The Role of NGAL as a Biomarker for Early Detection of Acute Kidney Injury

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Acute kidney injury (AKI) is a common complication in hospitalized patients, with mortality rates as high as 50% in intensive care unit patients; therefore, early detection of this condition is critical for patient survival and cost of care reduction. Finding reliable biomarkers for early AKI diagnosis has been a research priority in the past decade. Neutrophil gelatinase associated lipocalin (NGAL) is the most studied biomarker of AKI. Because plasma and urine NGAL levels increase within two hours after AKI, it was assumed to be due to enhanced kidney production of NGAL. However, the ability of NGAL to predict AKI in patients has been inconclusive suggesting that other mechanisms of NGAL production may also be involved. Other studies have shown that there could be a link between interleukin-6 (IL-6) and NGAL production in sepsis. In the present study, murine models of AKI induced by warm bilateral ischemia-reperfusion on wild type (WT) mice and IL-6 knock-out (KO) mice were utilized. My results demonstrated that after AKI, the main source of NGAL is the liver, and not the kidney. Furthermore, because elevated NGAL levels were only detected in WT mice and not in KO mice, suggests that the hepatic NGAL production is IL-6 regulated. These results provide significant insight into the mechanisms of NGAL production in AKI, and the role NGAL plays as an early AKI biomarker.

Awards Won:

Third Award of $1,000