Article published online: 2022-09-27

CASE REPORT

Intracranial metastasis of spinal intramedullary anaplastic astrocytoma

Rashim Kataria, Vishal Bhasme, Sanjeev Chopra, V. D. Sinha, Shashi Singhvi[1]
Departments of Neurosurgery and [1]Pathology, SMS Medical College, Jaipur, Rajasthan, India

ABSTRACT

Meningeal spread of spinal intramedullary astrocytoma into the cranium is rare. Only few case reports are available so far in the literature. We report a case of intramedullary high grade astrocytoma of the conus, developing intracranial metastasis after three months of partial excision of the spinal mass. The need for radical surgery, entire neuroaxis radiation, and adjuvant chemotherapy is suggested in the management of malignant spinal cord astrocytoma to prevent dissemination.

Key words: Astrocytoma, cranial metastases, intramedullary, spinal cord tumor

Introduction

Primary intracranial tumors such as ependymoma, medulloblastoma, germinoma, and less commonly glioblastoma and anaplastic astrocytoma are known to disseminate into the spinal cord. However, intracranial metastasis from a primary spinal tumor is rare. Spinal tumor can metastasize to the brain by direct leptomeningeal, cerebrospinal fluid (CSF) pathways and rarely by bloodstream.[1] Meningeal invasion paves the way for spread via CSF pathways. Tumor cells after coming in contact with the spinal subarachnoid space disseminate to the basal cisterns and the cistern magna causing communicating hydrocephalus. Abnormal direct flow of CSF from the basal cisterns to the ventricles occurs in the presence of communicating hydrocephalus leading to metastatic deposits in the basal cisterns, cistern magna, and the ventricles.

Case Report

A 15-year-old female presented with pain, progressive weakness, and numbness in the right lower limb for three months. There was brief history suggestive of bladder and bowel involvement. On examination, she was conscious, co-operative, oriented to time, place and person with normal higher motor functions, and cranial nerve examination. Motor examination revealed weakness of bilateral lower limbs (right more than left). Sensations were diminished below L1 dermatome. A magnetic resonance imaging (MRI) scan of the thoracolumbar spine [Figure 1a-c] revealed an intramedullary mass lesion extending from T11 to L1. The mass was partially suckable and was infiltrating into the cord; hence, complete removal could not be done. The histopathology [Figure 2a and b] was suggestive of Anaplastic astrocytoma. Post operative period was uneventful and patient was discharged after seven days with improvement in sensory, motor, and bladder bowel functions. The patient underwent local radiotherapy to the thoracolumbar spine and was doing fine till three months after the surgery, when she turned up in an unconscious state and poor general condition. MRI of brain [Figure 3] revealed enhancement in the cisterns around the brainstem. CSF examination was negative for malignant cells. The patient died after five days of admission.

Access this article online	
Quick Response Code:	Website: www.asianjns.org
	DOI: 10.4103/1793-5482.92179

Address for correspondence:
Dr. Rashim Kataria, Assistant Professor (Neurosurgery), 53, Mauji Colony, Malviya Nagar, Jaipur - 302 004, Rajasthan, India.
E-mail: rashim_kat@yahoo.com

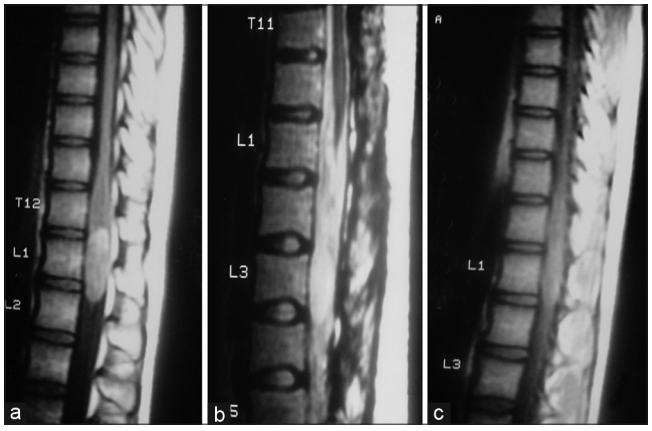

Figure 1: (a) MRI thoracolumbar spine T1W, (b) T2W and (c) contrast enhanced sequences showing intramedullary mass at conus taking up the contrast

