



NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN [NGAL] AS A DIAGNOSTIC AND PROGNOSTIC BIOMARKER

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ABSTRACT

Neutrophil gelatinase-associated lipocalin (NGAL), also known as lipocalin-2 or siderocalin, is a 25-kDa protein of the lipocalin superfamily that was first isolated from human neutrophil granules and subsequently identified as one of the most highly and rapidly induced genes in the kidney following ischaemic and nephrotoxic injury.^[4, 5] Over the past two decades, NGAL has emerged as one of the most extensively studied biomarkers in clinical medicine, with a diagnostic footprint that extends well beyond its original description as an early indicator of acute kidney injury (AKI). This review synthesises current evidence on the biology, physiological regulation, and diagnostic performance of NGAL across a range of clinical contexts, including cardiac-surgery-associated AKI, sepsis and critical illness, contrast-induced nephropathy, chronic kidney disease, cardiovascular disease, urinary tract infection, and malignancy. Particular attention is given to the analytical characteristics of NGAL assays, including point-of-care platforms, and to the confounding factors that limit its specificity, most notably systemic inflammation and pre-existing renal impairment. While NGAL consistently outperforms serum creatinine in the early detection of tubular injury, with pooled sensitivities and specificities in the range of 70-90% across multiple meta-analyses, its diagnostic accuracy is heterogeneous across clinical settings and is influenced by the timing of sampling, the specimen matrix used (serum, plasma, or urine), and the presence of concurrent inflammatory states. This review concludes that NGAL holds substantial value as an adjunctive diagnostic and prognostic biomarker, particularly when used within defined clinical contexts and in combination with other markers of kidney injury, but that its role as a stand-alone diagnostic test remains constrained by its lack of organ specificity.

KEYWORDS: NGAL; neutrophil gelatinase-associated lipocalin; lipocalin-2; acute kidney injury; biomarker; chronic kidney disease; sepsis; cardiovascular disease; urinary tract infection; cancer.

1. INTRODUCTION

Reliable, early, non-invasive biomarkers of organ injury have long been sought in clinical laboratory medicine, nowhere more urgently than in acute kidney injury (AKI). Serum creatinine, the long-standing reference standard, is a functional rather than an injury marker: it rises only after substantial nephron loss, is influenced by muscle mass, age, sex, hydration, and diet, and can remain normal for 24–72 hours after an acute insult.^[1,2]

Neutrophil gelatinase-associated lipocalin (NGAL) has become the prototype of this new generation of injury biomarkers. First characterised as a neutrophil granule

protein covalently associated with matrix metalloproteinase-9 (MMP-9)^[3,4], it was later identified as one of the earliest and most robustly upregulated transcripts in renal tubular epithelium after ischaemic or toxic injury, well before any rise in creatinine.^[5] This dual identity — innate immune effector and stress-induced epithelial protein — underlies both its diagnostic promise and its pitfalls.

Since Mishra and colleagues' 2005 study showing urinary NGAL predicted AKI after paediatric cardiac surgery hours before creatinine rose^[5], thousands of studies have evaluated NGAL across tubular stress in

cardiac surgery, sepsis, contrast exposure, and transplantation; chronic tubulointerstitial damage in CKD; myocardial and vascular injury; urinary tract infection (UTI); and tumour biology.^[6-9] This review synthesises this literature, describes the molecular basis of NGAL's utility, appraises its performance across major clinical domains, and outlines the limitations constraining its use as a stand-alone test.

2. METHODOLOGY

SEARCH STRATEGY

A comprehensive literature search was conducted to identify relevant publications on neutrophil gelatinase-associated lipocalin (NGAL) as a diagnostic and prognostic biomarker across renal and non-renal clinical contexts.

DATABASES SEARCH

The following electronic databases were searched: PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. Reference lists of retrieved articles, systematic reviews, and meta-analyses were also manually screened to identify additional eligible studies.

