Clinical Implementation of Novel Kidney Injury Biomarkers for Diagnosis Assessment; the NGAL in a Real-life AKI Setting

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Abstract

Acute kidney injury (AKI) is a complex syndrome. Novel kidney injury biomarkers have been shown to improve prognostic and diagnostic assessment, but their use has been mostly limited to research settings. Their true clinical pertinence in a clinical setting has been rarely reported. Our group has recently decided to implement the neutrophil gelatinase-associated lipocalin (NGAL) in standard practice in our academic hospital. Methodology: An audit on the first 250 AKI cases with NGAL measurement was performed with a specific focus on the diagnostic performance to discriminate functional from intrarenal AKI. A questionnaire was prospectively distributed for all cases to the consultant nephrologist aiming to evaluate pertinence and appreciation. NGAL could be measured on urine (uNGAL and uNGAL/Cr) and plasma (pNGAL). The final adjudication for all cases was performed by two independent nephrologists. Findings: 174 (70%) of the 250 AKI patients were males, and 54 (22%) were at the ICU. The median serum creatinine at the time of NGAL measurement was 229 (IQR: 172-327) umol/L with Stage 1, 2 and 3 KDIGO-AKI occurred in 99 (40%), 58 (23%) and 93 (37%) respectively. The median pNGAL was 238 (112-485) ng/mL for functional AKI and 363 (224-706) ng/mL for intrarenal AKI (p=.154). The median uNGAL was 62 (25-172) and 550 (191-1873) ng/mL respectively (p<.001), similar to uNGAL/Cr (71 [39-215] vs 917 [282-2715] ng/mL [p<.001]). The overall AUROC of uNGAL/Cr to discriminate intrarenal from pre-renal AKI cases was 0.825 (0.766-0.884, p<.001) and was improved when considering the presence of UTI (0.874 [0.810-0.937, p<.001]). Interestingly, nephrologists considered the NGAL useful or very useful in 69% of cases, leading to immediate modification in management in 42% of all cases. Conclusion: NGAL, especially uNGAL, can be successfully implemented in a clinical setting to improve AKI etiologic diagnosis and management. Clinical adoption of emerging AKI biomarkers should be further encouraged.

Table 1. Biomarkers according to the Final AKI Etiology

Biomarkers	n	Pre-renal (n=100)[a]	Intra-renal (n=139)	Post-renal (n=11)	p-value[b]
pNGAL, *ng/mL*	44	238 (112-485)	363 (224-706)	235 (123-235)	0.154
uNGAL, *ng/mL*	214	62 (25-172)	550 (191-1873)	60 (26-1556)	<0.001*
uNGAL/Cr, *ng/mmol*	213	71 (39-215)	917 (282-2715)	123 (36-1984)	<0.001*
Urine Sodium, *mmol/L*	243	30 (12-55)	42 (20-76)	49 (29-67)	0.021*
FENa, *%*	242	0.56 (0.25-1.57)	1.69 (0.52-3.99)	1.45 (1.04-1.96)	<0.001*
uAlb/Cr, *mg/mmol*	220	9.1 (2.8-28)	23 (7.5-58)	26 (1.7-115)	<0.001*
uProt/Cr, *g/mmol*	232	0.04 (0.02-0.09)	0.13 (0.05-0.26)	0.11 (0.02-0.36)	<0.001*

Using median (IQR)
[a] Pre-renal cases include hypovolemia, cardiorenal and hepatorenal syndromes.
[b] Using a Kruskal-Wallis Non-parametric Test

Keywords: •Dialysis •Kidney transplantation

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Biography

Jean Maxime Côté is a nephrologist and research investigator at the Centre hospitalier universitaire de Montréal. He has expertise in acute kidney injury and critical care nephrology. He has progressively developed an interest and expertise in novel AKI biomarkers, such as NGAL and TIMP2-IGFPB7 to improve diagnosis and prognostication of patients with AKI.

Notes/Comments

It will be a pleasure to present on AKI biomarkers in various clinical settings. Our center is the first in Canada to have implemented the NGAL (since end of 2019). I am also responsible of the implentation locally at the St-.Vincents' University Hospital (Dublin, Ireland)(see references), where I completed an additional research fellowship last year. I will summarize current evidence regarding AKI biomarkers as well as presenting our local data, and the future of biomarkers.

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