Case Report

Different Outcome of Myeloid Sarcoma with Spinal Cord Compression Preceding Acute Myeloid Leukemia: Report of Two Cases and Review of Literatures

Qiang Yin[1], Yang-yang Zhou[2*], Duo Chen[2], Wen-liang Li[1**]

[1]*Key Laboratory of Cancer Prevention and Therapy, Department of Neurosurgery and Neuro-oncology, Tianjin Medical University Cancer Hospital, Tianjin 300060, China;* [2]*Department of Neurosurgery, Shengjing Hospital Affiliated to China Medical University, Shenyang, China*

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ABSTRACT

Myeloid sarcomas (MS) preceding acute myeloid leukemia (AML) are rare, which presenting as acute spinal cord compression is even rare. Here we report two new cases of myeloid sarcoma patients, whose outcomes were different. Twenty-seven patients with spinal MS preceding AML have been reported to date, including the two cases presented in this article. Surgical decompression was performed in 25 of the 27 patients, and 23 of these received additional anti-AML therapy. Considering our patients and the published cases in the literature we suggest that immunohistochemical study plays an essential role in arriving at a correct diagnosis of MS, and that emergency surgery to resect spinal MS is an available treatment to make neural function recovery, and that the disease must be treated with intensive chemotherapy similar to that used to treat AML as soon as possible after resection or irradiation of the tumor.

Key words: Spine; Myeloid sarcoma; Immunhistochemistry; Acute myeloid leukemia;

INTRODUCTION

MS can arise anywhere, but usually occur in the bone, skin, and lymph nodes[1]. Occurrence in the central nervous system and spinal cord is uncommon[2]. Once the incident occurs, the neurological dysfunction becomes worse and worse. So, emergency surgical decompression is an available treatment to make neural function recovery. To reduce the risk of subsequent AML in patients with nonleukemic MS, it is important to emphasize the need to treat patients who had nonleukemic MS with AML-type therapy, except of resection or irradiation[3,4]. Herein, we report two cases of patients with MS with spinal cord compression preceding AML. They accepted different therapies and their outcomes were different.

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*Contributed equally to this study.
**Corresponding author.
E-mail: lwlyq1982@126.com

CASE REPORT

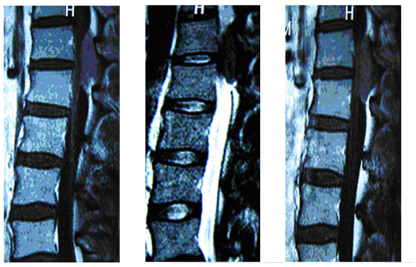
Case One

A 28-year-old previously healthy man presented with progressive low back pain and numbness of his legs for 10 days and bladder incontinence for 1 day. At physical examination, he was awake, alert, and fully oriented. His cranial nerves were intact. Sensory assessment revealed hypoesthesia below L12 and motor examination revealed 2/5 paraparesis. Deep tendon reflexes were hyperactive and Babinski signs were bilaterally positive. Laboratory evaluation revealed a white blood cell count of 3,900/mm^3, a hemoglobin level of 11.0 g/dl and a hematocrit of 38.2%. Magnetic resonance imaging of the spine revealed a posterior epidural mass between T12 and L1 (Figure 1). High-dose methylprednisolone was given initially, and he underwent emergent spinal cord decompression with T12-L1 laminectomy and tumor resection. A soft and grayish tumor compressing the

spinal cord was identified. Histologically the tumor was composed of a relatively uniform population of immature cells (Figure 2A). Immunohistochemical staining revealed the expressions of CD68, CD45, CD43, CD117 and lysozyme but not of MPO, CD20(Figure 2 B-H). The result suggested a diagnosis of MS in vertebral canal. There was nothing wrong with both bone marrow biopsy and his blood. After 10 days he gradually gained strength in his lower extremities and his bladder function was recovered. But the patient refused standard chemotherapy and radiation therapy to the spinal axis and tumor bed. After 4 months he presented with progressive low back pain and a high fever. At that time we found immature monoblasts from his blood (Figure 3A). And bone marrow biopsy results were consistent with acute myelocytic leukemia. The results of bone marrow slides showed that bone marrow hyperplasia was supreme, and most of the myeloid cells were immature monoblasts (Figure 3B). The patient was treated with anti-AML therapy, but died of sepsis finally.

