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Review Article

An Overview of contrast Induce Acute kidney injury

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Abstract:

The risk of contrast induced Acute kidney injury (CI-AKI) is high in CKD & diabetic patient as compared to general population. It is the third most cause of Hospital acquired AKI. Pathogenesis is not clear but there is role of direct & indirect cytotoxicity of contrast media, oxidative stress & as well as of micro-RNA. There is no specific therapy of CI-AKI, hence prevention is the cornerstone of management. Except hydration therapy all other pharmacotherapy has been failed to prove beneficial effect in preventing CI-AKI in many RCT & meta-analyses.

Keywords: contrast induced acute kidney injury (CI-AKI), pathogenesis, contrast media.

Introduction

With advancement in radiology/Cardiology, the use of iodinated-contrast medium, has increased for diagnostic accuracy & therapeutic procedure. Large number of patients are receiving contrast media for PCI. Contrast media have many adverse effects like nausea, vomiting, hyper sensitivity, thyroid dysfunction, acute kidney injury etc. There are 3 types of iodinated contrast media–1st generation- High osmolar contrast media (HOCM), 2nd generation- Low osmolar contrast media (LOCM) & 3rd generation - ISO-osmolar contrast media (IOCM). First article about contrast related nephrotoxicity was published in 1954 in a patient of multiple myeloma. Since then, many terminologies are in use to describe renal dysfunction due to contrast agent. Currently contrast induced acute kidney injury (CA-AKI) is the most accepted term given by KDIGO, replacing the earlier use term contrast induced nephropathy. ACR (American college of radiology) & ESUR (European society of urogenital radiology) proposed the term CA-AKI (Contrast associated acute kidney injury) to any AKI occurring 48hr after contrast administration. It is synonymous with PC-AKI (Post contrast AKI) which is a radiological term.

Incidence of CI-AKI is high in patient of CKD ranging from 10-40% in few studies. Even mild CI-AKI increases hospital stay, cost, mortality & morbidity. Few patients require RRT & one third of recovered patient may develop CKD in few years. Typically Sr. creatnine rises within 48hr after contrast exposure, peaks 3-5 days & return to base line in 7-10 days.

Definition:

According to ESUR contrast media safety committee guideline, contrast induced Acute kidney injury is defined as increase in sr. creatinine by 0.5mg/dl or more than 25% from base line within 72hrs after administration of contrast.

According to KDIGO (2012) CI-AKI is- An absolute increase in sr. creatinine of 0.3mg/dl within 48hr after contrast exposure or relative increase in sr. creatnine of 50% from base line within 7day. There should not be any influence of nephrotoxic drugs, surgery, septicemia or any other cause of AKI.

Pathogenesis: remains unclear. It involves direct cytotoxicity to contrast causing cellular damage to tubular epithelial cells & endothelial cells. Increase in blood viscosity causes decrease in medullary blood flow & reduction in eGFR. Excessive ROS production causes oxidative stress & inflammatory response leading to renal dysfunction. Recent study shows role of micro-RNA in apoptosis & cellullar damage.

Risk score for prevention of CI-AKI:

Patient related risk factors are Low eGFR, Diabetes, advance age, hypotension, dehydration anaemia, CHF etc. While contrast related risk factors depends on types, dose of contrast & route of administration. High osmolar contrast is highly nephrotoxic while LOCM (Low osmolar contrast media) is non inferior to IOCM (ISO- osmolar contrast media) in most of the studies. Higher & repeated dose of contrast within short period of time is related to higher incidence of CI-AKI. In one meta-analysis by Mccullogh et.al IOCM (ISO-osmolar contrast media) has lower chances of CI-AKI in patient of CKD with Diabetes as compared to LOCM.

Now a days different risk scores are available to assess the risk of CI-AKI. Mehran risk score is very popular in patient going for PCI to assess the risk of CI-AKI, unfortunately it is not validated for intravenous contrast administration. It includes eight variables (hypotension, IABP, CHF, Age >75yr, Anaemia, Diabetes, contrast media volume, sr. creatinine>1.5 /e GFR<60). Higher the risk score more will be chances of CI-AKI & risk of dialysis.

