Hyperacute vascular rejection is a rare serious event leading to graft loss. Not many reports describing its imaging findings are available in the literature due to its rapidity of onset. This review describes the Doppler imaging findings in hyperacute vascular rejection characterized by absent intrarenal arterial flow and graft edema. These findings were further corroborated with contrast enhanced ultrasound and CT angiography that demonstrated nil cortical perfusion. This condition necessitates immediate graft nephrectomy to prevent further sensitization and complications. Other vascular causes of graft dysfunction include acute tubular necrosis commonly associated with cadaveric donors (34%) and prolonged warm ischemia time. Increased intrarenal vascular resistive indices and reduced diastolic flow are the observed findings. Similar reduction in the diastolic flow and elevation of the intrarenal resistive indices are also noted in abdominal compartment syndrome a rare cause of acute graft dysfunction. These findings rapidly revert after surgical decompression. Renal vein thrombosis characterized by early rapid diastolic flow reversal in the renal arterial Doppler. It has reported prevalence of 0.1-4.2% and leads to graft loss. Early identification and institution of thrombolysis/thrombectomy may help in graft survival. Segmental infarction is being increasingly recognized as a cause of graft dysfunction.

**Conclusion:** Ultrasound Doppler is an important first-line modality for the evaluation of the renal transplants. It can accurately predict and characterize important causes of graft dysfunction, helping in early institution of therapy and prolonging graft survival.

**Clinical Brief:**

26 yrs male, case of chronic renal disease was posted for renal transplant (mother-son). Pre transplant work up patient had high serum creatinine 8.64. Serum electrolyte showed potassium of 4.8 and chloride of 101. His liver function test were normal with albumin 4.4. Patient was negative for viral markers.

He had functioning arteriovenous fistula. His 2D echo revealed grade 1 diastolic dysfunction.

Pre transplant colour Doppler of the iliac vessels showed normal caliber of the iliac vessels with no atherosclerotic changes.

Patient was dialyzed and prepared for the renal transplant. Post dialysis the serum creatinine was 3.53. Urine routine revealed presence of protein deposits. Urine routine cultures were negative.

His hemoglobin was 12.1. Patient was put on steroids. After obtaining the informed consent patient was taken for transplant.

Right iliac fossa incision was taken and vessels were harvested. Kidney received from mother to donor with single artery and vein. End to end anastomosis of the graft artery to the internal iliac artery performed. The vein anastomosed to the external iliac vein.

On reperfusion the kidney had turgid look. Intraoperative ultrasound was done using linear and endocavitary probes, GE, Milwaukee USA.

Doppler revealed high resistance flow in the renal artery with no diastolic component. Fig. 1. No intrarenal arterial signals was obtained. Pulsatile venous flow was noted in the insonated intralobar and segmental intrarenal veins.

Patient was started on heparin, 5000 units were given. No urine outflow was noted. The ureter was anastomosed to the bladder using Lish gregor technique.

Patient was shifted to the Intensive care unit in hemodynamically stable state with Foley catheter in situ. Patient had some urine output probably from the shrunken native kidney after Lask infusion.

Interval contrast enhanced ultrasound was done to look for the cortical perfusion. CEUS was performed using 5ml of levovist. There was no demonstration of any cortical perfusion or that of the renal artery. Fig 2. The above findings were confirmed with CT angiography in which no enhancement of the transplant renal parenchyma and that of the anastomosed artery were noted. Fig 2.

Patient underwent graft nephrectomy on the same day. Re exploration revealed dusky dark graft and no pulsation in the renal artery. The renal vein and the iliac vessels were patent. Fig 3.

Histopathology of the specimen showed glomerular and peritubular capillary thrombi. No polymorphonuclear margination seen, suggestive of vascular type of rejection. Fig 4.

**Discussion**

Hyperacute vascular rejection is rare event wherein the transplanted graft is rejected within minutes to hours. It is humorally mediated immune response by preexisting antibodies. The antibodies are the result of prior blood transfusions, pregnancies or prior transplantation. The antigen antibody complexes activate the complement system resulting in thrombosis of the capillaries and the graft vascularization.
The HLA and the endothelium associated donor antigens are the usual targets of the antibody mediated rejection.

Histopathologic changes in Hyperacute rejection include widespread capillary thrombosis and necrosis. (1)

Early recognition of the antibody mediated rejection and attempting to remove the allo antibodies by plasma pheresis, immunoadsorption and immunosuppressive agents may help in salvaging the graft in cases of Hyperacute rejection.

Doppler ultrasound remains an important non invasive modality for the evaluation of the renal grafts. Its ubiquitous presence makes it readily available for intraoperative and post operative bedside evaluation.

