Deceased donor kidney transplant complicated by spontaneous rupture of native kidney in a HIV patient

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Abstract

Spontaneous native kidney rupture (SNKR) is a rare occurrence, commonly associated with underlying renal tumors or acquired renal cystic disease in both the kidney transplant (KT) and non-KT populations. Herein, we present a 65-year-old African American man who experienced a non-malignant SNKR 6 days after a deceased donor KT and underwent emergent native nephrectomy. The patient's hospital course was complicated by thrombocytopenia and refractory hypertension. He experienced delayed graft function and was maintained on hemodialysis until post-operative day 30. This case demonstrates an unusual presentation of SNKR in the immediate post-KT setting and illustrates the clinical decision-making algorithm.

Keywords Spontaneous native kidney rupture · Kidney transplant · Delayed graft function

Introduction

Albeit rare, spontaneous native kidney rupture (SNKR) usually presents in patients with underlying renal tumors [1–3]. Risk factors include malignant lesions, nephritis, atherosclerosis, blood dyscrasias, concomitant anticoagulant therapy, and uncontrolled hypertension [4]. A non-traumatic SNKR in a patient without a reasonable explanation is extremely rare, with only a few reported cases in the literature [4, 5]. SNKR has also been described in kidney transplant (KT) recipients previously maintained on peritoneal (PD) or hemodialysis (HD) [1, 3].

In the non-KT and KT population, renal cell carcinomas and angiomyolipomas are the most common causes of SNKR [4, 6, 7]. Multiple case reports describe SNKR in

patients with acquired renal cystic disease [3, 8]. Hemorrhage from the native kidneys in the setting of a recent KT is extremely unusual and, to our knowledge, hitherto never described. Herein, we present the case of non-malignant SNKR in a human immunodeficiency virus (HIV)-positive deceased donor KT recipient that occurred 6 days after transplant. Informed consent was obtained from the individual participant included in the study.

Case report

A 65-year-old African American man with a history of end stage renal disease (ESRD) presumed to be from hypertension and HIV received a deceased donor KT. Prior to KT, he had been on PD for 8 years. His medical history included HIV, well-controlled on highly active antiretroviral therapy; anxiety, depression, chronic obstructive pulmonary disease, hyperlipidemia, hypertension, obstructive sleep apnea, and peripheral artery disease. His surgical history included multiple cardiac and lower extremity catheterizations with an iliac artery stent placement; multiple endoscopies for a gastrointestinal bleed, and a spontaneous pneumothorax requiring video-assisted thoracoscopic surgery. Pre-operatively, the patient's platelet count was within normal limits at 200,000/ μ L and he was anemic with a hemoglobin of 10.8 g/dL.

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He received a kidney allograft from a 33-year-old brain dead donor with a kidney donor profile index score of 22% who died from a motor vehicle accident. The terminal creatinine of the donor was 1.3 mg/dL. Donor serologies were negative with the exception of Epstein Barr virus. The cold ischemia time for this kidney was 6.33 h. The intraoperative course of the KT was complicated by an external iliac artery dissection for which arterial reconstruction with an interposition 8 mm Dacron graft was necessary. The aortic patch of the renal allograft was anastomosed to the interposition graft. Overall, the warm ischemia time was 135 min. Induction immunosuppression consisted of methylprednisolone 250 mg and antithymocyte globulin, rabbit (rATG) 2 mg/kg. An additional 2 mg/kg of rATG was given on POD 2 and 3 for a total of 6 mg/kg. He was placed on a steroid taper to prednisone 5 mg daily, mycophenolate mofetil 1000 mg twice daily, and tacrolimus beginning POD 3 to a goal trough of 8–12 ng/mL.

A transplant ultrasound (US) on POD 0 demonstrated a small complex fluid collection ($5.3 \times 4.1 \times 4.6$ cm) lateral to the KT suggestive of a hematoma; however, the transplanted kidney appeared normal. By POD 3, the patient was tolerating a diet, however hypertension management became challenging. His blood pressure was persistently elevated with systolic blood pressures of 180s mmHg despite treatment with metoprolol and amlodipine. The patient experienced delayed graft function (DGF) and received HD on POD 4 for volume overload. His systolic blood pressures remained

> 160 mmHg despite titration of antihypertensive therapy in the intensive care unit (ICU). He developed thrombocytopenia with a platelet count of $82,000/\mu\text{L}$. At this time, he was also initiated on aspirin 81 mg daily for patency maintenance of the Dacron graft. On POD 5, he received two units of packed red blood cells (PRBCs) for a hemoglobin of 6.5 g/dL. A transplant US redemonstrated the postoperative hematoma, which was stable in size, and good flow to the kidney.

On POD 6, the patient complained of right upper quadrant flank pain, which was not relieved by hydromorphone. He had become more distended and lethargic with changes in mentation, and his systolic blood pressure dropped from the 150s mmHg to the 90s mmHg. Overnight, his hemoglobin decreased from 8.1 to 6.3 g/dL despite receiving blood the day prior, and his platelet count had dropped to $56,000/\mu\text{L}$. Aspirin was held, and a computerized tomography (CT) scan of the abdomen was performed, which demonstrated a large complex fluid collection along the lateral aspect of the right native kidney (Fig. 1a, b), causing a mass effect and anterior displacement. Compared to a CT scan 17 months prior to transplant (Fig. 1c), the right native kidney appeared grossly enlarged and measured approximately 2006 cm^3 on volumetric assessment (318 cm^3 17 months prior to the event), accounting for a 6.3-fold increase in volume. Therefore, it is reasonable to infer the increase in size to be a perinephric subcapsular hematoma. The transplant kidney appeared unchanged from previous imaging with the exception of a disrupted ureteral anastomosis.