Case Two

A 20-year-old girl presented with progressive back pain and numbness of her legs for 3 days. Sensory assessment revealed hypoesthesia below T7 and motor examination revealed 3/5 paraparesis. She was unable to stand or walk independently. Deep tendon reflexes were hyperactive and Babinski signs were bilaterally positive. Laboratory evaluation revealed a white blood cell count of 4,900/mm^3, a hemogloin level of 12.2 g/dl and a hematocrit of 37.2%. Magnetic resonance imaging of the thoracic spine revealed a posterior epidural mass between T7 and T9 (Figure 4). She underwent emergent spinal cord decompression with T7-T9 and tumor resection. A soft tumor compressing the spinal cord was identified. Histologically the tumor was composed of a relatively uniform population of immature cells, most of them were monocaryon (Figure 5A). Immunohistochemical staining revealed the expressions of CD68, CD45, and lysozyme, but not of MPO, CD20 (Figure 5B-F). These findings were consistent with a diagnosis of MS. There was nothing wrong with both bone marrow biopsy and her blood. After 9 days she gradually gained strength in his lower extremities. The patient accepted standard chemotherapy and radiation therapy to the spinal axis and tumor bed. During the 20 months follow-up she was with no evidence of leukemia disease, and there was nothing wrong with results of her three bone marrow biopsy.



A B C

Figure 1. MRI studies at the level of the T12-L1 disc space demonstrating the posterior epidural mass with near total obliteration of the spinal canal. (A): T1-weighted MRI scan; (B): T2-weighted MRI scan; (C): T1-weightd gadolinium-enhanced MRI scan.

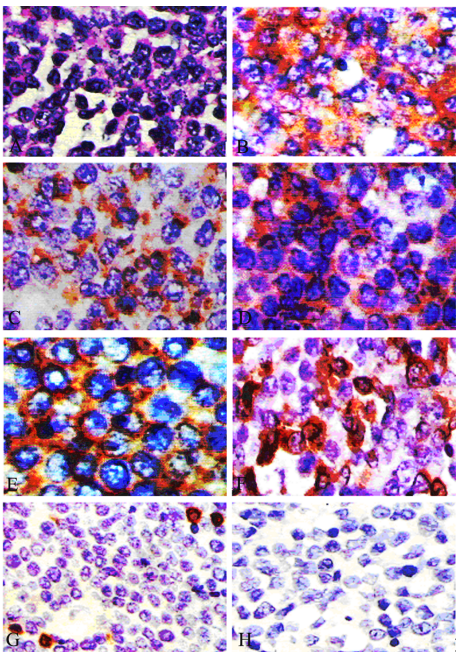
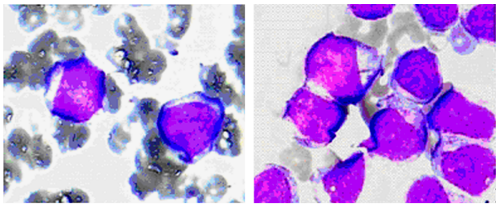


Figure 2. The histological and immunohistochemical study (case one). (A): Micrograph showing a meyloid sarcoma composed of atypical cells (Hematoxylin and eosin stain ×400); (B-H): Photomicrographs demonstrating positive immunohistochemical staining for CD68(B), CD45(C), CD43(D), CD117(E), Lysozyme(F) and negative for MPO(G), CD20(H).



A B

Figure 3. photomicrograph showing immature mono- blasts in peripheral blood. (A) and in bone marrow (B).

