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Case Study

Viral Malaria-Induced Pancytopenia in a G6PD-Deficient Patient: A Rare Clinical Intersection

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I N F O

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A B S T R A C T

In a rare instance, a 22-year-old woman with malaria who also had genetic glucose-6-phosphate dehydrogenase (G6PD) impairment developed pancytopenia. Pancytopenia is rare and led to more research, although anemia is a typical hematological result in malaria. Because of the danger of hemolysis, G6PD deficiency limited the use of common antimalarial medications, complicating the therapeutic course. Close hematologic monitoring and,-supportive treatment, including blood transfusions and G6PD-safe antimalarial medication, were used to treat the patient. Other potential causes of bone marrow suppression, including viral co-infections, were excluded. This situation highlights the need for personalized treatment plans in complex medical cases and the importance of considering enzyme disorders like G6PD deficiency in patients with unusual malaria symptoms.

Keywords: Malaria, G6PD, Pancytopenia, Hemolysis, Antimalarial Therapy, Haematological Complications

Introduction

The second most common cause of malaria, following Plasmodium falciparum (Pf), is Plasmodium vivax (Pv). The fact that Pv may cause substantial pregnancy difficulties, severe illness in children and adults, and a significant health, social, and financial cost is becoming more widely acknowledged. It can be challenging to diagnose and treat Pv malaria clinically, and controlling and getting rid of Pv poses unique problems.[1] Since trophozoite malaria is uncommon in peripheral blood, we were able to diagnose it in our instance. During an active infection, it is easier to see trophozoites, the parasite's feeding stage.[2] Trophozoites, however, can indicate a more severe stage of malaria, particularly in Plasmodium falciparum infections.[2]

While trophozoites are a crucial part of the parasite's lifecycle, they are not always easily detected in peripheral blood samples. Even though they are an essential component of the parasite's life cycle, trophozoites are not often readily identifiable within peripheral blood sample.[3] In blood smears, schizonts—the stage in which the parasitic organism develops within red blood cellsand forms rings are frequently more noticeable.[4]

Approximately 400 million individuals worldwide suffer from glucose-6-phosphate dehydrogenase (G6PD) insufficiency, an X-chromosome-transmitted erythrocyte condition.[5] Most cases of G6PD deficiency occur in regions in which malaria exists or has been prevalent. Drugs used to treat malaria in these regions have the potential to trigger (severe) haemolysis in people who are G6PD deficient.[6]

This instance illustrates how pancytopenia, G6PD deficiency, and malaria interact intricately in a young female individual. It was difficult to diagnose and treat the overlapping clinical symptoms, especially when choosing an antimalarial medication that was acceptable with G6PD deficiency. In this case, pancytopenia most likely came from a confluence of viral co-infection or haemolysis.

Case Report

A22-year-old female was admitted to a multispecialty hospital in India with complaints of persistent fever with chills and rigours for 4 days, vomiting, loose stool, burning micturition, cough and, yellowing of the eyes. She also reported recent gum bleeding, and easy bruising, and abdominal pain. Before being admitted to hospital, the patient has a past medical history of G6PD- and no any surgical history. She also didn't have any recent travelling history, and didn't even remember getting bitten by a mosquito or coming into touch with an infected person.

At the time of hospital admission, she was conscious and oriented-, she had a high fever (102°F) with a normal respiratory rate, pulse rate and blood pressure. On physical examination, she had yellowish eyes, mild puffiness of the face, pale skin and icteric, and multiple petechiae were noted over the lower limbs. Abdominal examination reveals a soft, mild tender abdomen.

As shown in Table 1, laboratory investigations reveal pancytopenia with red blood cell at 2.5x 10^6/cmm and hemoglobin at 6.9 g/dl, leucopenia (WBC 3,000/mm^3), and thrombocytopenia (platelets 41000/mm^3). The rapid diagnostic Test was done as a malaria antigen test on the 1st day and the result detected malaria antigens in blood. Then a peripheral blood smear was ordered to detect the species. On the next day, the test result confirmed the presence of a plasmodium vivax infection, with trophozoites seen on the peripheral smear.

Total serum bilirubin was markedly elevated at 8.5 mg/dL, with indirect bilirubin predominating at 6.8 mg/dL and direct bilirubin at 1.88 mg/dL, indicating indirect hyperbilirubinaemia. The G6PD test result was high (60 U/g Hb), indicating G6PD deficiency. USG report: spleen shows 14.6 mm mild splenomegaly.

The patient was isolated and treated carefully with constant monitoring. The pharmacotherapy for this patient is as follows: Antimalarial drug (artesunate) 120 mg twice a day, (primaquine) 7.5 mg, 2 tablets every morning for 7 days. The antibiotic doxycyclin was given at 500mg BD. Udiliv 300 mg TID as a hepatoprotective agent and IVIg 1gm/kg was given. Few multivitamins, such as B12, MVI, folic acid and vitamin-C were administered. Nutritional support RL and NS were given and paracetamol 500 mg TID for fever. On the 2nd day she had a requirement for a blood transfusion so whole blood transfusion was done and blood transfusion Inj. Avil and Inj. Hydrocort IV STAT were given.

