DOI: 10.7860/JCDR/2023/63027.17978

Case Report

Others Section

Seizure-Induced Coma in Patient with Haemoglobin 1.3 gram per decilitre and Multi-Organ Dysfunction: A Rare Case Report

SHRDHHA AGRAWAL[1], AKHILESH SINGH PARIHAR[2], CHARUTA GADKARI[3]

ABSTRACT

People of all ages are affected by anaemia which is a serious global health problem. Iron Deficiency Anaemia (IDA) seems to be the most commonly occurring type of anaemia in women. Elderly people with anaemia have higher morbidity and mortality. The purpose of treatment is to tackle the disorder's underlying cause. Older people with persistent anaemia may need regular blood transfusions. The authors present a case report of a 55-year-old woman who visited the Emergency Department with primary complaints of convulsions and subsequent unconsciousness. She had generalised oedema and weakness since eight days. On examination, she was afebrile with pulse rate of 92 beats/min, Systolic Blood Pressure (SBP) of 80 mmHg. Laboratory results showed haemoglobin of 1.3 g/dL, increased White Blood Cells (WBC) and peripheral smear showed pencil-shaped cells and teardrop cells. Radiological investigation showed grade III renal parenchymal disease, cirrhosis of liver, gross ascites with bilateral pleural effusion, and splenic cyst. A 2D Echocardiography was done, which was suggestive of left ventricular hypertrophy, mild to moderate systolic dysfunction, along with dilated left ventricle. She was intubated in view of low Glasgow Coma Scale (GCS) and unconsciousness. On further stay in the hospital, she received blood transfusion. Hence, this case is a rare finding of severe anaemia.

Keywords: Anaemia, Iron deficiency, Multiple organ dysfunction syndrome, Sepsis

CASE REPORT

A 55-year-old female patient, suffering from bronchial asthma since six years, was brought to the Emergency Department by the relatives with chief complaint of seizures, followed by unconsciousness. She had two episodes of convulsions on the same morning, which were associated with frothing, up-rolling of eyeball, and tonic clonic movement of both upper and lower limbs. There was a history of oedema all over the body, and generalised weakness since eight days. There was no history of chest pain, palpitations, cough, cold, fever, loose stools, or syncope. There was no information on whether the patient had hypertension, diabetes, or tuberculosis.

She was a known case of bronchial asthma and renal disease for which she was not taking any medication due to low socio-economic condition. There was a history of blood transfusion two years back, but no documentation was available.

On admission, the patient's Glasgow Coma Scale (GCS) was 5 (E1V1M3). She was afebrile, with pulse rate of 92/min, Systolic Blood Pressure (SBP) of 80 mmHg. There was pallor and generalised oedema all over the body. On auscultation, S1 and S2 heart sounds were normal, breath sounds were diminished but equal on both sides, bilateral crepitations were present. Abdomen was soft and non tender on palpation. Patient was drowsy, responding to deep pain stimulus (grimacing present), corneal reflex was absent, bilateral pupils were mid dilated and reactive to light, deep tendon reflexes were slow and sluggish.

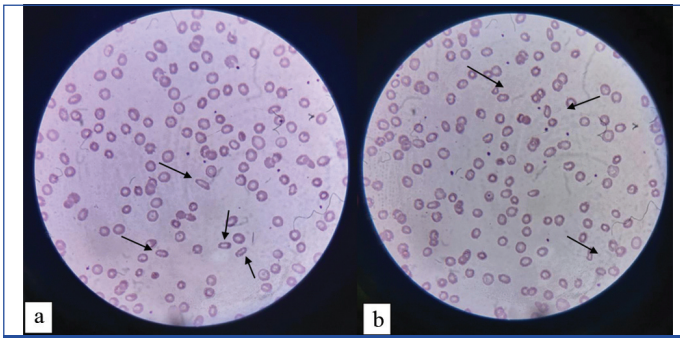
In view of low GCS 5, falling saturation, tachycardia, tachypnoea and gasping state, patient was intubated and taken on mechanical ventilator. A preliminary diagnosis of cardiogenic versus nephrogenic pulmonary oedema was considered at this point. Complete Blood Count (CBC), Liver Function Test (LFT), Kidney Function Test (KFT) and Arterial Blood Gas (ABG) analysis were done.

