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Case Report

A Case Report on Contrast Media Induced Acute Kidney Injury after Percutaneous Coronary Intervention

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ABSTRACT

Contrast Media-Induced Nephropathy (CIN) is a well-documented phenomenon characterized by a sudden decline in renal function following the administration of contrast medium. This article presents a case study of a 58-year-old male patient who developed CIN after undergoing emergency Coronary Angiography due to Acute STEMI with RVMI. The patient's renal parameters deteriorated rapidly, CIN is a multifaceted condition with various risk factors, including pre-existing renal impairment, diabetes mellitus, advanced age, and the use of specific medications. Its Pathogenesis involves direct Cytotoxicity, renal Vasoconstriction, and Oxidative stress. Early diagnosis is of paramount importance, relying on the detection of an increase in serum creatinine within 24 to 48 hr following contrast exposure. The management primarily revolves around hydration and the prudent avoidance of Nephrotoxic agents. This article underscores the significance of prevention strategies, which should encompass meticulous hydration, the judicious minimization of contrast usage, and the avoidance of nephrotoxic medications whenever feasible.

Keywords: Contrast media, Acute kidney injury, Tubular necrosis, Vasoconstriction.

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INTRODUCTION

Contrast Media-Induced Nephropathy (CIN) refers to the abrupt decline in renal function following the administration of contrast medium, without any other underlying cause.[1] A significant increase in serum creatinine concentration, exceeding 0.5 mg/dL (44 mol/L) or 25% above baseline within 48 hr, signifies deterioration in renal function.[2] The primary risk factors recognized for CIN include pre-existing renal impairment and diabetes mellitus. Additionally, other factors contribute to this risk, such as Anemia, hypercholesterolemia, peripheral vasculopathy, recent exposure to parenteral contrast medium within 72 hr, the use of diuretics, Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), and other Potential Kidney-damaging medications, along with the administration of a large volume of contrast medium. Advanced age, particularly beyond 75 years, also constitutes another risk factor.[3] The renal failure that ensues following exposure to radiocontrast agents is typically non-oliguric in nature. Creatinine levels rise within 48 hr of exposure, peak around 4-5 days later, and then gradually return to baseline within 7-10 days.[4] Approximately 10% of affected individuals may require dialysis, while more than 75% are expected to experience a complete recovery.[5] Notably, over the past two decades, clinical experience has shown a decrease in the incidence of contrast-induced nephropathy with the use of low- and Iso-Osmolar contrast media.[6] Iodixanol, a new-generation Iso-Osmolar contrast agent, has demonstrated safety even when administered to high-risk patients.[7] The Pathogenesis of CIN involves three pathways: direct effect, indirect effect, and the production of Reactive Oxygen Species (ROS). Direct effects encompass the cytotoxicity of Contrast Media (CM) on renal nephrons, leading to tubular damage and cell apoptosis or necrosis.[8] Indirect effects involve changes in renal Hemodynamic induced by Contrast Media (CM), resulting in intrarenal vasoconstriction and contributing to Medullary Hypoxia. Mediators like renin, Angiotensin II, and endothelin become more vasoconstrictive, while Nitric Oxide and PGI2 become less

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Vasodilatory, mediating this pathway.[9] Finally, Contrast Media (CM) can generate Reactive Oxygen Species (ROS) and Reduce Antioxidant enzyme activity through complex Mechanisms, ultimately leading to impaired renal function.[10]

Figure 1;[11] Illustrates a schematic representation of the impact of increased viscosity on blood and urine flow, leading to extended exposure to contrast media. This prolonged exposure directly harms renal tubular epithelial cells and vascular endothelial cells. Due to the presence of contrast medium, necrotic tubular epithelial cells become detached from the basement membranes and migrate into the urine space. Consequently, they obstruct the lumen, resulting in elevated tubular pressure and reduced glomerular filtration rate (GFR). Simultaneously, endothelial cell dysfunction consistently leads to a decline in vasodilatory effects and an increase in vasoconstriction, resulting in constriction of the vasa recta and ischemic conditions within the medulla. Importantly, all these pathways are associated with oxidative damage, inflammation, epigenetic regulation, and apoptosis.[11]

CASE PRESENTATION

A 58-year-old male patient presented with a sudden onset of sweating and chest pain radiating to the left arm with a past medical history of Type 2 Diabetes mellitus for 6 years, on treatment with Tablet. Metformin 500 mg OD, and had a social history of Smoker and Alcoholic for more than 8 years. The patient was diagnosed with Acute STEIWMI (ST-Elevation Inferior Wall Myocardial Infarction) + RVMI (Right Ventricular Myocardial Infarction) with mild LV (Left Ventricle) dysfunction and severe RV dysfunction. He was treated with the following medications as a stat, Tablet Ticagrelor 180 mg, Tablet Aspirin 300 mg, and Tablet Rosuvastatin 40 mg.

