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Myelophthisic marrow involved by breast cancer and acute myeloid leukemia

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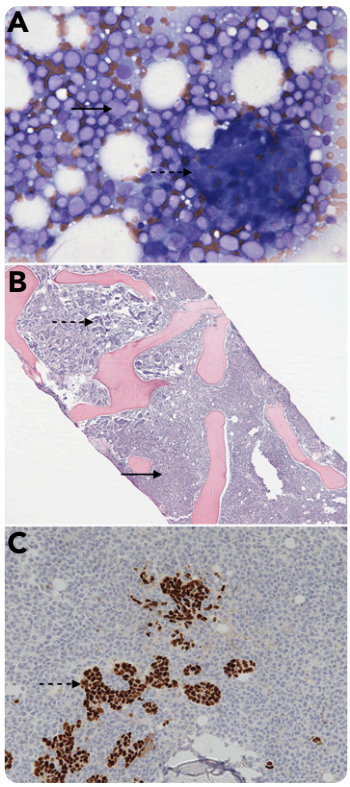

A 53-year-old woman was admitted with right thigh pain. She was diagnosed with estrogen receptor (ER)- and progesterone receptor (PR)-positive stage III adenocarcinoma of the left breast 3 years before. She was treated with lumpectomy and adjuvant doxorubicin and cyclophosphamide followed by paclitaxel and radiotherapy, but declined hormonal therapy. A right hip radiograph showed multiple lytic lesions, and positron emission tomography/computed tomography imaging confirmed widespread bony lesions. A complete blood count revealed a white blood cell count of 25 $\times$ 10^9/L with 20% blasts, a hemoglobin count of 11.1 g/dL, and a platelet count of 52 $\times$ 10^9/L. Bone marrow touch preparation (panel A; Wright's stain, original magnification $\times$400) and core biopsy (panel B; hematoxylin and eosin stain, original magnification $\times$40) were effaced by myeloblasts (solid arrows) and infiltrating adenocarcinoma arranged in clusters, ducts, and cords (dashed arrows) that were ER (panel C, dashed arrow; original magnification $\times$200) and PR positive and Her2 negative by immunohistochemistry. Cytogenetics revealed t(9;11)(p22;q23) and monosomy 7. Fluorescent in situ hybridization was positive for an *MLL* rearrangement. The patient was started on an aromatase inhibitor for her metastatic breast cancer and hypomethylating therapy for her acute myeloid leukemia (AML).

Rearrangements of the *MLL* (or *KMT2A*) gene are frequently seen in therapy-related AML after treatment with topoisomerase II inhibitors with a short latency of 2 to 3 years as compared with AML induced by alkylating agents, which has a latency of 5 to 7 years.

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