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Renal function and iodinated contrast in CT: beyond creatinine cutoffs

Abstract

Background: Iodinated contrast media remain essential to diagnostic computed tomography (CT), yet their use is frequently restricted by concern over renal injury. In clinical practice, this concern is often reduced to rigid laboratory thresholds, particularly serum creatinine values or eGFR cutoffs, sometimes applied without sufficient consideration of clinical context or diagnostic urgency.

Objective: To critically re-examine the conceptual basis of contrast-associated acute kidney injury, clarify current terminology, and explore whether contemporary evidence supports the routine avoidance of contrast-enhanced CT in patients with impaired renal function.

Methods: This narrative review integrates foundational imaging and renal physiology literature with contemporary guidelines, controlled observational studies, and meta-analyses. Emphasis is placed on the distinction between contrast-induced acute kidney injury (CI-AKI) and post-contrast acute kidney injury (PC-AKI), risk stratification using eGFR, and the balance between renal vulnerability and diagnostic necessity.

Results: Current evidence suggests that much of what has historically been labeled as "contrast nephropathy" reflects the natural course of critical illness rather than direct contrast toxicity. The use of eGFR <30 mL/min/1.73 m² as an absolute contraindication is not supported by renal physiology or outcome-based data. In many high-stakes clinical scenarios, contrast-enhanced CT remains decisive, while most post-contrast creatinine rises are transient and clinically manageable.

Conclusion: Contrast-associated renal risk should be framed as probabilistic rather than prohibitive. A structured risk–benefit approach, grounded in physiology, clinical urgency, and ethical proportionality, offers a more rational alternative to reflexive contrast avoidance and better aligns imaging decisions with patient-centered outcomes.

Volume 13 Issue 1 - 2026

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Received: January 22, 2026 | **Published:** February 9, 2026

Introduction

Contrast-enhanced computed tomography (CT) is central to modern diagnostic medicine not because of technical sophistication, but because it answers questions that unenhanced imaging simply cannot. Over time, CT has evolved from an adjunct modality into a decisive clinical instrument, particularly in acute care, oncology, and vascular medicine. This transformation was driven by faster scanners, refined reconstruction techniques, and, crucially, the ability of iodinated contrast media to convert subtle physiological differences into actionable diagnostic information.[1,2]

Alongside this progress, concern about renal safety has remained almost inseparable from contrast use. Early descriptions of contrast-associated renal dysfunction originated in an era characterized by high-osmolality contrast agents, prolonged acquisition times, limited hydration strategies, and incomplete understanding of renal physiology.[3] These early observations shaped a culture of caution that has largely persisted, often without adequate recalibration despite major advances in both imaging technology and contrast agent design.

At the center of this caution lies serum creatinine. Creatinine became a convenient surrogate for renal function not because of precision, but because of availability. Imaging and medical physics references have long emphasized that creatinine is influenced by age, muscle mass, sex, hydration status, and acute illness, making it an unstable marker when interpreted in isolation.[4,5] Despite this, clinical practice in many institutions has reduced renal assessment to a single number, commonly around 1.2 mg/dL, treated as an implicit threshold beyond which contrast administration becomes unacceptable.

From a physiological perspective, this threshold is arbitrary. Renal filtration does not collapse at a specific creatinine value. Kidney function exists along a continuum shaped by nephron reserve, renal perfusion, and systemic hemodynamics.[6] CT textbooks repeatedly stress that renal risk assessment should be individualized and contextual, yet in routine practice this nuance is frequently replaced by rigid numerical cutoffs.[2,7]

The consequences of this simplification are not theoretical. They are clinical and, in some cases, catastrophic. In acute vascular conditions such as pulmonary embolism, acute aortic syndromes, and mesenteric ischemia, contrast-enhanced CT is not optional; it is diagnostic. Non-contrast examinations may suggest abnormality, but they rarely provide the certainty required to initiate life-saving interventions.[2,8] Avoiding or delaying contrast-enhanced imaging in these scenarios, solely because of a marginally elevated creatinine value, risks prioritizing a potential and often reversible renal insult over an immediate threat to life.