He underwent an emergency Coronary Angiogram, which revealed 100% occlusion of the Right Coronary Artery (RCA). Primary PTCA (Percutaneous Transluminal Coronary Angioplasty) with the placement of 2 drug-eluting stents in the RCA was performed. The treatments given to the patient are mentioned below in Table 1.

The following Table 2 shows the trend of his renal parameters from the day admission to day 03.

Interpreting the data, it is evident that both blood urea and creatinine levels have increased significantly from their respective normal ranges over the five-day period. This could indicate a potential issue with Kidney function. He was diagnosed with Contrast Media induced Acute Kidney Injury and was continued on intravenous 0.9% normal Saline and N-Acetylcysteine infusions on day 03 "After administering Normal Saline and N-Acetylcysteine, the Blood Urea and Creatinine levels were slightly decreased on day 04 and 05." However, the patient succumbed and died due to severe RV dysfunction and post Myocardial Infarction (MI) Ventricular Tachycardia on day 6.

DISCUSSION

Intravenous administration of Contrast continues to be an important and often preventable cause of hospital-acquired ARF. Although Various Definitions of CN appear in the literature, it is commonly defined as an Acute decline in renal function after the Administration of Intravenous Contrast agents, and in the absence of other causes. Patients with CN typically present with an acute rise in Serum Creatinine 24-48 hr after the contrast study. Serum Creatinine usually peaks at 3-5 days and usually returns to normal after 7-10 days. Almost all patients revert to normal renal function, and dialysis is rarely required.[12]

In the case described, acute renal failure within a day of Contrast Medium administration in a patient with diabetes mellitus and there is no pre-existing renal disease. The patient had not received any other Nephrotoxic medication before the onset of acute renal failure. Hence, no other cause of acute renal failure could be identified.