Drugs used were Inj. Midazolam 3 mg, Inj. Vecuronium bromide 6 mg Intravenous (i.v.). The patient's blood investigations are shown in [Table/Fig-1].

Test	Value (units)
Haemoglobin (Hb)	1.3 gram per decilitre
White Blood Cells (WBC)	22900/cubic milimetre
Platelet	1.74 lacs/cubic milimetre
Peripheral smear	Leishman staining showing few pencil cells, occasional teardrop cells
Mean Corpuscular Volume (MCV)	89.3 femtolitres

[Table/Fig-1]: Complete Blood Count (CBC).

[Table/Fig-2] represents the peripheral smear which shows pencil-shaped cells (Elliptocytes) and teardrop shaped cell (Dacrocyte-A type of poikilocyte).



[Table/Fig-2]: a) Few pencil cells (type of elliptocytes); b) Teardrop cell-A dacrocyte (type of poikilocyte)-peripheral smear stained in Leishman stain (X100).

[Table/Fig-3] shows the results of the KFT and [Table/Fig-4] shows the results of the blood coagulation profile and LFT. The purpose of ABG analysis was to arrive at the cause of unconsciousness, which could be respiratory in the form of severe hypoxia or CO_2 narcosis or a metabolic cause.

Approximately two millilitre of yellowish ascitic fluid was withdrawn in a clot activator vacutainer. On wet mount, Red Blood Cells (RBCs) were 6.7 RBCs/High Power Field (HPF), WBCs-plenty of

Test	Value (units)
Urea	149 milligrams per decilitre
Creatinine	2.8 milligrams per decilitre
Sodium	142 milligrams per decilitre
Potassium	5.9 milligrams per decilitre

[Table/Fig-3]: Kidney Function Test (KFT).

Test	Value (units)
Prothrombin Time (PT)	11.9 seconds
Activated Partial Thromboplastin Time (aPTT)	30.1 seconds
International Normalised Ratio (INR)	2.48
Total bilirubin	2.2 grams per decilitre
Total protein	5.0 milligrams per decilitre
Conjugated bilirubin	1.2 milligrams per decilitre
Unconjugated bilirubin	1 milligrams per decilitre
Alanine Transaminase (ALT)	48 U/L
Aspartate Transaminase (AST)	194 IU/L
Alkaline Phosphatase (ALP)	260 U/L
Magnesium	2.3 milligrams per decilitre
Calcium	7.7 milligrams per decilitre
Phosphorus	5.0 milligrams per decilitre
C-Reactive Protein (CRP)	77 milligram per decilitre
Ferritin	25.1 nanogram per millilitre
Iron	145 microgram per decilitre
Uric acid	11.4 milligrams per decilitre
Vitamin B12	1000 picogram per millilitre

[Table/Fig-4]: Blood coagulation profile and LFT.

cells/HPF Total Leucocyte Count (TLC) was approximately 2992 cells/cu mm. Differential Leucocyte Count (DLC) have polymorphs 75%, lymphocytes 25%. The purpose of this investigation was to rule out sepsis.

Ultrasonography (USG) of the abdomen and pelvis was done, which was suggestive of grade III renal parenchymal disease according to USG grading of echogenicity of renal cortical disease, cirrhosis of liver, gross ascites with bilateral pleural effusion, and splenic cyst.

A 2D Echocardiography was done, which was suggestive of left ventricular hypertrophy, mild to moderate systolic dysfunction, along with dilated left ventricle.

Treatment at the Department of Emergency Medicine:

Initially, the patient was started on:

1) Inj. levetiracetam 1 g in 100 mL Normal Saline (NS)
2) Inj. noradrenaline 10 mL/hr.
3) Inj. hydrocortisone 100 mg i.v.
4) Inj. sodium bicarbonate 100 mL
5) Inj. calcium gluconate 10 mL was given for correction of acidosis and hyperkalaemia. Further, she was admitted to the Intensive Care Unit (ICU).

