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CASE REPORT

Salvaging kidneys with renal allograft compartment syndrome

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Summary

Renal allograft compartment syndrome is an under recognized cause of early allograft dysfunction which can be reversed by early intervention. It occurs early after renal transplantation where closure of the anterior abdominal wall seems to compress the transplant in the limited retroperitoneal space. The literature about this syndrome in renal transplantation is sparse. Our report describes the diagnostic criteria and the management of two renal transplant recipients with this syndrome. Its diagnosis depends upon duplex vascular scan findings of reversed or absent diastolic flow in the renal vasculature in the absence of any perigraft collection or severe acute tubular necrosis. In our hands emergency laparotomy, decompression of the transplant and closure with interposition mesh salvaged these kidneys.

Introduction

Early allograft dysfunction (EAD) after renal transplantation remains a challenging problem. Although some causes of EAD, like acute rejection, are readily diagnosed others like renal allograft compartment syndrome (RACS) [1] may be difficult to diagnose. Furthermore, if RACS is unrecognized the graft may be lost. Renal allograft compartment syndrome has been defined as EAD secondary to intra-abdominal hypertension leading to transplant ischaemia [1,2]. It occurs early after renal transplantation where closure of anterior abdominal wall seems to compress the transplant in the limited retroperitoneal space. Both the pathogenesis and treatment of RACS in renal transplantation are poorly understood [2]. Hence, our report describes the diagnostic features and management of RACS in two recipients.

Recipient 1

A 55-year-old male with end stage renal disease (ESRD) from adult polycystic kidney disease (ADPKD) received a live donor renal transplant. The graft had a single renal artery and vein; standard anastomoses were performed; and, the on table urine output was brisk. A routine renal perfusion scan using diethylenetriaminepentaacetic acid (DTPA) on the first postoperative day showed prompt uptake, extraction and excretion. However, during the next 36 h, the urine output decreased. A second DTPA scan showed decreased perfusion with no extraction and a duplex vascular scan revealed a poorly perfused kidney with reversed diastolic flow in the renal artery and absent flow in the renal vein. He underwent emergency laparotomy: the kidney was compressed by the wound; it was cyanotic, but the colour improved markedly once the

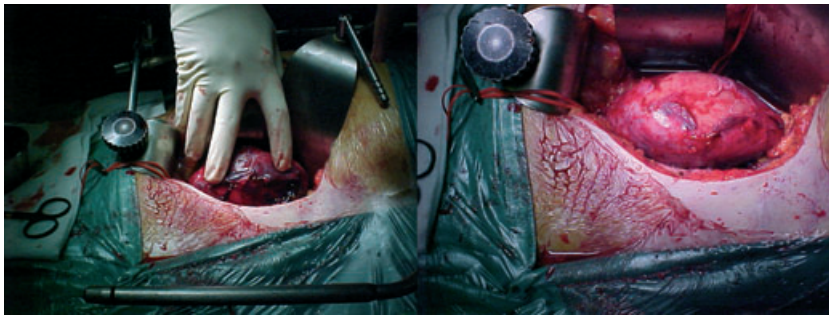

Figure 1 Intra-operative photographs. Left: small dusky graft just after opening the abdomen. Right: return of normal colour and turgidity a few minutes later.

wound was open and the compression released (Fig. 1). There was no perigraft haematoma and both renal vessels were patent.

The abdomen was closed with polytetrafluoroethylene (PTFE, Gore Dual Mesh Plus Biomaterial, Arizona) mesh which allowed the retroperitoneal space to increase. His urine output increased promptly over the next day and the third DTPA scan showed improved perfusion, extraction and excretion. The peroperative biopsy showed minor changes without rejection. He was discharged on day 14 with a serum creatinine of 160 μmol/l. His serum creatinine was normal (101 μmol/l) when last reviewed at 3 years.

