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A novel case of an intramedullary spinal cord metastatic lesion resulting from a gastric lymphoma

Benjamin L Judkins; Joseph A DeCosmo; Matthew D Bloom*; Coy D Heldermon

University of Florida College of Medicine, Gainesville, Florida, USA.

***Corresponding Author: Matthew D Bloom, MD**
University of Florida College of Medicine, Gainesville, Florida, USA.
Tel: (727) 259-3012, Fax: (607) 224-8397;
Email: mdbloom4@gmail.com

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Background

Gastric Lymphoma is a rare condition, accounting for only 2% of all lymphomas [1]. Intramedullary Spinal Cord lesions resulting from Metastases (ISCM) are also extremely uncommon, primarily arising from malignancies in the lung and breast. Only 2% of patients are reported to have contracted secondary spinal cord metastases from a primary lymphoma [2]. There has been some past evidence in the literature of patients contracting ISCM from non-Hodgkin's lymphoma but again, extremely rare (7 cases over 14 years at the Mayo Clinic [3]). Upon review of the literature, we believe this to be the first reported case of an ISCM from a gastric lymphoma.

Case presentation

Our patient, a 71 year-old male, originally presented with gastric lymphoma at a private oncology clinic. He was found to have multiple intra-abdominal node involvement including periportal, celiac, suprarenal, iliac, and paraesophageal nodes that may have been supradiaphragmatic. He began treatment with systemic chemotherapy and completed six rounds of R-CHOP (Rituximab, Cyclophosphamide, Doxorubicin Hydrochloride, Vincristine Sulfate, Prednisone) therapy. The patient stated he was told he was in remission and was started on maintenance chemotherapy (Rituxan) six months later.

One month later, following normal maintenance chemotherapy, the patient began complaining of numbness and lower extremity weakness. He was hospitalized for several weeks, at which time physicians discovered a T7-T8 intramedullary spinal cord lesion. He was started on Decadron and discharged to a rehabilitation facility. Over the next two weeks, he developed complete bilateral lower extremity paralysis and was sent to our facility for emergency evaluation. In this time, he lost all strength and sensation in his legs, his ability to walk, and bladder and bowel function. His past medical history is significant for hypertension, diabetes, which has been troublesome to

control, and childhood polio with sequelae of partial right hand paralysis.

At the time of admission, the patient was taking Neurontin, Reglan, Decadron, Glimepiride, Metformin and Benazepril HCTZ. He was non-ambulatory with an indwelling Foley in place. He also presented with a rash centralized on the chest and back storied to have started when he began his new medication regimen. Concerned with the possibility of UTI and potential drug reaction, all but his diabetes medication were withheld upon entry to our facility.

Neurosurgery and Neurology were consulted and polio reactivation syndrome and infection were ruled out. Analysis of cerebrospinal fluid showed a WBC of two, RBC of zero, HSV DNA PCR negative, and cryptococcal antigen negative. Probable differential diagnosis by these groups prior to biopsy included; chemotherapy-associated myelopathy, HTLV-associated tropical spastic paraparesis, transverse myelitis, or any other demyelinating diseases. However, the lesion's association with demyelinating diseases was deemed very unlikely because he had prior treatment with high-dose steroids and IVIG with no improvement. Upon conference with Neuroradiology, we also agreed that the lesion was very unlikely to be related to the patient's lymphoma, but we could not rule this out.

Radiation Oncology and Radiology reviewed and performed an MRI of the brain and cervical, lumbar, and thoracic spine. The intramedullary heterogeneously enhancing lesion was identified, expanding the thoracic cord from the level of T3-T12. Enhancement was greatest at T7-T8 and T9-T10. There was no extramedullary lesion. No evidence was found for intracranial or cervical cord involvement and no bony metastatic disease could be identified. CT of chest, abdomen, and pelvis was also negative of recurrent lymphoma.

Surgery (T7-T8 laminectomy with operating microscope) was performed to obtain biopsy samples of the lesion. There were notable abnormal exophytic lesions on the spinal cord at the area of the exit of multiple dural roots, which were sampled for biopsy. A small midline myelotomy was also performed to obtain a sample of the intramedullary area of the cord for biopsy. It was noted to have an abnormal, soft and mealy appearance.

The biopsies were negative for fungi, acid-fast bacilli, and showed no evidence of viral cytopathic effects. The specimens did show extensive necrosis of neural tissue with a small number of scattered lymphoid cells. There was also occasional perivascular infiltration by large atypical lymphoid cells that were strongly immunoreactive for CD20 and CD79a, suggestive of B-cell lineage. However, the biopsy sections that best demonstrated the large B-cell infiltrate were distal to the known intramedullary lesion.

Given that our patient had a history of large B-cell lymphoma and there is no other observed etiology known that could explain the pathological findings, the most likely conclusion is that our patient's lesion was caused by the previously diagnosed large B-cell lymphoma. While there is no definitive explanation for how such extensive necrosis could be caused by such a relatively small number of observed B-cells, the combination of treatment effect and vascular damage caused by the lymphoma cells may have lead to the intramedullary lesion formation.

The overall findings most likely represent involvement by previously diagnosed large B-cell lymphoma. In lieu of these findings, our patient elected to enter hospice care and passed away approximately six weeks later.

Treatment could possibly have been more effective to reverse the lower extremity paralysis if reported earlier. Review of the literature has shown that intramedullary lesions, when caught early enough, can be removed and some patients have regained some motor control [4]. This paralysis could have been further confused by his complications from childhood polio that continued throughout his life. The symptoms our patient complained of mimicked those of post-polio syndrome including progressive muscle and joint weakness and general fatigue [5]. Although this was eventually ruled out as a diagnosis, these symptoms could have prevented him from seeking treatment sooner, possibly leading to the inability to extract the metastases.

Conclusion

In reference to standards of care, this case reveals something very interesting. Normally, when a mass is found in the brain, it is automatically assumed to be cancerous and treatment is often started before histological confirmation. In the spinal cord, this mass was assumed to be non-cancerous and treatment was delayed until a later histological diagnosis of cancer. It is our recommendation that a diagnosis of cancer should be considered for masses of the spinal cord upon discovery.

Declarations

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References

1. Dawson IM, Cornes, JS, Morson BC. Primary malignant lymphoid tumours of the intestinal tract. Report of 37 cases with a study of factors influencing prognosis. Br J Surg. 1961; 49: 80-89.

2. Rykken JB, Diehn FE, Hunt CH, et al. Intramedullary spinal cord metastases: MRI and relevant clinical features from a 13-year institutional case series. Am J Neuroradiol. 2013; 34: 2043-2049.

3. Flanagan EP, O'Neill BP, Habermann TM, Porter AB, Keegan BM, et al. Secondary intramedullary spinal cord non-Hodgkin's lymphoma. J Neurooncol. 2012; 107: 575-580.

4. Boström A, Kanther NC, Grote A, Boström J. Management and outcome in adult intramedullary spinal cord tumours: A 20-year single institution experience. BMC Res Notes. 2014; 7: 908.

5. McNalley TE, Yorkston KM, Jensen MP, et al. Review of secondary health conditions in postpolio syndrome: Prevalence and effects of aging. Am J Phys Med Rehabil. 2015; 94: 139-145.