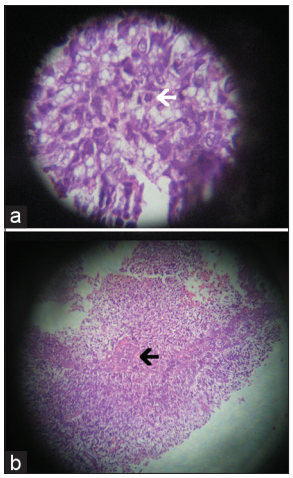

Figure 2: Photomicrograph of anaplastic asrocytoma showing increased cellularity, nuclear pleomorphisim and mitotic activity (white arrow) and endothelial proliferation (black arrow) (H and E, ×400)

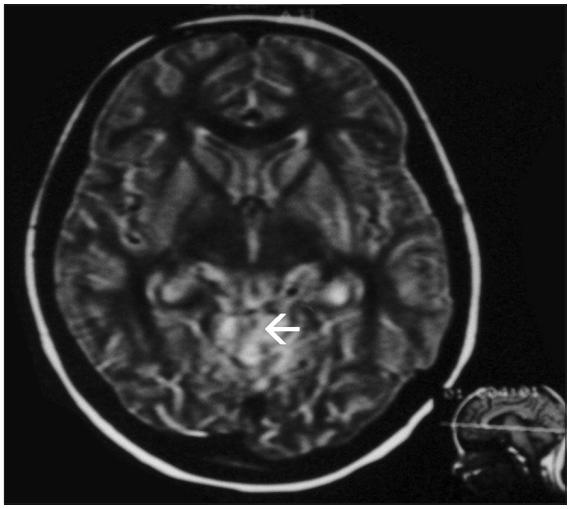

Figure 3: MRI Brain T1 Flair sequence showing altered signal intensity (arrow) in cisterns around the brainstem

Discussion

Spinal cord tumors account for 2-5% of central nervous systems neoplasms in adults.[2] Intramedullary spinal cord astrocytomas represent only 6-8% of spinal cord tumors.[3] Spinal cord gliomas accounts for only 1% of all primary neoplasms of the central nervous system.[4] Intracranial dissemination of spinal cord gliomas is rare; however, dissemination of tumor cells has been reported in virtually all types of primary central nervous system neoplasms. It is seen more commonly with primary brain tumors such as medulloblastomas, germ cell tumors, and high-grade gliomas and less commonly with low grade gliomas. Peraud *et al*. reported an incidence of dissemination of about 20 to 36% for supra tentorial tumors and up to 60% of infra tentorial lesions in an autopsy series.[5] When low-grade intramedullary astrocytomas metastasize intracranially, most of them have malignant transformation. Few case reports of dissemination of pilocytic astrocytoma have also been reported.[6,7] Intracranial dissemination is an ominous feature characterizing the terminal stage of the disease. Several factors are associated with tendency of the tumor to disseminate including young age, high histology grade, cellular anaplasia, oligodendroglial differentiation,[8] over expression of epidermal growth factor receptor,[9] increasing values of Ki-67/MIB-1 labeling index,[10] and immunosuppressed status.[11] It has also been hypothesized that malignant transformation, surgical manipulation or partial surgical excision, no post operative radiotherapy may contribute to dissemination of the tumor, but there is no level 1 evidence in the literature so far showing these variants can increase the chance of dissemination.

Surgical resection using an operating microscope often leads to an improvement in the resulting neurological deficits. But, the value of aggressive resection in high-grade spinal cord astrocytomas is unclear.[12] Infiltrating astrocytomas of the spinal cord pose a treatment challenge because they usually are not amenable to total resection due to the high morbidity of surgery. The tissue of low-grade astrocytoma is similar to normal tissue and difficult to differentiate grossly. Compared to the more circumscribed ependymoma, the infiltrative nature of spinal cord astrocytomas frequently limits the extent of resection. The majority of spinal cord astrocytomas tends to progress slowly, but are not well-demarcated and difficult to completely remove without neurological injury.[13] Even when gross total tumor removal is accomplished, there is no doubt that microscopically residual fragments remain.[3] Additional radiation or chemotherapy should be considered for tumors with clinically progressive lesions and in which a complete resection could not be achieved.[12] Its prognosis is dependent on the long-term behavior of the residual tumor.

