascites [1-4]. The pathophysiology of renal lymphangiectasia is poorly understood and it is extremely uncommon in native kidneys and even rarer following kidney transplantation (KT) [1-7]. The clinical presentation may range from relatively asymptomatic abdominal distension to markedly symptomatic abdominal pain and swelling, refractory ascites, renal dysfunction, renal vein thrombosis, hypertension, hematuria, and/or proteinuria. We present herein a singular case of renal lymphangiectasia

presenting several years following an uncomplicated deceased donor KT.

CASE PRESENTATION

A Jehovah's Witness black male in his 30s with end stage renal disease secondary to focal segmental glomerulosclerosis (FSGS), weight 142 kg, body mass index [BMI] 41.2 kg/m^2, on peritoneal dialysis for 8.3 years, underwent deceased donor KT in April 2009. The donor was a mid-20s white male, Kidney Donor Profile Index score 22%, with normal renal function who sustained head trauma but did not progress to brain death. Donation after circulatory death organ recovery was performed with a cold ischemia time of 22 hours. The donor left kidney was transplanted to the recipient's right iliac fossa through an extraperitoneal approach. He received alemtuzumab induction therapy (single intraoperative dose) in combination with triple maintenance therapy consisting of tacrolimus, mycophenolate, and prednisone. He experienced delayed graft function, requiring temporary hemodialysis for one week but was discharged on postoperative day 4. He subsequently did well with an established baseline serum creatinine level of 1.6-1.8 mg/dl (estimated glomerular filtration rate 55-60 ml/min/1.73m^2) and no readmissions for the next 8 years. Following his one-year visit to the Transplant Clinic, he was referred back to his local nephrologist and primary care physician.

Starting in 2017, he noticed fluid retention with lower extremity and abdominal swelling that improved with diuretic therapy and was not associated with proteinuria. However, he was not referred back to Transplant Clinic until 2023 at which time he presented with significant weight gain (179 kg, BMI 54.3 kg/m^2), abdominal wall hernias, and intermittent leakage of clear fluid from his right lower quadrant transplant incision. He responded well to diuretic therapy with resolution of incisional leakage and improvement in abdominal swelling and hernia symptoms. Serum creatinine levels remained in the 1.6-1.8 mg/dl and there was no evidence for significant proteinuria, thus ruling out recurrent FSGS as a cause for his symptoms. In addition, duplex ultrasonography was negative for transplant renal artery stenosis and the patient exhibited controlled hypertension. Given his Jehovah's Witness status, stable renal function, and high BMI, a decision was made neither to perform a kidney biopsy nor repair the abdominal wall hernias at this time.

He was referred back to his local physicians but eventually returned to Transplant Clinic in April 2026 at which time he presented with recurrent abdominal pain and swelling, enlarging hernias, distension, and weight gain (167 kg, BMI 48.6 kg/m^2). Physical examination revealed asymmetric abdominal swelling, easily reducible periumbilical and left inguinal hernias, a large diastasis recti, and trace peripheral edema. Renal transplant ultrasonography demonstrated allograft nephromegaly (16.2 x 9.7 x 9.0 cm, estimated volume 745 ml), perinephric fluid, and good perfusion to the kidney with normal resistive indices. Plain abdominal radiographs showed marked distension and asymmetry of the right side of the abdomen with absence of gas (Figure 1). Coronal and axial CT images revealed a markedly enlarged kidney allograft in the right lower quadrant surrounded by a rind of perinephric fluid with extension into the right psoas muscle and a mass effect with flattening of the inferior