Management:

There is no specific treatment of CI-AKI. So, prevention forms the foundation of effective management. First assess the risk & benefit of contrast administration, review the indication & explore if any alternative imaging available (for example Non contrast MRI, CO2 based angiography). Educate & counsel high risk patient regarding nephrotoxicity, risk factors & chances of CI-AKI. Prior to contrast adjust medication, stop nephrotoxic drugs & hold diuretics if possible. If patient is on metformin stop on the day of exposure to contrast & restart after 3-4 days, because it increases risk of lactic acidosis if AKI occurs. At present there is no guideline about stopping or reducing dose of ACEI/ARB. STATIN should be continued, it may be beneficial according to some studies. Use LOCM or IOCM, minimise the dose of contrast and avoid multiple repeated doses within 72hr.

A simple way to calculate the MAXIMUM DOSE of contrast is:

1) 5x wt (kg)/sr.creat
2) 4x e GFR
3) < 100ml if significant CKD & PCI planned.

REMEDIAL IV Trial (2023) is a contrast sparing trial, in which STEMI & NSTEMI patient were LVEDP guided hydrated & contrast is given by divert system, shows less chances of AKI as compared to control group.

Hydration therapy: is only proven effective preventive measures as compared to other pharmacological agents, none of which is proven effective in meta-analysis. Hydration improves renal blood flow, dilute contrast media, decreases activation of RAS, & reduces secretion of ADH. Intravenous hydration is preferred, because of difficulty in monitoring oral hydration. At present there is no consensus available regarding types of fluid, rate, volume & time of administration of fluid.

Individualized or personalised hydration is preferred because of different clinical scenario to minimise the side effects of fluid overload & symptomatic heart failure. POSEIDON TRIAL is a land mark trial, in which LVEDP guided fluid was administrated to prevent the risk of CI-AKI. AMACING trial is a negative trial in which No hydration was non inferior to hydration in patient of chronic kidney disease,also there was less risk of symptomatic heart failure with No hydration.

N- Acetyl cysteine (NAC): is scavenger of free radicals & antioxidant given oraly in a dose of 600mg BID, 24hr before & on the day of procedure along with hydration. Recent RCT & meta-analysis failed to prove its beneficial effect. PRESERVE Trial (2018) shows NAC is not superior to placebo also there was no benefit of sodium bicarbonate over sodium chloride in preventing, risk of CI-AKI.

STATINS- In recent study statin reduces CI-AKI by decreasing free radical oxygen production but general recommendation for statin is difficult, as these studied patients were already on statin. Also, study didn't include patient with eGFR<45.

SGLT2 inhibitors might prevent CI-AKI because of its anti-inflammatory & antioxidative effect. Data in human studies are awaited, Vit C, theophylline, diuretics and other pharmacological agents have no proven beneficial effect. Recent published trial, NITRATE CIN in 2024 shows inorganic nitrate decreases risk of CI-AKI as compared to placebo.

Hemodialysis:

Contrast media are dialyzable, but prophylactic HD cannot reduce the chance of CI-AKI so prophylactic HD is not recommended. Approx 12% patient of advance renal failure with diabetes may require HD, out of which 18% may require lifelong RRT. Those patients already on maintenance HD, doesn't require extra or change in HD schedule after contrast administration unless there is hyperkalemia or fluid overload.

Conclusion:

Incidence of CI-AKI is of great importance in patient of ckd & diabetes.

As there is no specific treatment of CI-AKI, a preventive approach is the hallmark of strategic management. Assess risk & benefit of contrast media, look for alternative, & identify high risk patient. Minimise dose of contrast media, hydrate well prior to exposure. Adjust medication& avoid nephrotoxic drugs. Prophylactic HD is not recommended. None of pharmacological agent have been proven efficacious in study though NAC is popular. Inorganic nitrates have some beneficial effect on preventing CI-AKI.

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