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Editorial on Myelodysplastic Syndrome

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Editorial

Secondary causes of PB cytopenia are much more common than primary BM neoplasms. Various nonclonal disorders affecting the BM, such as viral (particularly retrovirus, parvovirus, and hepatitis virus), bacterial and parasitic infections, autoimmune disorders (such as juvenile rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, immune thrombocytopenic purpura), nutritional deficiencies (such as malnutrition, iron deficiency anaemia, megalobl Diagnostic accuracy has practical implications, and disagreements over diagnosis are common. Cytopenia is a "must" for any MDS diagnosis; however, specific cytopenias have only a minor impact on classification. Furthermore, in individual MDS cases, the lineage exhibiting significant morphologic dysplasia frequently does not correlate with the specific cytopenia. The presence of an increased blast population in both the BM and PB, the evaluation of cytologic abnormalities [1-3] and dysplastic changes among the hematopoietic elements, the evaluation of cellular topography and cellularity, and the presence or absence of fibrosis all contribute to the morphologic classification of MDS.

Traditionally, the World Health Organization (WHO) defined MDS categories based on blast percentage in the PB and BM, but a cut-off of less than 20% is required. MDS with excess blast 1 (MDS-EB-1) is defined as an increased blast population of 2-4 percent in PB or 5-9 percent in BM, whereas MDS with excess blast 2 (MDS-EB-2) is defined as an increased blast population of 5-19 percent in PB or 10-19 percent in BM, or the presence of unequivocal Auer rods by morphologic examination (MDS-EB-2). Morphologic dysplasia can affect one or more hematopoietic lineages. Erythroid dysplastic features include megaloblastoid changes, multinuclearity in erythroid precursors, nuclear lobulation, the presence of pyknosis or chromatin condensation in the nuclei, cytoplasmic fraying involving 50% of the cellular membrane, internuclear bridging, cytoplasmic vacuolization, and the presence of ringed sideroblasts, which are five or more granules en Dyplastic features in the granulocytic lineage, on the other hand, can appear in the presence of myeloblasts and Auer rods, pseudo-Pelger-Hüet changes or nuclear hypo segmentation, and pseudo-Chediak-Higashi cytoplasmic inclusions, abnormal nuclear irregularities, abnormal cytoplasmic granules, and cytoplasmic hypo granulation [4,5].

Micro megakaryocytes, small mononucleotide, binucleated megakaryocytes, and the presence of separated nuclei are all described as megakaryocytic dysplastic features. It should be noted that the range of dysplastic changes can range from minor to outright bizarre. According to the WHO, at least 10% of the cells in a given lineage must be dysplastic to be considered a significant finding; however, interobserver variability is more problematic in cases where the degree of dysplasia is close to the 10% threshold. This cellular enumeration should be done in batches of 200 PB leukocytes and 500 BM nucleated cells. Changes in hematopoietic stem cells were widely regarded as one of the disease-initiating events in MDS, whether in primary de novo disease or secondary therapy-related causes, both of which result in disrupted hematopoiesis. Flow cytometry (FC) has been considered an important tool for the diagnosis, prognosis, and monitoring of the disease course, despite the fact that morphology is one of the key features in the diagnosis.

The International and European LeukemiaNet Working Group Guidelines (ELN IMDS Flow WG) suggested standardising protocols for using FC in MDS. Before incubating mature erythrocytes in a panel of antibodies, a bulk lysis method should be used, and a minimum of 100,000 CD45+ events should be acquired. The cellular dysplastic features can be used to detect surface antigen aberrations using multiparametric FC (MFC). The application of MFC in MDS focuses on the evaluation of precursor myeloid antigen abnormalities, abnormalities in myeloid maturational patterns among the granulocytic and monocytic lineages, the enumeration of blast immunophenotypes, and, to a lesser extent, the decrease in progenitor B-cells (hematogones). A four-parametric scoring system (referred to as the Ogata score) is widely used as a simple FC criterion for MDS diagnosis, with a reported specificity of 93 percent and sensitivity of 70 percent.

The following are the parameters described in this scoring scheme: (1) SSC of neutrophils defined as the lymphocyte SSC ratio (granulocyte/lymphocyte SSC); (2) percentage of CD34+ myeloid precursors among all viable nucleated cells (percent of CD34+ myeloblasts); (3) percentage of CD34+ precursor B-cells (hematogones) among all CD34+ cells (percent of CD34+ B-cells); and (4) CD45 antigenic expression of CD34+ myeloid progenitors as Cytogenetics has long been recognised as an important and necessary parameter in the diagnosis of MDS. In MDS, the WHO places a high value on cytogenetic abnormalities. Cytogenetics, in addition to establishing a clonal process in patients with peripheral blood cytopenia, plays a significant role in prognosis, clinical-morphologic correlation, therapeutic strategies, and predicting the likelihood of progression to AML. Unlike other myeloid malignancies where the diagnosis is defined by a single cytogenetic event (such as chronic myeloid leukemia and acute promyelocytic leukaemia), MDS has a wide range of cytogenetic-defining lesions, making diagnosis difficult. Nonetheless, approximately 50% of MDS patients have normal cytogenetics

Conflict of Interest

None.

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