Graft dysfunction and transplant renal artery stenosis

Sir,

The paper by Krishnamoorthy *et al.*[1] made interesting reading. It is a detailed analysis of a large prospective cohort of kidney transplant recipients with TRAS but the conclusion is disappointing.

We have a few comments and would like to share our views on the definition of significant TRAS. The lack of such a definition of significant TRAS needs to be addressed and should include both refractory hypertension and more importantly, graft dysfunction (of course, in the absence of rejection, obstruction, and infection). If TRAS is causing significant ischemia and hypoperfusion, it should cause graft dysfunction. Calculating the degree of stenosis as a percentage is subjective and prone to inaccuracies and reminiscent of the classification of Mirizzi syndrome based on the percentage of bile duct stenosis.[2] The increased availability of routine Doppler has increased the diagnosis of TRAS by 12.4% in totally asymptomatic cases who were probably wrongly labeled TRAS, and by only 2.4% in patients already suspected of having TRAS,[3] based on presence of refractory hypertension and renal dysfunction, highlighting the importance of clinical diagnosis. This increase in the above suspected TRAS cases have insignificant TRAS with normal renal function, and need only follow-up, like all transplant recipients.

The authors state that "an angiogram was performed in those with a strong clinical suspicion and/or with a radiological suspicion of significant stenosis." The question that needs to be answered is Would an angiogram be done on a recipient with refractory hypertension or Doppler findings without graft dysfunction? The authors do, however, admit that only symptomatic patients had significant stenosis and went on to receive treatment. In their study, a quarter of the 43 cases diagnosed with TRAS was based on high systolic velocities only and were totally asymptomatic and required no further evaluation. These patients should not be labeled as TRAS and should have only routine follow-up. This strengthens the case of graft dysfunction as vital to a diagnosis of TRAS which is considered significant.

Deceased donor allograft recipients are reported to have a higher incidence of TRAS because of prolonged cold ischemia and delayed graft function, but only two deceased donor recipients developed TRAS in the study.[4,5] Although the authors do not provide this data, but perhaps the cold ischemia in the study was short which

prevented DGF and reduced the incidence of TRAS. What is surprising then is its high incidence in the live donor population. Since all live donor allografts require back-table perfusion using some type of cannula which can cause intimal damage and perhaps result in TRAS. In comparison, the perfusion cannula in deceased donor recovery procedure is at quite a distance from the renal arteries.

An interesting issue not often discussed in the literature was raised in this paper about ischemia from TRAS. We feel that this is the crux of the matter – the greater the stenosis, greater the hypoperfusion, symptoms, graft dysfunction, and outcome. It is surprising that no downstream complications of allograft ischemia resulting from TRAS have been reported; we are preparing a report of a case of distal ureteric stenosis from significant TRAS that required treatment. We would like to suggest that graft dysfunction should be considered mandatory for the diagnosis of significant TRAS.

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