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Research Progress on Risk Prediction and Preventive Measures for Contrast-Induced Nephropathy During Cerebrovascular Interventional Procedures

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Abstract: To prevent the occurrence of contrast-induced nephropathy during cerebrovascular interventional procedures, this study systematically reviewed the relevant literature and delineated the pathogenesis of contrast-induced nephropathy, which mainly includes renal medullary ischemia and hypoxia, direct toxicity to renal tubular epithelial cells, inflammatory response and imbalance of vasoactive substances, and hemorheological alterations. It also summarizes the risk prediction measures commonly used in clinical practice, such as precise identification of risk factors for contrast-induced nephropathy, the Mehran risk score and other predictive models, and biomarkers. Furthermore, preventive strategies are proposed to avoid the development of contrast-induced nephropathy through preoperative risk assessment and stringent evaluation of indications, hydration therapy, careful selection of contrast agents and strict control of dosage, and the use of pharmacological prophylaxis, with the aim of enhancing the safety of cerebrovascular interventional procedures.

Keywords: Cerebrovascular intervention; Contrast-induced nephropathy; Risk prediction; Preventive measures

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1. Introduction

Cerebrovascular interventional procedures can be performed to diagnose and treat cerebral aneurysms, cerebrovascular stenosis, cerebrovascular malformations, and other conditions. The use of contrast agents during these procedures may induce contrast-induced nephropathy, which not only prolongs the length of hospital stay but also increases the probability of long-term adverse renal events in patients. Therefore, it is a complication of major concern in the perioperative period of cerebrovascular interventions. During cerebrovascular interventional procedures, the presence of underlying diseases, hemodynamic status, the type of contrast agent used, and the dose of contrast agent administered are all closely associated with the occurrence of contrast-induced nephropathy. To reduce the incidence of contrast-induced nephropathy and improve the safety of cerebrovascular interventional procedures, it is essential to perform risk prediction in advance and implement comprehensive preventive measures.

2. Pathogenesis of Contrast-Induced Nephropathy

2.1. Renal Medullary Ischemia and Hypoxia

Renal medullary ischemia and hypoxia is one of the core factors contributing to contrast-induced nephropathy during cerebrovascular interventional procedures. Ren Yuxiang et al. [1] have proposed that the anatomical structure of the human renal medulla is relatively specialized; it is supplied by small-diameter and elongated blood vessels, and active sodium transport in the medullary loop of Henle consumes a substantial amount of oxygen. This results in markedly reduced oxygen tension in the renal medulla, which is 30–40 mmHg lower than that in the renal cortex, rendering the renal medulla highly susceptible to hypoxic injury. During cerebrovascular interventional procedures, following the injection of contrast media, the renal vessels undergo initial dilation followed by persistent constriction, leading to a significant reduction in renal blood flow and glomerular filtration rate. Jing Shiwen et al. [2] have indicated that contrast media may further aggravate the imbalance between oxygen supply and demand in the renal medulla, indirectly causing renal vasoconstriction and promoting the redistribution of blood from the medulla to the cortex, thereby exacerbating medullary ischemia. Moreover, after the injection of contrast media, the tubular reabsorptive load increases, and renal oxygen consumption rises accordingly, resulting in ischemic and hypoxic injury to the renal medulla and thus inducing nephropathy.

2.2. Direct Toxicity to Renal Tubular Epithelial Cells

Direct toxic effects of contrast media administered during cerebrovascular interventional procedures on renal tubular epithelial cells can induce contrast-induced nephropathy. Currently, the iodinated contrast media commonly used in clinical practice are triiodobenzene ring derivatives. Whether in hydrated, ionic, or molecular form, they are toxic to human cells. They can undergo electrophilic reactions with amino acids in cell membrane proteins, such as histidine, tyrosine, and cysteine, causing damage to membrane proteins and loss of cell membrane integrity. As the contrast media pass through the renal tubules, water is reabsorbed, further increasing the intraluminal concentration of the contrast media and raising the osmotic pressure of the tubular fluid. This leads to enhanced vascular resistance and slowed blood flow. Concurrently, the contrast media exert cytotoxic effects: they not only adversely affect mitochondrial enzyme activity but also cause mitochondrial dysfunction, disruption of intracellular calcium homeostasis, and disturbances in energy metabolism, ultimately resulting in cell apoptosis.

