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Case Report

Hepatorenal Syndrome with Purpura Fulminans, Hypokalemic Periodic Paralysis, Prostatic Enlargement, Rheumatoid Arthritis, and Suspected COVID-19 Infection: Prognosis and Management

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Abstract

Introduction: Hepatorenal syndrome has been proposed by the Acute Dialysis Quality Initiative (ADQI), describing all patients with advanced cirrhosis and concurrent kidney dysfunction. Purpura fulminans is a hematological emergency in which there is skin necrosis and disseminated intravascular coagulation. Hypokalemic periodic paralysis (HypoPP) is a rare electrolyte disorder that has recurrent episodes of sudden-onset paralysis associated with low serum potassium levels. Prostate enlargement is a common condition in older men, characterized by a non-cancerous increase in the size of the prostate gland. Rheumatoid arthritis (RA) Rheumatoid arthritis (RA) is the most common chronic form of inflammatory arthritis, affecting approximately 1 percent of the population. Few reports noted the onset of hepatorenal syndrome (HRS) in the face of COVID-19 infection.

Case presentation: A 68-year-old male, Egyptian worker, was admitted to the intensive care unit (ICU) with hepatorenal syndrome, severe anemia, HypoPP, prostatic enlargement, suspected COVID-19 infection, and superadded hemorrhagic purpura.

Conclusion: Co-association of severe hypokalemia, severe anemia, renal impairment, advanced cirrhotic liver, superadded hemorrhagic purpura, and suspected COVID-19 infection is a hallmark for this case study. The severe hypokalemia, severe anemia, renal impairment, advanced cirrhotic liver, superadded hemorrhagic purpura, suspected COVID-19 infection, and elderly male are constellations of risk factors. Furthermore, future research will be recommended.

Keywords: Hepatorenal syndrome; purpura fulminans; hypokalemia; periodic paralysis; rheumatoid arthritis; COVID-19 infection

Abbreviations: ADQI: Acute dialysis quality initiative; ATN: Acute tubular necrosis; BPH: Benign prostatic hyperplasia; DIC: Disseminated intravascular coagulation; ECG: Electrocardiogram; HRS: Hepatorenal syndrome; HypoPP: Hypokalemic periodic paralysis; ICU: Intensive care unit; LAD: Left axis deviation; PF: Purpura fulminans; RA: Rheumatoid arthritis; VR: Ventricular rate

Introduction

Hepatorenal syndrome (HRS) is a severe sequel of end-stage cirrhosis characterized by increased splanchnic blood flow, hyperdynamic circulation, a state of decreased central volume, activation of vasoconstrictor systems, and extreme kidney vasoconstriction leading to decreased GFR [1,2]. HRS has been proposed by the Acute Dialysis Quality Initiative (ADQI) to describe all patients with advanced cirrhosis and concurrent kidney dysfunction [2]. Early diagnosis is pivotal in the management of HRS and helps in early identification of patients. Differentiation of hepatorenal syndrome from acute tubular necrosis (ATN) is essential. Hepatorenal syndrome and ATN may be considered as a continuum rather than distinct entities. Emerging biomarkers can differentiate between the two conditions. Vasoconstrictive drugs (especially terlipressin) and albumin are the initial drug of choice. Liver transplantation is the only curative treatment of hepatorenal syndrome [1]. The prognosis of patients with cirrhosis who have HRS is still poor, with a median survival without liver transplantation of less than six months [2]. Purpura fulminans (PF) is an acute, often fatal, hematological emergency and thrombotic disorder that manifests as blood spots, bruising, and skin discoloration resulting from coagulation in small blood vessels of the skin, resulting in skin necrosis and disseminated intravascular coagulation (DIC) [3,4].