Under all aseptic precautions, and USG guidance, a 3-lumen Central Venous Catheter (CVC) was inserted through the left internal jugular vein for guiding fluid management and administration of drugs.

Treatment in ICU: The patient was started with antibiotics such as inj. piperacillin and tazobactam 2.25 g i.v. TDS, inj. levofloxacin 500 mg i.v. OD. Acidosis and hyperkalaemia were corrected with i.v. glucose insulin infusion and inj. sodium bicarbonate 210 mEq, inj. calcium gluconate 20 mL were given. In view of severe anaemia, the patient received four units of packed Red Blood Cells (pRBC). In view of deranged coagulation profile, the patient was transfused with five units of Fresh Frozen Plasma (FFP). From the point of view of severe hypotension, inotropic supports were continued. She continued to be on mechanical ventilation and could not be considered for weaning as the condition kept on deteriorating in spite of the treatment and she died in the ICU on the 4th day with cardiogenic shock due to severe anaemia with cirrhosis of liver and Chronic Kidney Disease (CKD). The diagnosis of the patient was Multiple Organ Dysfunction Syndrome (MODS) secondary to cardiogenic shock with CKD along with underlying chronic severe anaemia.

DISCUSSION

Anaemia affects one third of the total community, contributing to increased morbidity and mortality, decreased work productivity, and impaired neurodevelopment [1]. Recently, research has increased the understanding of the complex aetiology of anaemia, including prevalence of anaemia due to Iron Deficiency (ID) and its effect on inflammation and infection. An approach for determining the aetiology, including the patient's past medical record as well as laboratory testing of the number of reticulocyte, serum ferritin and CRP, serum vitamin B12, RBC, folic acid, and serum creatinine. Microcytosis is an indication of inadequate haemoglobin synthesis, which can be brought about by either iron deficit or the congenital condition, haemoglobinopathy. Macrocytosis is the consequence of an interruption of division and maturation of pro-erythroblasts in the bone marrow, e.g., due to vitamin B12/folic acid deficiency or drinking too much alcohol [2].

This patient presented with severe life-threatening anaemia with Hb of 1.3 g/dL. To the best of the authors' knowledge, this is the minimum haemoglobin level reported in the literature. Chronic anaemia can be life-threatening and can even cause death. Untreated anaemia can lead to multiorgan failure. In early phases, body can compensate through non haemodynamic and haemodynamic mechanisms [3]. A case of severe anaemia with Hb of 1.8 g/dL had been reported in 2016 where the patient had generalised fatigue, and palpitations at presentation. He was diagnosed to have carcinoma of the colon with presence of occult blood in the stool [4]. There was no known source of bleeding in the present case. Another case reported with a Hb value of 1.4 g/dL where the patient had fatigue and dizziness and suffered a fall. She had irregular menstrual bleeding for over three decades and did not seek medical help [5]. Similarly in India, women tend to ignore seeking medical help. Nutritional factor is an important reason of anaemia in India.

Another case of severe anaemia with Hb of 1.7 g/dL has had been reported in 2005. However, the patient was a diagnosed case of bulimia nervosa with coeliac disease [6]. A study based on haemoglobin level and blood transfusion in septic patients showed that anaemia is very common in septic patients. There are a number of mechanisms that indicate a rapid drop in haemoglobin levels in patients with sepsis. The major mechanism consists of a decrease in erythropoietin production due to release of inflammatory cytokines, such as Tumour Necrosis Factor-α (TNF-α), Interleukin-1 (IL-1), and Interleukin-6 (IL-6). Other possible mechanisms that contribute to the reduction in haemoglobin in sepsis are gastrointestinal bleeding/stress-induced haemophilia due to fluid overload, withdraw for laboratory analysis, altered iron metabolism, haemolysis as part of pathogenesis of some infectious processes, bleeding due to Disseminated Intravascular Coagulation (DIC) and possibly erythrocytosis destruction due to damage to the RBC membrane [7]. A combination of anaemia and sepsis can impair tissue oxygenation.

