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Case Report

Acute Kidney Injury and Anemia in a Patient with HTN, found to be severe rheumatoid heart disease complicated by endocarditis with vegetation

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Abstract
We present a case of a 41-year-old woman with a history of hypertension and recent NSAID use, who developed acute kidney injury (AKI) on top of chronic kidney disease (CKD), (that is not recognized previously) , normocytic anemia, and suspected heart failure. The case highlights the potential risks of NSAID use in patients with underlying renal disease and the need for early recognition and intervention. Anemia complication on heart , fever can indicate serious undergoing disease , case found to be severe rheumatoid heart disease complicated by endocarditis and highly mobile valve vegetation.

Case Presentation

Initial Presentation (December 30, 2023)

A 41-year-old woman, a known case of hypertension for six years, presented with bilateral lower limb swelling more rt ankle. Despite being on oral iron therapy, her hemoglobin (Hb) was 8.4 g/dL. Her glomerular filtration rate (GFR) had declined to 53 mL/min/1.73 m². She had been taking NSAIDs for four months for right foot swelling, previously diagnosed as gout in a private clinic and xr done showed bone spire .

Medical and Social History:

Past medical history: Hypertension, chronic anemia not responding to oral iron , gout on NSAIDs.

Medications: NSAIDs for four months, oral iron, antihypertensive medication.
Allergies: No known allergies.
Social history: Works in a salon, single.

Examination in initial visit :

Vital signs:
Temperature: 36.8°C
Heart rate: 97 bpm
Blood pressure: 117/68 mmHg
SpO_2: 100%
Weight: 61.6 kg

Physical exam:

Bilateral hand dermatitis.
Cardiovascular: S1, S2, S3 present.

Respiratory: Bilateral lower zone crepitations, suggestive of pulmonary congestion or effusion.
Peripheral: Bilateral non-pitting lower limb edema up to the ankles more the right foot

Initial Impression and Referral:
Anemia with possible heart failure, referred for intravenous iron therapy and further evaluation for acute kidney injury. Advice was given to stop NSAIDs
The patient reported went to emergency department (ED) where she was discharged without assessment or documentation.

Follow-Up Presentation (January 14, 2024)
The patient returned with worsening symptoms, including severe fatigue, dizziness, one episode of fainting, worsening lower limb swelling, and a further drop in Hb to 8.1 g/dL. Her GFR had declined further from 53 to 33 mL/min in two weeks.

Additional Findings:
Electrolytes and Renal Function:
Sodium: 129 mmol/L (previously normal, now mildly low).
Potassium: 5.4 mmol/L (mild hyperkalemia).
Bicarbonate: 17 mmol/L (low).
Urea: 11.6 mmol/L (elevated).
Creatinine: 161 µmol/L (elevated).
eGFR: 33 mL/min/1.73 m².(declined)

Infection Markers:
CRP: 53.2 mg/L (elevated).
Urinalysis: White blood cells, bacteria, and blood present, suggestive of a urinary tract infection (UTI).
Uric acid: 530 µmol/L (elevated).

Examination:
General: Appeared ill, pale, with an earthy look
Cardiovascular: S1, S2, S3 present.
Respiratory: Bilateral lower zone crepitations.
Peripheral: Lower limb edema up to the ankles.

Referral to ED done with clear reason for referral to be admitted under medical team for anemia workup , AKI on top of CKD , and to rule out cardiac cause for her cvs examination .

ED Management (January 14, 2024):
Intravenous iron (ferric carboxymaltose).
IV fluids (0.9% sodium chloride).
IV paracetamol for symptomatic relief.
Chest X-ray: Mild prominence of perihilar bronchovascular markings, no consolidation.
The patient was admitted under medical team due to suspected decompensated heart failure secondary to anemia and AKI.

Hospital Admission (January 15, 2024)
The patient was admitted under the medical team with a diagnosis of AKI secondary to NSAID use on top of CKD.

Assessment and Plan:
1. Acute kidney injury on CKD:
Likely hypertensive nephropathy worsened by NSAID use.
Nephrology consultation arranged.
Avoid nephrotoxic medications.
Continue IV fluids cautiously.