PF often depends on the patient's age and the circumstances of presentation. PF may complicate severe sepsis or may occur as an autoimmune response to otherwise benign childhood infections. PF may also be the presenting symptom of severe heritable deficiency of the natural anticoagulant's protein C or protein S [3]. Inherited defects in protein C activity are autosomal dominant (AD) and may be partial or severe loss of function [3,5]. Hundreds of natural mutations of the protein C gene (PROC) have been identified [6,7]. Acquired protein C deficiency is caused by either depletion of available protein C in plasma or decreased protein C synthesis (caused by administration of vitamin K antagonists, severe liver failure [8]. Purpura fulminans is a presenting feature of severe acute sepsis. Sometimes, purpura fulminans has an unknown cause. Early recognition and treatment of PF are essential to reduce mortality and to prevent major long-term health sequelae. However, management strategies require accurate identification of the underlying cause. However, management strategies require accurate identification of the underlying cause [3]. Purpura fulminans with disseminated intravascular coagulation should be urgently treated with fresh frozen plasma (10–20 mL/kg every 8–12 hours) and/or protein C concentrate to replace pro-coagulant and anticoagulant plasma proteins that have been depleted by the disseminated intravascular coagulation process [3-5,8].

Hypokalemic periodic paralysis (HypoPP) is a rare electrolyte disorder that has recurrent episodes of sudden weakness associated with hypokalemia. It is usually triggered by arduous exercise or a high-carbohydrate diet. Patients with HypoPP experience a sudden generalized or local flaccid paralysis associated with hypokalemia. It can last for several hours to days. Both familial and acquired causes of HypoPP have been reported. Mostly, HypoPP cases are familial [9]. Type 1 HypoPP is the most common familial form, which is characterized by a mutation in the dihydropyridine-sensitive skeletal muscle calcium channel gene (CACNA1S). This genetic abnormality is present in about 70% of HypoPP cases. Type 2 familial HypoPP is associated with mutations in the voltage-sensitive skeletal muscle sodium channel gene (SCN4A) [10]. Thyrotoxicosis is implicated in acquired HypoPP. Provocative testing with potassium can be used to diagnose HypoPP. Potassium chloride (KCl) is recommended for acute treatment, typically beginning with incremental doses of oral KCl, starting at 0.5 to 1 mEq/kg [11]. Prostate enlargement or benign prostatic hyperplasia (BPH), is a common disease in older men with a non-cancerous increase in the size of the prostate gland.

The hallmark symptoms of BPH are urinary frequency, which leads to the need to urinate more often than usual, particularly during the day. Transrectal ultrasound visualizes prostate structure and evaluates size. Treatment options include lifestyle changes, medications, procedures, and surgery [12]. Rheumatoid arthritis (RA) Rheumatoid arthritis (RA) is the most common chronic form of inflammatory arthritis, affecting approximately 1 percent of the population [13]. The diagnosis is mostly based on a person's signs and symptoms [14]. RA may result in permanent joint damage and disability. RA may affect other organs, including the lungs, heart, blood vessels, skin, and eyes. Physical examination, blood tests such as rheumatoid factor (RF), anticyclic citrullinated protein (CCP) antibodies, and x-ray scans are tools for the diagnosis of RA. Disease-modifying antirheumatic drugs (DMARDs) are the mainstay of treatment for RA [15]. There have been numerous reviews evaluating the causative relationship between coronavirus disease 2019 (COVID-19) and liver pathology, with several emerging cases of COVID-19-induced liver injury. There are a few reports noting the onset of hepatorenal syndrome (HRS) post-COVID-19 infection [16].

In this manuscript, I report a hepatorenal syndrome, severe anemia, HypoPP, prostatic enlargement, suspected COVID-19 infection, and superadded hemorrhagic purpura in an elderly male patient.

Case presentation

History and Physical Examination

A 68-year-old male, Egyptian worker, was admitted to the intensive care unit (ICU) with severe pallor, weakness of four limbs, fever, and dizziness. He gave a history of liver cirrhosis 18 years ago on oral frusemide (40mg, OD) and oral spironolactone (25mg, OD). He gave a history of rheumatoid arthritis 23 years ago and episodic management with NSAIDs, steroids, and immunosuppressant medications. There was no history of the same attack. Currently, the patient denies a history of other relevant diseases, medications, or special habits. Upon general physical examination; generally, the patient show severe pallor, dyspnea, flaccid paralysis of four limbs, deformity of fingers (Figure 1A), abdominal enlargement with tense ascites, umbilical hernia (Figure 1B), Café au lait spot in below right knee (Figure 1C), pitting bilateral lower limb edema with a regular

pulse rate of VR; 60 bpm, blood pressure (BP) of 120/70 mmHg, respiratory rate of 18 bpm, a temperature of 38 °C, GCS of 15/15, and pulse oximeter of oxygen (O2) saturation of 95%.

