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Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

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Identification of DICAM's Role and Pathophysiological Mechanism in Acute Kidney Injury

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The pathogenesis of AKI is not limited to a single precipitating event. Instead, it results from multiple potential causes. Given the broad spectrum of precipitating factors and the potential for rapid progression, early detection and identification of the underlying etiology are of paramount importance in the management of AKI. Dual immunoglobulin domain-containing cell adhesion molecule (DICAM), also known as limitrin and matrix remodeling-associated 8, is a type I transmembrane protein with two V-type immunoglobulin domains. DICAM is ubiquitously expressed in a variety of cell types, including macrophages, Th17 cells, endothelial cells, epithelial cells and chondrocytes, and is known to play a role in regulating inflammatory responses, particularly by increasing under stress conditions, but its role in renal cell injury, such as AKI, remains unclear. In our previous study, we identified high expression of DICAM in renal proximal tubule cells, and this study confirms its role in AKI and its potential use as an early marker. We identified the role of DICAM in H_2O_2- and LPS-induced injury in in vitro experiments using HK2 cells, a human renal proximal tubular cell. DICAM increased in response to oxidative and inflammatory stress and exhibited antioxidant and anti-inflammatory activities. In addition, DICAM was observed to increase earlier than NGAL, a previously known marker of renal injury, in the early stages of renal tubular cell injury, demonstrating its potential as an early diagnostic marker of AKI. Based on these results, we measured DICAM in the blood of patients with severe AKI requiring CKRT to determine its prognostic relevance. Elevated DICAM levels are independently associated with an increased risk of in-hospital mortality in critically ill patients with AKI requiring CKRT. In conclusion, DICAM is elevated early in AKI and may play a protective role against kidney injury, and its mechanism of action requires further investigation.

Keywords: Acute kidney injury, DICAM, DIagnostic marker, Inflammation