Educational Material IQAP 1041

The blood film was taken from a 34-year patient complained of right thigh mass. A complete blood count showed WBC 385.7 $x10^9$/L, Hb 7.7 g/dL and platelet 106 $x10^9$/L. He was diagnosed of chronic myelogenous leukaemia (CML). The high white cell count, presence of circulating myeloid precursor cells, bi-modal prominence of neutrophils and myelocytes as well as basophilia are characteristic features (Figures 1-2). Blasts account for 1 to 2% of the white blood cells, compatible with chronic phase of CML. Cytogenetic analysis and molecular study confirm the diagnosis by showing the presence of Philadelphia chromosome t(9;22)(q34;q11.2) and *BCR-ABL1* fusion transcripts, respectively.

CML is a myeloproliferative neoplasm characterized by leukocytosis at different stages of maturation and the presence of *BCR-ABL1* fusion gene resulting from the translocation of the long arms of chromosome 9 and chromosome 22. Basophilia is inevitably present and eosinophilia is common. Monocytosis is rarely present at diagnosis. Splenomegaly is almost a constant clinical finding at presentation.

Evidence of t(9;22)(q34;q11.2) and *BCR-ABL1* fusion gene, as indicated by conventional cytogenetics, fluorescence in-situ hybridization or reverse-transcriptase polymerase chain reaction, are required to confirm the diagnosis according to the World Health Organisation Classification 2008. Such information is not only useful for diagnosis but also salient in therapeutic consideration of tyrosine kinase inhibitor.

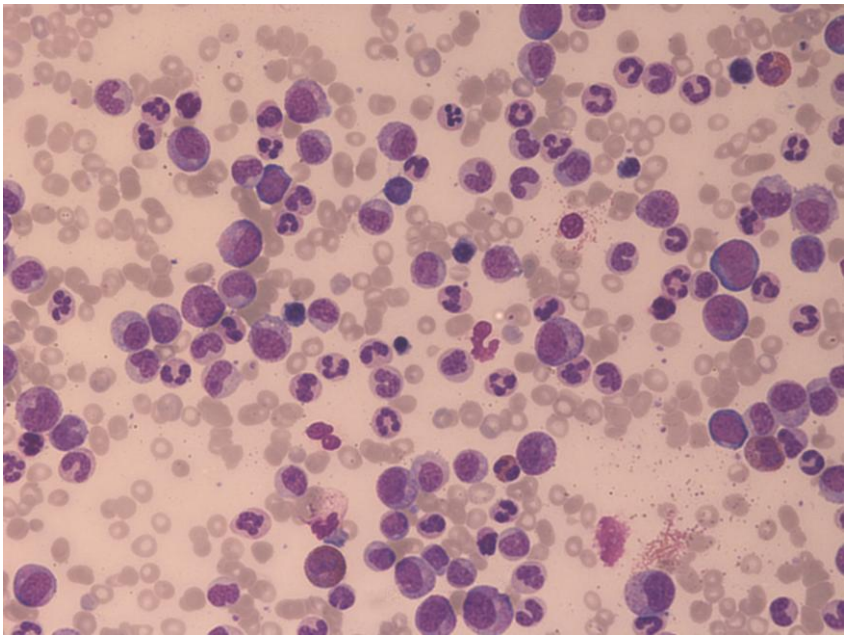

Figure 1. A marked leucocytosis with the predominance of myeloid progenitors and immature myeloid cells (400x magnification).

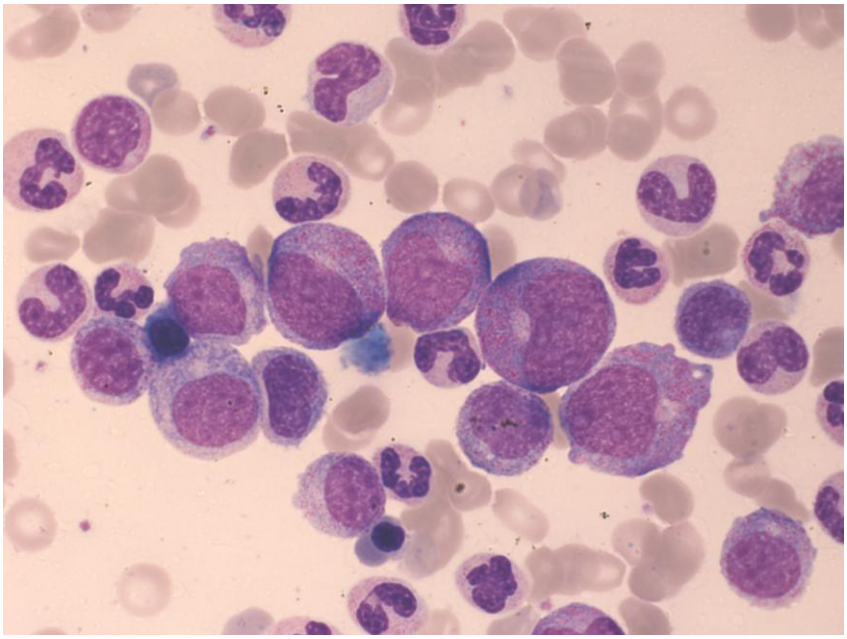

Figure 2. A hierarchy of myeloid linage cells (1,000x magnification).