SEARCH TERMS

Searches combined Medical Subject Headings (MeSH) and free-text keywords using Boolean operators ("AND"/"OR"), including combinations of: "neutrophil gelatinase-associated lipocalin," "NGAL," "lipocalin-2," "siderocalin," "acute kidney injury," "cardiac surgery-associated AKI," "sepsis," "contrast-induced nephropathy," "chronic kidney disease," "cardiovascular disease," "urinary tract infection," "biomarker," and "diagnostic accuracy."

INCLUSION CRITERIA

- Original research articles, systematic reviews, and meta-analyses evaluating serum, plasma, or urinary NGAL.
- Studies addressing the biology, structure, or molecular function of NGAL.
- Studies evaluating the diagnostic or prognostic performance of NGAL in acute kidney injury, chronic kidney disease, cardiovascular disease, urinary tract infection, or malignancy.
- Studies assessing analytical platforms, including point-of-care and laboratory-based NGAL assays.
- Studies published in peer-reviewed journals.
- Articles published in English.

EXCLUSION CRITERIA

- Case reports and case series.
- Conference abstracts, editorials, and letters without original data.
- Studies unrelated to the diagnostic or prognostic role of NGAL.
- Non-peer-reviewed sources.
- Duplicate publications or overlapping datasets, where the most comprehensive or recent report was retained.

TYPES OF STUDIES CONSIDERED

Eligible study designs included cross-sectional studies, cohort studies (prospective and retrospective), case-control studies, systematic reviews, and meta-analyses. Basic and translational studies elucidating NGAL's molecular biology and mechanism of action were also considered where they informed interpretation of its clinical performance.

STUDY SELECTION PROCESS

Titles and abstracts identified through the search were screened for relevance. Full texts of potentially eligible articles were retrieved and assessed against the inclusion criteria. Studies were selected based on relevance to the review's scope, methodological quality, and contribution to the synthesis of evidence on NGAL's biology, diagnostic accuracy, and prognostic value across clinical domains. Where multiple studies addressed the same clinical question, preference was given to larger, more recent, or higher-quality studies (e.g., meta-analyses over single cohort studies) to support key claims.

3. BIOLOGY, STRUCTURE AND MOLECULAR FUNCTION OF NGAL

3.1 DISCOVERY AND MOLECULAR STRUCTURE

NGAL was first isolated in 1992 as a 25-kDa glycoprotein bound to the 92-kDa form of MMP-9^[3], with its amino acid sequence determined the following year.^[4] It belongs to the lipocalin superfamily, characterised by an eight-stranded antiparallel beta-barrel ("lipocalin fold") forming a calyx-shaped ligand-binding pocket^[10], enabling it to bind small hydrophobic/hydrophilic ligands including bacterial siderophores complexed with ferric iron.

NGAL is stored preformed in neutrophil secondary granules and released upon degranulation.^[11] It is also constitutively expressed at low levels in epithelial tissues (kidney, lung, liver, stomach, colon), with marked upregulation after injury, infection, or malignant transformation. Circulating forms include a 25-kDa monomer (epithelial), a 45-kDa homodimer (neutrophil), and a 135-kDa MMP-9 heterodimer; some assays attempt to distinguish these to improve tissue specificity.

NGAL's best-characterised function is bacteriostatic: it binds catecholate-type bacterial siderophores (e.g., enterobactin) with high affinity, sequestering iron from invading bacteria^[12] — the basis for its alternative name "siderocalin" and its elevation during infection, including UTI.

In the kidney, NGAL participates in tubular iron trafficking, exerting anti-apoptotic and pro-proliferative effects during repair^[13], acting as an active participant rather than a passive marker. In the cardiovascular system, it is expressed by cardiomyocytes, endothelial cells, and plaque macrophages, interacting with MMP-9 to modulate matrix remodeling and plaque stability.^[14] In

malignancy, NGAL has context-dependent pro-tumorigenic effects (apoptosis inhibition, epithelial-to-mesenchymal transition, angiogenesis, metastasis), though some studies report tumour-suppressive effects instead.^[6,15]