Therefore, the treatment of patients with sepsis focuses on maximisation of oxygen delivery to tissues to treat cellular hypoxia and cell progression dysfunction for the purpose of preventing or delaying the onset of MODS [7]. Elderly patients with anaemia often

have significant co-morbidities, including heart failure and CKD [8]. A study has shown that 5-year mortality is 2.4 times higher in anaemic men and 1.6 times higher in women compared to non anaemic patients. It should be noted that the increase in anaemia-related mortality was found to remain unchanged after adaptation for pre-existing co-morbid conditions [9]. The treatment for anaemia includes oral iron therapy, intravenous iron therapy and in chronic cases, blood transfusion. In this case, the patient presented with severe anaemia of 1.3 g/dL. The patient continued to be on mechanical ventilation and could not be considered for weaning as her condition kept on deteriorating in spite of treatment.

CONCLUSION(S)

Severe anaemia can lead to life-threatening complications. This patient came in peri-arrest condition where oxygenation and circulatory support were a priority. Haemoglobin (Hb) of 1.3 g/dL could be a result of multiple medical factors, mainly nutritional, associated renal pathology, sepsis as well as social factors like lack of awareness and education, which are common in Indian scenario.

REFERENCES

[1] Chaparro CM, Suchdev PS. Anaemia epidemiology, pathophysiology, and etiology in low-and middle-income countries. Ann N Y Acad Sci. 2019;1450:15-31. https://doi.org/10.1111/nyas.14092.
[2] van der Lelie J, van Oers MH, von dem Borne AE. [Diagnostics for classification of anaemia]. Ned Tijdschr Geneeskd. 2001;145(18):866-69.
[3] Metivier F, Marchais SJ, Guerin AP, Pannier B, London GM. Pathophysiology of anaemia: Focus on the heart and blood vessels. Nephrol Dial Transplant. 2000;15:14-18. https://doi.org/10.1093/oxfordjournals.ndt.a027970.
[4] Schmitt RE, Buckley CJ. Extreme anaemia (hemoglobin 1.8 g/dL) secondary to colon cancer. Proc (Bayl Univ Med Cent). 2016;29(4):393-94.
[5] Chai AL, Huang OY, Rakočević R, Chung P. Critical iron deficiency anaemia with record low hemoglobin: A case report. J Med Case Rep. 2021;15:472. https://doi.org/10.1186/s13256-021-03024-9.
[6] Jost PJ, Stengel SM, Huber W, Sarbia M, Peschel C, Duyster J. Very severe iron-deficiency anaemia in a patient with celiac disease and bulimia nervosa: A case report. Int J Hematol. 2005;82:310-11. https://doi.org/10.1532/IJH97.E0505.
[7] Muady GF, Bitterman H, Laor A, Vardi M, Urin V, Ghanem-Zoubi N. Hemoglobin levels and blood transfusion in patients with sepsis in Internal Medicine Departments. BMC Infect Dis. 2016;16:569. https://doi.org/10.1186/s12879-016-1882-7.
[8] Price EA, Mehra R, Holmes TH, Schrier SL. Anaemia in older persons: Etiology and evaluation. Blood Cells Mol Dis. 2011;46:159-65. https://doi.org/10.1016/j.bcmd.2010.11.004.
[9] Izaks GJ, Westendorp RGJ, Knook DL. The definition of anaemia in older persons. JAMA. 1999;281:1714-17. https://doi.org/10.1001/jama.281.18.1714.

PARTICULARS OF CONTRIBUTORS:
1. Undergraduate, Department of Emergency Medicine, Jawaharlal Nehru Medical College, Wardha, Maharashtra, India.
2. Senior Resident, Department of Emergency Medicine, Acharya Vinobha Bhave Rural Hospital, Wardha, Maharashtra, India.
3. Professor, Department of Emergency Medicine, Acharya Vinobha Bhave Rural Hospital, Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Shrdhha Agrawal,
DMIMS, Indira Hostel, Sawangi, Wardha, Maharashtra, India.
E-mail: shrdhhaagrawal@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jan 25, 2023
- Manual Googling: Feb 20, 2023
- iThenticate Software: Mar 02, 2023 (4%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. No

Date of Submission: **Jan 21, 2023**
Date of Peer Review: **Feb 11, 2023**
Date of Acceptance: **Mar 03, 2023**
Date of Publishing: **Jun 01, 2023**