an increase in eosinophilic series cells. Blast ratio was detected as negative. ECHO findings were normal. No pathology was observed in the lung. Diagnostic bone marrow biopsy was performed.EMG revealed sensorimotor demyelination with block at the wrist levelin the right median and neuropathy with secondary axonal damage. It was evaluated as CTS. After the biopsy, corticosteroid treatment was startedOn the 2nd day of treatment, the patient's eosinophil count was 350μLShe was discharged with oral steroid treatment and discharged with oral steroid.In the control eosinophils decreased to2160 μL. In the pathology report of biopsy, hypereosinophilic syndrome was considered.No diagnostic findings were detected in favor of neoplastic/clonal eosinophil expansion

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PP 32

A RARE CAUSE OF THROMBOCYTOPENIA: MALARIA

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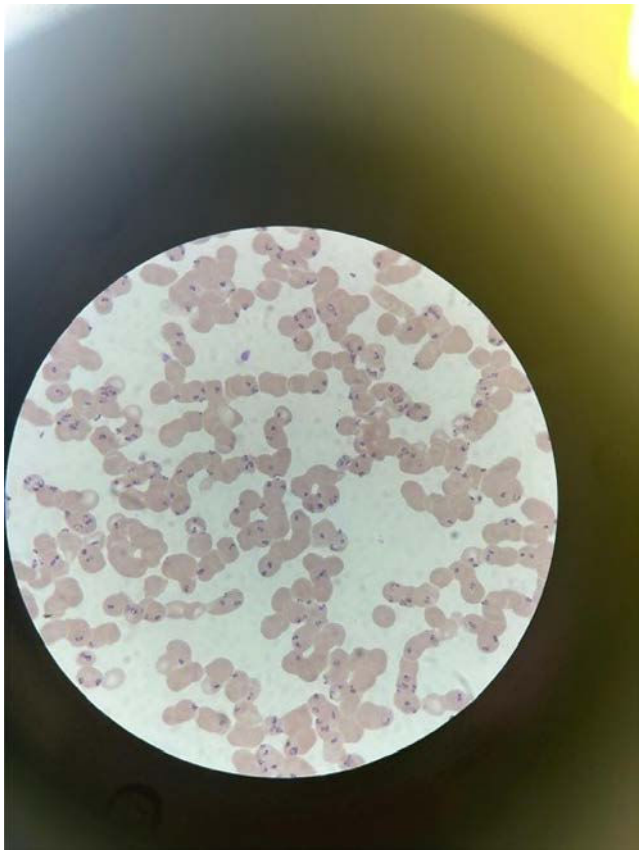
Objective: Malaria is a potentially fatal condition caused by parasites that are spread to humans through the bites of infected female Anopheles mosquitoes, according to the World Health Organization (WHO). Two parasite species, Plasmodium falciparum and Plasmodium vivax, are the most significant threats globally, both known to be infectious to humans. Hematological changes are the most frequent consequences of malaria and have a significant impact on the pathophysiology of the disease. Changes in platelet parameters are considered a hallmark of malaria infection. Often, these changes in malaria infection may be a result of higher levels of parasitemia. Thrombocytopenia is frequently observed in malaria infection. This report presents a case of malaria as a rare cause in a patient investigated for thrombocytopenia. **Case Report:** A 34-year-old male patient with no known medical history presented to the emergency department with complaints of fever, chills, and rigors. Upon admission, his lab results showed wbc: 3,2 thousand/ul, hgb:13.2 gr/dl, neutrophils: 2400, plt:12 thousand/ul, CRP: 232 mg/dl, creatinine: 0.9 mg/dl, AST: 100 u/l, total bilirubin: 2.7 mg/dl, ALT: 66 u/l. The patient was a sailor and had recently returned from the Ecuador Gine region 15 days ago. He had also stayed in Ghana for 40 days prior to that. The patient had taken prophylactic medication for malaria once. Physical examination revealed abdominal tenderness and fever. Peripheral blood smear evaluation revealed widespread ring forms (Figure 1). Following consultation with microbiology, the patient was diagnosed with malaria. The health authority was notified, artemether + lumefantrine medication was procured and the patient was referred to the tertiary care facility. It was later learned that the patient started IV treatment for malaria, but his condition deteriorated, he was intubated and subsequently expired. **Discussion:** Malaria remains a global public health concern considering the number of cases and death rate worldwide. Changes in platelet parameters are considered a hallmark of malaria infection, and often these changes in malaria infection may be a result of higher levels of parasitemia. Studies have shown that the median platelet count was significantly decreased in adult patients with malaria compared to apparently healthy individuals. Thrombocytopenia is one of the most frequent complications of malaria infection, though it is not a criterion for severe malaria, and it is commonly observed in both Plasmodium vivax and Plasmodium falciparum malaria. Previous studies have shown a correlation between parasite density and the severity of malaria infection complications. There is uncertainty regarding the degree of platelet parameter changes that occur during malaria infection and the underlying biological mechanisms associated with parasitemia levels. The speculated mechanisms leading to thrombocytopenia include coagulation disturbances, splenomegaly, bone marrow alterations, antibody-mediated platelet destruction, oxidative stress, and the role of platelets as cofactors in triggering severe malaria. There is no clear recommendation for the adequate management of these hematological complications. It is essential to consider thrombocytopenia and changes in platelet parameters in malaria patients. This report also highlights the need for further research on the subject.



