

A rare case of conus medullaris and cauda equina infiltration in acute lymphoblastic leukemia: MRI imaging features

Sun Yiqing¹ and Sun Shengjun^{2*}

¹Department of Radiology Liangxiang Hospital of Beijing Fangshan District, China

²Department of Neuroradiology, Beijing Tiantan Hospital Affiliated to Capital Medical University, 100050, China

Case report

The 40-year-old female patient had persistent soreness and pain in the lower limbs three months ago for unknown reasons. She was combined with weakness and mobility impairment in the left lower limb. Weakness in the right lower limb occurred two months ago, with progressive mobility impairment in both lower limbs. She cannot walk independently one month ago. The patient complained of soreness and pain in the left hip 10 days ago, with numbness in the distal ends of the lower limbs and hypoesthesia in the perineum. She received physical examination at our hospital, which revealed no specific abnormalities. Examination of the nervous system revealed diminished discriminative touch and superficial sensation in the waist and lower limbs. The muscle strength was of grade II+ in the proximal and distal ends of the lower limbs. Tendon reflex was absent, and pathological reflex was not elicited.

MRI manifestations

Plain MRI and contrast-enhanced MRI of the lumbar spine before surgery: strip-like isointense on T1WI and isointense on T2WI at the L1-S1 level (Figure 1). The signals were uniform, with unclear boundaries extending along the dural cavity. The spinal cord was apparently compressed, and the cauda equina was thickened and swollen with obscure margin. Upon the contrast-enhanced scan, the intramedullary lesions were uniformly enhanced, and the dura mater adjacent was linearly enhanced (Figure 1). Having all the clues, lymphoma was suspected. Plain and contrast-enhanced MRI scan of the lumbar spine was performed one week after surgery: The spinal cord was obviously thinner than before, with abnormally enhanced signals of the focal spinal cord at the L2 level, and the boundaries were less clear. The dura mater was linearly enhanced above the lesion (Figure 2).

Intraoperative findings

Under general anesthesia, a median straight incision was made at the L1-4 spinous process. The soft tissues were dissociated, and paraspinous muscles were retracted using a retractor to expose the L1-4 lamina. The dura mater had moderate tension. The dura mater was cut open, and the arachnoid membrane was apparently thickened and adhered to the cauda equina. The cauda equina was apparently thickened and compressed. The arachnoid membrane was cut open and the adhesion was loosened. The membranoid



Figure 1. Sagittal T1WI and T2WI images of lumbar vertebrae MRI scanning before surgery. The spinal cord was thickened, with even patchy signals filled in. Coronal T1WI image of enhanced scanning. Obvious even enhancement of the spinal cord lesion was observed, with linear enhancement of the adjacent dura mater and obvious edema of cauda equina.

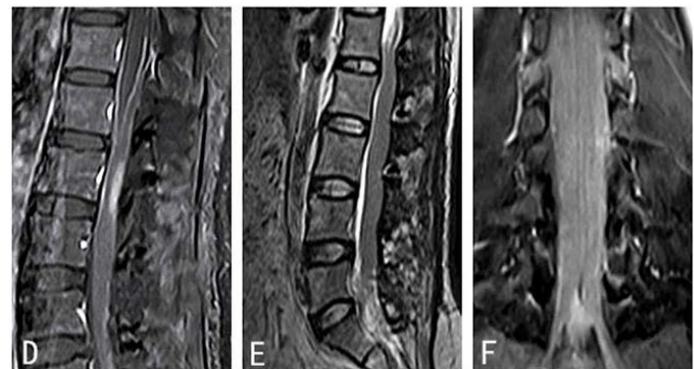


Figure 2. Sagittal T1WI image of enhanced MRI scanning of lumbar vertebrae at one week after surgery. The spinal cord was obviously thinner than before, with abnormally enhanced signals of the focal spinal cord at the L2 level and linear enhancement of the upper dura mater. Sagittal T2WI image and enhanced coronal T1WI image of lumbar MRI scanning at 3 months after surgery. The spinal cord was thickened, and fresh patchy isointense signals were observed in the spinal cord, which showed infiltrative growth. The abnormally enhanced shadow in the spinal cord was enlarged and widened compared to the previous images, and there were unclear structures of the inferior portion of the cone and cauda equina, with uneven enhancement of the local nerve roots.

substance adhering to the cauda equina was resected. The membranoid substance was white and semi-transparent, with a slightly tough texture and insufficient blood supply. The resected specimen was submitted for pathological examination.