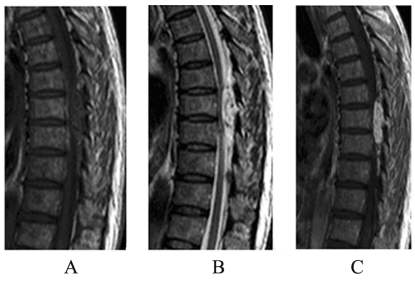


Figure 4. MRI studies at the level of the T7-T9 disc space demonstrating the posterior epidural mass with near total obliteration of the spinal canal. (A): T1-weighted MRI scan; (B): T2-weighted MRI scan; (C) T1-weighted gadolinium-enhanced MRI scan.

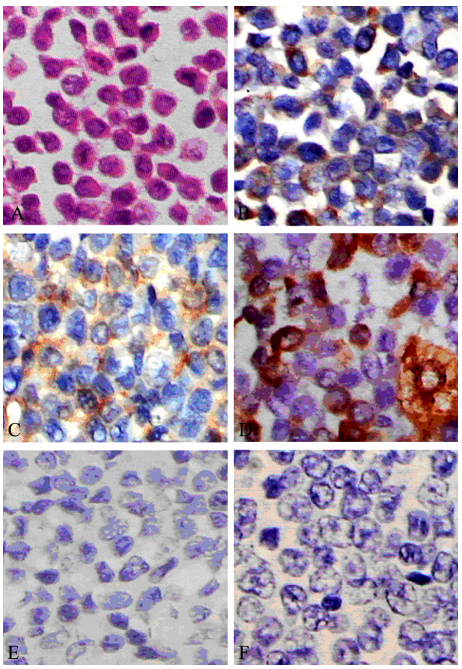


Figure 5. The histological and immunohistochemical study (case two). (A): Micrograph showing a myeloid sarcoma composed of atypical cells. (Hematoxylin and eosin stain ×400) (B-I): Photomicrographs demons- trating positive immunohistochemical staining for CD68(B), CD45(C), Lysozyme(D) and negatve for MPO(E), CD20(F).

DISCUSSION

Twenty-seven patients with spinal myeloid sarcoma preceding AML, 23 men and 4 women aged 2 to 74 years (median, 32 years), have been reported to date, including the two cases presented in this article[2,5-27] (Table1). The affected sites included 2 at cervical, 19 at thoracic, 10 at lumbar, and 4 at sacral spinal levels. The chief clinical symptoms were related to the affected sites. MS is a rare, solid, malignant extramedullary tumor of myeloid cells. MS is seen more often in men than in women as is generally the case in leukemia[18]. The male predominance could be associated with the higher incidence of acute myeloid leukemia in men[28]. The tumor may appear at any age or localisation, and usually occurs in the subperiost of the vertebra, sternum, orbits and skull. The involvement of the central nervous system and spinal cord is rare, myelopathy due to tumor compression is even rarer[29]. Most spinal myeloid sarcomas developed at the thoracic level, followed respectively by the lumbar, sacral and cervical level[30].

Immunohistochemical study played an essential role in arriving at a correct diagnosis of myeloid sarcoma. Nineteen of the spinal myeloid sarcomas were correctly diagnosed by immunohistochemical staining, including the two cases presented in this article[5,7-10,12-14,17-20,25,26]. The correct and prompt diagnosis of MS was important for adequate therapy, which was often delayed because of a high misdiagnosis rate[31]. The histological diagnosis of MS is difficult in the absence of evidence of AML. The differential diagnosis includes Ewing sarcoma, Langerhans histiocytosis, B-cell or T-cell lymphoma and metastatic tumors[1,32]. With extensive morphological and immunohistochemical analyses, the myeloid sarcoma were classified into five types: a) immature granulocytic sarcoma; b) differentiated granulocytic sarcoma; c) monoblastic sarcoma; d) monocytic myeloid sarcoma, and e) myelomonocytic sarcoma[15]. An immunohistochemical staining including lysozyme, CD43, CD45, MPO, CD117, CD68, CD3 and CD20 can successfully identify the majority of MS in formalin-fixed, paraffin-embedded tissue specimens[33-37]. The most common type of MS is the granulocytic sarcoma composed of myeloblasts and neutrophils. The less common type, monoblastic sarcomas have similar surface antigens to that of monoblasts in acute monoblastic leukemias. CD43 and lysozyme are the most sensitive markers staining a large proportion of neoplastic cells in all tumors examined. MPO and CD117 are the most sensitive of the markers for myeloid differentiation while monocytic precursors consistently strongly expressed CD68 and CD163[38].