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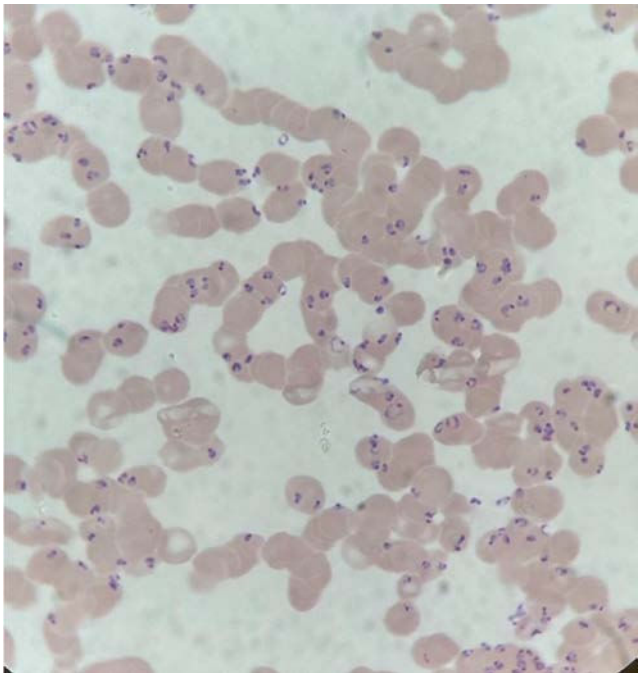

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PP 33

A CASE OF THROMBOTIC THROMBOCYTOPENIC PURPURA RELATED TO MALIGNITY AND CHEMOTHERAPY

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Objective: Thrombotic thrombocytopenic purpura (TTP) is a life-threatening multisystem disease. TTP progresses with Microangiopathic hemolytic anemia (MAHA), fever, thrombocytopenia, neurological symptoms, and renal failure. Due to microangiopathic hemolytic anemia, schistocytes are seen in the peripheral blood smear, resulting in thrombocytopenia. Damage to the brain and kidneys occurs due to microvascular thrombosis, and this is how symptoms appear. In pathogenesis, it is caused by the deficiency of ADAMTS 13 (a disintegrin and metalloproteinase with thrombospondin type 1 motif, member 13), which breaks down the von Willebrand factor (VWF) found in the endothelium into multimers, or the development of antibodies against it. Due to ADAMTS 13 deficiency or decrease in its activity, VWF cannot be separated into small pieces and is arranged in large pieces in the endothelium, causing widespread intravascular thrombosis. Many factors can be considered as triggers for the development of TTP, such as pregnancy, malignancy, medications, and autoimmune diseases. **Case Report:** A 59-year-old female patient was admitted due to thrombocytopenia, epileptic seizure, hematemesis and decreased consciousness while being followed up due to cholangiocellular carcinoma. Due to malignancy, 6 cycles of gemcitabine and carboplatin treatment were applied. The last cure was 6 months ago. In followers, WBC 3.24 10^3/uL (3.7-10 10^3/uL), Hbg 8g/dL(12.9-14.2 g/dL), MCV 84 f/L(81-96fL), platelet 27 10^3/uL (155-356 10^3u/L), total bilirubin 13 mg/dL (0.3-1.2 mg/dL), indirect bilirubin 5.36 mg/dL (0-1.5 mg/dL), LDH 408 U/L (0-247u) /L), creatinine 1.59 mg/dL (0.51-0.95 mg/dl), protein 1+ in full criterion examination, INR 1.36, PT 15.4 sec (10-15 sec), APTT 22.2 sec (21-29 sec) fibrinogen was 1.46 g/L (1.8-3.5 g/L), 3-5 schistocytes were seen in each area in the peripheral smear. Plasmapheresis treatment was started with the preliminary diagnosis of TTP and steroid 80 mg was given. ADAMTS 13 tests were requested. ADAMTS 13 level is 3.78% (40%-130%) low and ADAMTS 13 inhibitor > 80 U/mL (<12U/mL negative, 12-15 U/mL borderline >15U/mL positive), ADAMTS 13 antigen<0.01lU/ mL (0.19-0.81 lU/mL) was seen. As the patient's thrombocytopenia continued, plasmapheresis was started to be performed twice a day after a week. With this treatment, weekly treatment of medicinal rituximab, which could not be treated with platelets, was arranged. However, the patient did not respond to treatment and died. **Conclusion:** In cancer assosiated TTP, endothelial cells are damaged due to abnormal angiogenesis and tumor cell invasion, and vWF multimers in the endothelial wall are exposed. In addition, ADAMTS 13 activity decreases due to antibodies formed against ADAMTS 13. Some chemotherapeutics such as mitomycin c, gemcitabine can cause TTP. When a diagnosis of TTP is considered, plasma exchange should be started immediately. In addition to plasma exchange, steroids are given in the treatment and if there is no response, other immunosuppressive treatments are added. Our patient with high ADAMTS 13 inhibitors is a condition that is thought to contribute to the mortality of TTP. In a study, it was observed that low ADAMTS 13 activity, as well as high ADAMTS 13 inhibitor and low ADAMTS antigen, caused an increase in mortality.

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PP 34

A RARE DISEASE ASSOCIATED WITH IG4, CHARACTERIZED BY SYSTEMIC AMYLOIDOSIS AND LYMPHOPLASMACYTIC CELL DOMINANCE: A CASE PRESENTATION

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