2.3. Inflammatory Response and Imbalance of Vasoactive Substances

Imbalance of vasoactive substances is also a major factor in contrast-induced nephropathy during cerebrovascular interventional procedures. Prostaglandins and nitric oxide are endothelium-derived vasodilatory factors; under physiological conditions, their continuous release maintains the vasculature in a dilated state. Intrarenal production of these vasodilatory substances plays a critical role in preserving oxygen supply and blood perfusion in the renal medulla. However, when renal pathology occurs, the generation of vasoactive substances decreases, predisposing patients to contrast-induced nephropathy during cerebrovascular interventional procedures. Furthermore, under the toxic effects of contrast media, renal medullary epithelial cells and vascular endothelial cells are induced to generate oxygen free radicals, which can activate inflammatory responses. Through the interplay of these processes, the production of reactive oxygen species increases, enhancing the bioactivity of vasoconstrictive substances and further aggravating renal medullary ischemia. This leads to tubular cell injury and, consequently, contrast-induced nephropathy.

2.4. Hemorheological Changes

The influence of contrast media used during cerebrovascular interventional procedures on hemorheology is also a principal cause of contrast-induced nephropathy. After contrast injection, blood viscosity increases significantly, leading to impaired microcirculation and a markedly elevated risk of microvascular thrombosis. Specifically, when the contrast media enter the small renal vessels, the blood flow velocity within these vessels decreases, reducing oxygen delivery and rendering the kidney hypoxic. In addition, as the contrast media pass through the renal tubules, most of the water is reabsorbed,

progressively increasing the viscosity of the contrast media. This can easily result in the formation of plugs that obstruct the tubules and cause further tubular damage. Moreover, increased contrast media viscosity leads to the aggregation of erythrocytes within the renal tubules and other small renal vessels, substantially increasing the risk of contrast-induced nephropathy.

3. Risk Prediction of Contrast-Induced Nephropathy in Cerebrovascular Interventional Procedures

3.1. Identification of Risk Factors for Contrast-Induced Nephropathy

Accurate identification of the risk factors that trigger contrast-induced nephropathy (CIN) during cerebrovascular interventional procedures can lay a solid foundation for risk prediction. The risk factors for CIN are mainly divided into two categories: patient-related factors and procedure-related factors. Patient-related factors include age and the presence of underlying diseases such as diabetes mellitus, hypotension, renal insufficiency, and cardiac insufficiency, all of which are risk factors for CIN. Tilakezi Tuersun et al. [4] proposed that both an increase in the dose of contrast medium used during cerebrovascular intervention and an elevation of the serum NLRP3 level are risk factors for CIN. In addition, advanced age, heart failure, and anemia are also closely associated with the occurrence of CIN. Regarding procedure-related factors, the type of contrast medium selected and the dose administered are both linked to the development of CIN. Both excessively high and excessively low doses of contrast medium may induce CIN; therefore, no absolutely safe dose exists, and the dose should be determined according to the patient's actual condition.

3.2. Risk Prediction Using the Mehran Score

The Mehran score is currently a commonly used tool in clinical practice for predicting the risk of CIN. The variables of this scoring model mainly include the presence or absence of hypotension, NYHA class III/IV heart failure, pulmonary edema, anemia, diabetes mellitus, prior use of an intra-aortic balloon pump, as well as patient age, serum creatinine level, and contrast medium dose. Based on the score, patients can be divided into four risk categories—very high risk, high risk, intermediate risk, and low risk—with a decreasing probability of developing CIN across these groups. Wang Qi et al. [5] demonstrated that the Mehran score has good predictive value for adverse cardiovascular events after percutaneous coronary intervention in patients with acute myocardial infarction; the risks of adverse cardiovascular events in the high-risk and intermediate-risk groups were 3.095 times and 1.714 times that in the low-risk group, respectively. Wang Chenyu et al. [6] found that combining the Mehran score with seven different serum inflammatory cytokines—IL-2, IL-4, IL-6, IL-10, IL-17, TNF-α, and IFN-γ—can further improve the predictive performance for CIN. However, the Mehran score is derived from a post hoc analysis, and its variables do not include preoperative hydration, the type of contrast medium selected, or the use of nephrotoxic drugs; therefore, it has certain limitations in predicting the risk of renal complications related to contrast medium.

3.3. Other Prediction Models and Biomarkers

In addition to the Mehran score, other CIN risk prediction models used internationally in clinical practice include the Maioli CI-AKI risk assessment model, the Duan CI-AKI risk assessment model, and the Chen CI-AKI risk assessment model, all of which have shown certain application value in predicting CIN during cerebrovascular interventional procedures in specific populations. Moreover, nomogram prediction models established in recent years based on large samples have also demonstrated good predictive performance. Qi Xingmin et al. [7] found that the most extensively studied marker for the early diagnosis of CIN is neutrophil gelatinase-associated lipocalin (NGAL). Following injury to renal tubular epithelial cells, the level of this marker rises rapidly, and this change occurs earlier than the rise in serum creatinine.