3.2 SPECIMEN TYPES AND REFERENCE CONSIDERATIONS

NGAL can be measured in serum, plasma, urine, and other fluids (peritoneal dialysate, CSF, saliva, tears, breast milk).^[9] Urinary NGAL more directly reflects tubular production, since elevated levels with normal GFR suggest local tubular stress. Plasma/serum NGAL reflect a composite of renal handling, systemic neutrophil activation, and extrarenal production — more sensitive to early stress but less kidney-specific.^[1, 16]

4. NGAL AS A BIOMARKER OF ACUTE KIDNEY INJURY

AKI remains the clinical context in which NGAL has been most extensively validated. The rationale for its use rests on the observation that NGAL messenger RNA is one of the most highly upregulated transcripts in the thick ascending limb of the loop of Henle and the collecting duct within hours of ischaemic or nephrotoxic renal injury, well before histological or functional evidence of damage becomes apparent through conventional means.^[5]

4.1 CARDIAC SURGERY – ASSOCIATED AKI

Mishra and colleagues' foundational study in 71 children undergoing cardiopulmonary bypass found urinary NGAL rose sharply within two hours in those who developed AKI, with AUC 0.99 at two hours and 1.00 at four hours, versus creatinine which did not rise until 1–3 days postoperatively.^[5] Larger paediatric cohorts corroborated this, with urinary NGAL peaking around six hours.^[17]

Adult results are more variable, likely reflecting higher rates of pre-existing CKD, comorbidity, and perioperative confounders. One study of serial plasma NGAL in adults reported AUC of only 0.71 for a single measurement, improving to 86% sensitivity/specificity when two consecutive measurements were combined with creatinine and urine output.^[17]

4.2 CRITICAL ILLNESS, SEPSIS AND THE INTENSIVE CARE UNIT

In critically ill populations, NGAL performs less consistently, since systemic inflammation independently elevates it via neutrophil activation.^[18,2] Nonetheless, meta-analyses confirm a useful signal: plasma NGAL is an early, independent AKI predictor in mixed adult ICU populations^[16], with pooled AUC 0.70–0.91 across settings, reflecting heterogeneity in population, sampling timing, and AKI definition.^[19]

In sepsis-associated AKI specifically, a meta-analysis of 15 studies (1,478 patients) found pooled sensitivity/specificity of 0.83/0.57 for plasma NGAL (AUC 0.86) versus 0.80/0.80 for urinary NGAL (AUC 0.90), suggesting urinary NGAL retains more specificity in sepsis.^[20] A related meta-analysis reported pooled sensitivities of 0.87/0.83/0.80 and specificities of 0.84/0.79/0.74 for serum, plasma, and urine NGAL respectively, again favouring urine.

In the emergency department, point-of-care plasma NGAL predicted AKI within 3 days with 96% sensitivity and AUC 0.82 at a 150 ng/mL cutoff, replicated in an independent cohort^[21] — attractive for triage given creatinine's diagnostic delay.

4.3 CONTRAST – INDUCED NEPHROPATHY

In coronary angiography patients, a 25% rise in urinary NGAL from baseline predicted contrast-induced nephropathy (defined by cystatin C rise) with 80% sensitivity and 83% specificity, before creatinine changed. Similar early elevation (within two hours) has been reported in paediatric cardiac catheterization.

4.4 KIDNEY TRANSPLANTATION

Elevated post-transplant urinary/plasma NGAL correlates with subsequent delayed graft function and dialysis need, potentially identifying at-risk recipients before creatinine-based criteria are met.

4.5 DIAGNOSTIC ACCURACY SUMMARY

Table 1 summarises representative diagnostic accuracy estimates for NGAL across major clinical settings, drawn from key primary studies and meta-analyses discussed above.