Raised intracranial pressure is not always indicative of dissemination intracranially of spinal tumors and a cerebral CT scan should be performed in such cases. A patient of spinal tumor may also present with signs and symptoms of hydrocephalus or increased intracranial pressure without evidence of dissemination due to tumor mass interfering with CSF dynamics, arachnoid villi fluid absorption blocked by high CSF protein levels, and exudation of fluid from the tumor.[14]

The time course for the progression of symptoms in our patient makes it unlikely that the primary lesion were multifocal (spinal intramedullary and intracranial) in origin. Patient had dissemination even after local radiation. Hence, the need for entire neuroaxis radiation and adjuvant chemotherapy is stressed in high risk patients. CSF examination didn't reveal atypical cells in our case. Previous reports also suggest that CSF examination provides unreliable results. In subarachnoid metastatic disease in children, if a single CSF sample is employed, nearly 50% of the patients will not be diagnosed.[15]

Conclusion

Intracranial dissemination is an ominous feature characterizing the terminal stage of this disease. Intracranial dissemination is a major indicator of a poor prognosis. Chances of dissemination can be minimized by radical surgery of the primary high grade intramedullary astrocytoma, especially in the young. This should be followed by entire neuroaxis irradiation and adjuvant chemotherapy. Although there is no level 1 evidence that these factors do prevent dissemination, but these appear to be logically correct.

References

1. Rubinstein LJ, Logan WJ. Extraneural metastasis in ependymoma of the cauda equina. J Neurol Neurosurg Psychiatry 1970;33:763-70.
2. Abel TJ, Chowdhary A, Thapa M, Rutledge JC, Geyer JR, Ojemann J, *et al*. Spinal cord pilocytic astrocytoma with leptomeningeal dissemination to the brain. Case report and review of the literature. J Neurosurg 2006;105:508-14.
3. Bouffet E, Amat D, Devaux Y, Desuzinges C. Chemotherapy for spinal cord astrocytoma. Med Pediatr Oncol 1997;29:560-2.
4. Jeong SM, Chung YG, Lee JB, Shin IY. Intracranial dissemination from spinal cord anaplastic astrocytoma. J Korean Neurosurg Soc 2010;47:68-70.
5. Peraud A, Herms J, Schlegel J, Müller P, Kretzschmar H, Tonn JC. Recurrent spinal cord astrocytoma with intraventricular seeding. Childs Nerv Syst 2004;20:114-8.
6. Figueiredo EG, Matushita H, Machado AG, Plese JP, Rosemberg S, Marino R Jr. Leptomeningeal dissemination of pilocytic astrocytoma at diagnosis in childhood. Two cases report. Arq Neuropsiquiatr 2003;61:842-7.
7. Ng HK, Leung CH, Boet R, Poon WS. Spinal cord pilocytic astrocytoma with cranial meningeal metastases. Case report. J Clin Neurosci 2001;8:374-7.
8. Eade OE, Urich M. Metastasising gliomas in young subjects. J Pathol 1971;103:245-56.
9. Penar PL, Khoshyomn S, Bhushan A, Tritton TR. Inhibition of epidermal growth factor receptor-associated tyrosine kinase blocks glioblastoma invasion of the brain. Neurosurgery 1997;40:141-51.
10. Faria MH, Gonçalves BP, do Patrocínio RM, de Moraes-Filho MO, Rabenhorst SH. Expression of Ki-67, topoisomerase IIalpha and c-MYC in astrocytic tumors: Correlation with the histopathological grade and proliferative status. Neuropathology 2006;26:519-27.
11. Yamashita Y, Kumabe T, Jokura H, Tominaga T, Yoshimoto T. Intracranial dissemination from thoracic spinal cord anaplastic astrocytoma in a patient with idiopathic CD4-positive T lymphocytopenia: A case report. Surg Neurol 2006;56:39-41.
12. Henson JW. Spinal cord gliomas. Curr Opin Neurol 2001;14:679-82.
13. Aryan HE, Meltzer HS, Lu DC, Ozgur BM, Levy ML, Bruce DA. Management of pilocytic astrocytoma with diffuse leptomeningeal spread: Two cases and review of the literature. Childs Nerv Syst 2005;21:477-81.
14. Umezu H, Seki Y, Aiba T, Matsuya S. Intracranial seeding following surgery for spinal cord astrocytoma-case report. Neurol Med Chir (Tokyo) 1992;32:281-4.
15. Balhizen JC, Bots GT, Schaberg A, Bosman FT. value of cerebrospinal fluid cytology for the diagnosis of malignancies in the central nervous system. J Neurosurg 1978;48:747-53.

How to cite this article: Kataria R, Bhasme V, Chopra S, Sinha VD, Singhvi S. Intracranial metastasis of spinal intramedullary anaplastic astrocytoma. Asian J Neurosurg 2011;6:116-8.

Source of Support: Nil, **Conflict of Interest:** None declared.