Assessment of the renal graft is also facilitated by its location in the right iliac fossa unlike retroperitoneally located native kidneys. The normal graft has hyperechoic renal sinus with medullary pyramids being echoluent. Small amount of perirenal fluid is common post operative appearance. Color Doppler allows for rapid assessment of the graft vascularity evaluating both the arteries and the renal vein.

The anterior and the posterior divisions of the renal arteries as well as the segmental intralobular arteries can be identified.

The renal arterial flow has antegrade flow throughout the cardiac cycle due to low impedance capillary bed. As the impedance increases the diastolic flow decreases, this can be assessed by measuring the Resistive index.

Resistive index= peak systolic frequency shift-lowest diastolic frequency shift/peak systolic frequency shift. (2)(3)

In vascular rejection endovasculitis causes swelling of the endothelial cells and inflammatory infiltrate in the subendothelial spaces. This greatly increases the impedance resulting in severe reduction in the diastolic flow and elevation of the vascular resistance.

The author has encountered two such cases of Hyperacute vascular rejection where there was nil cortical perfusion in the immediate postoperative period. This second case due to delayed nephrectomy underwent emphysematous pyelonephritic changes. Fig. 5

Other abnormal Doppler findings in the transplant include, reduced diastolic flow and elevation of the resistive index in cases of acute tubular necrosis. ATN is a common cause of early post transplant renal impairment, more commonly associated with cadaveric grafts and prolonged warm ischemia time. The condition is usually self resolving. In severe cases there can also be absent to reversed diastolic flow seen on Doppler. Fig. 6

Other features observed on the ultrasound include altered echogenecity of the renal parenchyma and swollen renal pyramids. (4)

Renal vein thrombosis occurs in less than 5% of the patients in the immediate post operative period. It has reported prevalence of 0.1-4.2% and leads to graft loss. It manifests in form of graft dysfunction. Swelling and tenderness over the graft area also noted findings.

Doppler reveals absent flow in the renal vein with marked diastolic reversal in the main and intrarenal arteries. Fig. 7. Partial thrombosis can cause elevation in the resistive indices and high velocities in the renal vein.

Segmental infarcts in the the transplanted kidney are being increasingly recognized as cause of graft dysfunction. It is poorly characterized condition following renal transplantation. Doppler can reveal hypovascular area in segmental distribution. Fig. 8

Altered hemodynamics is also observed in abdominal compartment syndrome, one of the uncommon causes of graft dysfunction. Reduced diastolic perfusion and elevation of the resistive indices are the Doppler observations. These changes rapidly reverse after surgical pressure relief with improvement in the renal function. Fig. 9

Contrast enhanced ultrasound is being increasingly used for transplant evaluation. It can be used for both qualitative and quantitative analysis of cortical perfusion. It can provide exquisite details of perfusion defect and details of the Anastomotic vessels. Heterogenous enhancement is noted in allografts with parenchymal disease. Bolus tracking can provide alteration in time to peak, wash in washout slopes in the graft.

Apart from the vascular complications stressed in this article Ultrasound in itself can diagnose many other graft related issues like perirenal hematomas, lymphocele, urinomas and urinary tract obstruction.

Ultrasound is an important first line modality for evaluation of the renal transplant. It can accurately predict and characterize important causes of graft dysfunction and, can provide directions in the graft management and help in graft survival.

Fig.1 Intraoperative Doppler revealing no parenchymal flow in transplanted kidney and only systolic waveforms with absent diastolic flow in the anastomosed renal artery.

Fig.2 Contrast enhanced ultrasound and CT angiography revealing no cortical enhancement/perfusion of the renal graft.

Fig.3 Intraoperative photograph revealing congested dusky kidney.

Fig.4 Histopathological slide revealing glomerular and peritubular capillary thrombi in vascular rejection.
Fig. 5 Emphysematous pyelonephritis in the renal graft.

Fig. 6 Case of Acute tubular necrosis with elevated intraparenchymal resistive indices.

Fig. 7 Case of renal vein thrombosis with diastolic reversal of the flow. Note the hypoechoic cortical infarcts in the graft.

Fig. 8 Wedge shaped hypoechoic area of segmental infarct in the transplant kidney on power Doppler.

Fig. 9 Case of compartment syndrome revealing reduced diastolic flow. Normalization of the diastolic flow after surgical decompression in noted in the next image.

References:


8. Fredrick Lefavre, Jean Michel Correas, Serge Briancon, Olivier Helenon, Michelle Kessler, MICHEL Claudon.