Acute myeloid leukaemia masquerading as transverse myelitis
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Abstract
Acute myeloid leukaemia (AML) is known to cause paraneoplastic transverse myelitis. However, the direct invasion or extramedullary compression of the spinal cord is rare unlike in the case of chronic lymphocytic leukaemia. We report the case of a young man presenting with transverse myelitis who was found to have leukaemic infiltrates on neuroimaging. The uniqueness of this being the first presentation of the patient with AML in the form of epidural infiltrates in the absence of peripheral blood count abnormalities. This highlights the importance of neuroimaging to delineate associated conditions in a suspected patient with transverse myelitis.

Key words: Transverse myelitis, acute myeloid leukaemia, extramedullary spinal compression

Introduction
AML is a malignant proliferation of undifferentiated myeloid precursor cells in the bone marrow. The immature blast cells are seen in the peripheral blood smear and deposited in other tissues. The normal production of blood cells in the bone marrow is suppressed giving rise to symptomatic anaemia, bleeding diatheses and recurrent infections. AML is the commonest acute leukaemia among adults with a median age of onset at 65 to 70 years.¹ Central nervous system (CNS) involvement in leukaemia is multifaceted ranging from leukaemic infiltrations, granulocytic sarcomas (chloromas), bleeding and thrombotic manifestations and CNS infections to paraneoplastic phenomena and drug side effects.²,³

Case Report
Mr. C is a 27-year-old, a tractor driver presented with an episodic chest pain which was distributed in a band like pattern from the back to the local hospital. He was treated with non-steroidal anti-inflammatory drugs with no improvement. Over three days he developed a gradually worsening numbness and weakness of the bilateral lower limbs with bladder and bowel retention. The fourth day of the illness he was bed bound. He presented to us with a bilateral spastic paraparesis and acute urinary retention. He had bilateral extensor plantar reflex, with exaggerated knee jerk. Notably his ankle reflex was absent. There was loss of pain, light touch sensation and proprioception upto T2. There was no spinal tenderness. An evolving transverse myelitis, myeloradiculopathy or myeloneuropathy was suspected. Though rapidly progressed given the subacute nature of onset an inflammatory or infective pathology was suspected.

The full blood count revealed a white blood cell count of 4.75 x 10⁹/UL, a mild neutropenia of 1.46 x 10⁹/UL and a thrombocytopenia of 141 x 10⁹/UL. The Haemoglobin level was 11.5 g/dL. There were no abnormal or atypical cells in the peripheral blood. These finding were keeping with an ongoing viral infection or post viral syndrome. The inflammatory markers were however normal. After excluding an active sepsis Mr. C. was started on intravenous methyl prednisolone while awaiting the magnetic resonance imaging (MRI) of the spinal cord which revealed diffuse heterogeneously contrast enhancing marrow involving the whole spine and sternum with multiple enhancing epidural lesions suggestive of leukemic infiltrates.

An urgent peripheral blood smear revealed 23% blast cells, normochromic normocytic red cells and mild thrombocytopenia and moderate rouleaux formation. Intravenous steroids were withheld and he was transferred to the National Cancer Institute for further evaluation and interventions. Subsequently a bone marrow biopsy confirmed the diagnosis of acute myeloid leukaemia, and chemotherapy was commenced.

Discussion
The involvement of spine in acute myeloid leukaemia is a rare manifestation. Myelopathy in leukaemia can result from direct leukaemic infiltrates, compressive effects of bony metastases, paraneoplastic inflammation of the cord. Compressive lesions of the spine can occur in the form of both leukemic infiltrates as well as granulocytic sarcomas (chloroma) and bony metastases. In 2018 Lee

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et al. reported a rare case of an intramedullary myeloid sarcoma mimicking transverse myelitis. Leukaemia associated bleeding or ischemia, vitamin B12 or copper deficiency, infections or post-infectious states, and post-radiotherapy or chemotherapy can involve the spinal cord causing myelitis or myelopathy. There have been several reports of acute leukaemia presenting as para-neoplastic transverse myelitis.

The first presentation of an AML without overt clues in the peripheral blood counts is highlighted.

An extramedullary compression of the spinal cord results in findings that are similar to that of transverse myelitis. The temporal progression of the illness as well as the circumstances such as a concurrent or preceding viral infection or autoimmune disorder points towards a non-compressive inflammation. However, in our patient too, the temporal progression was misleadingly suggestive of a transverse myelitis rather than a compressive pathology. With regards to the management of this patient prompt treatment of the AML is vital in order to prevent the systemic complications. Furthermore, the need for a neurosurgical intervention such as a laminectomy was discussed and planned. The need for early intervention as well as long term rehabilitation of this young breadwinner is highlighted.

Conclusion

We report the rare case of a young adult who presented with a subacute myelopathy found to have acute myelocytic leukaemia with spinal infiltrates. The importance of broader aetiological differentials and prompt neuroimaging in a patient presenting with a transverse myelitis like picture and the need to screen for spinal involvement in a diagnosed patient with AML with suspicious symptoms are highlighted.
References