Table 1.Laboratory Findings

Investigation	Findings			Reference Range
	First Day	Second Day	Last Day	
HB	8.7	6.9	7.8	12-16 g/dL
RBC	2.5	3.1	2.	4.5 – 5.5 x 10^6/cmm
WBC	3000	3200	3400	4000 -11000/mcL
Platelets	41000	55000	53000	1.5 - 4.1 lacs/mcL
Neutrophils	39	42	50	50 – 62 %
Lymphocytes	50	55	20	20 – 40 %
Eosinophils	01	04	01	00 – 06 %
Basophils	00	00	00	00 – 02 %
HCT	20.4	24.4	24.6	36 – 46 %
MCV	70.1	69.7	70.1	80 – 100 FL
MCH	21.9	22.7	22.5	28 – 32 pg
MCHC	31.2	31.8	32.5	32 – 36 g/dl
RDW-CV	17.7	17.8	18.1	12.0 – 14.6 %
Malaria parasite	POSITIVE	+VE (Trophozoites seen)	POSITIVE	-
G6PD	60	-	-	1.5-9.03 U/g Hb
S. Creatinine	0.60	0.61	0.60	0.8 – 1.2 mg/dl

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S. Sodium	139.00	-	-	135 – 145 mEq/L
S. Potassium	4.10	-	-	3.5 – 5.1 mEq/L
S. Bilirubin	1.88	0.61	-	0 – 0.4 mg/dl
S. Indirect Bilirubin	6.8	4.4	4.1	0.1 – 0.8 mg/dl
S. Total Bilirubin	8.5	8.2	8.2	0.1 – 1.2 mg/dl
S. Total Protein	6.00	-	-	6 – 8 gm/dl
S. Albumin	2.60	-	-	3.5 – 5 g/dl

HB: Hemoglobin, WBC: White Blood Cells, HCT: Hematocrit, MCV: Mean Corpuscular Volume, MCH: Mean Corpuscular Hemoglobin, MCHC: Mean Corpuscular Hemoglobin Concentration, RDW-CV: Red Cell Distribution width – Coefficient of Variation

Discussion

The main issues with malaria coexisting with pancytopenia and G6PD deficiency are the higher likelihood of hemolysis and the requirement for cautious patient care. Given the possible consequences of pancytopenia and G6PD deficiency, the first priority should be managing the patient with the proper antimalarial medication.

Red blood cells with a G6PD deficiency are more vulnerable to oxidative damage, which might result in hemolysis. In those who lack G6PD, malaria, especially when the parasites Plasmodium vivax or Plasmodium falciparum are present, can cause oxidative stress, which exacerbates hemolysis.[7] A reduction in all of the blood cell types, known as pancytopenia, can be a consequence of malaria, particularly in severe instances.[7]

Malaria, which may cause everything from asymptomatic illness to serious, life-threatening consequences, continues to pose a serious danger to world health, particularly in endemic areas. Hemolysis and dyserythropoiesis are major causes of hematological abnormalities, with anemia being a well-known side effect. Nonetheless, pancytopenia, which is the concurrent decrease of red blood cells, white blood cells, and platelets, is a quite rare observation in malaria and frequently indicates the existence of other underlying or contributory conditions.[8]

The discovery of pancytopenia in this instance led to a more comprehensive diagnostic strategy. The X-linked enzymatic ailment G6PD deficiency, which makes people more vulnerable to oxidative stress, was one important coexisting condition. Hemolysis brought on by infections, certain drugs, or metabolic stress is especially dangerous for those with G6PD deficiency. In addition to making clinical presentation more difficult, this enzymopathy limits the use of primary antimalarials like dapsone and primaquine, which can cause hemolysis, in the setting of malaria.[9]

Pancytopenia in malaria has a complex pathogenesis. Possible explanations include immune-related hematological reduction, reticuloendothelial system hyperactivity, direct parasitic suppression of bone marrow, and hypersplenism that increases blood cell sequestration and destruction. Furthermore, when pancytopenia is present, viral coinfections such as Epstein-Barr virus infection (EBV), cytomegalovirus (CMV), and parvovirus B19 should be thoroughly examined since these may further impair bone marrow function.[10]

When a patient presents with pancytopenia, G6PD deficiency, and malaria, a thorough and interdisciplinary diagnostic approach is necessary. A smear of peripheral blood and quick diagnostic assays are used to confirm malaria initially; PCR is saved for species determination when required. A quantitative enzymatic assay is used to confirm G6PD deficiency- however acute hemolysis may result in misleading negative results. A whole blood count, reticulated cells, and peripheral blood test are used to assess pancytopenia; if a decrease continue or cannot be explained, a bone marrow biopsy may be undertaken. Secondary causes of bone marrow suppression or hemolysis can be found with the aid of other tests, such as nutritional panels (B12, folate), testing for liver function, LDH, Coombs test, and viral serologies (EBV, CMV, and parvovirus B19).[11]

In G6PD-deficient individuals, management necessitates cautious antimalarial medication selection, avoiding oxidant medications such as primaquine and dapsone. It is preferable and thought to be safe to use artemisinin-based combination treatments (ACT), such as artemether-lumefantrine. In cases of severe malaria, parenteral artesunate may be utilised. Transfusions in cases of severe anemia or thrombocytopenia, folate supplements, and water to avoid renal problems are all examples of supportive treatment. It is crucial to closely monitor blood counts, hepatic and renal function, and reevaluate G6PD status following recovery. Better results and fewer treatment-related issues are guaranteed when overlapping disorders are identified early.[12]

Conclusion

In conclusion, this case illustrates the uncommon but clinically relevant combination of pancytopenia, G6PD deficiency, and malaria. Both diagnosis and treatment are made more difficult by the overlapping pathophysiology, which necessitates cautious antimalarial drug selection and a comprehensive assessment for contributory variables

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such as bone marrow suppression and viral infections. To avoid problems and guarantee positive results, early detection and tailored treatment are crucial.

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