Solitary Intramedullary Spinal Cord Metastasis from Colon Carcinoma: A Case Report and Literature Review

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ABSTRACT

It is extremely rare for cancer to present as an intramedullary spinal cord metastasis. The authors report on a case of a 74-year-old woman presenting with progressive tetraparesis to metastatic colon adenocarcinoma in the spinal cord. A review of the literature reveals that intramedullary localization is associated with a very short life expectancy, with a median survival of 3 - 4 months from the time of the diagnosis; nevertheless microsurgical removal of tumor can improve quality of life.

KEYWORDS

Spinal Cord Metastasis

1. Introduction

Intramedullary spinal cord metastasis (ISCMs) is an unusual complication of the cancer, representing 8.5% of CNS metastasis [1]. Spinal colorectal carcinoma related metastasis is extremely rare with only few cases reported in the literature so far. We report on a patient with an intramedullary metastasis from colon carcinoma. Total resection of the tumor has improved the quality of life of the patient. The aim of our paper is to emphasize the role of surgery, when there is an indication for improving the quality of life in patients with a very short life expectancy.

2. Case Report

A 74-year-old woman had undergone colectomy, without adjuvant chemotherapy for adenocarcinoma 1 year previously. Two months before admission she developed progressive weakness of her left leg and urinary hesitancy, she also noted oppressive burning and dysesthetic pain in the neck radiating in to both arms, and back pain that persist in recumbency.

Over 3 - 4 days the patient reported progressive right lower limb weakness and a severe deterioration in the left leg, that makes impossible standing, also referred progressive weakness in the upper limbs.

Neurological findings included spastic tetraparesis with severe paresis on the left leg (2/5), the right leg had a marked weakness (3/5), in the arms she had slight bilateral weakness (4/5), hyperactive tendon reflex more prominent on the left than on the right, bilateral Babinski sign, and hypoesthesia to vibration on the left hemibody and for pinprick and temperature on the right hemibody below T1. Urinary retention and reduced anal sphincter tone were also noted.

MRI images of the spine in toto showed a solitary intramedullary lesion at C7 level, the lesion measured about 1.5 cm in cranio-caudal dimension, occupying almost the entire spinal cord (Figure 1(a)).

The lesion appeared slightly hypointense on T1-w image, and hyperintense on T2-w image associated with a large proximal and distal edema extending from C3 to T3 vertebral body (Figure 1(b)), contrast-enhanced T1-w image demonstrated homogeneous enhancement and the cord appeared expanded at the same level (Figure 1(c)).

A preoperative chest radiograph showed no abnormalities, and MRI of the brain was negative for other lesions. Cerebrospinal fluid (CSF) analysis showed an elevated
protein level, but cytology was negative. Based on the progressive neurological worsening, surgical treatment was performed.

The patient underwent C6-T1 laminectomy and the dural sac exposed was opened at the midline. Once exposed, the spinal cord appeared expanded.

A median posterior myelotomy was performed and disclosed the lesion as light yellow-reddish in color with a pseudocapsule distinguishing it from surrounding spinal cord parenchyma. It was sharply dissected out and totally removed.

The histological examination showed the pathological features of adenocarcinoma metastasizing from colon cancer. No radiotherapy was planned because adenocarcinoma is unlikely to be radiosensitive.

Postoperatively, the patient progressively showed improvement in neurological function. For both arms, the muscle strength rose to 5/5; the muscle strength of the right lower limb improved up to 4/5. However, the severe paresis on the left leg did not improve and her sphinctical disturbance and urinary hesitancy were only slightly improved. After 14 months, the patients died of local recurrence of the colon carcinoma, with invasion of sigmoid colon, uterus and bladder.

3. Discussion

ISCM is a rare clinical entity, comprising 1% - 3% of all intramedullary spinal cord tumors and accounting for only 0.1% - 0.4% of all cancer patients [2,3].

Furthermore, they account for 3% - 5% of cases of myelopathy in patients affected by cancer [4]. Most of ISCM patients have brain metastasis and systemic metastasis at the time of the diagnosis [5]. ISCM occurs in as few as 2% of cancer autopsy; on the other hand, brain metastasis is found in 20% of cases [2,6]. The lower incidence of ISCMs can be attributed only partially to the fact that the spinal cord is frequently omitted from routine autopsies [7].

