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MR of Staphylococcal Myelitis of the Cervical Spinal Cord

Henry M. Friess and John J. Wasenko

Summary: A homogeneously enhancing cervical cord lesion and multiple ring-enhancing brain lesions were detected with MR imaging in a 41-year-old woman with a staphylococcal septicemia. The brain and spinal cord lesions diminished in size after antibiotic therapy. Although no biopsy was performed, we believe, on the basis of the clinical outcome, that the spinal cord lesion represented a bacterial myelitis and that the lesion was prevented from developing into an abscess by early antibiotic therapy.

Index terms: Spinal cord, infection; Myelitis

Case Report

A 41-year-old woman had fever, chills, weakness, and decreased sensation in the right upper extremity. A motor vehicle accident sustained 3 years before admission had left her paraplegic. Physical examination revealed the presence of a T-9 sensory level. Other diagnostic tests showed a white blood cell count of 14 000 with a differential of 72% polymorphonuclear cells and 22% bands; blood cultures were positive for staphylococcus aureus. An echocardiogram showed a thickened mitral valve. Magnetic resonance (MR) studies of the brain (Fig 1A–C) revealed multiple ring-enhancing lesions, and an MR examination of the cervical spine (Fig D–G) showed an elongated, homogeneously enhancing intramedullary lesion of high T2 signal intensity extending from C-2 to C-7 and involving the central segment and right side of the spinal cord; there was no cord expansion or hemorrhage. No cerebrospinal fluid (CSF) samples were obtained.

Gentamicin and oxacillin were administered, resulting in improved function and sensation of the right upper extremity. A repeat MR examination 2 weeks later showed a decrease in the size and number of brain lesions and a decrease in the size of the spinal cord lesion (Fig 1H–K). The patient's right arm function returned to baseline. The presumptive clinical diagnosis was staphylococcal myelitis associated with staphylococcal brain abscesses.

Discussion

The occurrence of intramedullary infection is rare; a few cases have been described in the

literature (1). Bacteria such as staphylococcus and streptococcus are the most common organisms to invade the spinal cord (1), yet infections may also be caused by viruses, fungi, *Mycobacterium tuberculosis*, cysticercus, *Toxoplasma* and *Listeria* organisms, and parasites (1–6). Hematologic spread is the most common source of infection; however, lymphatic and direct extension may also occur (1). The most common sources are coexistent pulmonary, cardiac, skeletal, genitourinary, gastrointestinal, and cutaneous infections. The natural course of an intramedullary bacterial infection has not been formally described as far as we know. This is understandable, as a suspected abscess would be treated immediately with antibiotic therapy with or without surgical intervention.

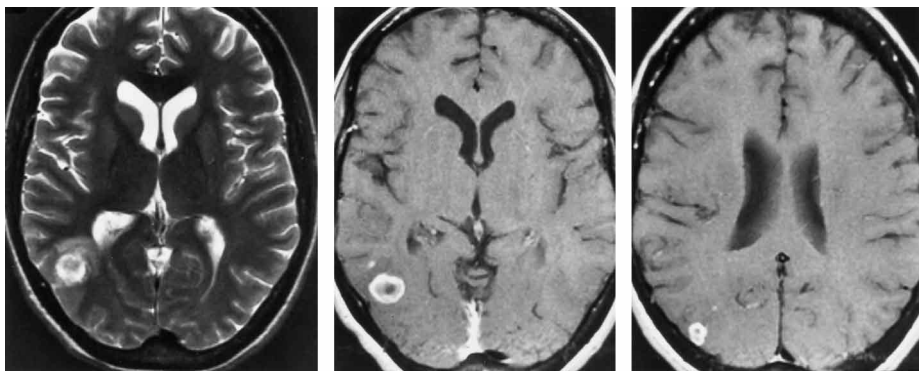
In this case, a thickened mitral valve suggested endocarditis as the source of the staphylococcal septicemia that resulted in infection of the brain and spinal cord. Ring-enhancing parenchymal lesions at the gray-white matter junction are characteristic of abscesses resulting from blood-borne infection. The spinal cord lesion enhanced homogeneously and showed no evidence of cavitation. Since the patient improved both clinically and radiologically after antibiotic treatment with gentamicin and oxacillin, we believe that the imaging findings represented an early bacterial myelitis that may have been pathophysiologically related to the early cerebritis stage of brain abscess prior to encapsulation and central liquefaction necrosis.

