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Case report

Anaplastic oligodendroglioma presenting with drop metastases in the cauda equina

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Abstract

Metastatic involvement of the cerebro-spinal fluid (CSF) pathway in oligodendrogliomas is not uncommon; however, symptomatic involvement of the spinal cord is very rare: less of 10 cases have been published. To our knowledge, an intracranial oligodendroglioma presenting with symptoms of drop metastases in the cauda equina has never been reported.

We report a case of 67-year-old woman who after 1 month of severe low back and legs pain developed symptoms of raised intracranial pressure. A spinal cord MRI showed multiple intradural nodular lesions at the level of the cauda equina, a MRI of the brain showed an intraventricular brain tumor. The histopathological diagnosis of both surgically treated lesions was anaplastic oligodendroglioma. The choices adopted in planning diagnostic and therapeutical procedures are discussed. The importance of the clinical and neuroradiological data in the diagnosis is stressed. Pathophysiology of the seeding of intracranial tumours via the cerebrospinal fluid is reviewed.

Keywords: Oligodendroglioma; Drop metastases; Low back pain; Spinal metastasis

1. Introduction

Metastatic involvement of the cerebro-spinal fluid (CSF) pathway in oligodendrogliomas is not uncommon occurring in up to 14% of cases [1]. Usually, metastases to the spinal meninges give rise to few clinical signs. To our knowledge, intradural drop metastases in the lumbar spine as first presentation of an intracranial oligodendroglioma has never been reported. We describe a case of multiple intradural drop metastases which had the macroscopical appearance of multiple nodules on the cauda equina roots in a woman with an initially asymptomatic intraventricular tumour (anaplastic oligodendroglioma).

2. Case report

A 67-year-old woman was admitted at the Department of Neurosurgery of the Second University of Naples, Italy, with a 1-month history of severe low back and legs pain. She also presented progressive weakness in left leg and paresthesias on both legs, anorexia and weight loss (about 10 kg in 2 months). Neurological examination revealed a mild paraparesis, worse on the left side, with partial loss of proprioception and pinprick sensation below the L2 dermatome and absence of deep tendon reflexes in both legs. There were not bowel and bladder dysfunction. A spinal MRI with gadolinium showed the presence of multiple enhancing nodules attached to the nerve roots with diffuse enhancement of the surrounding dura mater at L2–L3 and L5–S1 levels (Fig. 1). The night before the scheduled operation, she presented headache and vomiting. A CT scan of the head showed a solid tumor in and around

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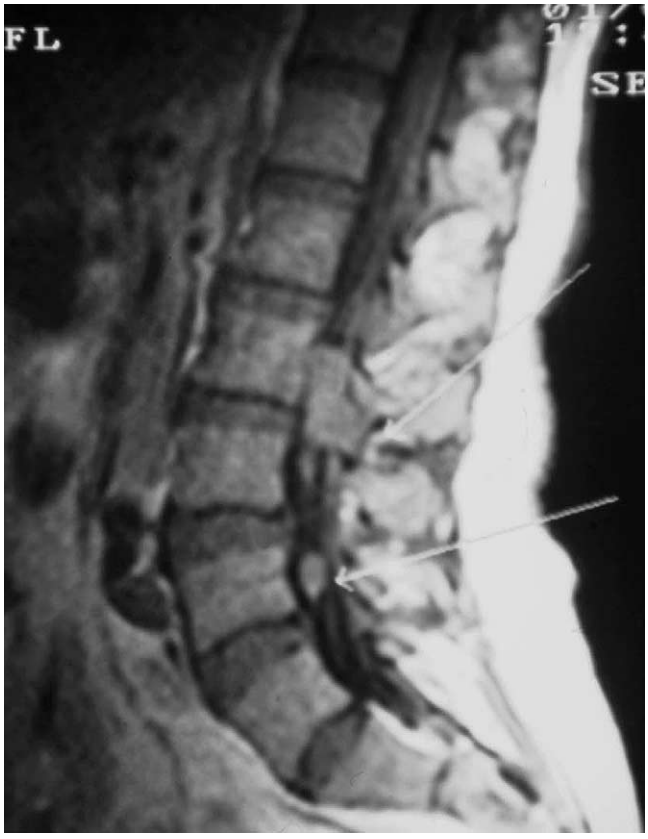

Fig. 1. Sagittal MRI of the lumbar spine revealing multiple contrast enhancing lesions in the cauda equina.

