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# Perirenal Fat and Renal Congestion: A Double Burden on Kidney Function in Acute Decompensated Heart Failure

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#### **Conflict of Interest**

The author has no financial conflicts of interests.

See the article "Perirenal Fat and Kidney Function Deterioration in Patients With Acute Decompensated Heart Failure" in volume 5 on page 36.

The perirenal fat and thick fibrous capsule surrounding and protecting the kidneys in the retroperitoneal space prevents them from expanding beyond the enclosed space. In acute decompensated heart failure (ADHF), increased interstitial fluid caused by elevated hydrostatic pressure leads to pitting edema in the skin and alveolar edema in the lungs.<sup>1)</sup> In kidney, renal congestion increases intracapsular pressure as the increased interstitial fluid cannot stretch the rigid capsule surrounding the kidneys. This reduces renal medulla perfusion and decreases the glomerular filtration rate (GFR).<sup>2)</sup> Additionally, perirenal fat accumulation increases perirenal pressure.<sup>1)</sup> Therefore, patients with a thick perirenal fat pad, especially those with ADHF, may be more vulnerable to renal congestion developing kidney dysfunction. Perirenal fat is also metabolically bioactive and a source of adipokines and pro-inflammatory molecules, which can worsen kidney function.<sup>3,4)</sup> Although perirenal fat accumulation is associated with renal dysfunction,<sup>3)</sup> it is unclear whether perirenal fat has an additive adverse impact on kidney function in patients with ADHF.

In this issue of the *International Journal of Heart Failure*, Cho et al.<sup>5)</sup> reported the results of a retrospective study of 266 patients with ADHF. Perirenal fat thickness was measured and averaged at the left and right posterior fat pads using computed tomography imaging. The authors investigated the association between perirenal fat thickness and kidney function by measuring the GFR. They showed that increased perirenal fat thickness, age, diabetes, and N-terminal pro b-type natriuretic peptide levels were independently associated with poor kidney function. The impact of perirenal fat thickness on kidney function was more prominent (odds ratio [OR], 1.15; 95% confidence interval [CI], 1.09–1.22; p<0.001) in patients with high left ventricular (LV) filling pressure (E/e' ratio >15) than in patients with low LV filling pressure (OR, 0.91; 95% CI, 0.79–1.06; p=0.347) (interaction p=0.019). These findings support the hypothesis that a thick perirenal fat pad and renal congestion worsen kidney function through 'renal compression' in patients with ADHF. Therefore, clinicians should evaluate metabolic profiles, including obesity, and closely monitor volume overloads to prevent worsening kidney function in patients with ADHF (especially those with a thick renal fat pad and evidence of elevated filling pressure).

The current study links perirenal fat, renal congestion, and poor kidney function. However, the pertinent question is "How can we manage patients with ADHF who have a thick perirenal fat pad and renal congestion?" Unfortunately, the authors could not answer this

question because of the limitations of their study. The hemodynamic profile, including the LV filling pressure, was assessed in the study using only the E/e' ratio. The E/e' ratio is a useful indirect indicator of LV filling pressure. However, LV filling pressure should be directly measured using invasive catheterization to accurately evaluate the impact of renal congestion on kidney function in patients with ADHF and a thick perirenal fat pad. Whether perirenal fat and renal congestion actually induce kidney dysfunction was also not confirmed probably because of the study's nature (cross-sectional and retrospective). To address this question, a longitudinal and prospective study is needed. The long-term outcomes of the adverse impacts of perirenal fat and renal congestion on kidney function in patients with ADHF also need to be assessed. Conversely, a study is needed to explore whether interventions to reduce the perirenal fat pad through metabolic profile improvements or the use of lipolysis drugs (such as sodium-glucose cotransporter inhibitors) could reverse renal deterioration in ADHF. Current practice guidelines in heart failure emphasize individualized treatment strategies according to the patient's phenotype.<sup>6)</sup> Further studies focusing on the double burdens of kidney function—perirenal fat and renal congestion may suggest a new treatment strategy for ameliorating renal compression in patients with ADHF.

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