A similar imbalance appears in the evaluation of sepsis with an unknown source. Contrast-enhanced CT plays a critical role in identifying abscesses, ischemic bowel, infected collections, or necrotizing soft-tissue infections. Procedural guides consistently emphasize that effective source control depends on accurate localization, something non-contrast CT frequently fails to achieve.[1,9] In such cases, the absence of enhancement does not protect the patient; it obscures the pathology.

Oncologic imaging further illustrates the cost of rigid creatinine-based exclusion. The characterization and staging of hepatocellular carcinoma, pancreatic adenocarcinoma, and hypervascular metastases rely fundamentally on enhancement patterns across arterial, portal, and delayed phases.[7,10] Without contrast, arterial hyperenhancement, washout, and vascular invasion become speculative. Even renal masses, where concern for kidney function appears most intuitive, cannot be reliably characterized without contrast-enhanced imaging.[6]

Neurological emergencies add another layer of complexity. In suspected large-vessel occlusion stroke, CT angiography determines eligibility for mechanical thrombectomy, a time-sensitive intervention with profound implications for functional outcome. Withholding contrast based on laboratory thresholds risks converting potentially reversible neurological injury into permanent disability.[8]

Revisiting the theoretical basis of contrast-associated kidney injury helps explain this imbalance. Classical models emphasized renal vasoconstriction and tubular toxicity, largely extrapolated from experimental settings using older contrast agents and relatively high doses.[3,5] Contemporary imaging literature places these mechanisms within a broader framework that includes baseline renal reserve, volume status, systemic inflammation, hypotension, and concurrent nephrotoxic exposures.[6,7] Contrast media is increasingly viewed as a contributing stressor rather than an isolated cause of injury.

Modern low- and iso-osmolality iodinated contrast agents demonstrate substantially improved safety profiles. When administered with optimized dosing, controlled injection rates, and basic preventive measures such as hydration, their association with clinically significant acute kidney injury appears far weaker than historically assumed.[1,6] Yet despite this, many imaging departments continue to apply absolute creatinine cutoffs driven more by policy and fear than by physiology.

This gap between theory and practice defines the problem addressed in this review. The question is not whether iodinated contrast can influence renal function. It can. The issue lies in how renal risk is conceptualized and operationalized. Elevating a single creatinine value to a quasi-absolute contraindication reflects a misunderstanding of renal physiology and an underestimation of the consequences of diagnostic failure. This review seeks to reframe contrast decision-making by grounding it in physiology, imaging principles, and clinical urgency, replacing numerical reflexes with proportional judgment.

Terminology: CI-AKI vs PC-AKI

Confusion around terminology has quietly shaped how radiologists think about renal risk, often more than the evidence itself. Two terms dominate the discussion: **contrast-induced acute kidney injury (CI-AKI) and post-contrast acute kidney injury (PC-AKI).** They sound similar, are frequently used interchangeably, and yet they represent very different conceptual positions. That difference matters, because it influences whether contrast is perceived as a proven cause of harm or merely a temporal association.

Historically, CI-AKI emerged from early observations where a rise in serum creatinine followed contrast administration. The assumption was causal. Contrast went in, creatinine went up, therefore contrast caused kidney injury. Early imaging textbooks adopted this framework at a time when high-osmolality contrast agents were common and supportive care was limited.[1,2] Within that context, the term *induced* felt reasonable, even cautious. Over time, however, the term hardened into dogma, often without sufficient scrutiny of confounding variables.

Modern nephrology and radiology literature increasingly challenge this causality. Hospitalized patients undergoing contrast-enhanced CT are rarely physiologically stable. Hypotension, sepsis, dehydration, heart failure, anemia, and concurrent nephrotoxic medications frequently coexist. All of these are independently capable of altering renal function. When creatinine rises after contrast in such patients, attribution becomes problematic. This realization led to the adoption of **PC-AKI**, a deliberately more neutral term that describes temporal association without asserting causation.[9,10]

The distinction is not semantic nitpicking. PC-AKI acknowledges uncertainty. It accepts that creatinine may rise after contrast exposure, but it refuses to automatically assign blame. Several contemporary reviews emphasize that when appropriate control groups are used, the incidence of AKI in contrast-exposed patients often mirrors that of non-contrast cohorts with similar clinical profiles.[13,14] In other words, kidneys fail in sick patients, sometimes with contrast, sometimes without it.