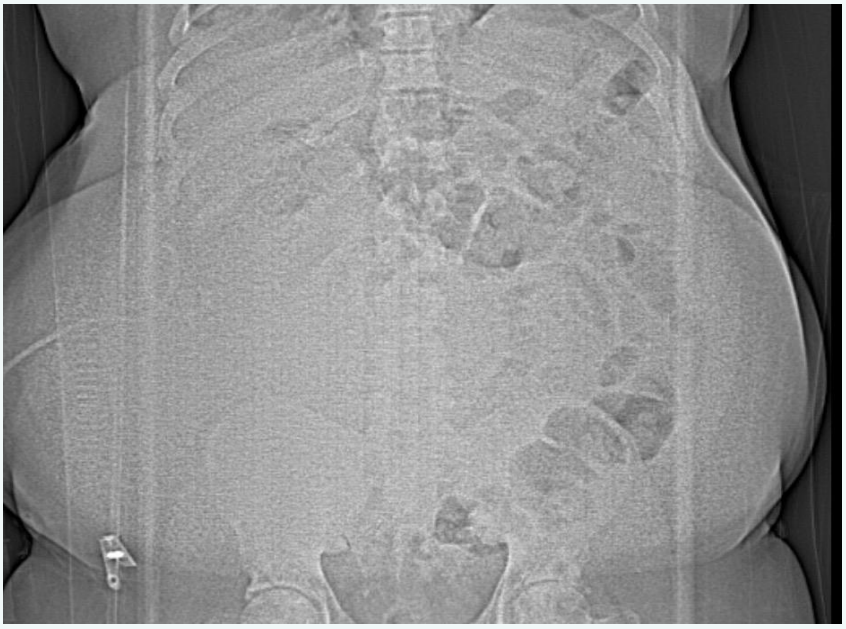


Figure 1: Plane abdominal radiograph depicting marked distension and asymmetry of the right side of the abdomen with absence of gas

vena cava (Figures 2a and 2b). Coronal and axial magnetic resonance imaging (MRI) showed similar findings with a complex perinephric fluid collection and renal sinus dilatation (Figures 3a and 3b). Coronal and axial Positron Emission Tomography (PET) scan displayed tracer uptake in the stomach, bladder, and renal transplant but no uptake in the perinephric fluid or evidence for tumor or inflammatory avid activity elsewhere (Figures 4a and 4b).

The patient was admitted to the hospital and subsequently underwent repeated percutaneous drainage procedures (with drain fluid analysis) as well as attempted lymphatic ablation, none of which revealed a specific etiology or completely resolved the clinical findings. Each attempted intervention resulted in treatment with intravenous antibiotic therapy for "peritonitis" pain. At one point, he had two percutaneous drains in place, each draining up to one liter per day of serous fluid that was negative for creatinine, triglycerides, cytology, gram stain, and culture.