Surgery is generally preferred for cases of acute spinal cord compression. Surgical decompression was performed in 25 of the 27 patients, including the two cases presented[5-21,24-27]. MS is a malignant tumor, and its growth speed is fast. When the tumor occurred in the small intraspinal space, it could result in spinal cord compression, and spinal cord function was damaged in a very short time. So emergency decompression is an available treatment to make neural function recovery, which is associated with high survival quality.

Table 1. *Summary of clinical findings in patients with non-leukemic spinal myeloid sarcoma*

Author (published years)	Age/sex	Location	Diagnosis	Treatment	Bone marrow involvement (months from diagnosis)	Outcome (months from diagnosis; cause of death)
Mason et al. (1973) (16)	20/M	T8	CES	SD, Rad, Che	AML (2)	Dead (20 leukemic relapse)
McCarty et al. (1980) (17)	33/M	T9-T12	MPO	SD, Rad, Che	Not described	Alive not described
Chan et al. (1986) (6)	13/M	T12-L1	CES	SD, Rad, Che	No (72)	Alive (72)
Meis et al. (1986) (15)	29/F	T9-T12	CES	SD, Rad, Che, BT	AML (7)	Dead (12, GVHD)
Zuiable et al. (1989) (27)	31/M	T12-L4	CES	SD, Rad, Che, BT	AML M3 (1)	Alive (18)
Ripp et al. (1989) (21)	58/M	T5-T8	CES	SD, Rad	Refractory anemia (1,5)	Dead (3 sepsis)
Kim et al. (1990) (11)	70/M	L2-L3	CES	SD, Rad, Che	No (3)	Alive (3)
Doshi et al. (1991) (7)	74/M	T9-T10	lysozyme and CES	SD, Rad	No (6 days)	Dead (6 days, DIC)
Kook et al. (1992) (12)	10/M	T5	Lysozyme	SD, Rad, Che	AML (14)	DEAD (17, ich)
Lagrange et al. (1992) (14)	22/M	T4	CD15, CD45	SD, Che BT	AML M2 (11)	Dead (20, leukemic relapse)
Sajjad et al. (1997) (23)	22/M	T4-S1	Not described	Not described	AML M4 (1)	Not descrived
Deme et al. (1997) (8)	47/M	C6-C7	CD45,CD43, MPO, lysozyme	SD, Rad, Che	No	Dead (12 MTX encephalophathy)
	49/M	L3-L4	CD45,CD43	SD, Che	No	Alive (3)
	15/M	L2	CD45, CD43, MPO, lysozyme	SD, Che, PB, CTE	AML, M5 (1.5)	Dead (12, leukemic relapse)
Sandhu et al. (1998) (24)	22/M	L3-S1	Not described	SD, Che	Not described	Dead (4 leukemic relapse)
Mostafavi et al. (2000) (18)	29/M	T2-T4	CD45, CD34	SD, Rad, Che	No (2)	Alive (114)
Machii et al. (2000) (19)	73/M	T4-T6	MPO	SD, Rad, Che	No (3)	Dead (4 sepsis)
Niedermayer et al. (2000) (20)	58/M	T3-T7	MPO	SD, Che	Not described	Not described
Ugras et al. (2001) (26)	13/M	T11-L1	CD45, CD43, CD68, MPO	SD	No (6)	Dead (not described)
Buckland et al. (2001) (5)	35/M	C4-C5	CD45, CES	SD, Rad, Che	No (3)	Alive (3)
Hamadani et al (2005) (9)	33/M	T6-T8	CD68	SD, Rad, Che, BT	No (7)	Alive (7)
Shiozawa et al. (2005) (2)	2/M	Below L3	MPO, CD56, CD68, CD13, CD33, CD45, CD64	Rad, Che	No (12)	Alive (12)
Kalayci et al. (2005) (13)	24/M	T3-T5	MPO, Lysozyme	SD, Rad, Che, BT	No (8)	Alive (8)