Imaging textbooks have gradually reflected this shift. Procedural guides now recommend abandoning rigid terminology in favor of risk stratification and clinical context.[2,4] Importantly, even when AKI occurs after contrast, it is often transient and clinically mild, particularly with modern low- or iso-osmolality agents.[1,18] Permanent dialysis-requiring injury attributable solely to intravenous contrast is, by current evidence, uncommon.

Yet, despite these insights, older terminology persists in daily practice. *CI-AKI* continues to appear in request forms, consent discussions, and institutional policies, subtly reinforcing the idea of inevitability and direct harm. This persistence feeds defensive decision-making, where contrast avoidance is framed as renal protection, even when diagnostic compromise is substantial.

Clarifying terminology is therefore foundational. Using **PC-AKI** does not deny risk; it places that risk in its proper probabilistic frame. It shifts the conversation from prohibition to balance, from fear to judgment. For radiology practice, this shift is essential. Without it, discussions around creatinine thresholds, eGFR cutoffs, and diagnostic urgency remain trapped in outdated causal assumptions that the evidence no longer fully supports (Table 1).

Table 1 Conceptual differences between CI-AKI and PC-AKI

Aspect	CI-AKI	PC-AKI
Terminology meaning	Implies direct causation	Describes temporal association
Historical origin	High-osmolality contrast era	Modern CT era
Assumption	Contrast is primary cause	Multifactorial AKI
Control groups	Often absent or biased	Propensity-matched cohorts
Evidence strength	Weak observational	Moderate–strong controlled
Guideline preference	Largely outdated	ACR / ESUR preferred
Clinical implication	Contrast avoidance	Risk–benefit assessment

Why most 'contrast nephropathy' would have happened anyway

The uncomfortable truth is that much of what has long been labeled *contrast nephropathy* was never truly caused by contrast. It simply appeared after it. In hospitalized patients, kidneys rarely fail in isolation, and almost never on a timetable that neatly matches imaging. The temporal proximity between contrast administration and a rise in serum creatinine has created a narrative of causation that feels intuitive, yet often collapses under closer scrutiny.

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Acute kidney injury is a syndrome, not a single-pathway disease. Renal perfusion fluctuates with blood pressure, cardiac output, and intravascular volume. Tubular oxygen demand rises in sepsis, anemia, and systemic inflammation, while neurohormonal responses in shock and heart failure redistribute blood flow away from the renal cortex. These processes may begin hours or days before imaging is even considered, while serum creatinine remains deceptively stable because of its delayed kinetics.[1,8] When creatinine eventually rises after contrast-enhanced CT, timing alone creates an illusion of causality.

Early descriptions of contrast-induced nephropathy relied heavily on uncontrolled observational data. Patients who received contrast were, by definition, sicker. They were hypotensive, septic, bleeding, or suspected of harboring life-threatening pathology. Control groups, when present, were often healthier by design. Under such conditions, attributing AKI to contrast exposure reflects selection bias more than renal physiology.[11]

This bias becomes evident in contemporary studies using more rigorous methods. When propensity score matching is applied, or when contrast-enhanced CT is compared with non-contrast CT in clinically similar cohorts, the excess risk attributed to contrast diminishes markedly and, in many analyses, disappears altogether.[14,15] Importantly, the baseline incidence of AKI in hospitalized patients remains high regardless of contrast exposure. Kidneys fail in critical illness because the system is failing, not because iodinated contrast briefly passed through the glomerulus.

Imaging textbooks have long suggested this reality, albeit cautiously. Procedural guides describe renal injury as multifactorial and emphasize that contrast should be viewed as a potential contributor, not an automatic culprit.[2,3] What has changed in recent years is the confidence with which this point is stated. If contrast were a dominant nephrotoxin, its signal would persist after controlling for illness severity. In many modern datasets, it does not.

This reframing does not imply that contrast is harmless. Rather, it restores proportion. Contrast may tip a vulnerable kidney toward measurable dysfunction, but it rarely initiates injury in a physiologically stable one. In patients with sepsis, hemorrhage, heart failure, or advanced malignancy, the trajectory toward AKI is often already established before the scanner table moves.[6,18] Blaming contrast is often easier than acknowledging inevitability.

