Ultrasonography diagnosis of renal arterial thrombosis: an important cause of renal allograft loss in children

An 11-year-old girl had end-stage renal failure secondary to primary hyperoxaluria type 1. She underwent cadaveric renal transplantation and had tenderness over the renal graft since the fifth postoperative day. Doppler ultrasonography performed on day 1 (Fig 1) and day 6 showed satisfactory blood flow over the renal graft, but there was a small amount of perinephric haematoma. Renal biopsy performed on day 7 showed acute tubular necrosis, but no features of acute rejection. The patient developed anuria on day 10 despite intravenous steroid therapy. Urgent Doppler ultrasonography showed an absence of arterial and venous blood flow over the renal graft (Fig 2). Normal renal artery and vein could not be identified.

FIG 1. Longitudinal colour Doppler ultrasound image obtained 1 day after renal transplant showing satisfactory blood flow over the renal graft down to the levels of the segmental renal arteries (arrows) and arcuate renal arteries (arrowheads).

FIG 2. Longitudinal power Doppler ultrasonography image performed 10 days after renal transplant showing absence of arterial or venous blood flow over the renal graft.

FIG 3. Transverse colour Doppler ultrasonography image performed 10 days after renal transplant showing absence of the normal renal artery and vein at the expected location of the graft renal hilum (arrows). No intrarenal blood flow could be detected.

FIG 4. Coronal reformatted computed tomographic image of the lower abdomen and pelvis after intravenous injection of contrast medium showing complete loss of perfusion over the graft kidney (black arrowheads). Satisfactory contrast enhancement was evident along the inferior vena cava (white arrowhead), bilateral internal arteries, and external iliac arteries (double white arrows).
at the graft renal hilum (Fig 3). Contrast-enhanced computed tomography performed on the same day showed similar features (Fig 4). The findings were compatible with acute graft renal artery thrombosis. Emergency laparotomy showed non-viable necrotic renal graft, and graft nephrectomy was performed.

Discussion
Graft renal artery thrombosis is uncommon, occurring in only 1% of all kidney transplant recipients. The condition is an important cause of renal allograft loss in children. Risk factors specific to paediatric patients include the young age of the recipient and donor, and intravascular volume depletion secondary to high urine production of the native kidneys. The clinical presentation includes sudden anuria and graft pain. A high index of suspicion is required to make this diagnosis because acute tubular necrosis and acute severe rejection may have a similar clinical presentation. Urgent duplex ultrasonography is the investigation of choice. Absence of blood flow in the renal parenchyma at Doppler examination, with appropriate settings of colour gain and pulse repetition frequency, is characteristic. This finding, however, may also be observed in other conditions such as hyperacute rejection and renal vein thrombosis. In situations other than arterial thrombosis, the main renal artery is patent and may exhibit reversal of diastolic flow on spectral Doppler examination. Surgical exploration is indicated once graft renal artery thrombosis is confirmed by Doppler ultrasonography. However, the prognosis is poor and loss of the allografted kidney is the usual outcome.

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References