2. Normocytic normochromic anemia (Hb 7.9 g/dL):
Monitor hemoglobin and ferritin.
Consider further iron supplementation and possible transfusion if Hb drops further.

3. Mild hyperkalemia (K^+ 5.4 mmol/L):
Treated with sodium polystyrene sulfonate.
Monitor potassium levels.

4. Mild hyponatremia (Na^+ 128 mmol/L):
Monitor sodium levels and adjust fluids accordingly.

5. UTI:
Started on IV ceftriaxone for seven days.

6. Suspected heart failure (NT-proBNP 1150 pg/mL):
Echocardiography:
TTE (15/01/2024):
– Normal global systolic LV function.
– Biplane LVEF is calculated at 58 %.
– No regional wall motion abnormality.
– Aneurysmal LA dilatation.
– Severe mitral regurgitation.
– Mild mitral valve stenosis.
– There is mild mitral valve thickening {MVTHICKSUB:MV Thickening subcatalog}.
– Mild aortic valve regurgitation.
– The aortic valve findings are consistent with rheumatic disease.
– Pulmonary artery pressure is normal

Hospital Course (January 16, 2024)
The patient developed a low-grade fever (37.8°C).
Blood culture reported Gram-positive cocci in pairs and chains (one bottle).
Urine WBC count: 12
No respiratory complaints.
Plan:
Continue IV ceftriaxone.
Repeat blood cultures.
Monitor inflammatory markers (CBC, CRP, procalcitonin).
Consider transesophageal echocardiography (TEE) to evaluate for infective endocarditis (IE), given anemia and borderline splenomegaly.

Discussion
This case illustrates several critical issues:
1. NSAID-Induced Acute Kidney Injury:
The patient had CKD (likely hypertensive nephropathy).
Prolonged NSAID use likely precipitated AKI, leading to a rapid decline in renal function.
The case underscores the importance of avoiding NSAIDs in CKD patients.

2. Decompensated Heart Failure Secondary to Anemia:
The presence of S3 heart sound and bilateral crepitations raised suspicion for heart failure exacerbation.
The patient's anemia (Hb 7.9 g/dL) may have contributed to high-output cardiac failure.
She received 2 units PRBC.

Echo done and cardiology assessment
Admitted initially with fever, found to have strept. gordonii bacteremia
TEE 18/01 Echo done showed mitral valve vegetations
Follow up with Cardiology clinic, after completion of the antibiotics for MV replacement.

3. Infectious Complications:
The Gram-positive cocci in blood culture raised concerns for infective endocarditis.
The borderline splenomegaly and persistent anemia may indicate an occult infection or chronic inflammatory response.

Antimicrobials:
Vancomycin 16/01 - 18/01
Ceftriaxone 18/01
4. Hyponatremia and Hyperkalemia:
Hyponatremia (Na^+ 128 mmol/L) was likely secondary to volume overload or CKD-related dysregulation.

Hyperkalemia (K^+ 5.4 mmol/L) required monitoring due to potential arrhythmic risks.

Us abdomen CONCLUSION:
No ultrasound evidence of obstructive uropathy.
Gallbladder polyp.
NM Whole body FDG PET CT

Conclusion
Increased metabolic activity at the apical and basal lateral wall with mild metabolically active

thoracic nodes. These are keeping with an inflammatory process.
Diffuse splenic metabolic activity can be seen with anaemia, sepsis (or combination of both).
No pathological metabolic activity in the cardiac valves.

Conclusion

This case highlights the complex interplay of NSAID-induced nephrotoxicity, anemia-related heart failure, CKD progression, and possible infective endocarditis. It emphasizes the need for early recognition and avoidance of nephrotoxic agents in CKD patients and the importance of comprehensive evaluation in patients presenting with anemia, kidney injury, and systemic symptoms.

Key Takeaways:

NSAIDs should be used cautiously in any patient and for short time, more caution in CKD patients.
Anemia can contribute to heart failure.
Persistent anemia with fever requires evaluation for heart condition, chronic infection or endocarditis.
Early intervention and close monitoring can prevent complications.