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Intravenous Iron Induced Pigment Nephropathy Leading To Severe Acute Necrosis: A Rare Cause of AKI

[1]Sandeep Kumar Garg, Consultant Nephrologist and Head, Department of Nephrology, Nutema Hospital, Meerut, India

[2]Shweta Garg, Consultant Pathologist and Head, Department of Pathology, Nutema Hospital, Meerut, India

[3]Kartikay Garg, MBBS, Maulana Azad Medical College, Delhi, India

Corresponding Author: Sandeep Kumar Garg, Consultant Nephrologist and Head, Department of Nephrology, Nutema Hospital, Meerut, India

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Abstract

Ferric carboxymaltose (FCM) is widely used for the treatment of iron deficiency anemia. Although generally safe, severe adverse reactions including muscle injury and pigment induced acute kidney injury (AKI) are rare but lead to clinically significant complications whenever they occur

Case Summary: We present a middle aged male who developed severe rigor, diffuse myalgia, dark colored urine, and oliguria on administration of ferric carboxymaltose which was administered by the local practitioner without any lab investigation beforehand. On admission to our center he presented with aforementioned symptoms pointing towards advanced renal failure, hyperkalemia, and with markedly elevated CPK and LDH levels, signifying rhabdomyolysis. Urine microscopy showed no RBCs but the dipstick test was positive for blood, signifying myoglobinuria. Serum myoglobin was elevated with normal haptoglobin.

The patient required multiple sessions of hemodialysis. A renal biopsy was performed to know the cause of persistent anuria which confirmed acute tubular necrosis with pigment-laden epithelial cells. He gradually regained urine output by day 10 and achieved complete renal recovery by 3 weeks.

Keywords: Acute Kidney Injury, Ferric Carboxymaltose, Haptoglobin Myoglobinuria

Introduction

Intravenous iron preparations, including ferric carboxymaltose (FCM), have become one of the go to therapies for moderate to severe iron deficiency anemia, also preferred when oral iron is ineffective or poorly tolerated. Although intravenous iron is generally considered safe, rare complications such as

hypersensitivity reactions, hypophosphatemia, rhabdomyolysis, and pigment-induced acute kidney injury (AKI) have been reported.

Case Presentation

A middle-aged male with no known comorbidities had complaints of malaise and generalized weakness. A local practitioner administered intravenous ferric carboxymaltose without prior laboratory evaluation, including hemoglobin, ferritin or iron studies.

Within hours of infusion, the patient developed severe rigor lasting 4-6 hours, requiring multiple blankets for warmth, he did not seek medical attention initially.

By the next morning, he experienced intense myalgia involving all major muscle groups, along with dark-colored urine. By evening, he noted progressive oliguria. A local physician detected deranged renal parameters, but despite symptomatic treatment, renal function did not improve. He was subsequently referred to our center (Nutema Hospital).

Clinical Findings

When the patient had presented to our center, his symptom complex & ECG findings pointed towards Severe renal dysfunction, Hyperkalemia, Oliguria progressing to anuria. However his Blood pressure and other vitals were stable

Investigations: A series of blood and urine investigations were performed.

Biochemistry

Table 1: Laboratory Investigation During Hospital Course

Parameter	Value on Admission	Peak Value	Discharge	Reference Values
Serum Creatinine (mg/dl)	3.8	6.9	1.5	0.6-1.2
Serum Urea (mg/dl)	92.0	208	30	7-20
Serum Potassium (mEq/L)	5.6	5.8	4.1	3.5-5.0
Sodium (mEq/L)	131	134	130	135-145
Bicarbonate (mmol/L)	15	24	22	22-28
LDH (U/L)	12680	12680	160	140-280
CPK (U/L)	4540	5000	150	<200
Serum Myoglobin (ng/mL)	4500	5200	100	<70
Serum haptoglobin (mg/dL)	170	180	150	30-200
Hb (g/dL)	7.4	12.8	12.4	12-17
TLC (cells/microL)	17,300	18,100	10,600	4k-11k
DLC (cells/microL) (N/L/M/E/B)	98/01	98/01	75/13/04	40-75/20-50/2-10/1 6/<1
Platelets (cells/microL)	2.15	4.76	4.2	1.5-4.5 (Lakhs)
Ca2+ (mg/dL)	8.0	9.4	9.0	9-11
AST (U/)L	3015	3015	35	0-45
ALT (U/L)	793	670	28	7-50
T. Bilirubin % (mg/dL)	0.86	0.9	0.53	0.5-1.1