The clinical cost of misattribution is defensive medicine. Contrast is withheld, diagnoses are delayed, and decisions are made with incomplete information to prevent an injury that, in many cases, would have occurred anyway. Recognizing this does not eliminate renal risk. It replaces reflexive avoidance with intellectual honesty and shifts the question to where it belongs: will missing this diagnosis cause more harm than a transient rise in creatinine?

eGFR <30: A risk marker, not a stop sign

The adoption of **eGFR <30 mL/min/1.73 m²** as a marker of renal vulnerability in contrast-enhanced CT was, in principle, a scientific step forward. Compared with serum creatinine alone, eGFR incorporates age and sex and offers a more realistic approximation of filtration capacity. The problem did not arise from the metric itself, but from how quietly it was converted into a prohibition.

From a nephrological standpoint, eGFR was never designed to function as a binary switch. An eGFR of 28 does not describe a kidney that is fundamentally different from one at 32. No physiological cliff exists at that boundary. What changes is probability, not certainty. Imaging and renal physiology texts consistently describe renal injury risk as continuous, shaped by hemodynamics, intravascular volume, nephron reserve, and systemic illness rather than by isolated numerical thresholds.[1,3]

Contemporary evidence supports this interpretation. Even among patients with eGFR below 30, the incidence of severe, dialysis-requiring AKI attributable solely to intravenous contrast remains low when modern low- or iso-osmolality agents are used alongside basic preventive measures.[13,15] What increases sharply in this population is baseline AKI risk, regardless of contrast exposure. These kidneys are already vulnerable. Contrast does not create fragility; it exposes it.

Misinterpreting eGFR <30 as a stop sign distorts its original intent. The threshold was introduced to trigger **caution**, not cancellation. It signals the need for dose optimization, careful timing of repeat studies, hydration when feasible, avoidance of additional nephrotoxins, and post-imaging monitoring.[2,9] When applied rigidly, however, it shifts clinical attention away from diagnosis and toward numerical reassurance.

For nephrology, this logic should feel familiar. No other major AKI risk factor is treated as an absolute contraindication to diagnostic necessity. Sepsis, hypotension, and heart failure raise renal risk far more than contrast exposure, yet they do not halt investigation. They prompt mitigation and closer observation. eGFR <30 deserves the same treatment: a warning light on the dashboard, not a locked door.

Framing it this way restores proportionality. It allows contrast decisions to be guided by clinical urgency and diagnostic consequence, while still respecting renal vulnerability. In doing so, it replaces reflexive avoidance with informed judgment, which is ultimately what both nephrology and radiology aim to protect.

When the risk of missing the diagnosis outweighs renal injury

t debatable. These are scenarios in which contrast-enhanced CT is not an adjunct or a refinement, but the decisive step that determines survival, organ viability, or irreversible outcome. In such contexts, withholding contrast does not eliminate harm. It merely shifts it, often from a manageable renal risk to a catastrophic diagnostic failure.[1,2,9]

Life-threatening vascular emergencies fall squarely into this category. Pulmonary embolism, acute aortic dissection including intramural hematoma, acute mesenteric ischemia, and active internal bleeding are diagnoses in which time is inseparable from outcome. Non-contrast CT may hint at abnormality, but it rarely delivers the certainty required to trigger anticoagulation, surgery, or endovascular intervention. In these cases, delayed or missed diagnosis carries a mortality risk that dwarfs the probability of transient post-contrast renal dysfunction.[2,5,6,9,13]

Sepsis represents a second domain where contrast avoidance becomes ethically indefensible. In the septic patient with an unknown source, contrast-enhanced CT is central to identifying abscesses, ischemic bowel, or deep infected collections. Similarly, necrotizing soft-tissue infections rely on enhancement patterns to delineate fascial plane involvement and guide urgent surgical decisions. A non-contrast examination may appear cautious, but often produces equivocal reports while the patient deteriorates clinically.[1,2,7,9]

Oncologic imaging introduces a quieter but equally consequential risk. Certain malignancies cannot be meaningfully staged or characterized without enhancement. Hepatocellular carcinoma depends on arterial hyperenhancement and washout for diagnosis.

