WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.084
Volume 12, Issue 17, 751-756. Case Report ISSN 2277– 7105

A COMPLEX CONFLUENCE: PARALYTIC ILEUS, ACUTE KIDNEY INJURY, AND HYPOKALEMIA – A CASE REPORT

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Article Received on
10 August 2023,

Revised on 30 August 2023,
Accepted on 20 Sept. 2023

DOI: 10.20959/wjpr202317-29720

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ABSTRACT

An 81-year-old male patient presented with complaints of abdominal distension lasting for 20 days, accompanied by nausea persisting for the same duration. Additionally, the patient had been experiencing bilateral lower limb swelling for the past month and appeared drowsy. Following admission, a comprehensive set of investigations was conducted, revealing elevated total white blood cell counts and abnormal renal function test results. Given the patient's deteriorating condition and drowsiness, he was admitted to the Medical Intensive Care Unit (MICU). Subsequent diagnosis indicated that the abdominal distension was a result of paralytic ileus, which was accompanied by acute kidney injury and hypokalemia. With appropriate treatment, the patient's symptoms gradually improved, leading to his eventual discharge. This case serves to highlight the intricate interplay between paralytic ileus, Acute Kidney Injury (AKI), and hypokalemia, as further elucidated in the subsequent case study.

KEYWORDS: Paralytic ileus, Hypokalemia, Acute kidney Injury, Abdominal distension.

INTRODUCTION

Hypokalemia has the potential to cause diminished neural transmission to and within the enteric nervous system (ENS), modifying the typically synchronized reflexes and rhythmic patterns of gastrointestinal (GI) motility. This mechanism serves as a connecting factor between potassium levels and the development of paralytic ileus. Paralytic ileus, acute kidney injury (AKI), and hypokalemia are interrelated disorders that can pose considerable obstacles in diagnosis and treatment. Grasping the interconnections among these ailments is vital for delivering prime patient care. This case study endeavors to offer an intricate narrative of how

a patient with these intertwined conditions was managed, accentuating the intricacies entailed.

CASE REPORT

The 81-year-old male patient presented with a history of abdominal distension spanning 20 days. Alongside this, he reported persistent nausea over the same period. It is important to note that the patient had been experiencing swelling in both lower limbs for the past month, and there was a noticeable decline in his level of alertness. Approximately a month ago, the patient was in a seemingly normal state, but then developed insidious-onset bilateral lower limb swelling. He sought medical attention from a local doctor nearby and received conservative management, resulting in reduced symptoms. However, the patient's condition subsequently progressed, leading to abdominal distension for the past 20 days. The patient also reported not having passed stools for the same duration and complained of nausea lasting 20 days. Additional symptoms included loss of appetite and reduced urine output over the past 5 days.

Physical examination: Pulse:102bpm, BP:150/90mmhg, RR:20cpm, B/L: pitting lower edema, CVS: S1S2 heard , no murmurs, CNS: drowsy , obey commands , RS: B/L entry present, PA: hard , bowel sound absent , guarding present ,rigidity present , stool not present, flatus tube inserted showed no air bubble, PR EXAMINATION: sphincter tone decreased , no fecal staining.

Laboratory investigations: HB:12.3g/dl, TLC: 14200cells, PLATELET:299*10^6/mm^3, HCT;37.0%, RBS:3.28*10^6/mm^3, K:3.99mEg/l, CL: 101.34mEq/l, CREATININE: 3.86mg/dl, INDIRECT BILIRUBIN: 0.2mg/dl, TOTAL BILIRUBIN:0.5mg/dl, DIRECT BILIRUBIN:0.2mg/dl, TOTAL PROTEIN:6.5mg/dl, ALBUMIN:3.1g/dl, GLOBULIN:3.4g/dl, MDCT OF ABDOMEN AND PELVIS DEMONSTRATES: MINIMAL BILATERAL PERINEPHRIC FAT STRANDING MILD PROSTOMEGALY, LEFT MILD TO MODERATE PLEURAL EFFUSION WITH BASAL ATELECTASIS, MILD PERICARDIAL EFFUSION. TRANS THORACIC ECHO: NORMAL CHAMBERS, MILD MR PRESENT, TR GRADE 1, MODERATE PAH, CONCENTRIC LVH, NORMAL LV SYSTOLIC FUNCTION, LVEF 60%

