and 1 had methylmalonic aciduria). Average duration of neutropenia was 6,43 months while average age at diagnosis was 19,6 months. During this study 227 (78,82%) examinees were cured, 34 (11,81%) were still being followed up and for 27 (9,38%) of them we didn't know outcome. 39,24% neutropenias were acute and 56,25% were chronic. According to the severity of neutropenia the distribution was 60,42% – 26,39% – 12,85% (severe, moderate, mild). Average duration of neutropenia in cases of severe neutropenia ($ANC < 0,5 \times 10^9/l$) was 9,14 months, in moderate neutropenia ($ANC = 0,5-1,0 \times 10^9/l$) 4,09 months and in mild neutropenia ($ANC > 1,0 \times 10^9/l$) 1,64 months. We recorded infection during neutropenia in 232 (80,56%) examinees while 88 (30,56%) had noted infections before the onset of neutropenia.

Conclusion This study showed that 97,57% children had benign neutropenia and 78,82% were spontaneously cured during the research which are encouraging results. We noted that children with mild neutropenias and those who had no recorded infections during neutropenia had shorter average duration of neutropenia. In conclusion, most neutropenias of the early childhood are benign and have favorable outcome.

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APLASTIC CRISIS INDUCED BY HUMAN PARVOVIRUS B19 AS AN INITIAL PRESENTATION OF HEREDITARY SPHEROCYTOSIS IN A CHILD – A CASE REPORT

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Background A transient, self-limiting aplastic crisis is a rare manifestation of parvovirus (PV) B19 infection, usually seen in patients with underlying haemolytic anaemias. The virus has a predilection for infecting the erythroid progenitor cells of the bone marrow resulting in their lysis and red cell aplasia, although white cell and platelet counts may also decline.

We herein report aplastic crisis induced by PV B19 infection unmasking hereditary spherocytosis in a boy.

Case Presentation A 11-year-old boy presented with high-grade fever, headache, drowsiness, sore throat, and a rash.

On admission he was conscious, but sleepy, drooling, with pale skin and conjunctivae. Physical examination revealed macular, somewhere petechial rash involving the neck and extremities, tachycardia, systolic murmur and hepatosplenomegaly.

Complete blood count revealed RBC of $2.34 \times 10^{12}/L$, with haemoglobin concentration of 64 g/L and reticulocytopenia, WBC $1.3 \times 10^9/L$, and platelet count $75 \times 10^9/L$. Spherocytosis was present on the peripheral blood smear.

Bone marrow aspirate showed suppressed erythropoiesis. The PV B19 infection was diagnosed by polymerase chain reaction (292 000 000 DNA copies/ml of blood) and positive serology for specific anti-PV B19 IgM.

Osmotic fragility testing showed increased fragility of erythrocytes, which was consistent with the diagnosis of hereditary spherocytosis.

The boy was treated by blood and platelet transfusions and supportive care, and was discharged after 9 days of hospital care with improved blood count.

Conclusions PV B19 induced aplastic crisis can be the first manifestation of hereditary spherocytosis. PV B19 infection must be considered in the differential diagnosis in patients with acquired aplastic anaemia.

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AUTOSPLENECTOMY – CASE REPORT

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Introduction Autosplenectomy defines spontaneous splenic infarction leading to hyposplenism. It occurs mainly as a complication of sickle cell anemia, pneumococcal sepsis or systemic lupus erythematosus, but may also be associated with various other conditions. An anatomical variation of the spleen's position, the wandering spleen, has been reported as an underlying cause in less than 0.5% of autosplenectomy cases.

Case Report A 9.5-month-old female was admitted to the Department of Oncology and Hematology of the Children's Hospital Zagreb due to a prolonged fever of an unknown origin and splenomegaly. Laboratory findings detected profound normocytic anaemia, attributed to splenic sequestration. Abdominal ultrasound detected multiple hypoechoic areas in the spleen, indicating splenic infarction, confirmed by MR angiography. An extensive work-up excluded sickle cell anemia, thalassemia, hematological malignancies, myeloproliferative disorders, thrombophilia, autoimmune vasculitis, Kawasaki disease and infection as a cause of the condition. However, repeated ultrasound examination revealed an aberrant, ventrally positioned spleen, which most likely led to an inadequate blood supply and consequently splenic infarction. Antiplatelet therapy and antibiotic prophylaxis were initiated, along with pneumococcal and meningococcal vaccines. The patient is still monitored regularly, currently with normal hematological laboratory findings and none of the complications possibly attributed to hyposplenism.

Conclusion Autosplenectomy in children, although rarely, may result from an aberrant spleen's position. Congenital weakness of the splenic ligaments, allowing its mobility, causes torsion of the splenic artery, leading to blood supply failure and tissue damage. Given the wide differential diagnosis of splenic infarction, it is necessary to exclude numerous underlying medical conditions, primarily sickle cell anemia. A conservative approach is currently the treatment of choice. Hyposplenism, leading to susceptibility to sepsis caused by encapsulated bacteria, requires appropriate antibiotic prophylaxis and vaccination of such patients.

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DIFFICULTIES EMERGING FROM THE END-OF-LIFE CARE IN THE PEDIATRIC INTENSIVE CARE UNITS

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Working in ICUs that involves care for critically ill children is inherently demanding. The intricacy of end-of-life issues in this setting adds additional layer of high demands that health care professionals are inadequately prepared for. An interpretative, qualitative inquiry based on thematic data analysis using focus groups as data collection method was used in order to