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Post-biopsy renal allograft compartment syndrome: Addressing the problem, illustrated with a case report

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ABSTRACT

INTRODUCTION: Renal allograft compartment syndrome (RACS) has recently been coined to describe early allograft dysfunction secondary to raised pressure in the retroperitoneal space. This may be caused by direct compression of the renal vessels or by a diffuse renal parenchymal compression. Herein, we report a renal allograft compartment syndrome secondary to a needle core transplant biopsy and discuss the management strategies in line with an updated literature review.
PRESENTATION OF CASE: A retrospective case-note review was carried out where a 45-year-old male had a transplant renal biopsy at 4-weeks after transplant for raising creatinine. Following biopsy patient developed abdominal discomfort and had haematuria.
DISCUSSION: Doppler ultrasound scanning of graft demonstrated good perfusion but a small haematoma ($2 \times 2 \times 2$ cm) in the upper pole of the kidney at the site of the biopsy. Patient was thereafter assessed conservatively with serial ultrasound monitoring. After 24 h, significant deterioration of graft function was observed. The third scan, demonstrated reversed flow in diastole in the upper pole of the kidney with a resistive index of 1.0 in the main renal vessel. With the above findings the kidney transplant was explored immediately and the transplant released from a 300 ml of liquefied haematoma, which was under considerable pressure. In the next 24-h, the patient showed an immediate return of graft function.
CONCLUSION: We recommend sequential ultrasound Doppler scanning as an invaluable tool to help identify early RACS. The surgical exploration and adequate heamostasis with surgical glue should be sought out in all RACS.

1. Introduction

The term 'renal allograft compartment syndrome' (RACS) has recently been coined to describe early allograft dysfunction secondary to raised pressure in the retroperitoneal space.[1] This may be caused by direct compression or kinking, of the renal vessels or by a more diffuse renal parenchymal compression. These mechanisms may result in decreased renal plasma flow and glomerular filtration rate causing subsequent graft ischemia.[2] In this report, we describe a case of renal allograft compartment syndrome secondary to a needle core transplant biopsy.

The incidence of RACS is about 2%, considering the impact on graft dysfunction and/or loss of graft, thus affecting both the patient themselves and the resources of a transplant program.[3] This is more often seen in cadaveric transplant and in male recipients.[3]

There are number of causes leading to RACS or acute page kidney (APK); first described in the 1930.[5,6] This can be caused by spontaneous or iatrogenic bleeding as well.[10,11] Early presentation of RACS is not easy to suspect or to identify before the irreversible damage happens to the graft. In post-biopsy scenario it is very difficult to anticipate RACS, as there may be other confounding factors like suspicion of acute rejection, delayed graft function or acute tubular necrosis prior to the biopsy especially in a new graft.

We report a case with presentation of acute page kidney or RACS and highlight the current literature with a view to address the importance of early diagnosis and management strategies.

2. Presentation of case

The patient was a highly sensitised 45-year old male, who had undergone two previous transplants. His 38-year old sister offered to donate a kidney but the pre-operative immunological cross-match demonstrated significant levels of donor specific IgG antibody against class I and II antigens. The potential transplant recipient received Rituximab and after desensitisation with five sessions of plasmapheresis, the immunological cross-match showed low levels of donor specific antibody and the live donor kidney was transplanted successfully. Two weeks post-operatively the serum creatinine fell to 140 μmol/l. Patient was continued on Tacrolimus, Mycophenolate Mofetil and prednisolone for immunosuppression.

At four weeks post-transplant, the creatinine rose to a level of 189 μmol/l and a needle core renal transplant biopsy was performed under ultrasound control. The biopsy demonstrated acute

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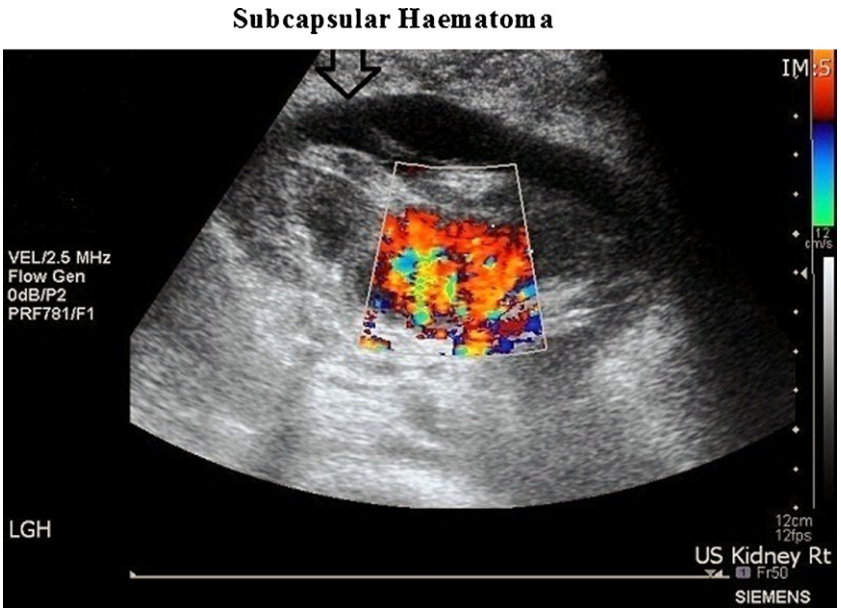

