

# Coexistence of Hemolytic Uremic Syndrome and Type 3 Cardiorenal Syndrome Due to Weil's Disease

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I read with interest the original article of plasma exchange (PE) in the treatment of anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) by Dr. Ebru Gök Oguz et al.<sup>1</sup> They emphasized that no additional benefit was gained by adding PE to standard therapy in AAV syndrome with or without pulmonary involvement.

Indeed, we have stressed the primary importance of PE in the treatment of patients with atypical hemolytic uremic syndrome admitted with sepsis, cardiorenal syndrome, anemia, thrombocytopenia and anuric acute kidney failure due to Weil's disease.

A 49-year-old female was transferred to our hospital with fever, myalgia, diarrheal illness, and chest pain. Due to impaired left ventricular function accompanied with high troponin values, myocardial infarction was excluded angiographically. Laboratory investigations revealed an acute kidney injury, acute liver injury, mild thrombocytopenia, and schistocytes on a blood film. A diagnosis of hemolytic uremic syndrome was made. Autoimmune markers were negative. The ANCA panel was negative. The C3 level was reduced, but the C4 level was normal. Leptospirosis was confirmed by the microscopic agglutination test. Kidney biopsy findings were intracapillary fibrin thrombi, glomerular congestion, and tubulointerstitial chronic inflammable cell infiltration. She received PE, glucocorticoids, and antibiotics

that made significant clinical, cardiological, and blood parameter improvement.

The risk for the development of kidney failure requiring kidney replacement therapy is higher in AAV patients with decreased estimated glomerular filtration rate (eGFR) levels and a low percentage of normal glomerulus as revealed by biopsy at the time of diagnosis.<sup>2</sup> Thrombotic microangiopathies due to leptospirosis infection may be seen in severe disease, which respond well to PE treatment.<sup>3</sup> While glomerular destruction is essential in AAV, hemolytic uremic syndrome (HUS) progresses with thrombotic microangiopathies and tubulointerstitial involvement. Therefore, the efficacy of PE in restoring kidney functions may be different.

The cardiorenal syndrome is a complex entity in which a primary heart dysfunction causes kidney injury (types 1 and 2) and vice versa (types 3 and 4), being either acute or chronic events, or may be the result of a systemic disease that involves both organs (type 5). Extrarenal effects of kidney injury especially on heart should be considered in designing therapies. Cardiac changes after experimental kidney ischemia include cytokine induction, leukocyte infiltration, cell death by apoptosis, and impaired function. Type 3 cardiorenal syndrome (acute cardiac dysfunction following acute nephropathy due to high systemic cytokine levels) is the scenario suitable for our patient.<sup>4</sup>



As acute kidney failure has systemic consequences and must be thought of as more than a kidney disease, PE can normalize immune flare with steroid and PE therapy by extracting the excess bacterial products, cytokines, and inflammatory mediators in severe systemic infections like leptospirosis.<sup>5</sup>

Cardiorenal syndrome should be kept in mind, especially when cardiac dysfunction is observed in patients with acute kidney failure who present to the emergency department with multi-organ failure. In the presence of cardiac dysfunction secondary to a condition related to nephropathy, we think that the addition of PE treatment may positively affect the clinical picture by improving cardiac functions, although its benefit in AAV alone is limited.

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