

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' 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.

- 1. Dupont V, Bonnet-Lebrun AS, Boileve A, et al. A pilot study on the association between early fluid status indicators after kidney transplantation and graft function recovery. *Kidney Int Rep.* 2022;7:1416–1419.
- Dupont V, Debrumetz A, Leguillou A, et al. Intra-abdominal hypertension in early post-kidney transplantation period is associated with impaired graft function. *Nephrol Dial Transplant.* 2020;35:1619–1628. https://doi.org/10.1093/ndt/gfaa104
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In Reply to "Abdominal Pressure and Fluid Status After Kidney Transplantation"

The Authors Reply: We thank Yehuda and Nicolau-Raducu¹ for their commentary on our recent publication.² They first question the use of intra-abdominal pressure (IAP) as a surrogate of hydration status. We previously established a positive correlation between IAP and both central venous pressure and weight gain in our cohort.³ We also reported that severe intraabdominal hypertension was associated with higher fluid balance.³ Others have previously revealed a correlation between IAP and other fluid status indicators, such as extravascular lung water index.^{4,5} These data suggest IAP as a fluid status indicator of interest, notably when it comes to optimizing kidney perfusion.

We agree with the authors that common risk factor of intra-abdominal hypertension include obesity (as we reported elsewhere³), postoperative ileus, and bed position.⁵ We would like to clarify that none of the patients included in our study developed postoperative ileus (median time to resumption of transit: 2 days) and that all IAP measurements were performed in supine position according to our local protocol.

Second, CVP, Δ weight, and IAP decreased between 36 hours and 72 hours from 9 ± 5 to 6 ± 4 cm H₂O, +8 \pm 4 to +6 \pm 4%, and 15 \pm 6 to 11 \pm 5 mm Hg, respectively. These findings are consistent with the positive correlation found between these 3 variables in a linear mixed effect model.³

Third, as pointed out by the authors, the pathophysiology of intra-abdominal hypertension-related acute kidney injury is multifactorial.^{S1} Although renal venous congestion represents the main driver of kidney failure in this setting,^{S2} Yehuda and Nicolau-Raducu¹ also suggest increased ureteral pressure as a trigger. However, anticipated ureteral stenting failed to prevent oliguria when IAP was mechanically raised in an animal model.^{S3}

Finally, we thank Yehuda and Nicolau-Raducu¹ for pointing out the risk of fluid restriction-induced hypovolemia. Moderate hyperhydration has been widely used after kidney transplantation, consistently with the idea that large fluid administration would improve graft function recovery.^{S4} However, we and others have now reported that these patients could develop fluid overload complications.^{S4–S6} Our findings suggest that IAP could be useful to help decision once the patient is out of operatory room to optimize graft perfusion while avoiding fluid overload.

SUPPLEMENTARY MATERIAL

Supplementary File (PDF) Supplementary References.

- 1. Yehuda R, Nicolau-Raducu R. Abdominal pressure and fluid status after kidney transplantation. *Kidney Int Rep.* 2022;7: 127.
- Dupont V, Bonnet-Lebrun AS, Boileve A, et al. A pilot study on the association between early fluid status indicators after kidney transplantation and graft function recovery. *Kidney Int Rep.* 2022;7:1416–1419.
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