Abdominal Pressure and Fluid Status After Kidney Transplantation

To the Editor: We read with great interest the article by Dupont *et al.*[1] who reported an association between elevated intra-abdominal pressure (IAP) and day 30 glomerular filtration rate. The authors concluded that the formers are clinically "relevant fluid status indicators" after kidney transplant. Although we congratulate the authors on these novel and important findings, we like to highlight several points. First, the notion that IAP is a surrogate of volume status is unsupported by the literature or authors' own findings.[1,2] More frequent etiologies for elevated IAP in the early postoperative period than volume overload include the following: ileus, obesity, and sliding down in bed from elevated head of bed position.[3,4] In addition, volume overload severe enough to cause intra-abdominal hydrostatic edema will usually have other signs of generalized edema. Second, as depicted in Figure 1 of Dupont *et al.*,[1] IAP further increased between 36 hours and 48 hours, despite a decrease in weight gain and central venous pressure.[1] This additional elevation in IAP cannot be ascribed to an increased fluid status. Third, we wholeheartedly agree with the authors' statement elsewhere[5] that IAH-induced decrease in glomerular filtration rate is multifactorial and insufficiently understood, with renal venous congestion playing an important, putative, pathophysiological role. In fact, the elevated IAP-mediated compression of the vena cava triggers pooling and increased pressure in upstream venous beds, with a simultaneous underfilling of the heart and ensuing multitude of compensatory mechanism that culminate in renal injury.[S1] As a result, renal venous congestion may occur in both extraperitoneally and intraperitoneally grafted kidneys. Last, an additional plausible mechanism for IAP-mediated kidney injury is that elevated IAP subsequently increases intravesical and ureteral pressures and diminishes urinary flow, with resultant kidney injury.

The differential diagnosis of elevated IAP is critical. Erroneously attributing elevated IAP to excess fluid may lead to fluid restriction, decreased preload, and exacerbation of acute kidney injury by prerenal azotemia.[S1] Volume status is better estimated by CVP, pulmonary artery occlusive pressure, or noninvasively with B-type natriuretic peptide, chest X-ray, or bedside echocardiography. Intraoperatively and in patients on mechanical ventilation, stroke volume and pulse pressure variation (e.g., FloTrac) may be used to guide fluid therapy.

SUPPLEMENTARY MATERIAL

Supplementary File (PDF)

Supplementary Reference.

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In Reply to "Abdominal Pressure and Fluid Status After Kidney Transplantation"

The Authors Reply: We thank Yehuda and Nicolau-Raducu[1] for their commentary on our recent publication.[2] They first question the use of intra-abdominal