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Isolated CNS leukaemic relapse in acute myeloid leukaemia

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DESCRIPTION

A 61-year-old woman with acute myeloid leukaemia (AML) without maturation (M1) with trisomy 13 who achieved complete remission after her initial diagnosis with cytarabine and idarubicin. Two years later, she relapsed and underwent successful haematopoietic stem cell transplantation afterwards, and she entered complete remission. Sixteen months later, she presented to our emergency department with a 2-week history of worsening fatigue, progressive expressive aphasia and then she lapsed coma. On the workup, there was no identifiable reversible metabolic or infectious cause of her presentation. Brain MRI revealed multifocal patchy abnormalities in the white matter (figure 1A,B). Her lumbar puncture (LP) revealed blasts on the cytocentrifuge preparations of cerebrospinal fluid (CSF) (figure 1C). Flow cytometry on the CSF confirmed the presence of a myeloblast population with 14% blasts with expression of CD13, CD33, CD34, CD45 and Human Leukocyte Antigen – DR isotype (HLADR). And fluorescence in situ hybridization test was negative for Promyelocytic Leukemia/Retinoic Acid Receptor alpha (PML/RARa) translocation. There was no evidence of blast cells on the peripheral blood smear. And, bone marrow aspirate and biopsy revealed no evidence of blasts, indicating isolated central nervous system (CNS) relapse as the patient had no signs of other extramedullary involvement manifestation. Despite treatment with intrathecal methotrexate, she continued to deteriorate and died.

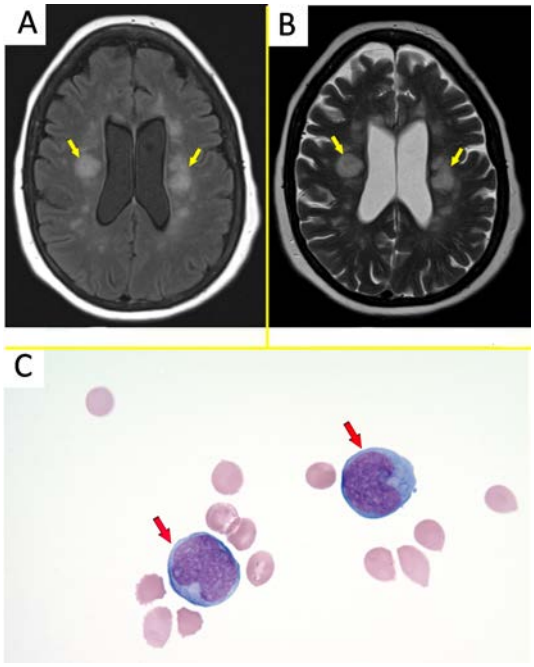

Figure 1 (A,B) Brain MRI T2 weighted sequence showing periventricular flair and hyperintensity (yellow arrows) with multifocal patchy abnormalities in the white matter characterised as restricted diffusion. (C) Cerebrospinal fluid showing large cells with increased nuclear size, increased nuclear/cytoplasmic ratio, fine chromatin and prominent nucleoli, consistent with blasts (red arrows).

CNS complication in patients with leukaemia can be related to several mechanisms; direct involvement, vascular complications and infectious complications related to the disease itself or the treatment. CNS involvement in adults with AML is less common when compared with acute lymphoblastic leukaemia and is rare in general, and routine evaluation for CNS involvement is not recommended in absence of CNS symptoms.[1] Symptoms of CNS involvement depends generally on the involved anatomy and pathology. LP should be the initial workup for suspected CNS involvement and identification of leukaemic blasts on CSF should confirm the diagnosis.[2] CNS imaging studies should be performed based on the manifestations and to exclude other suspected possible pathology (eg, stroke, inflammatory or vascular lesions). Treatment include intrathecal chemotherapy and radiation therapy. Particular clinical features associated with a higher risk for CNS involvement in AML have been reported in the literature.[1 3]

We thought the MRI finding, in this case, was related to myeloid sarcoma in the setting of isolated CNS leukaemic relapse. Myeloid sarcoma is a term for a tumour mass of myeloblast or immature myeloid cells occurring in an extramedullary site or the bone.[4]

This case demonstrates a good example of CNS imaging in CNS leukaemic relapse that can be an

Learning points

- Central nervous system (CNS) involvement in acute myeloid leukaemia is rare compared with acute lymphoblastic leukaemia.
- Lumbar puncture with cerebrospinal fluid analysis for blasts should be the initial workup once CNS involvement is suspected.
- CNS imaging should be directed towards a specific clinical presentation, and it can aid in the diagnosis.

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instructive case for other clinicians. Also, it highlights the importance of imaging and pathology along with other clinical data in yielding the final diagnosis.

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