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Contrast-Induced Nephropathy, Focus on Prevention

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Abstract
Contrast-induced nephropathy (CIN)- iatrogenic pathology that occurs afterintroduction of a contrast agent into the body.Known links in the pathogenesis of CIN are direct toxic effects in the renal tissues and decreased hemodynamics in the kidney. Other mechanisms of CIN pathogenesis are still studied not enough. To date,but many definitions of CIN, the most common of which consider an increase in serum creatinine greater than 0.5 mg/dL or more than 25% of the original level, determined during the first 2-3 days after the introduction of con-trust substance. CIN is quite rare is in the general population of patients,wearing contrast studies, but many fold increases in groups of patients with initial kidney disease in diabetic patients and the elderly. Multiple risk factors greatly increases the chance of developing CIN. The best ways to prevent CIN consider active identification of patients with risk factors, and adequate periprocedural hydration. The role of various drugs in prevention of contrast-induced nephro-pathy is still debatable and suggests a further research. In this clinical practice for the prevention of CIN should be pre-read isosmolar and low osmolar contrast agents, strictly avoiding highly osmolar contrast agents in patients with impaired renal function, despite the ongoing discrepancies regarding the degree of nephro-toxicity of various contrast agents.

Keywords: contrast agents, serum oral creatinine (Cr), creatinine clearance(CC), glomerular filtration rate (GFR),contrast-induced nephropathy (CIN).

Relevance of the Topic

Increased use of contrast substances for diagnostic and therapeutic radiological procedures has led to an increase number of cases of contrast-induced nephropathy (CIN) - iatrogenic pathology,associated with the introduction into the body of contrast-th substance.

CIN is a complex acute renal failure syndrome.Insufficiency, developing after the introduction of denia of iodine-containing contrast agents. The definition of CIN includes absolute or a relative increase in the level of creatinine on blood serum after contrast injection compared with baseline creatinine on, when other causes of violation are excluded renal function. Increasing creatinine levels observed within 24-48 hours after the introduction contrast ratio, peak creatinine noted 3-5 days after the procedure and return to normal or close to normal level occurs within 1-3 weeks (1). The size of the increase in the level of tinin, which determines CIN, differs from research to

research (from 20 to 50% or in absolute terms - from 0.5 to 1.0 mg / dl), making it difficult to compare their results. Most general definition, most commonly used recently, is more than 25% relative increase or more than 0.5 mg/dL absolute increase in serum creatinine from the initial value within 48-72 hours after injection of contrast into the body. Based on given this definition, the frequency of CIN in the total populations are reported to fluctuate from 1.2 to 1.6% (2.3). CIN frequency is even higher in patients with various cardiovascular pathology, which is not surprising, given the high how many risk factors for the development of CIN in this group of patients. According to the Mayo Clinic (Mayo) incidence of CIN in 7586 patients,who underwent percutaneous coronary interventions(PCI) was 3.3% (4). In relation very small study by McCullough and et al. (5) where the data was analyzed 1826 patients undergoing PCI, CIN was registered in 14.5% of cases. Hemodialysis in these two studies, it was required respectively, in 0.7% and 0.3%.

PATHOGENESIS OF CONTRAST-INDUCED NEPHROPATHY

The pathogenesis of CIN is not fully understood. Until now time, several pathophysiological biological mechanisms of CIN, including direct toxic effect on the renal canal annular epithelium, oxidative stress, ischemical injury and tubular obstruction(6.7). Domestic (8) and foreign nephro-logs (9) believe that radiopaque media-on the one hand, after a short period vasodilation is caused with the participation of renin-angiotensin system spasm leading arterioles, and on the other hand, increasing blood viscosity,disrupt microcirculation and have an adverse effect on medium toxic effect on the canal annular epithelium, probably by generating oxygen free radicals (8,9). Short blood flow in the medullary layer of the kidney, leading to leading to his hypoxia, could be the result of an increase peripheral hydrostatic pressure and secondary pressure increase in tubules due to contrast-induced diu-cut, vasoconstriction due to excess vasoac-active substances such as adenosine and endothelin and reduction of vasodilators - nitric oxide and prostaglandins (10,11). Excretion contrast-substance requires a significant amount urine to remove the osmotic load.The functioning of the kidney under conditions of high osmotic load leads to characteristic histopathological logical changes, called "osmotic sky nephrosis". Changes specific to osmotic nephrosis were detected in 22.3% biopsies performed on patients during 10 days after the introduction of a contrast agent properties (12). After the introduction of a contrast agent a transient increase in renal blood current followed by a longer its decrease is observed in animals and humans(13). Endothelin-1 is considered the most likely cause of changes in most research (14,15). Vasoactive the effect of adenosine on various organs depends on sieves on the ratio of its A1 and A2 receptors. IN kidneys, unlike the heart, adenosine causes causes vasoconstriction. They also think that he plays a role in the pathogenesis of CIN due to an increase its concentration in the kidney as a result of increased slow hydrolysis of adenosine