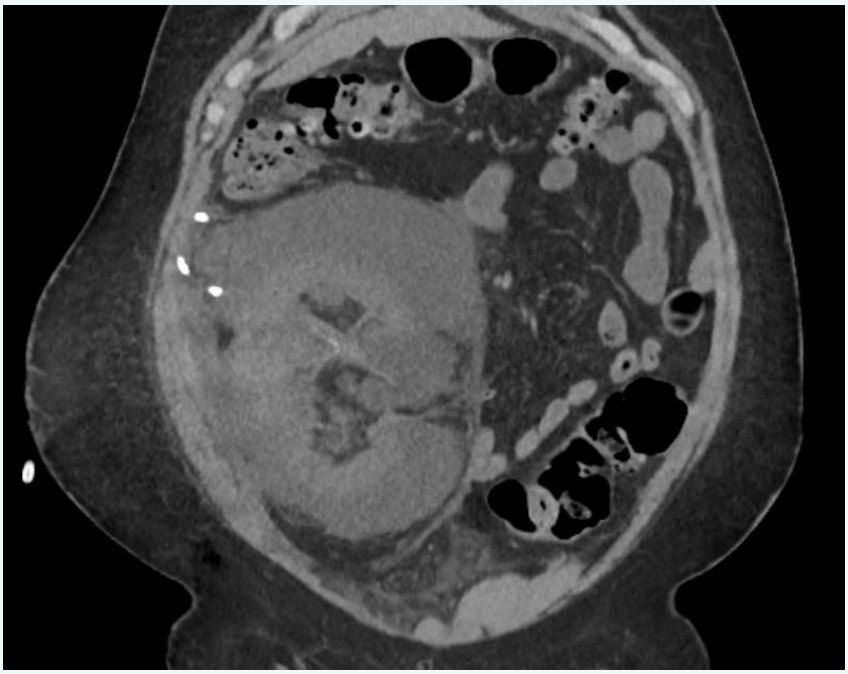


Figure 2a: CT scan coronal view post kidney transplant demonstrating a markedly enlarged kidney allograft in the right lower quadrant surrounded by a rind of perinephric fluid with extension into the right psoas muscle. A percutaneous drain can be seen superior-lateral to the kidney in the perinephric fluid collection.

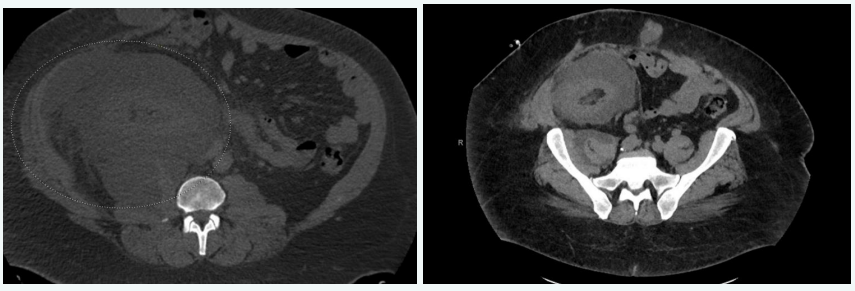


Figure 2b: CT scan axial views demonstrating allograft nephromegaly and a rind of perinephric fluid with psoas extension and a mass effect with flattening of the inferior vena cava.

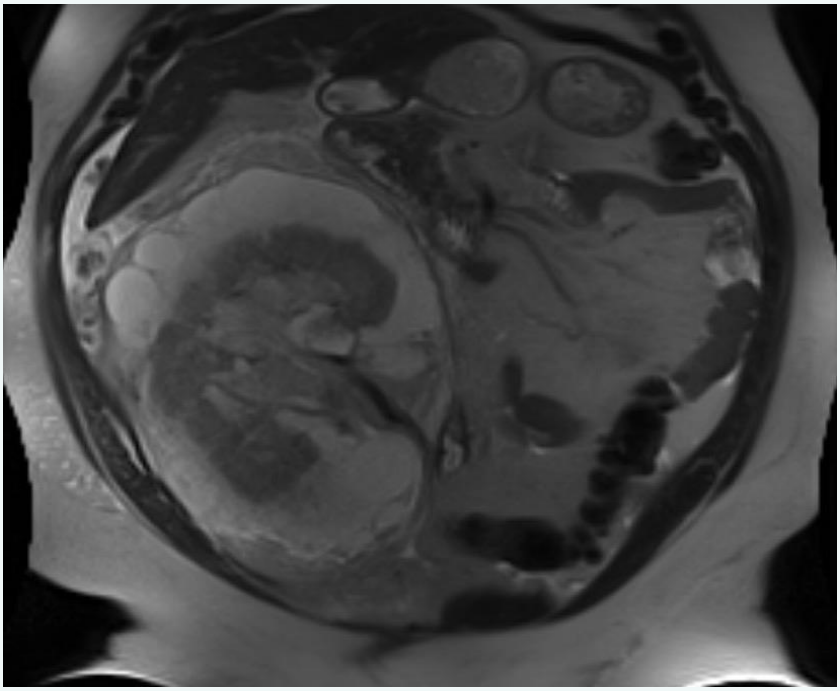


Figure 3a: Coronal magnetic resonance imaging (MRI) showing allograft nephromegaly with renal sinus dilatation, and a large complex perinephric fluid collection with associated surrounding mass effect.

