Letter to the Editor

Optimal Therapeutic Strategy for Children with Low Diuretic Responsiveness

To the Editor:

Responsiveness to diuretics is an important consideration when establishing a therapeutic strategy for patients with acute decompensated heart failure (ADHF). Price and colleagues investigated the mechanism of responsiveness to diuretics and its clinical impact in children hospitalized with ADHF.[1] I was interested in their finding that lower estimated glomerular filtration rate (GFR) was associated with augmented diuretic responsiveness in their single-center cohort, which contrasts with the physiologic response in adults who have low GFR. They hypothesized that low GFR may be a surrogate of fluid overload instead of intrinsic kidney injury, with a conclusion that greater intravascular congestion with depressed GFR may be associated with a better response to diuretics compared to those with normal GFR and ADHF.

In the clinical scenario of blunted diuretic responsiveness, a considerable number of high-dose diuretic regimens are often attempted in children presenting with suspected ADHF despite not always having clear signs of systemic congestion. This may reflect the challenges of noninvasive means of appropriately assessing for signs of systemic congestion in children. Right heart catheterization might be used seldomly in children due to its invasive nature, but other tools now exist to better assist the clinician in quantifying the degree of intravascular congestion. They include the remote dielectric system that our team recently reported as a method of quantifying congestion in ADHF[2] which, in turn, might be a reasonable tool to guide therapeutic strategies in children with ADHF. Furthermore, in the case of fluid redistribution instead of fluid accumulation (ie, low diuretic responsiveness), vasodilation therapy should be considered instead of standard diuretic therapies. In a case of low diuretic responsiveness despite obvious systemic congestion, it might be, rather, due to reduced renal function, and a vasopressin type-2 antagonist might be a better treatment strategy to potentiate greater natriuresis instead of increasing doses of conventional diuretics.[3]

Teruhiko Imamura, MD, PhD*
Nikhil Narang, MD
Division of Cardiology, University of Chicago Medical Center, Chicago, Illinois, USA
E-mail address: te.imamu@gmail.com (T. Imamura).

References

1. Price JF, Younan S, Cabrera AG, Denfield SW, Tunuguntla H, Choudhry S, et al. Diuretic responsiveness and its prognostic significance in children with heart failure. J Card Fail 2019. https://doi.org/10.1016/j.cardfail.2019.03.019.
2. Uriel N, Sayer G, Imamura T, Rodgers D, Kim G, Raikhelkar J, et al. Relationship between noninvasive assessment of lung fluid volume and invasively measured cardiac hemodynamics. J Am Heart Assoc 2018;7:e009175.
3. Imamura T, Kinugawa K. Update of acute and long-term tolvaptan therapy. J Cardiol. 2019;73:102–7.

https://doi.org/10.1016/j.cardfail.2019.07.006