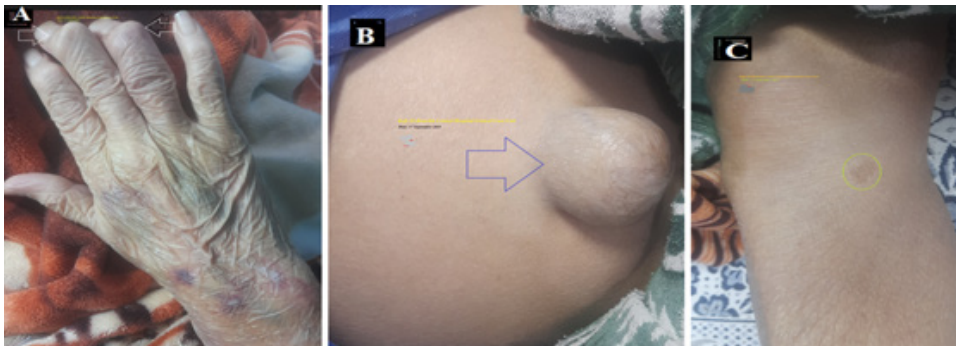
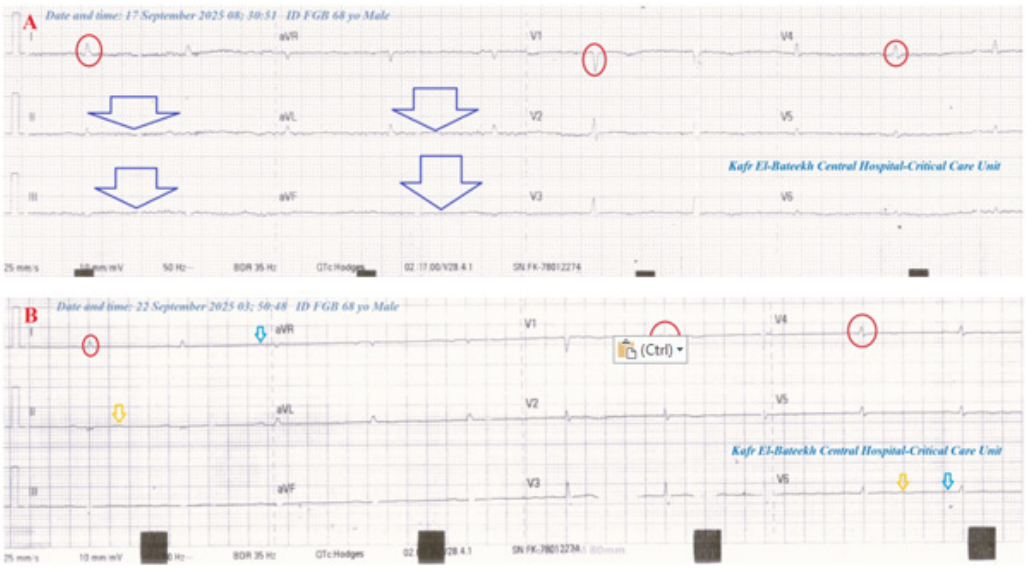


Figure 1: A. right hand showing peripheral finger deformity (white arrows). B. Abdominal enlargement and umbilical hernia (blue arrow). C. Café au lait spot below the right knee (yellow circle).

