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Perspective

Primary Plasma Cell Leukemia: An Uncommon Cause of Severe Leukocytosis

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DESCRIPTION

A rare and extremely aggressive plasma cell cancer is called Plasma Cell Leukemia (PCL). Primary PCL (pPCL) is a *de novo* leukemic progression of pre-existing Multiple Myeloma (MM) that typically occurs at an advanced and late stage. Secondary PCL (sPCL) is another option for PCL development. If there are less than 10,000 leukocytes/µl in peripheral blood, PCL is defined as having circulating plasma cells greater than 20%, or more than 2×10^9/l if there are more than 10,000 leukocytes/µl in peripheral blood. Recent study findings led to the suggestion of revising the PCL diagnostic criteria. The definition of PCL is based on the presence of ≥ 5% plasma cells in peripheral blood, which is considered to have a similar survival rate and prognostic outcome as the classically defined PCL. In comparison to MM, PCL is more likely to have bone marrow failure, extramedullary involvement, and renal impairment, elevated levels of beta-2-microglobulin and Lactate Dehydrogenase (LDH), as well as high-risk genetic events. It's not always the case that peripheral blood plasma cell presence indicates PCL. It is possible for reactive conditions like viral infections, autoimmune diseases, serum sickness, or even angioimmunoblastik T-cell lymphoma to cause marked peripheral blood polyclonal plasmacytosis mimicking PCL. Under those circumstances, plasma cells are not restricted by the kappa or lambda light chains and usually vanish when the underlying illness is properly treated. Flow cytometry and peripheral blood morphologic analysis are essential for making the right diagnosis. Flow cytometric analysis of PCL typically shows positive results for CD38 and CD138. In contrast to MM, PCL expresses HLA-DR, CD117, and CD56 less frequently, whereas pPCL is more frequently positive for CD20 and CD19. In PCL, numerous cytogenetic anomalies have been shown. None of them, though, are unique to PCL. The following have been identified in PCL: chromosomal loss of chromosome 17, chromosome 13 deletion, 1p loss or lq gains, 8q24 rearrangements, *TP53* inactivation, *K-RAS* and *N-RAS* mutations, and immunoglobulin heavy chain (IgH) translocations, including t(4;14), t(11;14), and t(14;16). In PCL, the t(14;20) is less common. However, t(14;20) has been linked to a worse prognosis, a higher frequency of lytic lesions, renal failure, and an increase in urine monoclonal protein in multiple myeloma patients. A concerning prognosis is associated with patients with PCL's increased propensity for extramedullary spread, including CNS. In PCL, hematogenous dissemination results in CNS involvement. Peripheral blood plasma cells may be noticeable before brain involvement. Cerebral dysfunction, spinal radiculopathies, and cranial nerve palsies are examples of clinical findings. Crucial to the diagnostic process are the CSF and MRI examinations. For PCL involving the central nervous system, there is no proven treatment. The literature has reported on various therapies, such as intrathecal methotrexate, methylprednisolone, and cytarabine, as well as combinations of systemic dexamethasone and pomalidomide. Patients with leukocytosis require clinical awareness of PCL because it is an uncommon cause of extreme leukocytosis. In PCL, extramedullary involvement, including the CNS, is fairly common. In the patient who is being presented, the t(14;20) positivity may also be linked to severe leukocytosis and CNS involvement. Diagnosing CNS involvement can be difficult because of the varied burden of symptoms. It's important to rule out common neurologic side effects from myeloma or treatment side effects. When PCL involves the central nervous system, treatment can be difficult, particularly for patients who are elderly and have poor performance.

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