Treatment given: INJ.VASIZONE (Cefoperazon and Tazobactum) 1.5g1V 1-0-1, INJ.METROGYL(Metronidazole) 500mg IV 1-1-1, INJ. TRAMADOL 50mg IN 100mlNS

IV 1-1-1, TAB. PRESMOVAC (Prucalopride) 2mg 1-0-1, SYP. POTCLOR 5ml P/O 1-1-1, INJ.HUMAN ALBUMIN IV, INJ. TELMIGET (Telmisartan) 40MG 0-0-1 P/O, IVF NS/DNS/RL 50ml/hr, INJ. PAUSE (Tranexamic Acid)500mg 1-1-1, ONABET CREAM (Sertaconazole)1-1-1 FOR LOCAL APPLICATION, NEB DUOLIN (Levosalbutamol + Ipratropium)1-1-1-1

Following admission, an extensive range of diagnostic assessments revealed elevated total white blood cell counts and anomalies in renal function tests. Due to the patient's deteriorating condition and somnolent state, the decision was made to transfer him to the Medical Intensive Care Unit (MICU). A pulmonologist was consulted after a CT scan of the abdomen and pelvis displayed mild to moderate left pleural effusion, minimal right pleural effusion with band atelectasis, and mild pericardial effusion. The recommended actions were duly pursued, and regular evaluations were conducted. In light of hypertension, a physician's input was sought and the prescribed guidance was adhered to. Anesthesia consultation was sought due to decreased SPO2 levels, resulting in the initiation of oxygen therapy at a rate of 2 liters per minute via nasal prongs. Continuous SPO2 monitoring was implemented. A nephrologist provided input regarding acute kidney injury (AKI), leading to ongoing monitoring and adherence to the provided advice. The patient was diagnosed with proteinuria and hypoalbuminemia, prompting a recommendation for albumin transfusion. Subsequently, the patient received 4 units of albumin through transfusion. A renal biopsy was suggested to ascertain the underlying cause of proteinuria, although the patient declined this procedure. Elevated levels of urine PCR indicated significant proteinuria. Consultation with a dermatologist was sought for diaper rash concerns, which was diagnosed as tinea cruris, and the recommended treatment was carried out. Given the patient's oxygen therapy at 2 liters per hour and the development of productive cough, the pulmonary status was reviewed daily and symptomatic treatment for the cough was administered. The patient demonstrated symptomatic improvement and was able to maintain oxygen saturation on room air. Consequently, the patient's request for discharge was honored, accompanied by the following recommendations.

Discharge medication: TAB.CEFUZACT(Cefuroxime) 500mg P/O for 5 days, TAB.XEPENTA DSR (domperidone+pantoprazole) 1-0-1 P/O for 1 week, TAB. MAXIVION FORTE(Coenzyme Q10 50 MG+Docosahexaenoic acid 25 MG+Elemental copper 1 MG+Elemental iron 15 MG+Elemental magnesium 10 MG+Elemental selenium 7.6

MCG+Folic acid 0.5 MG+L-arginine 50 MG+Levocarnitine 200 MG+Lycopene 2 MG+Riboflavine 1.7 MG+Thiamine 1 MG+Vitamin A 1250 IU+Vitamin B12 1 MCG+Vitamin C 30 MG+Vitamin D3 120 IU+Vitamin E 15 IU+Zinc oxide 12.5 MG) 0-1-0 P/O for 30 days, MALGIX PROTEIN POWDER P/O in one glass of water/ milk 0-1-0 for 30 days, TAB. CALCI M(Calcium Citrate Maleate, Vitamin D3, Magnesium & Zinc) P/O 1-0-0 for 30 days. ONABET CREAM (Sertacoazole Nitrate) 1-1-1 for local application, TAB. TELEKAST L (levocetirizine and montelukast) 0-0-1 for 5 days P/O, TAB.AB PHYLLINE N (Acebrophylline + Acetylcysteine)1-0-1 for 5 days, NEB FORMONIDE (Formoterol + Budesonide) 0.5MG 1-0-1 for 10 days.

