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PATHOMORPHOLOGICAL INDICATORS OF CHRONIC MYELOID LEUKEMIA

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Abstract: This article examines the pathomorphological indicators of chronic myeloid leukemia through blood smears, biopsies, and punctures. The causes and clinical manifestations of chronic myeloid leukemia are derived from both local and foreign articles.

Keywords: Chronic myeloid leukemia, Hemoblastosis, Metamyelocyte, Idiopathic myelofibrosis, Polycythemia.

Relevance of the study:Chronic myeloid leukemia (CML) is a clonal myeloproliferative disorder that develops as a result of the transformation of early hematopoietic progenitor cells. The acquired translocation of cytogenetic-Philadelphia chromosomes is central to CML. The emergence of the Philadelphia chromosome occurs due to the exchange of genetic material between chromosomes 9 and 22, resulting in the synthesis of the BCR-ABL gene on chromosome 22. In CML, there is an excessive production of myeloid progenitor cells. CML accounts for the fifth position (8.9% of cases) in the structure of hemoblastoses. The standardized average annual incidence is approximately one case per 100,000 people. It is equally common in men and women, typically affecting individuals aged 30-70, with rare occurrences in childhood and adolescence. The clinical manifestation of CML is characterized by symptom heterogeneity and various manifestations associated with aggressive therapy. The early stage of the disease can often be asymptomatic for several years. Symptoms are often identified during routine check-ups or clinical blood tests for concomitant pathology. Clinical manifestations include multiple syndromes:

- **Intoxication syndrome:** Progressive weakness, loss of appetite, weight loss, sweating, low-grade fever, bone and joint pain, skin itching, and exacerbation of concurrent diseases.

- **Proliferation syndrome**:Pain and heaviness in the left side due to an enlarged spleen, often accompanied by liver enlargement.

- **Anemia syndrome**: General weakness, shortness of breath, pale skin and mucous membranes, severe tachycardia, hypotension, and cardiovascular diseases.

- **Thrombotic complications:** Thrombosis and thromboembolism in various organs and tissues during thrombocytosis, with peripheral vein thrombophlebitis,

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myocardial infarction, and cerebrovascular disorders being common reasons for examination and diagnosis.

- **Hemorrhagic syndrome**: Spontaneous bleeding with petechial rash, often due to thrombocytopenia in the acceleration and blast crisis phases.In 86-88% of CML cases, granulocytes, monocytes, erythrocytes, and megakaryocytes in the bone marrow exhibit the Ph chromosome. Cells with the Ph chromosome constitute 98-100% of bone marrow cells. The variant of CML without the Philadelphia chromosome is rare, with a shorter average life expectancy. Diagnosis is confirmed by the identification of the Philadelphia chromosome (22q-) resulting from the balanced translocation t (9;22) (q34; q11) or the oncogene BCR-ABL in peripheral blood or bone marrow. CML progresses through three stages, each characterized by a specific set of symptoms: chronic, progressive (acceleration phase), and acute (blast crisis).

- Chronic phase: The initial stage of CML, diagnosed in over 80% of newly diagnosed patients. The acceleration phase is identified in 8-10% of patients with CML. The blast crisis is the most aggressive stage, with an unfavorable prognosis, occurring in 1-2% of patients.

- Research objective: To study the specific pathomorphological characteristics of chronic myeloid leukemia.

Research methods:Blood smears, biopsies, and punctured biopsies.

Research results:

- Chronic phase: Peripheral blood: Mild normochromic anemia, leukocytosis 50-1000x10^9/l, increased band neutrophils, presence of metamyelocytes, myelocytes, and promyelocytes. Granulocyte anisocytosis, nuclear and cytoplasmic vacuolization, nuclear polymorphism, absence of neutrophil granules (hypo- and agranulocytosis), possible emergence of blasts, eosinophil-basophil association, decreased lymphocytes, thrombocytosis in 40% of cases (600-1000x10^9/l).

- **Myelogram**: Hypercellular bone marrow, sharp increase in granulocyte lineage cells, eosinophil-basophil association, blasts up to 10%, numerous megakaryocytes, decreased erythrocytes.

- Acceleration phase: Peripheral blood: Moderate to severe normochromic anemia, leukocytosis 50-1000x10^9/l, increased band neutrophils, presence of metamyelocytes, myelocytes, and promyelocytes, blasts up to 15%, eosinophil-basophil association, decreased thrombocytes.

- Myelogram: Hypercellular bone marrow, sharp increase in granulocyte lineage cells, eosinophil-basophil association, blasts up to 15%, decreased megakaryocytes.

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