4. Preventive Measures for Contrast-Induced Nephropathy in Cerebrovascular Interventional Procedures

4.1. Preoperative Risk Assessment and Precise Determination of Indications

A comprehensive evaluation of the patient's condition before cerebrovascular interventional procedures is the primary step in preventing contrast-induced nephropathy (CIN). The assessment mainly includes the patient's age, baseline renal function, history of diabetes mellitus, cardiac function, anemia status, and the use of nephrotoxic drugs. If high-risk factors for CIN are identified through this evaluation, the therapeutic benefits of the cerebrovascular intervention must be weighed against the risk of CIN. When the risk of CIN is considerably high, alternative treatment modalities should be selected whenever possible to avoid cerebrovascular interventional procedures. If the interventional procedure is unavoidable, comprehensive preventive measures must be implemented, including the selection of the lowest possible concentration of non-ionic contrast medium and intensified perioperative management.

4.2. Hydration Therapy

Currently, hydration therapy is recognized as the cornerstone for preventing CIN and is a widely applied, low-cost preventive measure in clinical practice. The preventive mechanisms of hydration therapy are as follows: it increases renal perfusion pressure and expands circulating blood volume, thereby diluting the contrast medium concentration and facilitating its excretion; it also shortens the retention time of contrast medium in the renal tubules through its diuretic effect, thus reducing toxicity to renal tubular epithelial cells. Commonly used hydration fluids in clinical settings include sodium bicarbonate solution and isotonic sodium chloride solution. When administered before and after cerebrovascular interventional procedures, sodium bicarbonate solution is superior to sodium chloride solution in preventing CIN. The use of sodium bicarbonate solution can elevate the urine pH, increase the production of renoprotective substances, and simultaneously prevent excessive generation of reactive oxygen species. However, close monitoring of the patient's serum potassium level and urine pH is necessary during sodium bicarbonate administration to avoid inducing hypokalemia.

4.3. Strict Control of the Selection and Dosage of Contrast Media

The choice of contrast medium in cerebrovascular interventional procedures is crucial for the prevention of CIN. Based on osmolality, contrast media can be classified into high-osmolar, low-osmolar, and iso-osmolar types. High-osmolar contrast media have a high incidence of adverse reactions in clinical use and have therefore been largely replaced by low-osmolar and iso-osmolar contrast media. Low-osmolar and iso-osmolar contrast media are associated with a lower incidence of CIN, and they are particularly safer for patients with diabetes mellitus or pre-existing renal insufficiency. Since there is no significant difference in the incidence of renal injury between low-osmolar iodinated contrast media and iso-osmolar iodinated contrast media, either can be selected based on the actual clinical situation. Furthermore, the dosage of contrast medium should be minimized, adhering to the basic principle of "as low as reasonably achievable." The occurrence of CIN is closely related to the administered dose of contrast medium, and the maximum safe dose can be calculated using the following formula: contrast medium volume (mL) ≤ 5 mL × body weight (kg) / serum creatinine (mg/dL).

4.4. Pharmacological Prevention of Contrast-Induced Nephropathy

Although no guideline currently recommends a definitively effective pharmacological agent for the prevention of CIN, the preventive effects of various drugs have been explored in numerous studies. First, N-acetylcysteine: administering this drug orally at a dose of 600 mg twice daily before cerebrovascular interventional procedures, in combination with hydration therapy, can significantly reduce the incidence of CIN in patients with renal insufficiency. Second, statins: Zhou Lu et al. [8] demonstrated in their study that in addition to lipid-lowering effects, statins possess anti-inflammatory, antioxidant, anti-apoptotic, and hemodynamic-improving properties, thereby indirectly protecting the kidneys. Preoperative administration of statins can reduce the probability of renal injury following high-dose contrast medium exposure. Third, prostaglandin E1: this agent not only increases renal blood flow and dilates renal vessels but also inhibits platelet aggregation and inflammatory responses, and increases creatinine clearance rate, thereby protecting the kidneys; it is regarded as one of the important drugs for CIN prevention. In addition, ascorbic acid exerts antioxidant effects that can reduce apoptosis of renal

tubular epithelial cells, providing indirect protection for renal function.

5. Conclusion

In summary, contrast-induced nephropathy in cerebrovascular interventional procedures typically results not from a single mechanism but from the superimposition of multiple mechanisms. In clinical practice, the risk of CIN can be stratified through comprehensive patient assessment, screening of procedural risk factors, and the application of Mehran risk score or other predictive models and biomarkers. Based on this stratification, an individualized hydration therapy can be implemented preoperatively, contrast medium can be selected rationally and its dose calculated accurately during the procedure, and preventive pharmacological agents (such as N-acetylcysteine, statins, and prostaglandin E1) can be administered pre- and postoperatively. These measures effectively protect the kidneys and prevent the occurrence of CIN. Future research on the prediction and prevention of CIN in cerebrovascular interventions should include large-sample, randomized controlled trials to further determine the appropriate target population and optimal timing for various preventive strategies, thereby better reducing the incidence of contrast-induced nephropathy.

Disclosure statement

The author declares no conflict of interest.

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