DISCUSSION

Paralytic ileus is a state in which the normal motor functions of the intestines are compromised, typically lacking a mechanical origin. While this condition might resolve on its own, it is grave, and if it persists without treatment, it can lead to fatality, similar to the outcome seen in cases of acute mechanical obstruction.[1] Ileus is categorized into two types: uncomplicated and complicated. Uncomplicated ileus typically lasts for a duration of three days or less and resolves on its own. On the other hand, complicated ileus involves a prolonged halt in gastrointestinal motility, lasting beyond three days. Common symptoms encompass abdominal distention, bloating, nausea, vomiting, pain, and constipation.[2] In the timeline of events, it is noted that the paralytic ileus preceded the acute kidney injury. The fluid sequestration in the bowels would have led to severe dehydration, hyponatraemia and hypokalaemia.[3]

The interrelation among paralytic ileus, AKI, and hypokalemia can be ascribed to several underlying mechanisms. Primarily, the compromised motility of the intestines caused by paralytic ileus can induce fluid buildup and disturbances in electrolyte equilibrium. This encompasses the retention of potassium within the gastrointestinal tract, thereby contributing to the onset of hypokalemia. Furthermore, the absence of bowel movement can impede the elimination of potassium from the body, exacerbating the potassium imbalance.

Secondarily, AKI can play a role in precipitating hypokalemia through diverse pathways. The deterioration in renal function impairs the kidneys' capacity to effectively regulate electrolyte concentrations, including potassium. Reduced renal excretion of potassium can lead to its accumulation in the bloodstream, intensifying the state of hypokalemia. Additionally, AKI

can prompt an increase in urine output due to compromised water reabsorption, further depleting the body's potassium reserves.

Furthermore, the inflammation and systemic response triggered by AKI can disrupt the normal physiological processes responsible for maintaining electrolyte balance. The inflammatory condition can facilitate the movement of potassium from intracellular reservoirs to the extracellular fluid, thereby exacerbating the severity of hypokalemia.

CONCLUSION

To sum up, the interrelated states of paralytic ileus, AKI, and hypokalemia necessitate a thorough strategy for both diagnosis and treatment. Grasping the fundamental mechanisms and effectively managing each ailment is essential for providing the best possible patient wellbeing. A multidisciplinary team collaboration, attentive surveillance of electrolyte disruptions, and focused interventions all assume crucial parts in guaranteeing the effective management and averting complications linked to these intricate disorders.

AKNOWLEDGMENTS

The author would like to thank the Department of Surgery, SS Institute of Medical Science and Research, Davangere and Department of Pharmacy Practice, Bapuji Pharmacy College , Davangere for helping in reporting this case.

ABBRIVIATIONS

1. Medical Intensive Care Unit (MICU)
2. Acute Kidney Injury (AKI)
3. Enteric nervous system (ENS)
4. Gastrointestinal (GI)
5. Hb: Hemoglobin
6. TLC: Total Leukocyte Count
7. PLT: platelet count
8. PS: Peripheral Smear
9. AST: Aspartate Aminotransferase
10. ALT: Alanine Aminotransferase
11. ALP: Alkaline Phosphatase
12. MDCT: Multidetector Computed Tomography
13. K: potassium

14. CL: chlorine
15. MR: Mitral Regurgitation
16. TR: Tricuspid Regurgitation
17. PAH: Pulmonary Arterial Hypertension
18. LVH: Left Ventricular Hypertrophy
19. LV: Left Ventricle
20. LVEF: Left Ventricular Ejection Fraction.
21. PCR: Protein/Creatinine Ratio

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