

Acute Page Kidney Phenomenon Secondary to Lymphocele Compression in Renal Allograft Recipient: A Case Report

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Abstract

Acute renal failure after kidney transplantation frequently occurs and often represents a real challenge for the clinician. Acute rejection is the first diagnostic suspect but many other conditions, mimicking rejection, should be considered. A prompt diagnosis is recommended to avoid treatment delay or mistreatments. Page kidney phenomenon is a well-known condition in which an extrinsically compressed kidney results in hypertension and loss of renal function. It should be always considered among the acute complications of renal transplantation. Prompt recognition and early intervention are essential to restore renal function before irreversible damage occurs. We describe a Page kidney phenomenon as an acute complication due to a lymphocele compression. Proximal TEC-CD44 influenced the renal inflammatory milieu and TEC-CD44v3 associated with expression of anti-inflammatory molecules.

Keywords: Acute rejection; Lymphocele; Page phenomenon
Introduction

Acute renal failure after kidney transplant is a frequent complication. Most commonly, acute rejection is the first diagnostic hypothesis taken into consideration, but many other conditions, mimicking rejection, should be examined. We describe a Page kidney phenomenon as an acute complication following a kidney transplant, secondary to lymphocele compression.

Case Report

We present the case of a 61-year-old man who underwent two deceased heart beating donor kidney transplantations, with end-stage renal failure secondary to focal segmental glomerulosclerosis. A first transplant was performed in 2003 but the kidney was immediately removed due to an acute renal vein thrombosis. In March 2014, after the second transplantation, the graft functioned well, with a serum creatinine of 76.92 $\mu\text{mol/l}$ one week after surgery.

Immunosuppressive treatment consisted of a “double” induction therapy with thymoglobulin, immunoglobulin, basiliximab and steroids, in the suspect of hyperimmunisation secondary to the previous transplant. Maintenance therapy was tacrolimus, mycophenolato and steroids. Bladder catheter and ureteral stent were removed on day VII postoperatively without complications. The following day we also removed the surgical perirenal drain tube and performed Doppler ultrasound (DUS), according to our Hospital internal protocol for postoperative exams. DUS revealed a 7.5 by 1.5 cm fluid collection suggestive for lymphocele, without signs of urinary tract obstruction.

Two weeks after transplantation serum creatinine level raised to 150.28 $\mu\text{mol/l}$ therefore an urgent DUS was performed revealing increase of the renal resistive index to 0.8-0.86 without signs of urinary tract obstruction. Both blood and urine based laboratory tests were negative for infection. Acute rejection was suspected and high-dose steroid therapy was administered to the patient. Despite renal biopsy nowadays represents a necessary element to confirm transplanted kidney acute rejection, we opted not to perform it because of the known large fluid collection surrounding the kidney which could be potentially associated with an increased risk of uncontrollable bleeding. Only a slight reduction of serum creatinine was obtained 3 days after treatment. However, serum creatinine remained stable and the patient was discharged on day XII postoperatively. Three days later the patient

was readmitted to our hospital for a recurrent increase in the serum creatinine that reached up to 206.86 $\mu\text{mol/l}$. An urgent Doppler ultrasonography was performed again and revealed increased kidney echogenicity with reduced cortico-medullary differentiation. Organ volume was normal and no signs of urinary tract obstruction were identified. The already known lymphocele was only slightly increased in size. A new episode of acute rejection was suspected, but, bearing in mind the recent steroid therapy, different diagnoses were considered. In particular, an abdominal computerized tomography (CT)-scan without contrast was carried out in order to explore the morphology of perirenal fluid collection and to highlight its relationship with the graft in the suspect of a Page kidney phenomenon. The CT scan showed a fluid collection surrounding the kidney, which was compressed against the psoas muscle and medially displaced (Figure 1). The lymphocele was percutaneously drained and 750 milliliters of clear fluid were promptly evacuated. A fluid sample was sent for biochemical and microbiological analysis and lymphocele diagnosis was thus confirmed. Five hundred more millilitres were drained during the following 24 hours. The following day the serum creatinine was 163.54 $\mu\text{mol/l}$. Lymph drainage continued, gradually reduced and drain tube was removed one month later. Serum creatinine level remained stable during the following days and the patient was therefore discharged (Figure 2). A three months follow-up revealed normal serum creatinine levels (106.08 $\mu\text{mol/l}$); an ultrasound of the transplanted kidney was also performed and confirmed the absence of peri-renal fluid (Figure 3). To date, considering such positive follow up findings, no further treatment was needed or considered.

