Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
KSN SEOUL, KOREA 2026
The 46th Annual Meeting of the Korean Society of Nephrology
THE KOREAN SOCIETY OF NEPHROLOGY
Jun 11 (Thu) – 14 (Sun), 2026
COEX, Seoul, Korea

Abstract Type : Oral presentation
Abstract Submission No.: A-0302
Abstract Topic : Acute Kidney Injury

Polo-Like Kinase 1 Is Upregulated in Experimental Acute Kidney Injury Induced by Ischemia-Reperfusion and Cisplatin

Beamjun Park[1], Dong Seop Kim[2], TaeWan Kim[3], Kwang Seock Kim[3], Hyo-wook Gill[2]
[1]Department of Future Innovation Medical Research Center, Soonchunhyang University Cheonan Hospital, Korea, Republic of
[2]Department of Internal Medicine-Nephrology, Soonchunhyang University Cheonan Hospital, Korea, Republic of
[3]Department of Future Innovation Medical Research Center, Soonchunhyang University Cheonan Hospital, Korea, Republic of

Objectives : Acute kidney injury is a major clinical problem characterized by abrupt loss of renal function and high risk of progression to chronic kidney disease. Despite extensive research, the molecular regulators driving maladaptive repair after acute kidney injury remain incompletely understood. Polo-like kinase 1(PLK1) is a key cell cycle regulator implicated in cellular stress responses, but its role in acute kidney injury has not been fully elucidated. This study investigated the expression and potential involvement of PLK1 in experimental acute kidney injury models.
Methods : Acute kidney injury was induced in C57BL/6 mice using two established models: renal ischemia-reperfusion injury and cisplatin-induced nephrotoxicity. For ischemia-reperfusion injury, bilateral renal pedicles were clamped for a defined period followed by reperfusion. Cisplatin-induced acute kidney injury was established by a single intraperitoneal injection of cisplatin. Renal function was assessed by measuring serum blood urea nitrogen and creatinine levels. Kidney tissues were harvested at defined time points for molecular analyses. PLK1 expression was evaluated using quantitative polymerase chain reaction and enzyme-linked immunosorbent assay.
Results : Both ischemia-reperfusion and cisplatin administration resulted in significant renal dysfunction, as evidenced by elevated blood urea nitrogen and creatinine levels compared with control groups. In the ischemia-reperfusion model, PLK1 mRNA and protein expression were significantly increased in injured kidneys. Similarly, cisplatin-induced acute kidney injury showed marked upregulation of PLK1 expression at both transcriptional and protein levels. These changes were consistently observed alongside biochemical confirmation of acute kidney injury, indicating a robust association between PLK1 expression and renal injury severity.
Conclusions : PLK1 is consistently upregulated in experimental acute kidney injury induced by ischemia-reperfusion and cisplatin. These findings suggest that PLK1 may play an important role in acute kidney injury pathophysiology and could serve as a potential biomarker or therapeutic target in acute kidney injury.