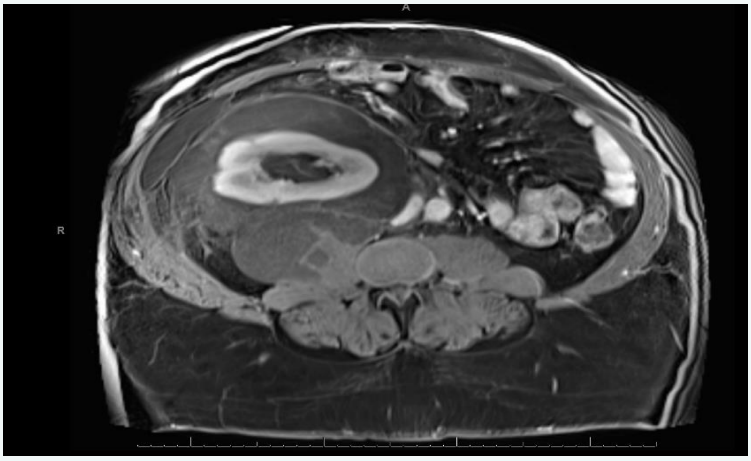


Figure 3b: Axial magnetic resonance imaging (MRI) showing similar findings with allograft nephromegaly, renal sinus dilatation, and a large complex perinephric fluid collection with associated surrounding mass effect

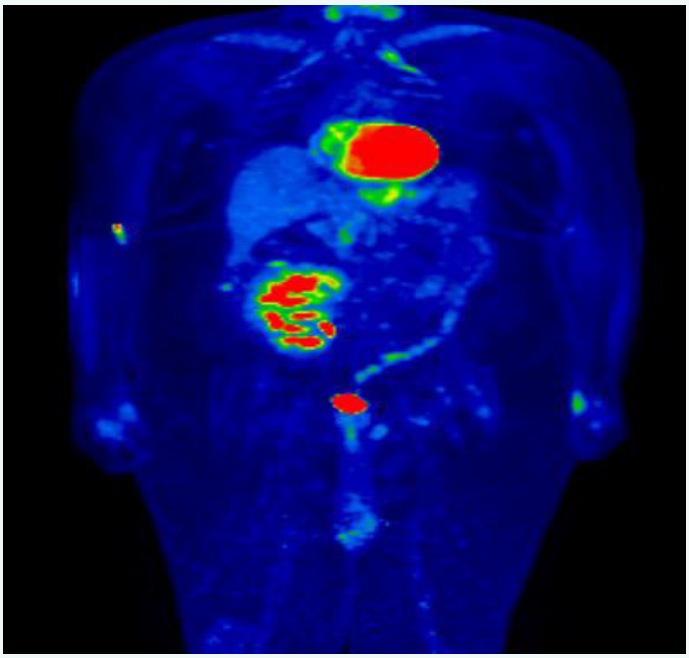


Figure 4a: Coronal Positron Emission Tomography (PET) scan displaying physiologic tracer uptake in the stomach, renal transplant, and bladder but no uptake in the perinephric fluid or evidence for tumor or inflammatory avid activity elsewhere.

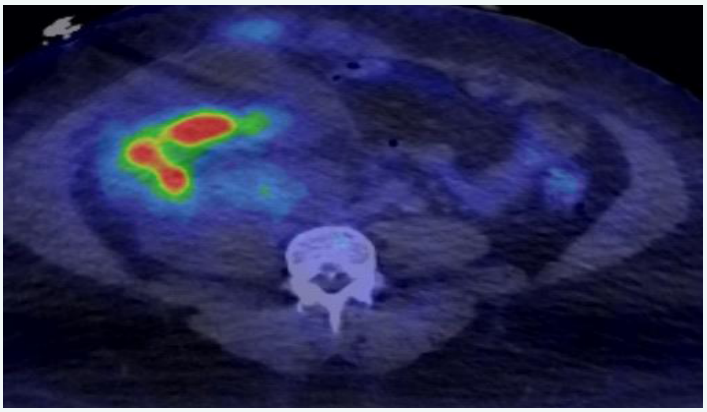


Figure 4b: Axial Positron Emission Tomography (PET) scan displaying tracer uptake in the renal transplant but no evidence for tumor or inflammatory avid activity elsewhere.

