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Images

An unusual case of posterior column myelopathy

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A 51 year-old Caucasian male with past medical history of stage IIIA IgG kappa multiple myeloma with progression to plasma cell leukemia, presented with new progressive lower extremity sensory impairment. He received intrathecal methotrexate therapy two months prior to his presentation. Sensory exam was notable for allodynia, decreased pinprick sensation, vibration and position sense in the lower extremity more than the upper extremity without clear sensory level. Motor exam showed profound lower extremity weakness, which was present prior to his presentation and it was attributed to polyradiculoneuropathy secondary to neoplastic processes. MRI of the brain, cervical, thoracic and lumbar spine demonstrated longitudinally extensive T2 hyperintensity from the level of C1 of the cervical cord to L1 of the lumbar cord without enhancement. The T2 hyperintensity was restricted to the posterior column similar to that of subacute combined degeneration (Fig. 1). Workup for longitudinally extensive myelopathy, including CSF studies (WBC 0, glucose 89, protein 22), flow cytometry, protein electrophoresis, aquaporin 4 antibody, serum and CSF autoimmune paraneoplastic panels, vitamin B1, B12, E, folate, copper, RPR, HIV, CSF EBV/HSV/CMV PCR, were non-diagnostic. The serum zinc and vitamin B6 were noted to be mildly low.

What is the most likely cause?

A. Spinal cord infarction
B. Vitamin B12 deficiency
C. Nitrous oxide abuse
D. Intrathecal methotrexate
E. Copper deficiency

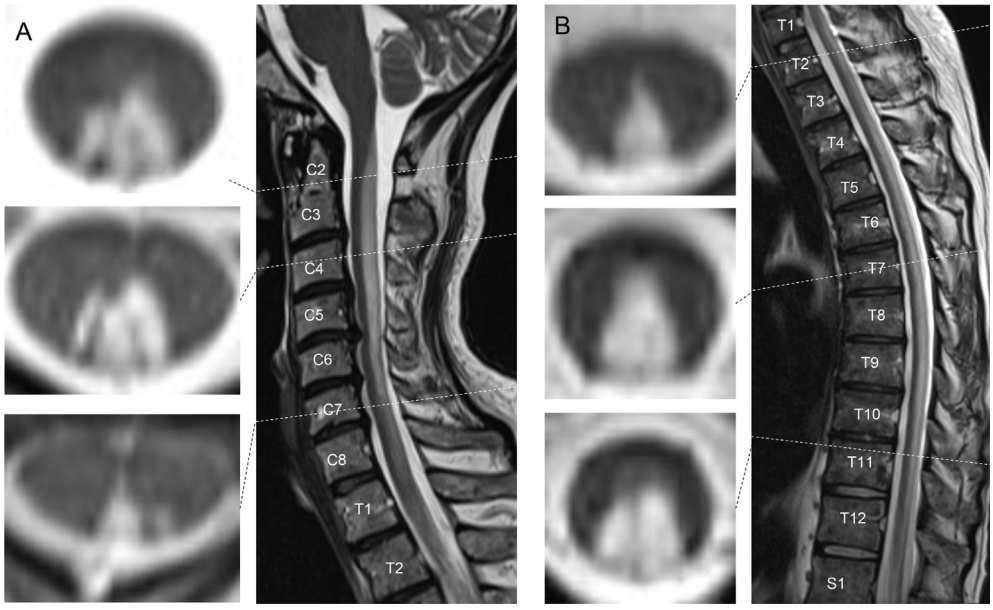

Fig. 1. MRI of the cervical (A) and thoracic cord (B) showed longitudinally extensive T2 hyperintensity along the posterior column. Notice that the shape of the hyperintensity can vary at different cord levels, but they are all restricted to the posterior column.

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1. Discussion

The MRI image showed hyperintensity in the posterior column on the T2-weighted image. Vitamin B12 deficiency, nitrous oxide abuse, and intrathecal methotrexate can all produce posterior column T2 hyperintensity [1–3]. However, in the setting of leukemia and chemotherapy, intrathecal methotrexate should be suspected as the most likely etiology. Spinal cord infarction can produce T2 hyperintensity involving the central cord. However, the hyperintensity does not follow a particular vascular territory, making it an unlikely diagnosis [4].

The imaging findings of methotrexate-induced myelopathy often resemble that of subacute combined degeneration from B12 and folate deficiency [3]. Time between treatment and development of myelopathy can be weeks to months. Even though serum vitamin levels, including folate and B12, can be normal, direct exposure to methotrexate in the spinal cord can cause a process similar to subacute combined degeneration. There is currently no standardized treatment for such a disease process. High doses of S-adenosylmethionine, folinate, vitamin B12 and methionine have been observed to improve paraparesis in a single case [5]. The case presented here was treated with folate, vitamin B1, B6 and B12, but there was no reported improvement in sensory or motor function.

Pathology of the spinal cord in an immunosuppressed patient carries a very broad differential diagnosis. Infectious, inflammatory, autoimmune, vascular, neoplastic, paraneoplastic, and toxic metabolic etiologies all must be considered. However, in the setting of intrathecal methotrexate administration and findings of longitudinally extensive T2 hyperintensity restricted to the posterior column, intrathecal methotrexate-induced myelopathy should be high on the differential diagnosis.

Author contributions

Kwo Wei David Ho conceptualized and drafted the manuscript. Christine Smith, Miguel Chuquilin and Maria Jose Bruzzone critically reviewed the article and provided revisions for intellectual content.

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