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DOI: 10.15406/ijrrt.2026.13.00448

Pancreatic adenocarcinoma requires contrast to assess vascular invasion and resectability. Hypervascular metastases, including those from neuroendocrine tumors, renal cell carcinoma, and melanoma, may be visible only during arterial phases. Even renal masses themselves often require contrast to distinguish cystic from solid disease, exposing the paradox of renal over-protection undermining renal diagnosis.[4,5,6]

Neurological emergencies further clarify this imbalance. In suspected large-vessel occlusion stroke, CT angiography determines eligibility for mechanical thrombectomy. Denying contrast in this setting risks converting a potentially reversible neurological deficit into permanent disability.[5,6,9] Across these scenarios, the ethical question is not whether iodinated contrast may influence renal function. That risk is acknowledged and well described.[9,13,18] The question is whether avoiding contrast compromises survival, organ preservation, or meaningful clinical outcome. When diagnostic certainty directly determines management, renal risk must be contextualized rather than absolutized.

Conditions where contrast avoidance is not defensible

- √ Pulmonary embolism
- √ Acute aortic dissection / intramural aortic hematoma
- √ Acute mesenteric ischemia
- √ Active internal bleeding (trauma or gastrointestinal)
- √ Sepsis with unknown source
- √ Necrotizing soft-tissue infection
- √ Hepatocellular carcinoma
- √ Pancreatic adenocarcinoma
- √ Hypervascular metastases (e.g., neuroendocrine tumors, RCC, melanoma)
- √ Acute ischemic stroke with suspected large-vessel occlusion

Ethical framing: when avoiding contrast becomes clinical neglect

At a certain point, avoiding contrast stops being renal protection and starts becoming clinical neglect. That boundary is rarely named explicitly, yet it defines the ethical tension at the core of contrast decision-making. The discomfort comes from the fact that both choices carry risk, but only one of them is visible on a laboratory report.

Medicine accepts risk as part of practice. Central venous lines cause infections, anticoagulation causes bleeding, surgery causes harm. These risks are tolerated because the alternative is worse. Contrast-enhanced CT belongs in the same category. When withholding contrast delays the diagnosis of pulmonary embolism, mesenteric ischemia, occult sepsis, or the resectability of malignancy, the ethical balance shifts decisively. At that moment, the question is no longer whether contrast might harm the kidney, but whether avoiding it harms the patient more.

Imaging textbooks consistently describe contrast as an enabling tool rather than an optional luxury.[1,6]. The ethical obligation of the clinician is not to eliminate all risk, but to choose the risk that best serves the patient's overall outcome. Protecting the kidney at the expense of the brain, bowel, or heart is not neutrality. It is a choice, and often the wrong one.

What makes this ethically difficult is the asymmetry of harm. A creatinine rise is measurable, documentable, and easily attributed. A missed diagnosis is abstract until it declares itself catastrophically. This imbalance biases decision-making toward inaction, even when inaction carries greater and less reversible harm. Defensive avoidance feels safer because it leaves a paper trail, while diagnostic failure often reveals itself later, when responsibility is diffuse.

From an ethical perspective, refusing contrast without integrating diagnostic urgency, reversibility of renal injury, and availability of alternatives violates the principle of proportionality. It prioritizes avoidance of a measurable but frequently transient insult over prevention of irreversible injury or death. That is not caution. It is a misalignment of responsibility.

Reframing contrast decisions ethically does not dismiss renal risk. It demands honesty about competing harms. In many clinical scenarios, the ethically defensible choice is not the one that preserves creatinine stability, but the one that preserves life, function, or meaningful diagnosis. When contrast is withheld reflexively, without this balance, the intention may be protection, but the outcome can be neglect.

A practical risk–benefit framework for contrast decision-making

In everyday practice, contrast decision-making fails not because clinicians lack data, but because they lack a usable mental model. Numbers are readily available, guidelines are published and updated, yet decisions are often reduced to a reflex: check creatinine, read eGFR, approve or cancel. What is missing is a structured way to weigh renal vulnerability against diagnostic consequence, using variables clinicians already understand.

The first step is redefining renal risk as probabilistic rather than absolute. An eGFR below 30 does not predict inevitable injury; it signals increased vulnerability. Imaging and nephrology references consistently emphasize that vulnerability modifies risk magnitude, not causality.[1,2] Renal impairment should therefore trigger mitigation strategies and closer monitoring, not automatic exclusion from contrast-enhanced imaging.