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Received: January 13, 2015; **Accepted:** March 28, 2015; **Published:** April 05, 2015

Citation: Letto G, Amico F, Soldini G, Chiappa C, Franchin M, et al. (2015) Acute Page Kidney Phenomenon Secondary to Lymphocele Compression in Renal Allograft Recipient: A Case Report. J Nephrol Ther 5: 199. doi:10.4172/2161-0959.1000199

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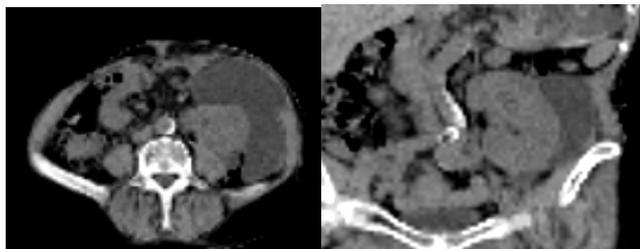


Figure 1: The Kidneys Compressed against the Psoas muscle (axial view) and Medially Displaced (frontal view) by a Large Fluid Collection.

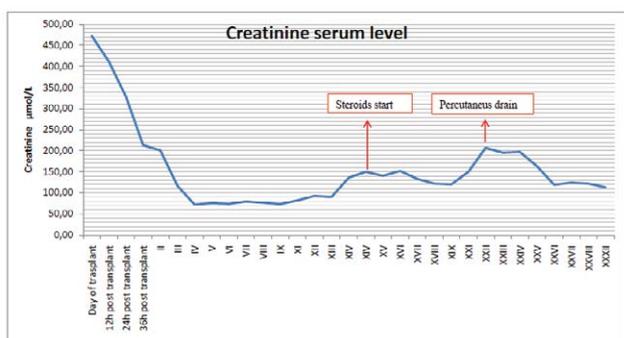


Figure 2: Creatinine Serum Level through Post-Transplant Period.

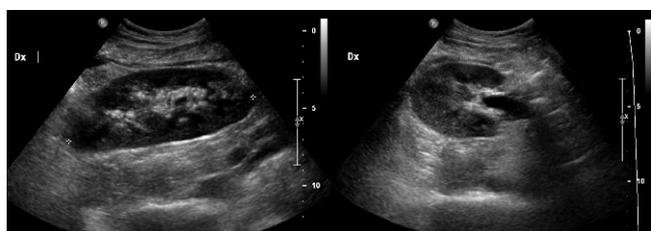


Figure 3: Follow-up Ultrasound of the Transplanted Kidney showed the absence of Peri-Renal Fluid.

Discussion

The Page phenomenon was described in 1939 when Page reported arterial hypertension in animals in which perinephritis was induced by wrapping their kidneys in Cellophane. Cellophane induced an

inflammatory response which resulted in a fibrocollagenous hull 3-to4 mm thick and this held under tension the renal parenchyma [1]. Compressive perinephritis causes renal ischemia that is responsible for the subsequent functional impairment and the hyperreninemic hypertension [2]. To date, only three cases of Page phenomenon occurring after kidney transplantation have been reported: the first caused by a haematoma [3], the second occurred after the marsupialization of a lymphocele and the compression related to the subsequent constrictive pericapsular fibrosis [4] (in these two cases, renal function deterioration described as pseudorejection was the main finding); the last case was the only one where arterial hypertension was reported and led the suspects towards the hypothesis of Page phenomenon [5]. Page phenomenon was previously described as a complication after kidney transplant, but never as an acute one. Incidence of Page phenomenon might be higher than expected and often undiagnosed: patients often develop hypertension and electrolyte abnormalities after kidney transplant but these are usually considered consequences of tubular impairment related to the ischemia or the pharmacological damage. Furthermore, except for obvious cases, Page phenomenon might be responsible for mild chronic renal impairment after transplantation in numerous patients. Prompt recognition and treatment might improve graft function and long-term graft survival. More panoramic images highlighting the relations between the transplanted kidney and the adjacent structures could be critical for this purpose.

Acknowledgment

We thank Emily Amico, Marta Zanzi, Emilia Ietto and Melissa Borroni for their kind contribution to the realization of this manuscript.

Conflict of interest

All authors declare no conflict of interest.

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