Despite high volume drainage, serial imaging revealed persistence of loculated perinephric fluid collections such that sclerotherapy was not a consideration. There was no evidence for liver disease, urinary tract infection, hydronephrosis, malignancy, subcapsular hematoma, cystic kidney disease, or transplant renal artery stenosis. Due to the infiltrative nature of the imaging findings, a kidney biopsy was recommended but the patient refused because of a concern for bleeding and potential need for blood products. Serum creatinine has remained stable in the 1.6 mg/dl range with no significant proteinuria. Symptoms have improved with diuresis and intermittent drainage.

DISCUSSION

The pathophysiology of renal lymphangiectasia, also known as renal lymphangiomatosis, is unclear. Renal lymphatic capillaries are distributed around the interlobular and arcuate arteries of the cortex and drain into hilar collector vessels [4]. However, misconnection in this drainage pathway can result from congenital factors such as lymphatic malformations or acquired factors from inflammatory reactions, infections, tumors, or trauma [2-4]. In this setting, renal lymphangiectasia may occur. In our patient, it is interesting to speculate whether long-term pretransplant peritoneal dialysis may have contributed to an impairment in lymphatic drainage, predisposing to the

development of this disorder. Renal lymphangiectasia is an extremely uncommon benign condition that is largely a diagnosis of exclusion. Differential diagnosis includes hydronephrosis, seroma, urinoma, posttransplant lymphoproliferative disease, lymphocele, infection or abscess, (subcapsular) hematoma, renal vein thrombosis, and renal/pelvic cystic disease. Clinical presentation is nonspecific and indolent with symptoms of abdominal or flank pain, distension due to ascites, hematuria, proteinuria, and/or hypertension.

The few cases reported following kidney transplantation have occurred many years later. Imaging modalities play a key role in the diagnostic process [1-7]. Management is primarily conservative (medical and expectant) although symptomatic patients can be treated with repeated aspiration, drainage, ablation, attempted sclerotherapy, or surgical peritoneal fenestration. Our patient had a clinical presentation and course similar to the few other transplant cases reported in the literature [1-7]. Despite attempts to diagnose the cause of nephromegaly and drain the perinephric fluid, we were unable to establish a specific etiology or implement a treatment that resulted in complete resolution of symptoms. Ideally, a kidney biopsy would be indicated to rule out an infectious, inflammatory, malignant, or disease recurrence process. However, given that this process dated back to 2017 and imaging modalities ruled out remote abnormalities, it is unlikely that the patient has a malignancy or that a kidney biopsy would have changed the management algorithm. In the absence of proteinuria, renal allograft dysfunction, hydronephrosis, pyelonephritis, renal artery stenosis, liver disease, adenopathy, or urinary tract infection, we are left with a presumptive diagnosis of renal lymphangiectasia that is currently being managed medically. However, if the patient presents again with worsening signs and symptoms, our next step is to consider surgical peritoneal fenestration. Allograft nephrectomy or other invasive interventions are rarely indicated in this setting in the absence of suspected malignancy or infection provided that renal function remains adequate.

CONFLICT OF INTERESTS/DISCLOSURE

The authors do not have any conflicts of interest to disclose pursuant to this study. This study was approved by the Atrium Health Wake Forest Baptist Institutional Review Board under the auspices of the Cortex Study, Protocol Number 00084139.

DATA AVAILABILITY STATEMENT

Data available upon request from the authors.

AUTHOR CONTRIBUTIONS

Concept/design: RK, RS, EMM, ACF, RJS

Data analysis/interpretation/collection: RK, RS, EMM, RJS

Drafting article: RK, RS, EMM, RJS

Critical revision and approval of manuscript: RK, RS, EMM, ACF, GO, EM, CW, RJS

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