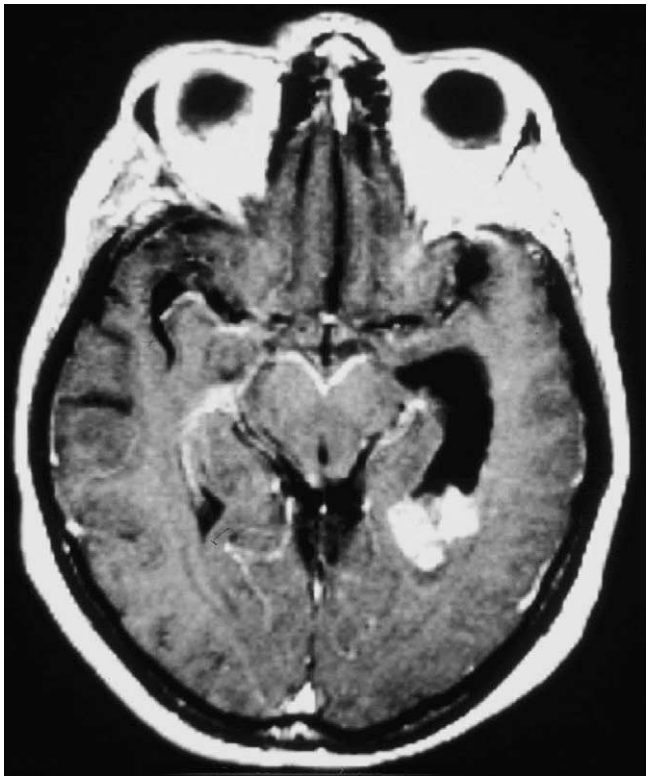

Fig. 2. Axial MRI of the head showing a tumor in the trigone of the left lateral ventricle, associated with enlargement of the temporal horn of the left ventricle.

the trigone of the left lateral ventricle and the enlargement of the temporal horn of the left ventricle. A MRI, performed in the next day, confirmed the presence of a left lateral ventricle mass, hypointense on TI weighted images, with intense contrast enhancement (Fig. 2), and inhomogeneously hyperintense on PD and T2 weighted scans. Chest and abdomen CT scans and total-body bone scintigraphy excluded extra-axial involvement. Medical therapy (steroids and diuretics) gave a good control of intracranial hypertension, while intractable back pain and progressive paraparesis required surgical treatment of the spinal lesions.

A L2–L5 laminectomy was performed to decompress the cauda equina roots, to remove the neoplastic nodules and obtain histological samples. At the surgery the thecal sac was tense and enlarged. Following opening of the dura, the cauda equina roots appeared bearing multiple tumoral nodules. A friable, dark reddish tumoral tissue enveloped in a "sleeve-like" manner the nerve roots. Microscopic dissection revealed extensive infiltration of the cauda equina roots. Subtotal resection was achieved. Morphological examination showed focal or diffuse histological features of malignancy, such as increased cellularity, marked cytological atypia, high mitotic activity and microvascular proliferation; necrosis was absent (Fig. 3).

Immunohistochemical examination showed positivity to S-100 protein and Ki-67 MIB1 labeling (40%), while it was low to GFAP (10%) and negative to synaptophysin. So, histopathological diagnosis of anaplastic oligodendroglioma (WHO grade III, St. Anne and Mayo grade 3) was done. Postoperatively the patient was relieved by legs and back pain with no further neurological deficits. Two weeks later the patient underwent surgical removal of the intracranial tumor, through a left parieto-occipital craniotomy, transcortical approach. Following the opening of the dura, the intraparietal sulcus was opened and in its depth a 3-cm long incision was done. The white matter was dissected until dark reddish tumoral tissue appeared. The tumor was internally debulked and cleaved from the ventricular walls. Gross total removal was achieved and the CSF pathway restored. At histological examination, it had the same features of the cauda equina tumour.

The early post-operative course was uneventful. Cranial and spinal fractioned irradiation (total 50 Gy on the brain and 45 Gy on L2–L5) was administered followed by chemotherapy with temozolomide (250 mg/day for 5 days/month). Our oncologists did not advice standard procarbazine–carmustine–vincristine protocol because of low Karnofsky performance status. The patient was referred to a rehabilitation center for further management, but she continued to deteriorate in her general status. Following the third cycle, chemotherapy was stopped because of hematological complications. She died 8 months after diagnosis because of broncopneumonia.