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Contrast-Associated Nephropathy: A Comprehensive Review

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REVIEW

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Contrast-Associated Nephropathy: A Comprehensive Review

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Abstract

Acute renal failure can be attributed to Contrast-Associated Nephropathy (CAN), which remains a threat due to population variation and the increasing use of contrast media in various techniques. Thus, this review aims to discuss the pathophysiology, factors and risk factors for CAN, measures for prevention, and approaches to its management. The pathophysiology of CAN is extensive and encompasses various factors. Vasoconstriction and medullary hypoxia in the renovascular bed area are crucial in the pathogenesis of CAN. Several contrast media can provoke the formation of free radicals and reactive oxygen species (ROS), leading to oxidative stress, an inflammatory response, and enhancing renal injury. Hypotoxicity to tubular cells and osmotic stress contribute to the damage caused by contrast agents. Further, metabolic changes such as alterations in nitric oxide synthesis pathways and imbalances in antioxidant enzymes also contribute to the cause of CAN. It is noted that when the concentration of contrast media is high and the glomerular filtration rate is low, the condition is often seen in patients with compromised kidneys. The factors that predispose a patient to CAN are compromised renal function, diabetes mellitus, the patient's age, lack of adequate hydration, and the administration of a high volume of contrast media. Possible measures involve managing these risks, promoting adequate hydration, and avoiding unnecessary translational imaging whenever possible. Early diagnosis and management of CAN components are crucial to decrease the risk of worsening renal dysfunction and other severe complications. This may involve prescribing suitable medications such as intravenous fluids, diuretics, and other nephroprotective drugs.

Further research and enhanced understanding of the mechanisms will advance the improvement of prevention and treatment of this challenging condition. Collaborative care efforts among healthcare practitioners should enhance decision-making to reduce the high risk associated with this severe condition.

Keywords: Contrast-Associated Nephropathy (CAN), Renal hemodynamics, Reactive oxygen species, Acute kidney injury, Contrast media

1. Introduction

Contrast-Associated Nephropathy (CAN) is still inadequately defined and represents an area of inconsistent management in emergency settings. Now, as a definition of CAN, there is the impairment of renal function measured by a rise of at least 25% of serum creatinine from the baseline or a rise of 0.5 mg/dL (44 μmol/L) of an absolute serum creatinine within 48–72 hrs after IV contrast [1–3].

This renal impairment associated with contrast administration is usually acute, often appearing 2–3 days after the administration. Nevertheless, some recommendations suggest that renal impairment occurring within the first week after contrast administration should be considered as CAN if linked to other potential causes of acute kidney injury (AKI). Hence, there is an implication of a temporal relationship [4]. Following contrast exposure, serum creatinine reaches its highest levels between the third and fifth

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day and return to baseline within a two-week period after the critical event.

Iodine contrast medium is considered mandatory in invasive and interventional cardiac procedures. The ongoing increase in the utilization of contrast media, particularly in the growing number of coronary angiography and coronary interventional procedures, along with the rising number of invasive cardiac procedures in patients with chronic kidney disease (CKD), diabetes mellitus, hypertension, and kidney failure patients, all present contrast-induced nephropathy as an enduring concern. An increase in serum creatinine levels is a typical consequence of coronary angiography and percutaneous coronary intervention, primarily caused by contrast-media-induced acute kidney injury or contrast-media-induced nephropathy [5–7].

Basically, a proper knowledge of pathophysiology, risk factors, prevention, and management of CAN assist different healthcare providers, especially those using contrast media, to enhance decisions and actions taken to reduce the occurrence of this severe complication.

2. Pathophysiology

The pathophysiology of CAN is not fully understood, but it is believed to involve a combination of several interrelated mechanisms that ultimately lead to renal dysfunction [8]. Understanding these underlying processes is crucial for the prevention, early detection, and effective management of this condition.

2.1. Renal hemodynamic changes

a. Vasoconstriction: Among the contrast media, some contrast agents cause constriction of the renal vasculature, mostly the renal afferent arterioles. This leads to a decrease in renal blood flow, which in turn causes a decrease in the delivery of oxygen to the kidneys and thus ischemic injury [9].
b. Medullary hypoxia: Medullary hypoxia plays a crucial role in the pathogenesis of contrast-induced acute kidney injury (CI-AKI), mainly attributed to decreased medullary blood flow causing a subsequent reduction in oxygen delivery to the renal inner medulla. The contrast media (CM) initiation causes vasoconstriction of the renal blood vessels with resultant low blood supply and ischemia in the tubular structures. Such an ischemic condition, along with the cytotoxic effects of CM on renal tubular cells, triggers the formation of reactive oxygen species (ROS) which further worsen oxidative damage and tubular necrosis [10].

