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Intrasinusoidal Erythroblast Localization in Case of Secondary Acute Myeloid Leukemia

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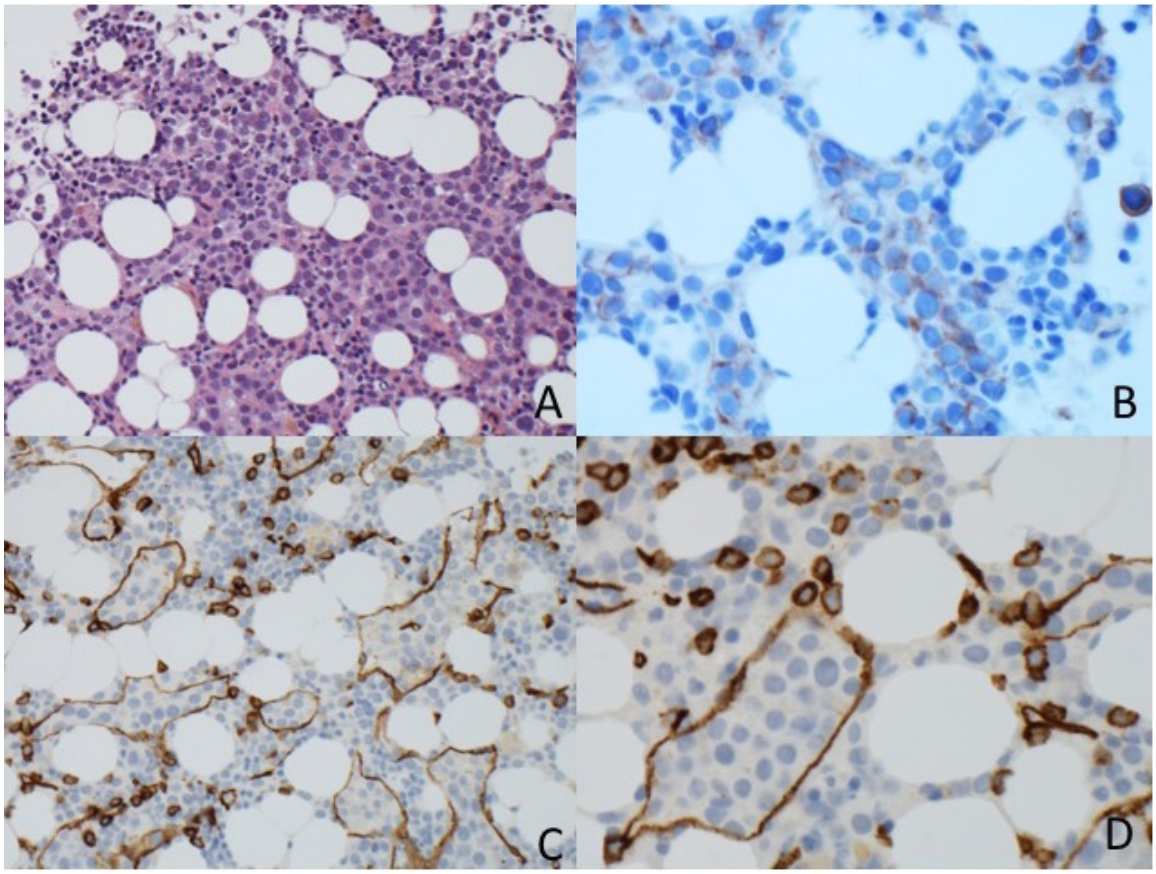

Figure 1: Intrasinusoidal erythroblasts in bone marrow. (A) H&E, x200; (B) E-cadherin (IHC), x400; (C) CD34 (IHC), x200; (D) CD34 (IHC), x400.

A 68-year-old man, on disease modifying medications for inflammatory arthritis, was referred for investigation of neutropenia and thrombocytopenia. Bone marrow trephine section demonstrated myelodysplasia with excess blasts (MDS-EB). Cytogenetics were complex and highly adverse with multiple chromosomal deletions and additions (43∼ 44,XY,?add(3)(q2?7),add(4)(p14),del(5)(q?q33),?add(6)(q?2),del(7)(q?q?),add(11)(p1?),-12,add(15)(q2?2),-16,-20,add(21)(p10),+1∼ 5mar[cp10]). He commenced azaciditine.

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After ten cycles he developed worsening cytopenias, not responsive to cessation of azacitidine. He underwent further bone marrow examination which showed a markedly dysplastic aspirate with myeloblasts quantified morphologically at 23% of all nucleated cells (ANCs) confirming disease progression to secondary acute myeloid leukemia (sAML). He commenced Vyxeos (liposomal daunorubicin and cytarabine). There was no improvement in his counts at day 53 (haemoglobin 86 g/L, white cell count 0.7 x 10^9/L, neutrophils 0.36 x 10^9/L, platelets 6 x 10^9/L). Bone marrow trephine section revealed a hypercellular marrow with extensive remodelling of the bony trabeculae. Erythropoiesis was abnormal with erythroid islands of atypical erythroblasts located within the sinusoids (Figure 1, Panel A & B). Blasts were elevated at 15% of all nucleated cells (Figure 1, Panel C & D).

Cytogenetics supported clonal evolution with the emergence of a hypertriploid population in addition to the abnormalities present at diagnosis (71∼ 73,XX,-Y,add(1)(q4?),add(2)(p2?),add(3)(q2?5),-4, del(5)(q1?),+6,del(7)(q1?),+8,del(9)(q2?),+add (9)(q2?),del(10)(q2?),add(11)(p1?4),+add(11)(p1?4)-12,i(13)(q10), +14,add(15)(q2?2)-16,der(21)?t(1;21)(p1?;q1)-22,+3∼ 8). He declined further therapy.

Erythroblastic islands are composed of erythroid progenitor cells encircling a central macrophage. As erythropoiesis progresses, erythroblastic islands migrate toward the sinusoids to allow reticulocytes to be released into peripheral blood. The role of these erythroblastic islands in AML is not well elucidated, although the macrophage population may deliver pro-survival signals to the surrounding leukemic blasts.

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