A. Urine Analysis

Dipstick: positive for blood

However, Microscopy showed No RBCs

This combination along with serum myoglobinemia and normal serum haptoglobin is suggestive of myoglobinuria.

B. Renal imaging

Ultrasound: normal sized kidneys, maintained corticomedullary differentiation which ruled out pre-existing chronic kidney disease.

C. Renal Biopsy was performed on day 9 of treatment in view of anuria, which had the following impression:-

1. Acute tubular necrosis
2. Pigment laden tubular epithelial cells
3. No evidence of glomerulonephritis or interstitial nephritis

Thus a Diagnosis of pigment induced AKI secondary to rhabdomyolysis is proven.

Management

On presentation, the patient's Serum potassium was 5.6 mEq/L along with tall peaked T waves on ECG, indicating hyperkalemia and worsening renal failure for which hemodiaysis was initiated. This was followed by Alternate day hemodialysis for 8-9 days due to hypercatabolic state

Patient was given Aggressive hydration and supportive therapy along with Monitoring of electrolytes, CPK, and renal parameters.

Renal biopsy to establish definitive etiology was performed on day 9

Hospital Course

Patient remained anuric for 8 days for which Renal Biopsy was performed.

- Gradual improvement in clinical status with supportive management was eventually achieved.
- Urine output started on day 10 ultimately followed by Steady improvement in renal function with decreasing creatinine levels, as a result of which No further dialysis was required after renal recovery began.

Eventually, Patient was Discharged with normal renal function after 3 weeks

Outcome

The patient achieved complete renal recovery with normalization of kidney function at 3 weeks. Follow up at 1 and 3 months showed stable renal function with no residual impairment.

Discussion

This case emphasizes several important clinical lessons. Intravenous iron can rarely induce rhabdomyolysis [1,2,7], potentially through hypersensitivity or direct muscle toxicity. due to which it should not be given indiscriminately. However, no studies have been present linking FCM specifically to these complications.

Rhabdomyolysis leads to myoglobin release, causing tubular obstruction [4,5,6], oxidative damage, and acute tubular necrosis, Which manifests as typical dark coloured urine with weakness. Diagnostic clues include Elevated CPK and LDH, Myoglobinuria with dipstick positive for blood but no RBCS, Elevated serum myoglobin[5,6]

Early volume resuscitation and, when needed dialysis can improve outcomes; in myoglobin cast AKI series many required dialysis but recovered.[3,6]

Dialysis is crucial for managing hyperkalemia, acidosis, and toxin clearance in pigment nephropathy. This case highlights the importance of urgent dialysis in acute settings. If oliguria persists or diagnosis is unclear, renal biopsy may reveal myoglobin casts and guide therapy and prognosis.[3,6]

Full recovery is achievable with timely intervention, although some cases progress to CKD.[3,6]

This case highlights the importance of Proper pre-infusion screening before IV iron therapy, Avoidance of empiric IV iron without laboratory confirmation, Educating practitioners about potential severe adverse effect

Conclusion

Ferric carboxymaltose induced rhabdomyolsis leading to pigment nephropathy and acute tubular necrosis is a rare but serious complication. Early recognition, aggressive supportive care and timely dialysis are essential for renal recovery. Clinicians must ensure appropriate evaluation before administering IV iron to prevent such avoidable events.

Renal Biopsy should be considered in cases of absence or in irregular clinical improvement to determine definite causes.

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