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3094 – AGGRESSIVE NK CELL LEUKEMIA IN A CAUCASIAN PATIENT

Aggressive NK cell leukemia (ANKL) is a rare malignancy characterized by a rapid progression and poor prognosis. Most cases are associated with an Epstein-Barr virus (EBV) infection. Typically, the disease is observed among Asian populations, Caucasian patients are very uncommon. We present a male, 39-year-old, Caucasian patient referred to our hospital with pancytopenia and hepatosplenomegaly. The white blood count (WBC) was 1.55 (reference range: 3.90-9.90) $\times 10^9/L$, hemoglobin (Hb) was 118 (135-175) g/L and platelets were 41 (140-440) $\times 10^9/L$. Further laboratory investigations revealed elevations of lactate dehydrogenase (LDH) up to 3922 (0-250) U/L, ferritin up to >40,000 (50-360) $\mu g/L$ and C-reactive protein (CRP) up to 13 (0-5) mg/L. In the microscopic evaluation of the peripheral blood, lymphocytes with dark-blue granules and partially with a nucleolus were found. The bone marrow cytology showed the presence of similar-looking lymphocytes. They accounted for approximately 12% of all nucleated cells. In the flow cytometric analysis, the lymphocytes were CD56bright and CD16neg, indicating NK cell lineage. Other positive markers were CD2, CD38 and cytoplasmic CD3. However, surface CD3 and B cell markers were negative. Staining with Epstein-Barr virus-encoded small RNA (EBER) in the histopathological workup was positive. Accordingly, the patient was diagnosed with ANKL. The clinical course was complicated by a hemophagocytic syndrome and by disseminated intravascular coagulopathy (DIC), conditions frequently observed in ANKL. The therapy consisted of chemotherapy containing L-asparaginase and subsequent allogeneic stem cell transplantation was planned. The first two cycles of the chemotherapy showed a good response, but the patient refused the continuation of the therapy. Soon after he developed sepsis and DIC and succumbed to the disease.

3095 – IMMUNE THROMBOCYTOPENIA AND BONE MARROW HEMOPHAGOCYTOSIS ASSOCIATED WITH SARS COV-2

Simon Michaelis¹, Angelika Pichler², Ingeborg Stelzer³, Christoph Tinchon², Dietmar Enko⁴

¹Institute of Clinical Chemistry and Laboratory Medicine, General Hospital Hochsteiermark, Austria, Leoben, Austria; ²Department of Hemato-Oncology, General Hospital Hochsteiermark, Austria; ³Institute of Clinical Chemistry and Laboratory Medicine, General Hospital Hochsteiermark, Austria; ⁴Clinical Institute of Medical and Chemical Laboratory Diagnostics, Medical University of Graz, Austria

The clinical presentation of patients infected with SARS-CoV-2 is remarkably diverse. Likewise, the underlying pathophysiological mechanisms are proving complex. Disturbances in the blood coagulation system and cytokine storm, such as seen in hemophagocytic syndrome, are among the most serious ones. We present the case of a female 79-year-old patient with marked thrombocytopenia of 4 (150-450) $\times 10^9/L$ occurring in the context of a confirmed SARS-CoV-2 infection. Clinically, one episode of epistaxis and petechiae was observed, otherwise no signs of bleeding occurred. Diagnostic workup included microscopic blood smear analysis, bone marrow cytologic evaluation and flow cytometric immunophenotyping. Hematological malignancies, thrombotic microangiopathies and common infections were excluded as cause of the low platelet count. In the bone marrow, cytology, the megakaryocytic lineage presented normocellular. However, several large hemophagocytes with engulfed hematopoietic cells were detected. A further evaluation of markers frequently associated with hemophagocytic syndrome was performed. Ferritin was 2888 (0-150) ng/mL, CRP and GOT were slightly elevated. The white blood count was normal with a marked decrease of lymphocytes to 0.13 (1.10-3.60) $\times 10^9/L$. There was no fever or organomegaly and the patient was in good clinical constitution. Thus, we did not diagnose hemophagocytic syndrome. Due to no other explanation for the clinical and laboratory findings, the patient was diagnosed with immune thrombocytopenia and concomitant bone marrow hemophagocytosis associated to SARS-CoV-2. The first-line treatment consisting of prednisolone and intravenous immunoglobulins failed to induce an increase in the platelet count. As second-line treatment therapy with Eltrombopag, a TPO-agonist, was started and a sustainable response with platelets in the normal range was achieved.