2.2. Oxidative stress and inflammation

a. Free Radical Generation: Contrast media can cause the production of free radicals and ROS with toxicity on renal cells through direct toxicity and alteration of their functions [2]. The oxidative stress experienced is worsened by the hypoxia resulting from contrast agents, thus increasing the generation of ROS and being involved in the damage to the renal parenchyma [11].
b. Inflammatory Response: The contribution of inflammation is crucial in enhancing renal damage, as the introduction of CM started bitterness inflammatory process, accompanied by the release of cytokines and the activation of leukocytes which have negative impact on the renal parenchyma [12]. When introducing CM to the patient, it triggers the secretion of inflammatory agents like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNFα), which are agents that stimulate inflammation. The aforementioned inflammatory mediators possess thrombotic properties, indicating their ability to induce the formation of blood clots, subsequently worsening renal functionality by reducing blood circulation to the renal organs [2].

2.3. Direct cytotoxicity

a. Tubular Cell Injury: Contrast agents have the ability to directly initiate apoptosis and necrosis of renal tubular cells, particularly in the proximal and distal convoluted tubules, which exhibit a higher susceptibility to harm. Exposure to contrast media may lead to the release of free iodine, causing necrosis of certain tubular epithelial cells and subsequent harm to the surrounding endothelium. This mechanism initiates cellular apoptosis, inflammation, and oxidative stress, all of which contribute to the onset of CAN. The susceptibility of these tubular cells to injury depends on factors including the cell type, the nephron's segment, and type of injurious stimulus [13].
b. Osmotic Stress: High osmolarity of some contrast agents may raise the osmotic pressure, which in turn, causes cells to shrink and die, further affecting renal cells [14].

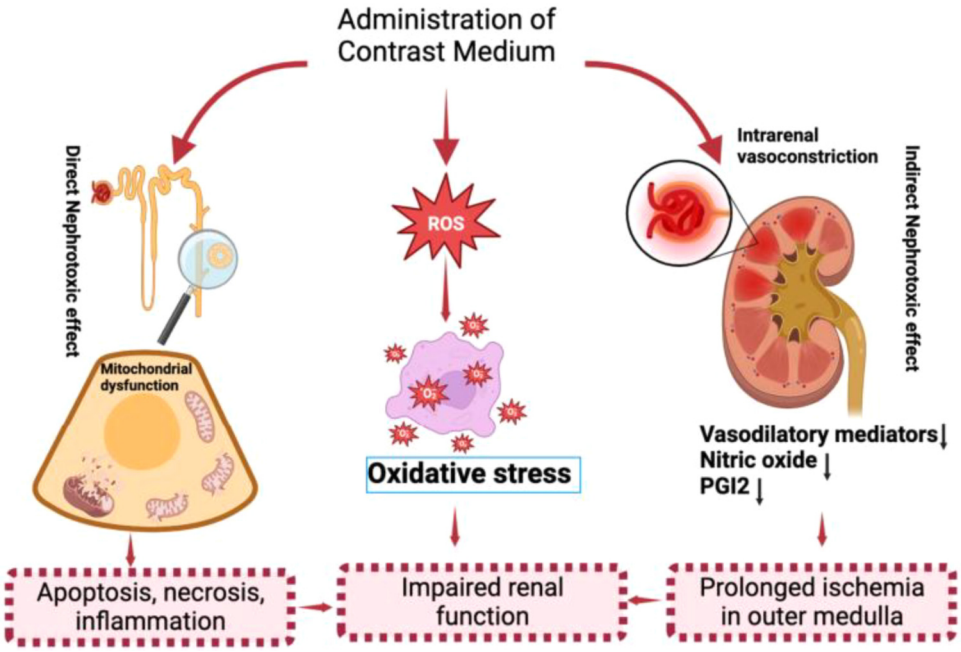

Fig. 1. The pathophysiology of CAN involves three primary mechanisms: direct effect, indirect effect, and the production of ROS.

2.4. Metabolic derangements

a. Altered Nitric Oxide Signaling: CM administration can exert its effect on the regulation of most NO mediated signaling and its implication in the maintenance of renal blood flow and prevention of ischemic injury. NO acts as an effective vasodilator, which plays a role in configuring different physiological events, among which it is possible to mention the regulation of the glomerular filtration coefficient and inflammation. Reduced bioavailability of NO especially in conditions such as diabetes and hypertension, leads to podocyte damage, proteinuria, and aggressive progression of CKD [15]. The utilization of CM, particularly those containing iodine, has the potential to elicit intrarenal vasoconstriction, hyperosmolality, and the production of ROS. These factors play a direct role in the development of nephrotoxicity and acute kidney injury (AKI) [16].
b. Disruption of Antioxidant Defenses: Contrast agents have the capability to diminish or hinder the efficiency of innate antioxidant mechanisms, thereby increasing the susceptibility of the kidneys to oxidative stress and injury [17].