triphosphate (16).Free oxygen radicals that form during hypoxia, are also likely to contribute contribution to kidney damage (17).

RISK FACTORS OF CONTRAST INDUCED NEPHROPATHY

Risk factors for the development of CIN have been well studied cheny in several studies and summarized in final table (Table 1). They can be di-divided into two categories: fixed (nemomodified) and modifiable.The following non-modified cited risk factors for CIN: elderly growth, diabetes mellitus, previous renal insufficiency, congestive heart failureт stagnation, hemodynamic instability, and nephrotic syndrome.

1.Age

Performing contrast studies in elderly patients is associated with increased risk of contrast-induced nephropathy. In a study by Rich et al. in patients older than 70 years, CIN developed in 11% of the study data (3). Reasons for higher risk development of CIN in the elderly have not been studied and probably have multifactorial toric nature, including age-related changes decrease in renal function with a decrease in the rate glomerular filtration rate (GFR), tubular secretion and concentration ability, as well as a more difficult puncture of the vessel, requiring giving more contrast, the presencemultivessel lesion, etc. Important, that in multivariate analysis, by itself growing older than 70 in some studies was an independent predictor of the development of CIN(18,19,20).

2.Previous kidney failure

Chronic renal failure (CRF),now included in the supranosological concept -chronic kidney disease (CKD), with increased serum creatinine level – critical risk factor for the development of CIN, the frequency of which swarm is extremely high, ranging from 14.8% up to 55% (4,5,21). With multivariate analysis baseline creatinine levels in most studies turned out to be an independent predictor rum CIN (3,4,5,21). As opposed to risk development of oil recovery factor turned out to be minimal (less than 10%) in patients who had a normal function the kidneys during the study with the introduction contrast agent.

High baseline creatinine values were associated with an increased risk of CIN (22). As shown in in their study by Hall et al. (23), at initial bottom creatinine level less than 1.2 mg/dl frequency ORF was only 2%. However, in patients with the level of initial creatinine from 1.4 to 1.9 mg /for the frequency of CIN increased to 10.4%, and in patients those who had initial creatinine more than 2.0 mg /dl, after angiography CIN developed in 62%.CIN risk prediction model according to the level of initial serum creatinine shows exponential social increase in the frequency of nephrotoxicity at baseline levels greater than 1.2 mg/dL (24). IN in general, the estimated rate of

glomerular filtration rate (GFR), which is less than 60 ml / min / 1.73m2, is considered borderline in relation to increase in the development of CIN (25).

