

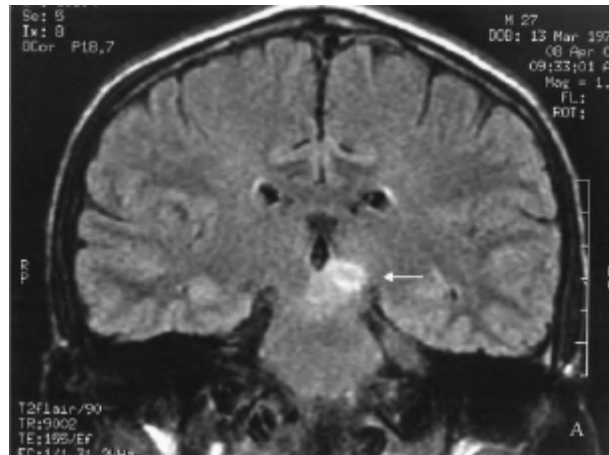
## Ophthalmic zoster sine herpette presenting as oculomotor palsy after marrow transplantation for acute myeloid leukemia

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A 34-year-old man relapsed 11 months after matched sibling stem cell transplantation (SCT) for acute myeloid leukemia (AML). A second remission was obtained with chemotherapy and additional stem cells. Four months later, isolated extramedullary relapse occurred in the tonsils and lymph nodes. A third remission was obtained after withdrawal of cyclosporin and radiotherapy. At 17-month, he complained of diplopia on right lateral gaze. Physical examination showed left third nerve palsy with subclinical uveitis but normal visual acuity. A lumbar puncture showed acellular cerebrospinal fluid (CSF) with normal protein levels and negative for herpes simplex and zoster viruses by culture and polymerase chain reaction (PCR). A cranial magnetic resonance imaging (MRI) showed abnormal hyperintense signal on T2-weighted scan in the left side of the midbrain extending into the inferior aspects of the adjacent left thalamus (Figure 1A). The lesion was contrast enhancing on T1-weighted imaging and was compatible with leukemic infiltration. However, there was no clinical response to intrathecal methotrexate (12 mg) or external radiotherapy (10 Gy). One week later, painful zoster eruption occurred (Figure 1B) with ciliary and corneal involvement, with positive zoster immunofluorescence. There was complete oculomotor recovery after treatment with acyclovir (500 mg every 8 hours x 7 days) and intravenous immunoglobulin (21g x 2 days). Isolated CNS relapse is not uncommon after hemopoietic SCT, especially after repeated marrow and extramedullary AML relapse. However, the focal nature of the lesion, negative CSF finding and lack of response to intrathecal therapy are all unusual. In our case, initial confusion was caused by initial absence of skin lesions (zoster sine herpette), isolated oculomotor inflammation on neuroimaging and negative CSF findings. Zoster ophthalmicus seldom causes extraocular muscle palsy, although subclinical IV and VI nerve compromise have been reported.<sup>1</sup> The largest series of post-SCT zoster ophthalmitis reported uveitis and keratitis in all seven cases, but not ocular palsy or delayed eruptions.<sup>2</sup> Neuroimaging abnormalities may precede skin eruptions for post SCT zoster encephalitis, but inflammation is usually extensive and CSF study is usually diagnostic.<sup>3</sup> In our case, the negative CSF detection of zoster virus may be due to early lumbar puncture, very localized reactivation or the presence of PCR inhibitors.<sup>4</sup> Hence, in SCT recipients, although imaging may help localize CNS lesions, diagnosis is seldom certain without biopsy. The use of molecular tests and empirical therapy cannot replace the role of continued clinical assessment. Atypical presentation of common conditions is still the most common culprit, and prompt treatment may prevent irreversible damage.

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**Figure 1. A** Coronal T2-weighted magnetic resonance scan showing hyperintense area (arrow) within the left side of the midbrain extending to the inferior aspects of the left thalamus. No mass effect is noted.

**Figure 1. B** Delayed onset of pain and vesicles on V1 dermatome one week after initial presentation.

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