2.5. Impaired renal function

Decreased Glomerular Filtration Rate (GFR): Exposure to contrast media has the potential to play a role in the decrease of GFR, thus affecting the functionality of the kidneys. An examination revealed a direct association between more frequent exposure to contrast media and a greater reduction in GFR over a period of time among individuals with impaired renal function [18]. Fig. 1 illustrates the pathophysiology of contrast-associated acute kidney injury.

The direct impact is characterized by the cytotoxicity of CM on the nephron, resulting in mitochondrial dysfunction, cellular apoptosis or necrosis, and interstitial inflammation, which ultimately leads to tubular injury. It is testified that CM-CAN affects the renal hemodynamics indirectly: intrarenal vasoconstriction that contributes to medullary hypoxia. This is achieved through alteration in the balance of vasoconstrictor hormones such as renin, angiotensin II and endothelin and vasodilatory hormones including NO and PGI2 and changes in renal circulation, intrarenal vasoconstriction, and medullary hypoxia. Moreover, through complex pathways that affect renal function, CM has the ability to generate ROS, which affects the activity of antioxidant enzymes due to oxidative stress [13].

3. Risk factors

Different issues connected with patients and certain procedures that may be implemented have been seen to be risk factors for CAN. These risk factors should be well understood in order to identify those patients who are most vulnerable and to use the adequate prophylactic strategy [19].

3.1. Patient-related risk factors

a. Preexisting Renal Impairment: Patients who have preexisting renal impairment, especially individuals with CKD and an estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73m^2, face a notably elevated likelihood of developing CAN [20].
b. Diabetes Mellitus: Diabetic patients,especially those with an ambulatory blood pressure of over 130/85 or those with poor glycemic control, are at a higher risk of CAN. Therefore, inadequate glycemic control or high HbA1c levels are considered factors that increase the risk of different complications, including infections and metabolic disorders. Studies, in fact, have shown that inadequate glycemic control means that clients' immunocompetence is lowered,thus increasing their susceptibilities to infections, such as cellulite and pneumonia, as signs of insufficiency of the immune system insufficiency [21]. Poor immune function may also fuel the risk of CAN since the body's ability to modulate oxidative stress and inflammation is impaired. Furthermore, hyperglycemia can initiate various alterations in the immune system,, affecting immune system cells,environmental conditions, supply to pathogenic bacteria, and inflammation. Such alterations can collectively increase susceptibility to infections and lead to CAN [22].
c. Advanced Age: CAN in patients with the aforementioned prevalence is more probable due to age-related changes in renal function and the presence of numerous comorbidities in people over 65 years old. The process of aging brings about both structural and functional modifications in the kidneys, which include a gradual reduction in GFR, a phenomenon that occurs irrespective of the presence of comorbid conditions. As a result, elderly kidneys are more susceptible to additional stressors like contrast agents used in different imaging techniques [23]. The vulnerability observed is exacerbated by the considerable prevalence of CKD among the elderly population. Studies show that nearly one-third of individuals aged over 65 have CKD, often coexisting with other chronic conditions like congestive heart failure, diabetes, and hypertension [24].
d. Dehydration: When the mass of the body mass decreases, leading to a reduction in the organism's renal circulation. This reduces, and the body's ability to filter waste and regulate fluid balance, posing a threat. Still, other decreased renal function might increase the risk for CAN [25].
e. Congestive Heart Failure: Heart failure (HF) is considered a specific risk factor for CAN because the pathophysiological relationships between the cardiac and renal systems are rather complex and closely intertwined. The symptoms of heart failure usually present with a reduction in the levels of cardiac output and systemic hypoperfusion; a decrease in renal perfusion may, therefore, lead to chronic renal disease [26]. This intricate pathophysiological correlation is further complicated by venous congestion, which worsens renal dysfunction by elevating central venous pressure and disrupting renal hemodynamics [27, 28].

3.2. Procedure-related risk factors

a. Type and Volume of Contrast Media: The incidence of CAN is greatly impacted by the osmolality, viscosity, and volume of contrast agents used in procedures such as coronary angiography and percutaneous coronary intervention (PCI). The use of higher-osmolarity contrast media (HOCM) has largely fallen out of favor due to its correlation with heightened CAN risk, resulting in a preference for low-osmolarity contrast media (LOCM) and iso-osmolar contrast media (IOCM) in the realm of clinical practice [14].
b. Timing of Contrast Administration: The risk of CAN significantly increases when contrast media are co-administered with other nephrotoxic substances or in close temporal proximity to other radiological procedures. The main pathophysiological process responsible for CAN includes direct toxicity to tubular cells, oxidative stress, and changes in hemodynamics, which are worsened by the presence of other nephrotoxic agents like nonsteroidal anti-inflammatory drugs (NSAIDs), ACE inhibitors, and specific antibiotics [16].