The second step is explicitly defining diagnostic urgency. Not all CT indications carry equal weight. Contrast-enhanced CT for suspected pulmonary embolism, acute mesenteric ischemia, aortic syndromes, septic source localization, or oncologic staging often determines immediate management or survival. In these settings, the cost of diagnostic failure is high and frequently irreversible. By contrast, elective follow-up imaging or screening studies usually tolerate delay or protocol modification with minimal harm.[3,4] Any rational framework must separate these categories early, before laboratory values dominate the discussion.

The third step involves weighing renal reversibility against diagnostic irreversibility. Most post-contrast creatinine rises, when they occur, are transient and manageable with supportive care. Dialysis-requiring injury attributable solely to intravenous contrast remains uncommon in contemporary practice.[13,15] Missed or delayed diagnoses, however, often carry permanent consequences. This asymmetry should be acknowledged openly rather than obscured by conservative language.

Mitigation, rather than avoidance, forms the fourth pillar of the framework. Procedural guides repeatedly emphasize dose optimization, avoidance of closely spaced repeat contrast exposure, withholding concurrent nephrotoxins, and ensuring adequate

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hydration when feasible.[2,9] These measures meaningfully reduce renal stress without sacrificing diagnostic yield, transforming contrast administration from an uncontrolled risk into a managed one.

Finally, decision ownership must be explicit. When contrast is withheld, it should be documented as a deliberate clinical choice, not an automatic laboratory response. When contrast is administered despite renal impairment, the rationale should clearly reflect diagnostic necessity and proportionality. In this framework, contrast-enhanced CT is neither recklessly normalized nor reflexively denied. It becomes what it should be: a clinical tool whose risks are acknowledged, contextualized, and balanced through informed judgment.

Conclusion

The debate surrounding iodinated contrast and renal injury has persisted for far longer than the evidence justifies. What began as a reasonable concern in an earlier technological era gradually hardened into a culture of avoidance, driven more by habit and fear than by physiology or outcome-based reasoning. In this context, serum creatinine and later eGFR thresholds evolved from risk indicators into de facto prohibitions, often applied without regard to clinical urgency or diagnostic consequence.

This narrative review argues that such an approach is no longer defensible. Contemporary understanding of renal physiology, combined with modern contrast agents and imaging protocols, makes it clear that most cases historically labeled as "contrast nephropathy" reflect the natural course of critical illness rather than a direct toxic effect of contrast media. The kidney, particularly in hospitalized or unstable patients, fails as part of a systemic process. Contrast exposure is frequently coincidental, occasionally contributory, but rarely singularly causative.

Reframing CI-AKI as PC-AKI is not an exercise in terminology for its own sake. It represents an intellectual correction. It forces clinicians to confront attribution bias, to separate temporal association from causation, and to acknowledge that avoiding contrast does not equate to eliminating risk. Instead, it often shifts risk elsewhere, into delayed diagnosis, inappropriate management, or irreversible organ damage.

The use of eGFR <30 mL/min/1.73 m² as a rigid stop sign exemplifies this misalignment. As shown, eGFR is a marker of vulnerability, not a physiological boundary. Treating it as an absolute contraindication misunderstands its purpose and ignores the continuous nature of renal risk. In high-stakes conditions, where contrast-enhanced CT directly alters outcomes, the ethical and clinical justification for avoidance collapses under scrutiny.

What emerges from this synthesis is not a call for reckless contrast use, nor a dismissal of renal safety. Rather, it is an argument for proportionality and judgment. Contrast administration should be guided by a structured risk–benefit framework that integrates renal vulnerability, diagnostic urgency, reversibility of harm, and available mitigation strategies. Such an approach aligns radiology with how risk is already managed across medicine.

Ultimately, protecting patients does not mean preserving laboratory values at all costs. It means choosing the path that maximizes meaningful diagnosis while managing, not fearing, collateral risk. When contrast is withheld reflexively, the intent may be caution, but the outcome may be neglect. When it is used thoughtfully, even in the presence of renal impairment, it becomes what it was always meant to be: a tool in service of clinical truth, not a trigger for automatic refusal.

Conflict of Interest

The authors declare no conflicts of interest.

Funding Statement

This review received no external funding.

Ethics and Consent

Not applicable; this review is based on previously published literature only.

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