The lesion begins after bacteria arrive in the cortex. Polymorphonuclear cells migrate through the swollen endothelium of local capillaries to form an area of suppurative cerebritis. Necrosis and hemorrhage follow (2). Some authors think that the lesion begins as a focus of

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A

B

C



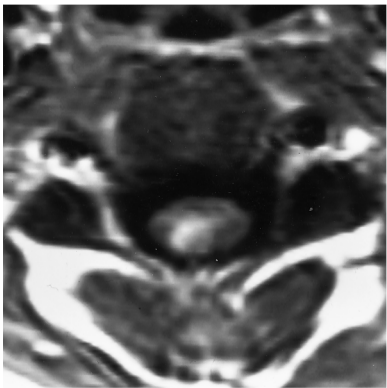
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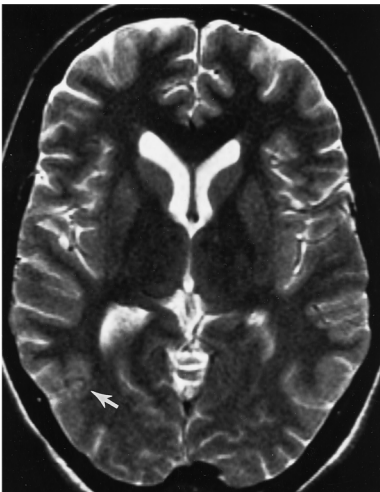
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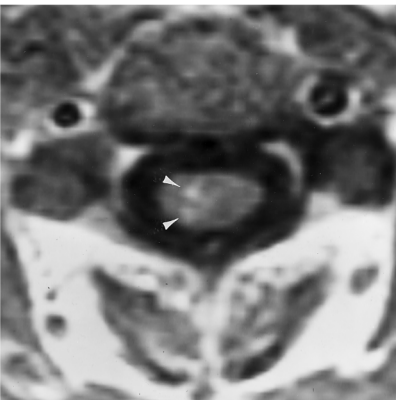
H



I



J



K

venous thrombosis and infarction with subsequent bacterial invasion (1).

The differential diagnosis of intramedullary spinal cord lesions with increased signal intensity on T2-weighted MR images is extensive. In this instance, most diagnoses were excluded by the patient's history and laboratory findings. It is difficult to differentiate bacterial myelitis from primary neoplasms, infarction, and transverse myelitis by radiologic findings alone, since the lesions may have similar imaging characteristics. Intramedullary neoplasms usually result in spinal cord enlargement and a slow progression of neurologic deficits. They may also be infiltrative without spinal cord expansion. The infectious process in this case must be infiltrative in nature as there was no spinal cord enlargement. The differentiating point is that neoplastic processes would not be expected to improve with antibiotic therapy.

An infarction involving the anterior spinal artery would be expected to involve the spinal cord bilaterally with variable contrast enhancement (7, 8) and should result in tissue loss, but no atrophy was seen on the repeat MR study. Acute transverse myelitis is characterized by loss of motor and sensory function and is preceded by a viral illness. Typical findings include cord swelling, increased signal intensity, and variable enhancement (9–11).

In our case, there was no history of recent trauma, multiple sclerosis, sarcoid, infection with the human immunodeficiency virus, lupus, radiation treatment, or exposure to toxins. There were no areas of signal void or hemorrhage to suggest a vascular malformation. No characteristic herpetic skin rash was present, and presuming the brain and spinal cord lesions

were of the same cause, the ring-enhancing lesions would be most uncharacteristic of a herpetic infection (6). Acute disseminated encephalomyelitis occurs after a viral illness or vaccination and usually affects the brain and occasionally the spinal cord. Typical intracerebral abnormalities include multiple areas of increased signal intensity with variable contrast enhancement in the periventricular and subcortical white matter (12, 13). Spinal cord sarcoid is characterized by cord expansion and patchy parenchymal and leptomeningeal enhancement (14). Multiple sclerosis lesions in the spinal cord are typically less than two vertebral bodies in length and located at the periphery of the cord (15, 16). Subacute necrotizing myelopathy is believed to be caused by dural arteriovenous fistulas. Patients present with progressive motor and sensory deterioration (17). MR findings include abnormal T2 signal, variable enhancement, and spinal cord enlargement.

Bacterial myelitis should be included in the differential diagnosis of an enhancing intramedullary spinal cord lesion in the proper clinical setting. The early diagnosis of this entity may prevent progression into a frank abscess and resultant permanent neurologic deficits.

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References

1. Sharif HS. Role of MR imaging in the management of spinal infections. *AJR Am J Roentgenol* 1992;158:1333–1345

Fig 1. A 41-year-old woman with staphylococcal septicemia.

