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BONE MARROW MICROARCHITECTURE ALTERATIONS IN ACUTE AND CHRONIC LEUKEMIAS

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Annotation: The bone marrow is a highly specialized and dynamic organ that serves as the primary site of hematopoiesis and plays a central role in maintaining systemic homeostasis. In leukemias, whether acute or chronic, the normal microarchitectural integrity of the bone marrow is profoundly disrupted as malignant hematopoietic cells proliferate uncontrollably and infiltrate the marrow space. This pathological transformation affects every structural component — the hematopoietic cords, stromal framework, adipose tissue, and sinusoidal vasculature — leading to a complete reorganization of the bone marrow microenvironment.

In acute leukemias, the rapid expansion of immature blast cells causes a total effacement of the marrow's normal architecture. Fat spaces disappear, sinusoids become compressed, and normal erythroid, myeloid, and megakaryocytic precursors are severely depleted. The resulting marrow is hypercellular, monotonous, and morphologically dominated by undifferentiated cells with high mitotic activity. These histopathological changes correlate with the clinical manifestations of anemia, thrombocytopenia, and immunosuppression.

In chronic leukemias, the microarchitectural alteration occurs more gradually. The marrow demonstrates marked hypercellularity with preservation of partial structural organization. Myeloid or lymphoid hyperplasia predominates depending on the disease subtype. Over time, progressive fibrosis, vascular proliferation, and stromal remodeling develop, leading to reduced marrow elasticity and impaired hematopoietic function. These changes are not only diagnostic hallmarks but also indicators of disease stage and transformation potential.

At the microscopic level, leukemic infiltration alters the delicate interaction between hematopoietic stem cells and their stromal niche, disrupting cytokine signaling, adhesion molecule expression, and extracellular matrix composition. This transformation establishes a self-sustaining leukemic microenvironment that promotes malignant survival and therapeutic resistance.

Understanding the morphological and structural remodeling of bone marrow in leukemias provides essential insights into the pathophysiology of these diseases. The assessment of microarchitectural alterations through histology, reticulin and collagen staining, and immunohistochemical techniques remains indispensable for accurate diagnosis, classification, and prognosis. Moreover, the integration of histopathological findings with molecular and

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cytogenetic data enables a comprehensive evaluation of leukemic progression.

This article analyzes the structural, vascular, and stromal modifications of bone marrow microarchitecture in acute and chronic leukemias, emphasizing their diagnostic, prognostic, and functional significance in the context of modern hematopathology.

Key words: bone marrow, microarchitecture, leukemia, hematopoiesis, blasts, histopathology, fibrosis

Main Part

The bone marrow consists of hematopoietic tissue, adipocytes, reticular fibers, and vascular sinusoids embedded in a supportive stroma. Its balanced architecture enables the production of all blood cell lineages within well-defined niches regulated by stromal and endothelial interactions. In leukemia, this structure is destroyed as malignant hematopoietic cells expand uncontrollably, infiltrating the marrow and adjacent tissues.

Microarchitecture in Normal Bone Marrow

Healthy bone marrow demonstrates a mosaic pattern of hematopoietic cords interspersed with adipose tissue. The ratio of fat to hematopoietic elements varies with age, but normal adult marrow retains 40–60% cellularity. The stroma, composed of fibroblasts, macrophages, reticular cells, and endothelial networks, forms a three-dimensional scaffold that maintains stem cell niches. Sinusoidal capillaries with thin endothelial linings facilitate the release of mature cells into circulation.

Alterations in Acute Leukemia

Acute leukemias are dominated by the explosive proliferation of undifferentiated blasts. Histologically, the marrow becomes hypercellular, with nearly complete replacement of adipose tissue by sheets of blasts. The normal architecture—composed of erythroid islands, myeloid clusters, and megakaryocytes—is obliterated. Adipocytes shrink or disappear, and the marrow appears dense and monotonous under light microscopy.

Blasts are large cells with prominent nucleoli, delicate chromatin, and scant basophilic cytoplasm. Sinusoids may become compressed or distorted, and vascular congestion is common. Reticulin and collagen fibers increase as part of a reactive stromal response, leading to focal or diffuse fibrosis**.** This fibrosis contributes to bone marrow stiffness and impedes normal hematopoiesis.
Functionally, these structural changes explain anemia, thrombocytopenia, and neutropenia seen clinically, as normal progenitor cells are displaced by malignant blasts.

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Alterations in Chronic Leukemia

Chronic leukemias progress more slowly and exhibit a different architectural pattern. The marrow remains hypercellular but contains predominantly mature and maturing cells.

In chronic myeloid leukemia (CML), there is marked myeloid hyperplasia extending into the endosteum and sinusoids. Erythroid and megakaryocytic elements are reduced but not absent. The marrow fat decreases gradually, and the architecture remains partially preserved in early stages. With disease progression, fibrosis increases and clusters of atypical megakaryocytes appear. Sinusoidal dilation and vascular proliferation may also be observed.
In chronic lymphocytic leukemia (CLL), infiltration occurs in a nodular or diffuse pattern composed of small, mature lymphocytes. These infiltrates may eventually coalesce, replacing normal marrow elements. The stroma becomes fibrotic, and trabecular thickening is occasionally seen due to secondary sclerosis.

Vascular and Stromal Changes

The leukemic process significantly alters the microvascular network. The number of marrow capillaries may increase, but their architecture becomes irregular and leaky. The endothelium shows hypertrophy and loss of integrity, allowing blasts to enter circulation prematurely. Increased angiogenesis, mediated by vascular endothelial growth factor (VEGF), is commonly observed in both acute and chronic leukemias.

Stromal cells, including fibroblasts and reticular cells, respond to leukemic infiltration by secreting extracellular matrix components and cytokines that further support malignant proliferation. This creates a self-sustaining leukemic microenvironment that promotes tumor survival and resistance to therapy.

Functional and Diagnostic Implications

The histological assessment of bone marrow architecture provides essential diagnostic clues. The degree of cellularity, pattern of infiltration, and extent of fibrosis are evaluated in trephine biopsies using hematoxylin–eosin and silver staining. Acute leukemias typically show diffuse blast replacement with high mitotic activity, whereas chronic leukemias demonstrate patchy or diffuse infiltration by mature cells.

Recognizing these patterns assists in disease classification and staging. Moreover, the assessment of microarchitectural remodeling can predict therapeutic response and potential transformation from chronic to acute phases.

Conclusion

Leukemia profoundly disrupts the delicate microarchitecture of the bone marrow. The transition

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from a balanced, multifunctional hematopoietic organ to a hypercellular malignant tissue reflects the aggressive nature of leukemogenesis. In acute leukemia, massive infiltration by undifferentiated blasts obliterates normal hematopoietic niches, compresses sinusoids, and induces stromal fibrosis. In contrast, chronic leukemia preserves partial marrow organization during early stages but gradually leads to hypercellularity, vascular proliferation, and fibrotic remodeling as disease advances.

These structural changes have significant functional consequences, including suppression of normal hematopoiesis, increased angiogenesis, and alteration of the stromal microenvironment, all of which contribute to disease progression and resistance to therapy.

Understanding the histopathological and microarchitectural alterations in the bone marrow provides critical insights into the biology of leukemia and supports accurate diagnosis, prognostication, and treatment planning. Future studies combining histology, immunohistochemistry, and molecular profiling will deepen our knowledge of leukemic remodeling and may guide new strategies for restoring normal marrow function.

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