(*to be continued*)

(*connected*)

Author (published years)	Age/sex	Location	Diagnosis	Treatment	Bone marrow involvement (months from diagnosis)	Outcome (months from diagnosis; cause of death)
Howell et al. (2005) (10)	11/M	T1-T5	MPO, lysozyme, CD68	SD, Rad	Not described	Alive (Not described)
Tomoo et al. (2008) (25)	26/F	L5-S2	MPO, lysozyme, CD15, CD45	SD, Rad, Che, BT	No (12)	Alive (12)
Present cases	20/F	T7-T9	CD68, CD45, CD43, CD117, lysozyme	SD, Rad, Che	No (16)	Alive (20)
	28/M	T12-L1	CD68, CD45, CD43, CD117, lysozyme	SD	AML, M5(4)	Dead (4 sepsis)

CES: chloroacetate esterase staining; AML: acute myeloid leukemia; BT: bonemarrow transplantation; Che: chemotherapy; DIC: disseminated intravascular coagulation disorder; GVHD: graft versus host disease; ICH: intracranial hemorrhage; MTX: methotrexate; PBSCT: peripheral blood stem cell transplantation; Rad: radiotherapy, SD: surgical decompressing; MPO: (myeloperoxidese) is a poroxidase enzyme most abundantly present in neutrophil granulocytes.

However, local treatments for MS, such as radiation therapy or surgical resection, are less effective than chemotherapy at improving the disease-free interval or disease-free survival. MS occurs most commonly in patients with previous or current AML or in patients with chronic myeloproliferative disorders as a sign of blast transformation[39,40]. MS may be also encountered in patients with no history of hematologic disorder[15]. For patients without leukemia, MS is frequently a herald for AML. In one study, 71% of the patients diagnosed with MS who did not receive antileukemic chemotherapy subsequently developed AML within a median time of 7 months[41]. A review of non-leukaemic MS concluded that chemotherapy used for acute non-lymphoblastic leukaemia was more effective than local treatment modalities specifically directed at myeloid sarcoma[3]. A current study demonstrated that anti-AML therapy is highly effective in patients with nonleukemic MS and is associated with higher rates of event-free survival and overall survival in MS than in AML after matching MS patients with comparable, typical AML patients[4]. Bonemarrow transplantation has also shown promise. Some cases of primary myeloid sarcoma treated with autologous bonemarrow transplantation secondary to radical surgery, chemotherapy, and local prophylactic irradiation, has also been reported to have a long period of disease-free survival[25,42].

In this article, 21 of the 27 patients received anti-AML therapy, consisting of chemotherapy, or bone marrow transplantation, including the one case presented , and the survival time was 3 to72 months (median, 14 months)[5,6,8-20,22,24,25,27]. Between the two new presented cases there were some similar pionts, but their outcomes were very different. They presented with back pain as the first symptom, and developed into spinal cord dysfunction at a short period of time. Their same immunohistochemical staining revealed the expressions of CD68, CD45, and lysozyme. We did not find any leukemia cells in their bone marrow at first. But one refused chemotherapy, and developed into AML after 4 months, and died of sepsis finally. The other case accepted the standard anti-AML therapy, and during 20 months follow-up she was still alive. The totally different outcome may be associated with whether or not accepting the anti-AML therapy.

Considering our patients and the published cases in the literature we suggest that immunohistochemical study plays an essential role in arriving at a correct diagnosis of myeloid sarcoma, and the combination therapy of emergency surgery and anti-AML therapy is an available treatment to spinal MS preceding AML.

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