Workup and Management

Currently, the patient is admitted to the ICU with hepatorenal syndrome, severe anemia, HypoPP, prostatic enlargement, and superadded hemorrhagic purpura. Initially, the patient was treated in the ICU with blood transfusion (4 units, single unit daily). Potassium chloride IVI ampoules (15%, 10 meq/h, for 0.5 to 1 mEq/kg) were added. Low-dose oral frusemide (40 mg, fasting, OD), spironolactone (25 mg, fasting, OD), paracetamol IVI (500mg TID, as needed), and ceftriaxone vials (IM/IV, 2gm daily for 5 days) were added. The patient was monitored hourly for vital signs, serial potassium levels, and electrolytes. The initial ECG tracing obtained at the initial presentation in the ICU showed slight sinus bradycardia with low-voltage QRS complexes, P waves, T waves, and tremor artifacts (Figure 2A). The ECG tracing was repeated within 6 days of the initial ECG in the ICU, showing normal sinus rhythm (NSR) with low-voltage QRS complexes, P waves, and T waves (Figure 2B). The initial workup on the ICU admission was: CBC: Hb: 7.0 g/dl, RBCs: 2.28*103/mm^3, WBCs; 11.3*103/mm^3 (Neutrophils: 76.3 %, Lymphocytes: 15.1%, Monocytes: 7.0%, Eosinophils: 1.0% and Basophils: 0.6%), and Platelets: 64*103/mm^3. Serum potassium was (1.7mmol/L). CRP was (48g/dl). Serum albumen was (3.3g/dl). Serum creatinine was (3.8mg/dl). Blood urea was (79mg/dl). Uric acid was (12.5mg/dl). SGPT was (10 U/L). SGOT was (25 U/L). Total bilirubin was (0.7 mg/dl). Fasting blood sugar was (73 mg/dl). Hb A1C was (5.2%). ESR, first hour was (140 mm/hr). ASOT was (99 IU/ml). INR was (1.17). PT was (13.5 sec). Screening for HCVAb was positive, HBsAg and HIVAb were negative. Within 12 hours of the above management, serum potassium was (3.55mmol/L). Plasma sodium was (141.1mmol/L). Serum chloride was (101.1mmol/L). Ionized calcium was (1.09mmol/L). Urine analysis on the second day of ICU admission showed RBCs over 100/HPF and pus 15-20/HPF. Within 3 days of the ICU admission, serum creatinine was 3.0 mg/dl. Blood urea was (70mg/dl). Bleeding time was 3 minutes. Clotting time was 9 minutes. APTT was 28 seconds. The CBC repeated within 4 days of the ICU admission was: Hb was 9.0 g/dl, RBCs; 3.2*103/mm^3, WBCs: 4.7*103/mm^3 (Neutrophils; 78 %, Lymphocytes: 17 %, Monocytes; 3%, Eosinophils; 2% and Basophils 0%), and Platelets: 59*103/mm^3. The current abdominal ultrasound showed liver cirrhosis, multiple gall-bladder stones, bulky splenomegaly, right grade II-III nephropathy with multiple cysts in both kidneys, moderate to severe ascites, and mild prostatic enlargement. Within 5 days of the ICU admission, there were extensive brown to blackish ecchymosis discoloration in the different body parts, mostly in the forearms (Figures 2C&2D). Fresh frozen plasma and platelet transfusions were urgently added. A dramatic clinical response for the flaccid paralysis with a gradual response for hemorrhagic thrombocytopenia, had happened. Hepatorenal syndrome, severe anemia, HypoPP, prostatic enlargement, suspected COVID-19 infection, and superadded hemorrhagic purpura were the most probable diagnoses. Within a few days of starting the medications, the patient showed dramatic clinical improvement. He was discharged and maintained on oral frusemide (40 mg, on fasting, OD), and spironolactone (25 mg, on fasting, OD). There is a recommendation for future urological, hepatic, and hematological follow-up.