Several theories have been postulated to explain the discrepancy between the frequencies of brain and intramedullary metastasis. Arterial seeding is believed to be the most common mechanism of intramedullary metastasis. Indeed, the brain receives about one-third of the cardiac output through large vessels under high pressure, while the spinal cord receives its arterial supply from small vessels under low pressure [8,9]. Moreover, the medullary arteries branch off the aorta at the right angles, while the cerebral arteries are almost a direct extension of the aorta, thus favoring embolic seeding [10]. Metastasis spreading may also occur through direct extension along the nerve roots, perineural sheaths and via CSF pathways [11,20].

The most common origin of the ISCMs is lung cancer, especially small cell carcinoma followed, by breast cancer, melanoma, lymphoma, and renal cell carcinoma [5,7,12]. ISCM from colorectal adenocarcinoma is extremely rare, with only few cases reported in the literature so far [4,8,11,13-20].

Table 1 summarizes the principal findings of these cases. ISCMs most commonly occur in the setting of advanced disease, but may exceptionally account for the initial presentation [4,5]. Pain and weakness were more common early in the course of disease, with sensory loss reported somewhat later. Bowel and bladder dysfunction most typically presented late [5]. Pseudo-Brown-Séquard or Brown-Séquard syndrome occurs in 30% - 45% of patients with ISCM, with progression to a cord transection syndrome [3,7,12,16].

An exact diagnosis of an ISCM can be difficult even when the primary tumor is known, because clinical findings do not help to distinguish ISCM from other spinal cord lesions or non compressive myelopathies that can occur in cancer patients [4].

The differential diagnosis should include primary intramedullary tumor, spinal epidural metastasis (SEM), radiation myelopathy, paraneoplastic necrotizing myelopa-
Table 1. Review of cases reports of patients with ISCM from colon carcinoma.

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Sex &amp; Age</th>
<th>Level</th>
<th>Symptoms</th>
<th>Time interval</th>
<th>Other Metastasis</th>
<th>Surgery</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sansoy et al.</td>
<td>1955</td>
<td>F, 65</td>
<td>T4</td>
<td>Right lower limb monoparesis</td>
<td>NS</td>
<td>No</td>
<td>No</td>
<td>NS</td>
</tr>
<tr>
<td>Silva &amp; McSwain</td>
<td>1967</td>
<td>F, 55</td>
<td>C5-C6</td>
<td>Left neck-arm pain followed by weakness of lower limbs</td>
<td>15 months</td>
<td>Lung</td>
<td>Yes</td>
<td>Died 15 days postoperatively</td>
</tr>
<tr>
<td>Walker et al.</td>
<td>1967</td>
<td>M, 56</td>
<td>L1-L2</td>
<td>NS</td>
<td>NS</td>
<td>Lung</td>
<td>No</td>
<td>NS</td>
</tr>
<tr>
<td>Jellinger et al.</td>
<td>1979</td>
<td>M, 77</td>
<td>T11-L1</td>
<td>Bilateral hip and leg pain with overnight paraplegia</td>
<td>5 years</td>
<td>Brain, Lung</td>
<td>No</td>
<td>Died 3 weeks after admission</td>
</tr>
<tr>
<td>F, 74</td>
<td></td>
<td>C2-C4</td>
<td></td>
<td>Neck pain followed by lower limb weakness, urinary hesitancy</td>
<td>6 years</td>
<td>Brain</td>
<td>No</td>
<td>Died 9 days after admission</td>
</tr>
<tr>
<td>Walker et al.</td>
<td>1987</td>
<td>M, 66</td>
<td>C4</td>
<td>Right lower limb monoparesis and right upper hyperesthesia</td>
<td>4 years</td>
<td>NS</td>
<td>Yes</td>
<td>Died 2 months postoperatively</td>
</tr>
<tr>
<td>Schiff &amp; O’Neil</td>
<td>1996</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Ogino et al.</td>
<td>2001</td>
<td>M, 69</td>
<td>C5-C6</td>
<td>Right hemiparesis, hypalgiesia below left L1 level, constipation, urinary hesitancy</td>
<td>4 months</td>
<td>Lung</td>
<td>Yes</td>
<td>Died 3 months postoperatively</td>
</tr>
<tr>
<td>Yano et al.</td>
<td>2002</td>
<td>NS</td>
<td>Thoracic</td>
<td>NS</td>
<td>NS</td>
<td>No</td>
<td>Yes</td>
<td>NS</td>
</tr>
<tr>
<td>Kaya et al.</td>
<td>2003</td>
<td>F, 44</td>
<td>T6</td>
<td>Lower limbs weakness, over 1 day paraplegia</td>
<td>2 years</td>
<td>Brain, Mediastinum</td>
<td>Yes</td>
<td>Died 14 months postoperatively</td>
</tr>
<tr>
<td>Grasso et al.</td>
<td>2007</td>
<td>F, 61</td>
<td>C3-C4</td>
<td>Right hemibody numbness lower limbs and upper limbs weakness</td>
<td>NS</td>
<td>No</td>
<td>Yes</td>
<td>Died 2 months postoperatively</td>
</tr>
<tr>
<td>Kaballo et al.</td>
<td>2011</td>
<td>M, 71</td>
<td>C2-C3</td>
<td>Right upper limb and lower limb feel pins and needles; over 5 days developed Brown-Séquard syndrome</td>
<td>16 months</td>
<td>Liver, Lung</td>
<td>No</td>
<td>NS</td>
</tr>
<tr>
<td>Present case</td>
<td>2012</td>
<td>F, 74</td>
<td>C7</td>
<td>Left lower limb weakness, urinary hesitancy, pain during recumbency; over 3 - 4 days severe tetraparesis</td>
<td>1 years</td>
<td>No</td>
<td>Yes</td>
<td>Died 14 months postoperatively</td>
</tr>
</tbody>
</table>