Fig. 1. Doppler ultrasonography of the renal allograft showing a haematoma; and a normal blood flow in the hilum.

tubular injury, possibly due to Tacrolimus nephrotoxicity. After this biopsy, the patient developed abdominal discomfort and had an episode of haematuria. Doppler ultrasound scanning of the kidney transplant demonstrated good perfusion of the transplant but a small haematoma (2 cm × 2 cm × 2 cm, Fig. 1) was noted in relation to the upper pole of the kidney at the site of the biopsy. The pelvicalyceal system and ureter were normal. A 3-way bladder catheter was passed but over the next 24 h, little urine was produced. At this point, the serum creatinine was 519 μmol/l. The kidney transplant was scanned by Doppler ultrasound sequentially by a member of the transplant surgical team over a 24-h period post-biopsy. The first two scans showed good global perfusion of the kidney with a biphasic arterial Doppler waveform. However, the third scan, performed 24-h post-biopsy, demonstrated elevated resistive index (RI – 1.0) in the main renal vessel. Perfusion in the lower pole of the kidney was normal. In view of the elevated resistive index with a deterioration of graft function, the kidney transplant was explored as an emergency.

Opening the muscle layer over the transplant released approximately 300 ml of liquefied haematoma, which was under considerable pressure. Intra-operative Doppler ultrasound scanning demonstrated good global perfusion of the kidney and an immediate return of diastolic blood flow to the upper pole with a resistive index of 0.5. The renal artery, vein and ureter were found to be normal. We have used surgical glue (TISSEL, Baxter Healthcare Corporation, CA, USA) to achieve haemostasis, especially over the raw surface of the kidney following the capsular stripping secondary to the haematoma.

There was considerable oedema of the tissue surrounding the kidney and the wound was closed with Redivac drain deep tension sutures (Unomedical, Birkerod, Denmark). In the next 24-h, the patient had a good diuresis, passing 2500 ml of urine and there was an immediate return of graft function, with a creatinine fall to 218 μmol/l. The transplant is working well six-months post-operatively.

3. Discussion

In RACS, the common risk factors are a significant weight discrepancy between donor and recipient, a non-compliant extra peritoneal compartment and paediatric recipients.[2,3] It is also seen more often in transplants from deceased donors and male recipients.[7] The pathophysiology is similar to an abdominal compartment syndrome with decreased organ perfusion, which damages the renal parenchymal well before clinical signs are evident.[8,9]

The risk factors for the development of APK are still not established.[5,12] Hence the suspicion must be raised in a coagulapathic patient, uncontrolled hypertension, patient experiencing a acute pain over graft, alteration in blood pressure, oliguria, Doppler evidence of subcapsular haematoma, elevated resistive index or reversal of diastolic flow.[5,13,14] The post-biopsy RACS scenario is rare and it is imperative to keep a high suspicion in any unexplained immediate graft deterioration of graft function following biopsy.

The incidence of peri-nephric haematoma range from 0.7% to 30% in a biopsied renal allograft.[5,15] Although the incidence of APK is about 1% repeat Doppler ultrasonographic examination[5] is a key in nailing the diagnosis and in our centre are carried out by surgical registrars trained by radiologists. Ultrasonography of the graft sometimes can struggle to identify RACS earlier, because even a small haematoma can cause crucial raise in the extraperitoneal pressure ending up in RACS.

The importance of ultrasonography has been highlighted by Chung et al.,[5] but more importantly serial ultrasound examinations by a trained surgical registrar add to this invaluable tool in monitoring and management of renal transplants. Similarly a non enhanced CT scan also demonstrate a subcapsular haematoma compressing the allograft but lacks of Doppler and serial investigation.

Treatment strategies described range from mere observation to medical followed by surgical management. Review of literature and success of treatment in our case clearly favour prompt surgical decompression, evacuation of haematoma and achievement of haemostasis. The use of mesh to achieve the haemostasis has been described to reduce the incidence of secondary RACS. We describe the use of surgical glue (TISSELL, Baxter Healthcare Corporation, CA, USA) to achieve haemostasis which can be easily applied and does not hamper with further follow up Ultrasound; where mesh can lead to an elevated resistive index[16] causing compression of renal parenchyma or main vessels.[17] On evaluation of the subcapsular haematoma, constant oozing is prompt from the renal parenchyma due to capsular stripping and tissue glue plays a significant in achieving adequate haemostasis.

4. Conclusion

In our patient, liquefied haematoma dispersed in to tissue planes which caused the raise in extraperitoneal compartment pressure followed by RACS. Hence we strongly recommend that an early clinical suspicion should be urgently followed by serial ultrasonography by a trained sonologist, the surgical registrar in our case. The ultimate diagnosis not solely on ultrasound findings, as it will represent a rather late diagnosis of an acute complication. The surgical exploration and adequate heamostasis with surgical glue should be sought out in all RACS.

Conflict of interest

None.

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None.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Authors' contribution

UMT and AB were involved in drafting the manuscript; IHM involved in retrieval of Doppler image and editing; MLN revised the manuscript; all authors have read and approved the final manuscript.

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