Biological pictures

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Do not forget megakaryocytes morphology when you deal with chronic myeloid leukaemia

N'oubliez pas de regarder la morphologie des mégacaryocytes lorsque vous êtes face à une leucémie myéloïde chronique

Jean-Baptiste Rieu[1]
Suzanne Tavitian[2]
Francois Vergez[1]
Laetitia Largeaud[1]

[1] Haematology laboratory, Cancer University Institute of Toulouse, Oncopole, France

[2] Clinical haematology unit, Cancer University Institute of Toulouse, Oncopole, France

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A 40-year-old man treated for chronic myeloid leukaemia (CML) was referred for investigation of persistent pancytopenia (hemoglobin: 96 g/L, platelets: 72×10^9/L, polymorphonuclear neutrophils: 0.9×10^9/L versus hemoglobin: 114 g/L, platelets: 370×10^9/L, polymorphonuclear neutrophils: 74.3×10^9/L at diagnosis). After 2 years of treatment with tyrosine kinase inhibitors (TKI), dasatinib then imatinib, complete cytogenetic response was achieved but *BCR-ABL1* transcript was still positive in blood (1.6%). TKI dose reduction has been performed one year before but without effect on pancytopenia. The reaching of major molecular response was the principal therapeutic issue for this patient; therefore TKI interruption was not considered an acceptable option. Bone marrow aspirate examination showed hypercellular marrow with marked dysmegakaryopoieisis with more than 50% of large multinucleated megakaryocytes (*figure 1A*). This morphological feature, uncommon in CML, was absent at diagnosis, where marrow was hypercellular but megakaryocytes were smaller with hypolobed nuclei (*figure 1B*) as conventionally expected in CML. The karyotype was normal. Next generation sequencing (NGS) revealed three *STAG2* mutations (variant allele frequencies of 70%, 4% and 3%). These results suggested a myelodysplastic syndrome with single lineage dysplasia (MDS-SLD) and not toxicity of TKI. Higher doses of imatinib were well tolerated and associated with a better molecular response. However *BCR-ABL1* level remained above major molecular response threshold. Imatinib has been replaced by nilotinib and allogeneic hematopoietic stem cells transplantation was discussed to treat the MDS.

Megakaryocyte morphology is an important feature in CML. In case of isolated thrombocytosis, the presence of megakaryocytes with hypolobed nuclei is more in favor of CML than essential thrombocythaemia where large megakaryocytes with hyperlobed nuclei (staghorn-like nuclei) are expected (*figure 1C*). It can also be useful in case of cytopenias to differentiate TKI toxicity and the occurrence of another malignancy such as MDS, especially in cases where TKI interruption is not considered as an acceptable option.

Correspondence: J.-B. Rieu
<rieu.jeanbaptiste@iuct-oncopole.fr>

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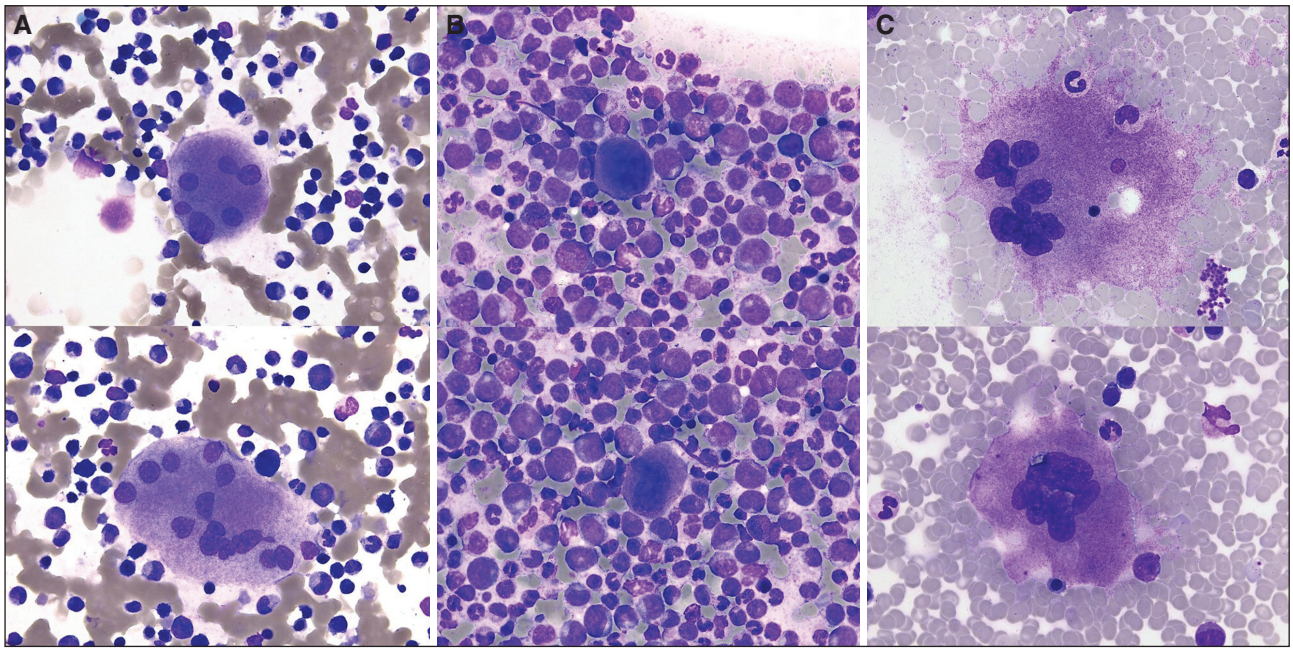

Figure 1. A. Large multinucleated megakaryocytes in myelodysplastic syndrome. **B.** Small megakaryocytes with hypolobed nuclei in chronic myeloid leukaemia. **C.** Large megakaryocytes with hyperlobed nuclei (staghorn-like nuclei) in essential thrombocythaemia.

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