Pathological examination and laboratory test

Pathology: The arachnoid membrane was submitted for pathological examination. Fibroblast proliferation was observed under the microscope. The arachnoid membrane was apparently thickened and locally covered by simple squamous epithelium, which expressed CK. The mesenchyme was infiltrated by a large amount of lymphocytes. Routine blood test revealed WBC $8.6 \times 10^9/L$, Hb 122 g/L, PLT $261 \times 10^9/L$. Blood smear: no significant increase or decrease of platelets, with the observation of juvenile cells and normocytic mature cells.

Discussion

The central nervous system is involved in less than 10% of the adult ALL patients [1]. The pathogenesis of this condition is considered to be relevant to the blood-brain barrier. Infiltration of the arachnoid membrane and dura mater is the most common, followed by infiltration of the brain parenchyma, choroid plexus and cranial nerves; however, the infiltration of the spinal cord is most rarely observed [2]. That is why the infiltration of the spinal cord in ALL is frequently misdiagnosed as the primary lesion of the spinal cord. Following causes have been proposed for the infiltration of the spinal cord in ALL [3]: (1) Direct infiltration and injury of the spinal meninges and nerve root by leukemia cells; (2) Leukemia cells present with nodular hyperplasia or form chloroleukemia that compresses the spinal cord, leading to obstructed blood flow to extradural venous plexus and hence to vasogenic edema; (3) Leukemia cells cause blockage of the vessels of the spinal cord, which leads to secondary ischemic changes and abnormal blood perfusion of the spinal cord, further resulting in the degenerative softening of the spinal cord.

Upon MRI scan, infiltration of the spinal cord in ALL mainly presents as the infiltration of the pia mater and dura mater. Infiltration of the pia mater is featured by non-uniform thickening of the pia

mater, with linear or patchy abnormal signals in the spinal cord [4]. The lesion is generally shown as isointense or slightly hypointense T1WI signals and as slightly hyperintense T2WI signals. The contrast-enhanced MRI scan usually reveals apparent thickening and enhancement of the pia mater. If thickening of the pia mater is absent, the contrast-enhanced MRI scan generally reveals linear enhancement of the pia mater at the lesions. Infiltration of the dura mater is mainly presented as a soft tissue mass, which has a wide-base connection with the dura mater. It is usually shown as slightly hypointense T1WI signals and slightly hyperintense T2WI signals with unclear boundaries and uniform enhancement upon the contrast-enhanced MRI scan. When the mass penetrates the dura mater, the adjacent vertebra will be involved and shown as non-uniform signals in the vertebral body. The boundaries are irregular and enhanced upon the contrast-enhanced scan.

Our reported case exemplifies typical diagnostic and treatment procedures for the infiltration of the spinal cord in ALL. The patient had spinal cord lesion as the initial presentation with sub-acute onset but no other specific manifestations. MRI should be performed to find out the causes after excluding other diseases, especially after the failure to explain the symptoms by the primary lesions of the spinal cord. Infiltration of the spinal cord related to ALL is probable in the presence of such conditions. The diagnosis should be confirmed by early bone marrow biopsy to prevent missed diagnosis and misdiagnosis.

References

1. Pan Y, Wang CH, Wang PH, Tao Q, Xiong S, et al. Transverse myelopathy occurring with intrathecal administration of methotrexate and cytarabine chemotherapy: A case report. *Oncol Lett.* 2016; 11: 4066-4068.
2. Prathap JJ, Maria RR. Dorsal column myelopathy following intrathecal chemotherapy for acute lymphoblastic leukemia. *J Spinal Cord Med.* 2014; 37: 107-113.
3. Jianxiang W. Precise diagnosis and standard treatment for adult ALL. Proceeding of the Third Chinese Symposium on Medical Oncology, 2009: 102-114.
4. Zhuzhen L. CT and MRI manifestations of central nervous system leukemia. *J Med Radiol Technol.* 2004; 227: 40-41.

***Correspondence:** Sun Shengjun, Department of Neuroradiology, Beijing Tiantan Hospital Affiliated to Capital Medical University, 100050, China, Tel: 13611293369; E-mail: sunshengjun0212@163.com

Rec: May 25, 2018; Acc: Jun 11, 2018; Pub Jun 15, 2018

J Clin Med Imag. 2018;1(1):3
DOI: [gsl.jcmi.2018.00003](https://doi.org/10.21960/jcmi.2018.00003)

Copyright © 2018 The Author(s). This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC-BY).