Fig. 1 Coronal (a) and cross-sectional (b) imaging demonstrated a large complex fluid collection along the lateral aspect of the right native kidney (red arrow), causing mass effect on the right native kidney with anterior displacement. Additionally, there is complex fluid in the posterior pararenal space (asterisk) and anterior pararenal space (asterisk) on the right. Compared to an abdominal CT from 17 months prior to the event (c), the right kidney is significantly larger in size and much more heterogeneous

Given these findings, the patient underwent emergent surgery via a midline incision. Intraoperatively, a large right perinephric hematoma was discovered. Consequently, the right kidney was removed. Additionally, the ureteral anastomosis was revised. The right native kidney weighed 1670 g (dimensions: 22 × 15 × 9 cm) and it demonstrated cystic changes, chronic inflammation, fibrosis, and extensive vascular tubular and glomerular sclerosis (Fig. 2). There was a rupture of the lower pole and extensive hemorrhage extending into the upper pole and mid kidney. Cysts were noted to contain serosanguineous fluid and lithiasis. The corticomedullary junction was indistinct and no normal kidney parenchyma was seen.

His postoperative course was complicated by refractory hypertension requiring a prolonged ICU stay for a nicardipine infusion. In addition, he required additional transfusions of four PRBCs and two units of fresh frozen plasma over the remainder of his admission. His hemoglobin and platelet count stabilized to 7.9 g/dL and 224,000/μL, respectively, and the patient was eventually discharged to a skilled nursing facility on POD 21/POD 15. At the time of discharge, he remained dependent on intermittent HD however urine production had increased to 1–2 L per day. Eventually, he was weaned off HD by POD 30 and his creatinine and glomerular filtration rate on POD 80 were 1.6 mg/dL and 53 mL/min/1.73 m², respectively.

Discussion

The mechanism of SNKR in the non-KT patient remains unknown. Speculation supports that rupture could be secondary to increased pressure in the renal venous system from tumor mass effect or an embolus. Other theories implicate renal hemangiomas and other kidney tumors or nephrogenic hypertension as culprits [4]. In ESRD patients, acquired renal cystic disease (ARCD) has been correlated with SNKR [3]. Up to 95% patients with prolonged courses of HD may develop ARCD, which is a progressive disease that has been shown to lead to renal cell neoplasm in 5–25%

of patients, further increasing the risk of cyst hemorrhage [3]. SNKR has also been observed in a patient treated with PD for Good Pasture Syndrome [1]. Although inferential, it does not appear that PD places patients at increased risk of SNKR, but rather PD appears to be an underlying disease state and risk factor [1].

Although SNKR has previously been reported in the KT patient, to our knowledge, this is the first report of a SNKR in the perioperative setting of a KT recipient from a non-neoplastic etiology. In a similar experience to the case presented herein, Garcia-Covarrubias et al. report SNKR on POD 4 in a 44-year-old African American due to an angiomyolipoma [9]. However, by contrast, the SNKR in our patient is not related to an underlying angiomyolipoma but rather a true spontaneous parenchymal rupture.

The diagnosis of SNKR may be challenging in the perioperative setting of a KT recipient. Anemia is a common complication post-KT, especially if the kidney suffers from DGF. A sudden drop in hemoglobin may be suggestive of surgery-related blood loss, large fluid shifts, and volume overload, which can be further muddled in the setting of DGF, immunosuppression, perioperative inflammation, and pain [10–12]. Given any or a combination of any of these potential sources, eliciting the cause of post-KT anemia can be challenging. Refractory hypertension may have also contributed to the development of SNKR [4]. This patient had numerous risk factors for post-operative refractory hypertension, including pre-transplant hypertension, prolonged warm ischemia time, delayed graft function, and immunosuppressive medications [13]. Once active large-volume hemorrhage is excluded, the focus is on the transplanted kidney to eliminate surgical bleeding as the source. In the case of this patient, the hemoglobin dropped significantly on POD6 with an inappropriate response to blood transfusions. Thus, after a stable US excluded the transplanted kidney as the bleeding source, a CT was obtained, obviating the decision to take him back to the OR for a native nephrectomy. Intraoperatively, because there was no active bleeding, consideration was given to not performing the nephrectomy given the risk of worsening anemia, surgical blood loss,

Fig. 2 The right native kidney (a) weighed 1670 g and measured 22 × 15 × 9 cm. Histologic assessment (b) demonstrated cystic changes, chronic inflammation, fibrosis, and extensive vascular tubular and glomerular sclerosis

postoperative hypotension and the transfusion-related complications. However, ultimately the decision was made to remove the kidney, given the high possibility of malignancy as a potential source of the SNKR [14].

Herein, we present an unusual presentation of a non-malignant SNKR in the immediate post-KT setting. Because the literature on this topic is scant, we advocate for prompt surgical exploration to exclude malignancy as a possible source of the SNKR.

Compliance with ethical standards

Conflict of interest The authors have declared that no conflict of interest exists.

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