A–G, Pretreatment MR studies.

A, Axial T2-weighted image shows a mixed-signal-intensity lesion with surrounding edema in the right posterior temporal lobe.

B and C, Contrast-enhanced T1-weighted images show ring-enhancing and nodular lesions in the posterior right temporal lobe.

D, Sagittal T2-weighted image of the spinal cord shows diffuse increased signal intensity extending from C-2 inferiorly to C-7.

E, Sagittal T1-weighted image shows diffuse low signal intensity in the cervical cord. Several punctate foci of high signal probably represent petechial hemorrhage.

F, Contrast-enhanced sagittal T1-weighted image shows diffuse intense enhancement within the spinal cord.

G, Contrast-enhanced axial T1-weighted image shows lesion to be located in the central and right side of the spinal cord.

H–K, MR studies 2 weeks after antibiotic therapy.

H, Axial T2-weighted image shows decrease in size of lesion and surrounding edema (arrow).

I, Sagittal T2-weighted image reveals a marked decrease in the abnormal signal intensity in the spinal cord. (The high signal abnormality at the T-4 level is volume averaging of CSF in a left posterolateral arachnoid cyst. The spinal cord is displaced to the right side.)

J, Contrast-enhanced sagittal T1-weighted image shows no detectable enhancement.

K, Contrast-enhanced axial T1-weighted image shows small residual foci of enhancement in the right side of the spinal cord (arrowheads).

2. Greenfield's Neuropathology. 5th ed. New York, NY: Oxford University Press; 1992
3. Harris TM, Smith RR, Bognanno JR, Edwards MK. Toxoplasmic myelitis in AIDS: gadolinium-enhanced MR. *J Comput Assist Tomogr* 1990;14:809-811
4. Melhem ER, Wang H. Intramedullary spinal cord tuberculoma in a patient with AIDS. *AJNR Am J Neuroradiol* 1992;13:986-988
5. King SJ, Jeffree MA. MRI of an abscess of the cervical spinal cord in a case of *Listeria* meningoencephalomyelitis. *Neuroradiology* 1993;35:495-496
6. Esposito MB, Arrington JA, Murtaugh FR, Coleman JM, Sergay SM. MR of the spinal cord in a patient with herpes zoster. *AJNR Am J Neuroradiol* 1993;14:203-204
7. Elksnis SM, Hogg JP, Cunningham ME. MR imaging of spontaneous spinal cord infarction. *J Comput Assist Tomogr* 1991;15:228-232
8. Yuh WTC, Marsh EE III, Wang AK, et al. MR imaging of spinal cord and vertebral body infarction. *AJNR Am J Neuroradiol* 1992;13:145-154
9. Merine D, Wang H, Kumar AJ, Zinreich SJ, Rosenbaum AE. CT myelography and MR imaging of acute transverse myelitis. *J Comput Assist Tomogr* 1987;11:606-608
10. Barakos JA, Mark AS, Dillon WP, Norman D. MR imaging of acute transverse myelitis and AIDS myelopathy. *J Comput Assist Tomogr* 1990;14:45-50
11. Holtås S, Basibüyük N, Fredriksson K. MRI in acute transverse myelopathy. *Neuroradiology* 1993;35:221-226
12. Atlas SW, Grossman RI, Goldberg HI, Hackney DB, Bilaniuk LT, Zimmerman RA. MR diagnosis of acute disseminated encephalomyelitis. *J Comput Assist Tomogr* 1986;10:798-801
13. Caldemeyer KS, Smith RR, Harris TM, Edwards MK. MRI in acute disseminated encephalomyelitis. *Neuroradiology* 1994;36:216-220
14. Nesbit GM, Miller GM, Baker HL Jr, Ebersold MJ, Scheithauer BW. Spinal cord sarcoidosis: a new finding at MR imaging with Gd-DTPA enhancement. *Radiology* 1989;173:839-843
15. Maravilla KR, Weinreb JC, Suss R, Nunnally RL. Magnetic resonance demonstration of multiple sclerosis plaques in the cervical cord. *AJNR Am J Neuroradiol* 1984;5:685-689
16. Tartaglino LM, Friedman DP, Flanders AE, Lublin FD, Knobler, Liem M. Multiple sclerosis in the spinal cord: MR appearance and correlation with clinical parameters. *Radiology* 1995;195:725-732
17. Mirich DR, Kucharczyk W, Keller MA, Deck J. Subacute necrotizing myelopathy: MR imaging in four pathologically proved cases. *AJNR Am J Neuroradiol* 1991;12:1077-1083