NS: not specified; Time interval: interval between colon surgery and onset of spinal symptoms; Symptoms: symptoms at onset.

thy, vascular malformations, syringomyelia, demyelinating plaques and meningeal carcinomatosis [11,18].

The most important factors to consider in diagnosis are time of clinical course, pain, and CSF cytology [4]. The rapid progression of symptoms less than 1 month distinguishes ISCM from primary intramedullary tumors such as gliomas, which typically present with a slower progression of symptoms [5,7,11]. Muscle atrophy and fasciculation commonly accompany the long-standing muscle weakness reported to accompany primary intramedullary tumors, whereas muscle atrophy has rarely been reported metastatic intramedullary disease [16].

ISCMs are much less common than metastatic epidural disease, which involves the dura in 10% of all cancer patients [5]. Both SEM and ISCM can occur with pain, and can be complicated by rapid deterioration in neurologic status. In ISCM pain tends to be a little less ubiquitous and severe characterized by a strong asymmetry of myelopathy. This is seen in almost half of patients with ISCM and only 3% of patients with SEM [21].

The clinical pictures of radiation myelopathy and necrotizing myelopathy are usually that of a painless hemicord syndrome [22]. Neoplastic meningitis is usually suspected on the basis of multifocal neurologic deficits, including cranial nerve palsies and spinal root deficit. Signs and symptoms of increased intracranial pressure are frequently observed secondary to obstruction of CSF pathway. CSF analysis reveals pleocytosis and malignant cells in the majority of patients with leptomeningeal disease [7].

CSF findings in ISCM are usually negative or show only an increase in protein and a mild pleocytosis. If positive, meningeal involvement may have occurred [4].

However when a spinal lesion is suspected in a patient with a history of malignancy, MRI with gadolinium enhancement is mandatory. PET has a sensitivity of 96% in detecting spinal metastasis, and this is even more sensitive when combined with CT scan [19].

The prognosis of a patient who has an intramedullary spinal cord lesion is grave. Intramedullary localization is associated with a very short life expectancy with a median survival of 3 - 4 months from the time of the diagnosis [7,11]. Death is due to complications of neurologic damage and progressive systemic cancer.
The management of ISCM remains controversial since the recurrent recommendations are based on anecdotal experiences described on retrospective reports [4]. The standard therapy is radiation treatment, with or without steroids [7,11,23].

Modern radiotherapy techniques, such as intensity-modulated radiation therapy, cyberknife, and tomotherapy can hold promise in efficacy; however, so far, no considerable data support their use [4]. It must be noted, however, that only radiosensitive metastasis, such as small cell carcinoma, breast cancer, or lymphoma respond to radiation therapy.

Several factors are important when considering surgical treatment such as patient’s age, physical condition, location, and severity of the primary neoplasm, as well as other metastasis, and surgical risk [4]. To date, in selected cases, the prognosis may be improved by early diagnosis and surgical removal using microsurgical techniques.

Like intracranial metastasis, ISCMs are often discrete, well-circumscribed deposits and subject to gross total resection. One should consider microsurgical resection of ISCMs in patients with well-controlled radiosensitive primary tumors a discrete, solitary, intramedullary metastasis, without leptomeningeal involvement [16]. In these cases, microsurgical removal of ISCM can improve the quality of life.

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