Recipient 2

A 61-year-old woman with ESRD from ADPKD received a second live donor renal transplant. She had factor V Leiden mutation, anti-cardiolipin antibodies and anti-HLA antibodies. The kidney had two renal arteries, which were reconstructed by an end to side anastomosis, and a single renal vein. The transplant functioned immediately, but on the second day she suddenly became anuric. A duplex vascular scan showed a patent renal artery with absent diastolic flow, a patent renal vein and a high resistive index in the intrarenal arteries. Emergency laparotomy was therefore performed: the transplant was compressed by the wound; the transplant vessels were patent; there were two small subcapsular haematomata, but no perigraft haematoma. The wound was closed with PTFE mesh after removing the small subcapsular haematomata. The peroperative biopsy showed normal architecture without rejection. Her urine output steadily increased over next 3 days and she was discharged on postoperative day 9 with serum creatinine of 122 μmol/l.

Discussion

These two recipients demonstrate that early allograft dysfunction can arise from external compression of the transplant without the presence of an extrinsic mass lesion, such as a haematoma or fluid collection. Compression led to reduced blood flow and then decreased urine output. It was relieved promptly by surgical decompression, leading to restoration of renal blood flow and graft recovery in these recipients. The key to diagnosis was a high index of clinical suspicion combined with duplex vascular scan findings of absent or reversed diastolic flow in the renal artery in the absence of extrinsic space occupying lesions or severe ATN [3].

The pathogenesis of RACS is not completely understood. It is possible that external pressure upon the transplant kidney increases the intracapsular pressure reducing its blood flow and function. Initially the intrarenal vasculature is at risk of thrombosis whereas the extrarenal artery and vein are patent: the reversed diastolic component of the renal artery waveform detected on scanning occurs because of high resistance within the kidney. Evidence for this mechanism is derived from the work of Herrler *et al.* [4] using a murine renal transplant model of ischaemia reperfusion injury (IRI). They showed that IRI leads to progressive increase in intracapsular pressures, reduction in glomerular blood flow and tubular function. With prolonged ischaemia times tubular function was irreversibly lost when measured by ^{99m}technetium mercaptoacetyltriglycine (Tc MAG-3) excretion scans. Interestingly, polar capsulotomy alleviated the long-term ischaemic damage in this model. It is possible that external compression of the kidney seen in RACS works in similar way, but there are no human mechanistic studies reported to date.

Renal allograft compression syndrome may be considered a variant of abdominal compartment syndrome [5]. In RACS the factors that can lead to increased external pressure are kidney-recipient size mismatch, a shallow pelvis leading to tight fascial closures and intravenous fluid resuscitation. As a result of its mass effect, renal transplantation may increase abdominal pressure as observed by Ferris *et al.* [6]. Compartment pressure may

also be increased by ascites and bowel oedema related to intravenous fluids and preoperative hypoalbuminaemia [7]. The diagnosis of RACS may become masked by the coexistence of severe ATN.

Renal allograft compression syndrome is an under recognized surgical adverse event with few reports [1,2,7,8]. Ball *et al.* [1] reported a series of 11 cases in 458 adult recipients. They were all diagnosed on the basis of duplex vascular scans, intra-operative findings and the response to decompression. In our view effective treatment for RACS is emergency laparotomy, decompression and mesh closure of abdominal incision. Other measures that may be used are intraperitonealization of the graft and a relaxing incision in the external and internal oblique fascia. Beasley *et al.* [9] have used a polypropylene mesh for primary closure of abdomen in all seventeen renal transplant recipients where fascial closure required excessive force and led to diminished graft arterial flow. With this approach four recipients were re-explored with the loss of one graft and the mesh did not confer an increased risk of wound infection.

Conclusions

Renal allograft compression syndrome is a reversible cause of early graft dysfunction if the diagnosis is made promptly. The diagnosis is made by clinical suspicion combined with duplex vascular scan findings of reversed or absent diastolic flow in the renal artery in absence of any perigraft collection. Emergency laparotomy, decompression and mesh closure provide definitive treatment. A prospective longitudinal study correlating pressures within the abdomen and iliac fossa with the findings of duplex vascular scanning is now required.

Authorship

MKH: wrote the article. PRT: critical review of manuscript. DBH: duplex scanning and interpretation, review of manuscript. ADH: conceived the idea, designed study, critical review of manuscript.

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Ethics

The two recipients have consented to the use of their de-identified data for the purpose of research.

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