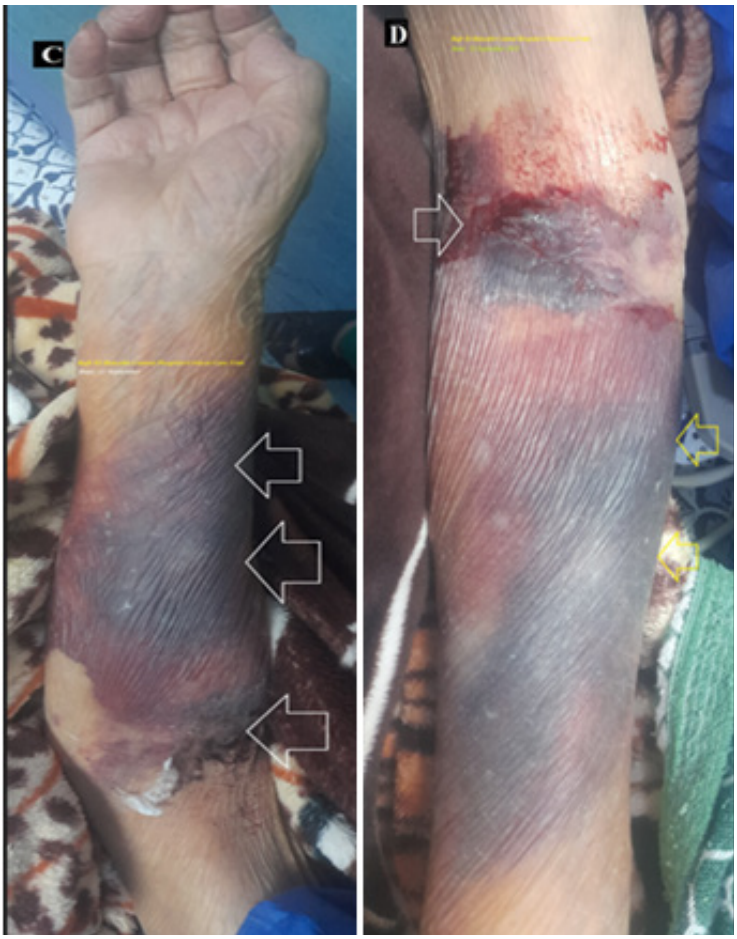


Figure 2: A. ECG tracing obtained at the initial presentation in the ICU showed slight sinus bradycardia with low-voltage QRS complexes (red circles), P waves, T waves, left axis deviation (LAD), and tremor artifacts (large blue arrows). B. ECG tracing was repeated within 6 days of the initial ECG in the ICU, showing normal sinus rhythm (NSR) with LAD, low-voltage QRS complexes (red circles), P waves, and T waves. Right (C: white arrows) and left forearm (D: white and yellow arrows) showing extensive brown to blackish ecchymosis discoloration.

Discussion

a) Overview: An elderly male, Egyptian worker, was admitted to the intensive care unit (ICU) with hepatorenal syndrome, severe anemia, HypoPP, prostatic enlargement, suspected COVID-19 infection, and superadded hemorrhagic purpura.

b) The primary objective for my case study was to manage the presence of hepatorenal syndrome, severe anemia, HypoPP, prostatic enlargement, suspected COVID-19 infection, and superadded hemorrhagic purpura in an admitted patient to the ICU.

c) The secondary objective for my case study was how you would manage this case in the ICU.

d) Presence of renal impairment on top of advanced liver cirrhosis strongly suggests the diagnosis of hepatorenal syndrome.

e) A quadrant flaccid paralysis with normal GCS and severe low serum potassium will be directed to the diagnosis of hypokalemic periodic paralysis (HypoPP). Acquired causes, such as malnutrition and furosemide, are mostly implicated.

f) Highly elevated ESR and CRP are parallel with the activity of established rheumatoid arthritis.

g) Also, elevated CRP, neutrophilia, and lymphocytopenia in CBC with fever may give a suspicion of associated COVID-19 infection. Unfortunately, a PCR assessment for COVID-19 infection wasn't done.

h) Low-voltage QRS complexes, P waves, and T waves in ECG may be due to advanced liver disease.

i) Severe hypokalemia may cause low-voltage P waves and T waves without affecting QRS complexes in the ECG.

j) An extensive brown to blackish ecchymosis discoloration with low platelets is strongly suggestive of purpura fulminans. Associated urinary tract infection, suspected COVID-19 infection, protein C deficiency, protein S deficiency, and DIC are possible causes. Unfortunately, there is no full and advanced laboratory supporting the causes of PF.

k) Dry gangrene is the most implicated differential diagnosis for purpura fulminans. But the presence of a peripheral pulse with hot extremities against it.

l) Interestingly, the severe hypokalemia, severe anemia, renal impairment, advanced cirrhotic liver, superadded hemorrhagic purpura, suspected COVID-19 infection, and elderly male are constellations of risk factors.

m) I can't compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison.

n) The only limitations of the current study were the unavailability of some laboratory testing due to the high costs.

Conclusion and Recommendations

a) Co-association of severe hypokalemia, severe anemia, renal impairment, advanced cirrhotic liver, superadded hemorrhagic purpura, and suspected COVID-19 infection is a hallmark for this case study.

b) The severe hypokalemia, severe anemia, renal impairment, advanced cirrhotic liver, superadded hemorrhagic purpura, suspected COVID-19 infection, and elderly male are constellations of risk factors.

c) Furthermore, future research will be recommended.

Conflicts of Interest

There are no conflicts of interest.

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