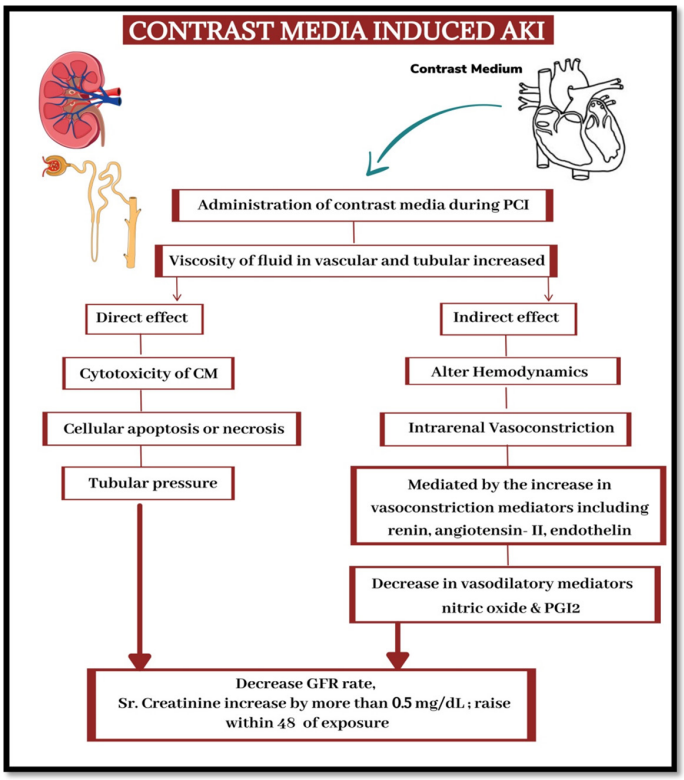

Figure 1: Illustrates a schematic representation of the impact of increased viscosity on blood and urine flow, leading to extended exposure to contrast media. This prolonged exposure directly harms renal tubular epithelial cells and vascular endothelial cells. Due to the presence of contrast medium, necrotic tubular epithelial cells become detached from the basement membranes and migrate into the urine space. Consequently, they obstruct the lumen, resulting in elevated tubular pressure and reduced Glomerular Filtration Rate (GFR). Simultaneously, Endothelial Cell Dysfunction consistently leads to a decline in vasodilatory effects and an increase in Vasoconstriction, resulting in constriction of the vasa recta and ischemic conditions within the medulla. Importantly, all these pathways are associated with Oxidative Damage, Inflammation, Epigenetic Regulation, and Apoptosis.[11]

Table 1: Treatment chart.

Medication	Dosage	Frequency	Day 01	Day 02	Day 03	Day 04	Day 05
Tablet Ticagrelor	90 mg	Twice Daily (BID)					
Tablet Aspirin	75 mg	Once Daily (OD)					
Tablet Rosuvastatin	40 mg	Once Daily (OD)					
Tablet Trimetazidine	35 mg	Twice Daily (BID)					
Tablet Nicorandil	5 mg	Twice Daily (BID)					
Tablet Nitroglycerin	2.5 mg	Twice Daily (BID)					
Tablet Paracetamol	650 mg	Three Times Daily (TDS)					
Tablet Alprazolam	0.5 mg	Once Daily (OD)					
Tablet Dapagliflozin and Sitagliptin	10 mg/100 mg	Once Daily (OD)					
Tablet Glimepiride	1 mg	Twice Daily (BID)					
Tablet Metformin Hydrochloride (SR)	500 mg	Twice Daily (BID)					
Normal Saline	0.9%	Continuous infusion	X	X			
N-Acetylcysteine	200 mg/mL	Continuous infusion	X	X			

Table 2: Renal parameters: This table presents renal parameters measured over a period of five days, with corresponding values for each day, as well as the normal reference range for each parameter.

Renal parameters	DAY-01	DAY-02	DAY 03	DAY 04	DAY 05	Normal range
Blood urea	33 mg/dL	62 mg/dL	93 mg/dL	86 mg/dL	72 mg/dL	19-43 mg/dL
Creatinine	0.8 mg/dL	2.3 mg/dL	3.8 mg/dL	3.5 mg/dL	2.8 mg/dL	0.6-1.2 mg/dL

Two of the most widely used new Contrast Media, Iodixanol (a Non-Ionic, Iso-Osmolar Dimer) and Iohexol (a Non-Ionic Low-Osmolar Monomer) have consistently been found adequately safe, both in Intraarterial and in Intravenous administration to high–risk patients. Chalmers and Jackson found that Iodixanol was Less nephrotoxic than Iohexol when administered intra-arterially to patients with renal impairment. This superior safety of Iodixanol as compared to Iohexol was confirmed in the NEPHRIC study.[13]

Saline Hydration has been shown to be effective in preventing CN, and other preventive strategies have also been used to reduce the chance of developing CN. Saline Hydration may correct any Pre-Existing Dehydration, and may also counter the osmotic diuresis attributable to the contrast agent.[14]

Liu, Raymond, *et al*. analysis suggests that administration of NAC around the time of contrast administration prevents renal injury. Patients treated with NAC had both a lower mean Creatinine (difference in mean ΔCr=−0.27 mg/dL; 95% CI, −0.43 to −0.11) and a reduced risk of developing CIN compared with control patients (RR, 0.43; 95% CI, 0.24 to 0.75). In their sensitivity analyses, the use of NAC was protective against renal injury in almost all subgroups that included at least 3 studies.[15]

In the majority of the cases, the renal function starts to improve within 3 to 7 days of contrast exposure and returns to normal by 3 weeks. Less than 1 percent of patients require dialysis on short-term or long-term.[7]

CONCLUSION

In conclusion, Patients having cardiac catheterization continue to have a high prevalence of contrast-induced acute kidney injury. An efficient preventive strategy should focus on IV fluid hydration and minimizing contrast administration. It is essential to maintain appropriate volume expansion during the peri-procedural period, reduce the volume of contrast media utilization, and avoid using Nephrotoxic drugs whenever possible since contrast-induced nephrotoxic cannot be effectively treated.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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