Clinical Setting	Specimen	Sensitivity	Specificity	AUC	Ref.
AKI after cardiac surgery (paediatric)	Urine	~90-100%	~90-98%	0.99-1.00	[5]
AKI, general meta-analysis (adult & paediatric)	Serum/Plasma /Urine	65-89%	60-85%	0.70-0.91	[19]
Sepsis-associated AKI (plasma)	Plasma	83%	57%	0.86	[20]
Sepsis-associated AKI (urine)	Urine	80%	80%	0.90	[20]
AKI in Emergency Department	Plasma	96%	-	0.82	[21]
Urinary tract infection (children)	Urine	90.3%	93.7%	0.95	[8]

5. NGAL IN CHRONIC KIDNEY DISEASE

NGAL is also chronically elevated in CKD, rising progressively as eGFR declines, reflecting reduced

clearance plus ongoing tubular injury and interstitial inflammation.^[9] Elevated levels are associated with faster progression, more cardiovascular complications,

and higher mortality, including in dialysis patients. In diabetic nephropathy, serum NGAL has been proposed as a marker of early tubular injury preceding detectable albuminuria, though albuminuria remains the better-validated predictor.

A key limitation is that NGAL cannot easily distinguish acute-on-chronic insult from stable CKD, since baseline levels are already elevated proportionate to renal impairment — necessitating patient-specific baseline values rather than fixed population cutoffs.^[2]

6. NGAL IN CARDIOVASCULAR DISEASE

NGAL is highly expressed in injured/failing myocardium, myocarditis, and atherosclerotic plaque, with systemic levels correlating with heart failure, coronary artery disease, and stroke severity.^[7] In carotid atherosclerosis, NGAL co-localises with MMP-9 in symptomatic plaque and drives a pro-inflammatory phenotype in vitro, correlating with plaque instability and cardiovascular risk factors.^[14]

In heart failure, urinary/plasma NGAL has been studied as a marker of cardiorenal syndrome and a predictor of worsening renal function during diuretic therapy, capturing tubular damage independent of glomerular markers. In STEMI patients, elevated admission plasma NGAL independently predicted AKI or 30-day mortality, suggesting NGAL captures broader physiological stress beyond the kidney.

A systematic review of 22 cardiovascular NGAL studies concluded that despite pathophysiological support, clinical utility as an outcome predictor remains limited, partly due to inadequate adjustment for renal function and inflammation.^[7]

7. NGAL IN URINARY TRACT INFECTION

Urinary NGAL is among the most promising rapid, non-invasive UTI biomarkers in febrile infants and young children, where conventional urinalysis can be falsely negative. An individual patient data meta-analysis of 845 children found urinary NGAL (cutoff 32.7 ng/mL) achieved 90.3% sensitivity, 93.7% specificity, and AUC 0.95 for UTI diagnosis, outperforming leucocyte esterase in sensitivity though not specificity.^[8]

Plasma NGAL also independently predicts pyelonephritis versus lower UTI, outperforming procalcitonin, CRP, and white blood cell count in several paediatric cohorts, and has been investigated as a non-invasive predictor of subsequent renal scarring, potentially reducing reliance on DMSA scintigraphy pending further validation.

8. NGAL IN CANCER

NGAL (lipocalin-2) is dysregulated across malignancies including breast, colorectal, ovarian, pancreatic, gastric, thyroid, oral, and nasopharyngeal cancers.^[6,15] Its role is tissue- and context-dependent: in several tumour types it

correlates with invasiveness, angiogenesis, and metastasis via MMP-9 stabilisation and iron modulation, while in others (some oral and pancreatic tumours) higher expression correlates inversely with invasiveness, suggesting a possible tumour-suppressive role.^[6]

Elevated tissue/circulating NGAL has been investigated for distinguishing malignant from benign lesions and as a prognostic indicator; nasopharyngeal carcinoma studies show significantly higher lipocalin-2 expression in tumour versus normal tissue. Despite this preclinical and correlative evidence, NGAL has not achieved validated stand-alone diagnostic status for any cancer, remaining primarily a mechanistic research tool and candidate for multi-marker panels.^[6,15]

9. ANALYTICAL PLATFORMS AND ASSAY CONSIDER

NGAL is measured via conventional ELISA, automated chemiluminescent/turbidimetric immunoassays, and point-of-care devices. The Triage NGAL Test, a fluorescence immunoassay, was among the first cleared platforms for plasma NGAL and is widely used in emergency/critical care settings.^[21] Newer lateral-flow/cassette point-of-care tests trade turnaround speed for accuracy — one cassette-based urinary NGAL device reported 59.3% sensitivity and 95.8% specificity for AKI against KDIGO criteria.