3.3. Prevention and management of contrast-associated nephropathy

Given the potential consequences of CAN, various strategies have been proposed to prevent and manage this condition.

3.4. Prevention strategies

a. Hydration: Adequate hydration plays a crucial role in the prevention of CAN, particularly among high-risk patients undergoing procedures involving contrast media. The intravenous (IV) administration of fluids, like

normal saline and sodium bicarbonate solutions, has been thoroughly studied for their efficacy in decreasing the incidence of CAN. Current literature presents compelling evidence supporting IV hydration over no fluid intake, particularly for high-risk patients exposed to intra-arterial contrast media, favoring isotonic saline over 0.45% saline or isotonic sodium bicarbonate [19].

b. Minimizing Contrast Volume: Studies have indicated that it is imperative to reduce the amount of CM administered during diagnostic and therapeutic procedures in order to decrease the likelihood of CAN. Research findings have shown that the amount of CM used is a significant prognostic factor for CAN, with a higher volume of CM linked to an increased risk, especially in individuals with pre-existing renal dysfunction [30]. The utilization of ultra-low contrast volume methodologies has demonstrated a notable decrease in the occurrence of AKI) and the necessity for dialysis, particularly in patients at high risk [31].
c. Use of Low-Osmolarity or Iso-Osmolar Contrast Agents: Research indicates that both LOCM and IOCM are linked to a reduced occurrence of CAN in comparison to HOCM. For example, a study conducted by Lee et al. revealed no statistically significant disparity in the prevalence of CAN between LOCM and IOCM, proposing that both varieties of contrast agents present as safer options to HOCM [14].
d. Prophylactic Medications: Prophylactic medications, such as N-acetylcysteine (NAC) and statins, have exhibited potential protective effects against the manifestation of CAN in high-risk individuals. The efficacy of NAC in averting CAN has been extensively investigated. Numerous studies have indicated that NAC can markedly diminish the occurrence of CAN in patients undergoing coronary angiography and computed tomography, with a meta-analysis revealing a relative risk decrease ranging from 24% to 49% in these clinical scenarios [32, 33]. The antioxidant properties of NAC are responsible for its protective mechanism, reducing oxidative stress and improving mitochondrial function, ultimately mitigating renal injury [34].
e. Timing of Contrast Administration: Minimizing the administration of contrast media near other potentially nephrotoxic substances or interventions may help reduce the likelihood of AKI or worsening of CKD [35].

3.5. Management of contrast-associated nephropathy

a. Early Identification: Vigilantly observing renal function, as assessed through serum creatinine levels, within the 48-72 hours subsequent to contrast exposure is imperative for the timely identification of CAN [36].
b. Supportive Care: Positive care dealing with CAN is helpful as it involves care that goes beyond the mere disease treatment to include a caring approach to the patient. Another factor that should be properly managed is water balance as it affects kidney function and contributes to further renal deterioration in patients with CKD and those who received the treatments influencing the state of their kidneys [37]. Another important part of the process is cessation of nephrotoxic agents since such preparations can severely impair kidney function and foster the development of CAN [38].
c. Dialysis: In cases of severe CAN with a significant and persistent decline in renal function, dialysis options such as intermittent hemodialysis or continuous renal replacement therapy may be necessary to support the kidneys and prevent the onset of fatal complications [39].
d. Prevention of Complications: It is necessary to reduce and control the complications of CAN to improve the life quality of the patients, and the pertinent steps, including maintenance and regulation of fluids and electrolytes, are remarkable for this. Standardizing the assessment and reporting of complications can lead to a better understanding and management of such events [40].

4. Conclusion

Contrast-associated nephropathy remains an issue of concern due to the tendency of improvement in the usage of contrast media in different medical practices. To prevent and manage the problem, one must understand the mechanism behind this process. The mechanism is quite complex, involving includes changes in renal hemodynamics, oxidative stress, inflammation, direct cytotoxic effects, and alterations in metabolism. Hypotension leads to proportional vasoconstriction, medullary hypoxia, increased free radical production, along tubulointerstitial damage leading to the development of CAN. Given this scenario, a more rigorous approach is required, consisting of key steps such as controlling risk factors, optimizing fluid balance, and utilizing imaging studies whenever feasible. Managing hypertension in

CAN intervention is crucial to reduce the likelihood of future acute onchronic renal disease and associated complications. Additional studies and collaboration among professionals are crucial for enhancing choices and reducing risks associated with the development of this severe consequence.

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