PP 29

A RARE CASE OF PANCREATOBLASTOMA IN A PEDIATRIC PATIENT

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Case Report: Pancreatoblastoma (PB) is a rare malignant neoplasm. PB is frequently detected in children under 10 years of age. Symptoms are nonspecific. When diagnosed, most tumors are enlarged (> 5 cm). Abdominal pain is often the first complaint (44%). Alpha-fetoprotein (AFP) levels are high. Provides long-term survival with surgical resection. It has been reported that the prognosis is poor if metastases are detected. Here we present a seven-year-old female PB case. She applied with the complaint of abdominal pain. On physical examination, a mass was palpated in the epigastric region. The changed laboratory findings were an increased serum AFP level of 171.1 micrograms/L (normal range 0-9 micrograms/L). Abdominal computed tomography (CT) examination revealed a solitary mass of approximately 6 × 4 cm in the tail of the pancreas. Multiple mass lesions were observed in the liver. These lesions were evaluated as compatible with metastasis. She was diagnosed with PB histopathologically after Tru-cut biopsy. Pathologically increased Fluorodeoxyglucose (FDG) uptake (SUVmax: 9.99) was observed in the mass lesion around the right upper quadrant gastric corpus in F-18-Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography (^{18}F-FDG PET/CT). Malignant hypermetabolic metastatic multiple hypodense mass lesions (SUVmax:7.7) were seen in the liver. OPEC chemotherapy was given. In the evaluation performed after 5 cycles of chemotherapy, a decrease in FDG uptake ^{18}F-FDG PET/CT was detected. The patient was evaluated as responsive to treatment. This case report may contribute to the literature with its rarity and treatment approach.

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PP 30

RPL5 NOVEL MUTATION IN A PATIENT WITH DIAMOND BLACKFAN ANEMIA

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Case Report: Diamond-Blackfan anemia (DBA) is a rare inherited disorder characterized by macrocytic anemia, congenital malformations, and growth retardation, typically presenting in the first year of life. RPL5 encodes a component of the 60S ribosomal subunit, and mutations in this gene are associated with DBA, which is usually inherited in an autosomal dominant. Our case is presented after identifying a novel mutation that lacks the typical phenotypic features associated with the condition. A 17-month-old female patient was sent to our hospital because she was pale and had anemia. During the physical examination, the patient exhibited growth and developmental retardation (height in the 3rd to 10th percentile, weight in the 3rd percentile) and a pointed nose; however, no organomegaly or congenital malformations were detected. Laboratory results showed a hemoglobin level of 4.9 g/dL, an MCV of 90 fL, and a corrected reticulocyte count of 0.8%. HbF level in hemoglobin electrophoresis was 3.5%. Bone marrow examination revealed severe hypoplasia in the erythroid series. Genetic examination using next-generation sequencing detected a novel mutation in the RPL5 gene c. 10G>C (p. Val4Leu) (Heterozygous)). Although RPL5 mutations show more severe phenotypic features in DBA, the new mutation detected in our patient caused anemia and developmental and growth retardation without congenital malformation. This genetic change has not been previously reported in the literature as a novel mutation. However, according to the American College of Medical Genetics and Genomics (ACMG) criteria, this variant has been classified as a "variant of uncertain significance". Given that no additional mutations were identified in the whole exome sequencing (WES) analysis conducted on our patient, the hematological and bone marrow findings were consistent with a diagnosis of DBA. **Methodology:** A blood transfusion was administered to the patient, and steroid treatment was started. Our patient responded to steroid treatment during follow-up. WES analysis was also requested for our patient's mother, father, and sibling. Based on the results, donor screening for bone marrow transplantation will be initiated. Once the results are available, the phenotype-genotype relationship can be interpreted more accurately.

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PP 31

HYPEREOSINOPHILIC SYNDROME

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Case Report: A 58-year-old female patient was referred to the hematology clinic in July for examination after leukocytosis was detected in her tests. She has a known history of CKD, Type 2 DM, HT, hyperlipidemia. The patient's general condition is good, and she had a complaint of numbness in her hands. First 1.5 months ago, numbness in her right hand up to the wrist began,especially severe in the first 3 fingers The same complaints started in her left hand 1 month ago. Physical examination findings were within normal limits. On sensory examination, there was hypoesthesia in the first 3 fingers of both hands, especially on the right. In the blood tests performed at the time of admission, leukocytes were 34.440μL, neutrophils 8.840μL, eosinophils 20.540μL, absolute lymphocyte count 3.890μL, monocytes 1.080 μL, hemoglobin 12 g/dL, platelets 348.000 μL, creatinine 1.07 mg/d/L, CRP: 56.6 mg/dL, sedimentation - 10 mm/h were measured. The patient has had borderline eosinophilia (1510μL) since 2022. Flow cytometry was performed on peripheral blood. 11% lymphoid series and 89% myeloid series cells were seen. No abnormality was observed in the lymphoid series. A slight regression in maturation was seen in myeloid series cells and

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.