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Letter to the Editor

LETTER TO THE EDITOR

Role of leukapheresis in the management of acute kidney injury associated with hyperleukocytosis of acute myeloid leukemia

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To the Editor,

Hyperleukocytosis of acute myeloid leukemia (AML) is a hematologic emergency with early mortality rates as high as 20% during the first week of presentation and requires prompt treatment, especially if complicated by leukostasis [1–3]. The role of leukapheresis remains controversial [4]. Recently, Stahl *et al*. reported in their large, multicenter, retrospective study that leukapheresis may not be universally beneficial in all patients with AML-associated hyperleukocytosis [5]. However, it did suggest the possibility of benefit in a carefully selected group of patients (i.e. presence of cytostasis). Here, to the best of our knowledge, we report the first case of dramatic benefit associated with leukapheresis in the management of severe acute kidney injury (AKI) related to hyperleukocytosis of AML.

A 73-year-old male presented with generalized fatigue of 2 weeks duration. He was found to have a white blood cell (WBC) count of 159 000/μL with 98% peripheral blasts, and peripheral flow cytometry was consistent with acute myeloid leukemia with monocytic differentiation. On admission, the patient had hypoxia requiring 2 liters per minute of oxygen (O_2) and oliguric AKI without evidence of sepsis, hypotension, tumor lysis syndrome (TLS), or disseminated intravascular coagulation (DIC). Computerized tomography of the chest was negative for any acute process. Urine microscopy revealed a few granular casts. The patient was initiated on aggressive hydration, allopurinol, and hydroxyurea 3 grams twice daily. Due to a lack of improvement in oliguric AKI after four days from the presentation and without any other identifiable cause of AKI than cytostasis, he underwent one session of leukapheresis. This was followed by two consecutive sessions of hemodialysis for uremic encephalopathy. Within two days after leukapheresis, his urine output increased, and he didn't require further dialysis (Fig. 1). Serum creatinine, which had peaked up to 4.5 mg/dL prior to initiation of hemodialysis, returned to 1 mg/dL within seven days following leukapheresis and remained stable for the remainder of the hospital stay. Initiation of intensive chemotherapy was delayed due to patient consent. Unfortunately, the patient passed away after six weeks of a complicated hospital course.

Kidney failure can occur due to leukostasis from hyperleukocytosis because of tubular and glomerular dysfunction. The exact mechanism is unknown but possibly due to mechanical obstruction from less deformable leukemic blasts, the release of proinflammatory cytokines, and matrix metalloproteinases that damage endothelial cells. No current guidelines exist for the treatment of kidney injury in this setting. Our case suggests leukapheresis should be considered a therapeutic option for such patients.

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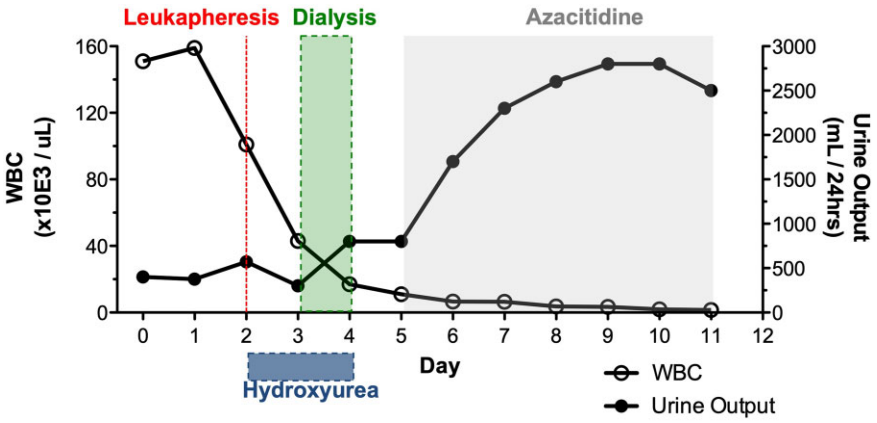

Figure 1: Leukapheresis in the management of acute kidney injury associated with hyperleukocytosis of acute myeloid leukemia. Leukapheresis was performed four days after the presentation of oliguric acute kidney injury. Within 48 hours after leukapheresis, the patient became non-oliguric, followed by a dramatic improvement in urine output and kidney function. WBC, white blood cells.

CONFLICT OF INTEREST STATEMENT

None declared.

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