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Expanding the living renal donor pool by using a horseshoe kidney

Sir,

In India, deceased kidney transplantation accounts for <2% of all transplants.[1] Shortage of donors has led to kidneys with congenital anatomical variations being used for renal transplantation. The most common anatomical variation of kidney is the horseshoe kidney.

A 49-year-old diabetic and hypertensive man on hemodialysis were referred to us for a renal transplantation. The only donor was his 44-year-old wife with a horseshoe kidney with double renal arteries and veins. The isthmus was supplied by the left lower polar artery arising from left common iliac artery and was draining through both side lower polar veins. A diethylene triamine penta-acetic acid scan showed a glomerular filtration rate of 89 ml/min and non-obstructed normal functioning kidneys.

Surgical approach was via a left flank incision. The vascular pedicle was dissected, and the lower polar artery and vein were isolated. The isthmus was transacted, and suture ligated [Figure 1]. After perfusion, the kidney was transplanted, with the renal vein anastomosed to the external iliac vein and the main renal artery to the internal iliac artery. The lower renal artery was anastomosed to the right deep inferior epigastric artery. There was no intraoperative or postoperative complication. Patient was discharged on the 7th day after surgery with a creatinine level of 1.0 mg/dl.

Surgery of horseshoe kidney is generally complex as it is frequently associated with vascular and ureteral abnormalities. Owing to the complex vascular and urinary collection system abnormalities, 17 of 80 kidneys in deceased donors could not be transplanted after division.[2] Only a third of all horseshoe kidneys contain a single renal artery per side.[3] During the separation of a fused renal isthmus, the urinary collection system may be injured, and a urinary fistula may develop.[2] All of these factors make separation of horseshoe kidney difficult in living donors.

In literature, there are only a few case reports of using horseshoe kidney in living donors.[4,5] In living donors, the site to divide the kidney should be made only after the vascular and collecting system anatomy have been meticulously evaluated preoperatively using CT angiography.[5]

The routine use of CT angiograms helps in the delineation of vascular and urological anatomy in the evaluation of donors. Horseshoe kidney can be successfully used as donor kidney for live renal transplantation with good results if vascular and collecting system anatomy is amenable to transplantation. This may expand the living donor pool.

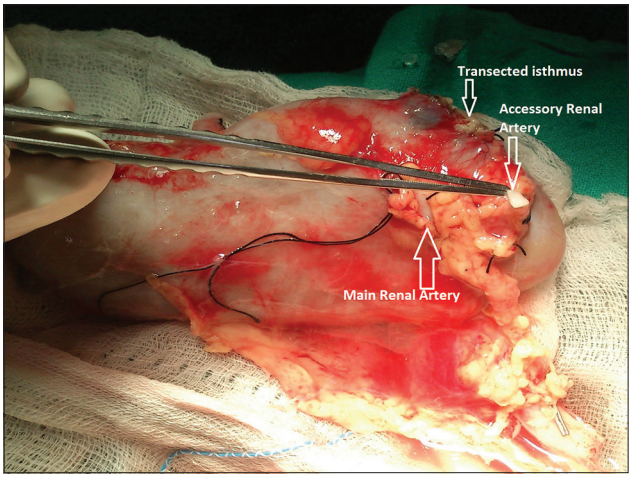

Figure 1: Donor nephrectomy specimen showing transected isthmus and two renal arteries

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Tubercular constrictive pericarditis after renal transplantation

Sir,

Constrictive pericarditis (CP) is reported in 2.4% of renal recipients.[1] A 40-year-old teacher, with presumed chronic interstitial nephritis underwent transplantation in 2004 (donor-6 antigen matched brother). Immunosuppression was mycophenolate mofetil (MMF), cyclosporine and steroids till 2012; MMF and steroids later. There were no rejections; serum creatinine was 0.8 mg/dl.

Two months after a febrile illness she noticed exhaustion, pedal edema and 6 kg weight gain. She had no pallor, was afebrile, BP was 100/70 mmHg. Heart sounds were muffled, jugular venous pressure elevated and there was hepatomegaly. Investigations showed creatinine 1.13 mg/dl, trace proteinuria without active sediments, hypoalbuminemia (2.1 g/dl). Clinical suspicion of CP was confirmed by cardiomegaly on X-ray, pericardial thickening on echocardiogram, 6 mm effusion on computed tomography scan with mediastinal lymphadenopathy.

Pericardiectomy tissue was TB – polymerase chain reaction (PCR) positive; histology showed caseating necrosis [Figure 1]. She received anti-tubercular treatment (ATT) (isoniazid, rifampicin, ethambutol, pyrazinamide); MMF was changed to azathioprine. After 3 months, she was asymptomatic with normal graft and liver functions.

Chronic CP, a sequelae of healing pericarditis, obliterates pericardial cavity, interferes with ventricular filling and cardiac output. TB remains common etiology, especially in developing countries.[2] Clinical features are weakness, cachexia; edema and ascites. Examination findings are feeble apical pulse, muffled heart sounds, distended neck veins, hepatomegaly and jaundice. Electrocardiography displays low voltage complexes, flattened or inverted T-waves and atrial fibrillation. X-ray demonstrates cardiomegaly and pericardial calcification. Echocardiogram shows pericardial thickening and effusion, distended inferior vena cava and hepatic veins, left shift of the ventricular septum during inspiration. Pericardiectomy relieves constriction; operative mortality is 5–10%; histopathology may reveal the etiology.

In patients with chronic kidney disease, uremia or effusion due to under-dialysis, hypoalbuminemia or volume overload contribute to pericarditis. TB remains a differential diagnosis. However, CP is rare.

Tuberculosis occurs in 10–13% of renal recipients.[3] CP is reported in about 2.4%.[1] Calcineurin inhibitors/sirolimus and oppurtunistic infections should be considered as etiologies.[4]

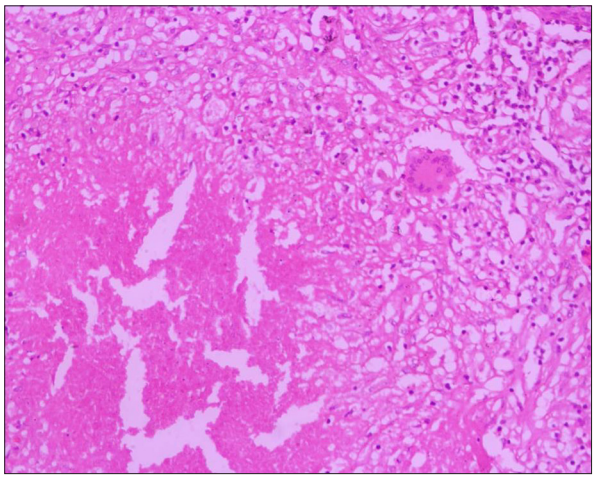

Figure 1: Pericardial tissue showing granuloma with central coagulative necrosis along with langhans' giant cell (x20 H and E stain)