3.Diabetes

In numerous studies (3,4,5,26) diabetes mellitus was defined as independent the most predictor of the risk of developing CIN.The incidence of CIN in patients with diabetes mellitus flashes from 5.7 to 29.4% (2,27,28). What is the prevalence of diabetes in the general population and its ability to cause a wide range of cardiovascular disease requiring imaging studies to correct diagnostics and treatment, patients suffering from DM represent a significant proportion among individuals undergoing contrast studies.It is interesting to note that the risk of CIN increases in patients with diabetes even with preserved kidney function(26.29). The presence of other risk factors such as like kidney failure or proteinuria,patients with diabetes further increases the likelihood development of CIN. In a study by Berns et al. (1)CIN occurred in 27% of DM patients with baseline creatinine level from 2.0 to 4.0 mg/dl, and had 81% of diabetic patients with baseline creatinine more than 4.0 mg/dl. In a study by Toprak et al. (30) in 421 patients, the estimated level was reduced creatinine clearance value, being in the range from 15 to 60 ml / min according to the Cockcroft-Gault formula.When dividing these patients into 3 groups according to fasting blood glucose levels it turned out that in the group with a normal value "fasting" glucose (n = 144; glucose less than 100mg/dl) CIN (determined within 48 hours after angiography as an increase in the level of creatinine more than 25% of the initial value) was in 5.5% patients, while those with prediabetic state (n = 140; glucose from 100 to 125mg/dl) CIN was in 11.4%, and in patients with DM (n =137; glucose over 125 mg/dl) CIN was observed in 20% of cases (30).

4.Anemia

In a large register, consisting of 6773 pain-patients who have consecutively undergone percutaneous coronary intervention, with multifactorial nom regression analysis initially low hematocrit was identified as an independent pre-CIN development speaker (29). CIN (defined as an increase over the next 48 hours after serum creatinine angiography25% or 0.5 mg/dl or more) increased from 10.3% in the highest hematocrit quintile before 23.3% in the lowest hematocrit quintile (P for trend <0.0001).

5.Condition after transplant kidneys.

Concomitant use of nephrotoxic drugs (eg, cyclosporine) along with a high incidence of diabetes and renal insufficiency in patients undergoing kidney transplantation is “favorable background for a high risk of developing CIN.Ahuja et al. (33) retrospectively estimated results of studies with the introduction of con-trust in 144 patients with functioning renal allograft. It turned out,that the overall frequency

of CIN in the group was 21.2%, and it was especially high (42.8%) among those who were not hydrated before con-trust research.

6.The amount of injected contrast.

The volume of contrast injected during the study substance is of paramount importance in the development of CIN (28). This is the main submitter-change in the risk factor for CIN. However, growth the complexity of coronary interventions is inevitable causes an increase in the use of con-trust substances during the procedure, and, consequently,importantly, increases the risk of CIN. Correlation the relationship between the amount of injected contrast of matter and an increase in the frequency of SIF was a has been reported in many studies (34,35). According to McCullough et al. (5) risk CIN is minimal in patients who received less 100 ml of contrast agent. induced nephropathy.

Focus on prevention.

High baseline creatinine values were associated with an increased risk of CIN (22). As shown in in their study by Hall et al. (23), at initial bottom creatinine level less than 1.2 mg/dl frequency ORF was only 2%. However, in patients with the level of initial creatinine from 1.4 to 1.9 mg /for the frequency of CIN increased to 10.4%, and in patients those who had initial creatinine more than 2.0 mg /dl, after angiography CIN developed in 62%.CIN risk prediction model according to the level of initialth serum creatinine shows exponential social increase in the frequency of nephrotoxicity at baseline levels greater than 1.2 mg/dL (24). IN in general, the estimated rate of glomerular filtration rate (GFR), which is less than 60 ml / min / 1.73m2, is considered borderline in relation to increase in the development of CIN (25).

CONCLUSION

Contrast-induced nephropathy - iatro-gene pathology that develops after the introduction of niya in the body of contrast agents. Straight cytotoxic effect of renal tissue, induced by a contrast agent, along with decrease in renal blood flow are the most more obvious links in the pathogenesis of CIN,while other arrangements are still poorly studied. Although the frequency of CIN in the general population treatment of patients receiving contrast is definitely low, it manifests itself in a significant the number of patients with existing chronic kidney disease, in patients with diabetes even in the elderly. The presence of several factors risk of CIN increases the likelihood of acute kidney injury after administration of contrast nogo substance. Currently the best CIN prevention strategy is to identify treatment of patients with risk factors, and also conduct adequate periprocedural hydration. The role of various drugs in pro-prevention of CIN is still controversial and suggests future research. Despite the remaining Xia discrepancies regarding the degree of nephro-toxicity of various

contrast agents, in present clinical practice should be read isosmolar and low osmolar contrast agents, strictly avoiding highly osmolar contrast agents in patients with impaired kidney function.

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