Commercial NGAL assays lack full harmonisation, differing in antibodies, epitopes, and sensitivity to monomeric, homodimeric, and MMP-9-bound forms, contributing to variable concentrations and cutoffs across studies and complicating universal reference intervals.

10. LIMITATIONS AND CONFOUNDING FACTORS

NGAL's clinical application as a stand-alone test is constrained by several factors. First, lack of renal specificity: systemic inflammation, infection, and malignancy all elevate circulating and urinary NGAL independent of true renal insult^[2], particularly problematic in septic/critically ill patients where inflammation and AKI commonly coexist.^[18]

Second, pre-existing CKD raises baseline NGAL, reducing the utility of fixed cutoffs and requiring individualized, trend-based interpretation. Third, heterogeneity in sampling timing, specimen type, analytical platform, and AKI reference standard contributes to the wide range of reported accuracy and complicates cross-study comparison.^[19] Fourth, most large validation studies come from relatively homogeneous, single-centre populations (e.g., elective paediatric cardiac surgery), limiting generalizability to real-world heterogeneous populations. Finally, cost, limited platform availability, and lack of universally agreed decision thresholds have slowed clinical adoption relative to the pace of research publication.

11. FUTURE DIRECTIONS

Several directions are likely to shape the future clinical role of NGAL. First, there is growing interest in combining NGAL with other kidney injury and stress biomarkers, such as kidney injury molecule-1 (KIM-1), interleukin-18, liver-type fatty acid-binding protein, and the combined tissue inhibitor of metalloproteinases-2 and insulin-like growth factor-binding protein-7 (TIMP-2-IGFBP7) assay, into multi-marker panels that may improve diagnostic accuracy and better distinguish true tubular injury from extrarenal inflammatory elevation; a single urinary TIMP-2-IGFBP7 measurement predicted moderate-to-severe AKI with an AUC of 0.84 in high-risk surgical patients^[22], and complementary damage markers have similarly outperformed single analytes in paediatric cardiac surgery.^[23] Second, rapid, low-cost point-of-care and nanoparticle-enhanced lateral-flow platforms, which can now approach laboratory-grade analytical performance^[24], may extend the use of NGAL to resource-limited settings where the burden of AKI is highest. Third, larger prospective studies are needed to confirm the incremental prognostic value of NGAL for hard outcomes such as renal replacement therapy, progression to CKD, and mortality; existing meta-analyses are encouraging, with pooled AUCs of 0.82 to 0.84 for predicting the need for dialysis^[25, 26], but both emphasised that higher-quality evidence is required before prognostic thresholds can be recommended for routine practice.^[25, 26] Finally, continued mechanistic research into the context-dependent roles of NGAL in tumour biology may clarify its utility in specific cancer subtypes, although this application remains far less mature than its role in renal and cardiovascular medicine.^[6, 15]

12. CONCLUSION

NGAL is among the most thoroughly investigated and clinically promising biomarkers of the past two decades, detecting renal tubular stress hours to days earlier than creatinine across contexts from paediatric cardiac surgery to the emergency department. Its utility extends beyond the kidney to cardiovascular disease, UTI, and, more speculatively, malignancy — reflecting its dual identity as innate immune effector and stress-response protein. This same versatility is also its principal limitation: a lack of renal specificity most apparent with concurrent systemic inflammation, sepsis, or pre-existing CKD. Current evidence supports NGAL as a valuable adjunctive diagnostic and prognostic tool within well-defined contexts — post-cardiac-surgery AKI surveillance and paediatric UTI screening — rather than as a universal stand-alone replacement for existing diagnostic paradigms. Continued assay standardisation, multi-marker panel development, and outcome-based validation will be essential to fully realize its clinical potential.

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