

J Korean Neurosurg Soc 40: 289-292, 2006

Thoracic Actinomycosis Causing Spinal Cord Compression

In-Soo Kim, M.D.

Department of Neurosurgery, Dongsan Medical Center, Keimyung University School of Medicine, Daegu, Korea

Thoracic spinal actinomycosis causing epidural abscess and significant spinal cord compression is very rare. A case is presented of a 56-year-old woman with rapid progressive upper back pain and weakness in both legs without evidence of systemic infection. Magnetic resonance imaging revealed a thoracic epidural enhancing lesion at the T1-T5 level. After decompression by laminectomy, precise diagnosis was accomplished using specific histopathological studies of the surgical specimens. A histopathologic findings showing typical Actinomyces sulfur granules surrounded by acute inflammatory cells. The clinical and radiological findings of spinal actinomycosis closely resemble metastatic tumors and other infectious processes. Delay in diagnosis and treatment can significantly worsen the condition of patient.

KEY WORDS: Spinal actinomycosis · Cord compression.

Introduction

A ctinomycosis is an uncommon chronic suppurative infection that generally involves the cervicofacial, thoracic and abdominal regions that caused by the Actinomyces species. Actinomyces are part of the normal gastrointestinal flora and its common primary sources are the oral cavity, the gastrointestinal system, the lungs, or the paranasal sinuses.

Spinal actinomycosis is an unusual infection and spread of actinomycosis occurs either by direct invasion or hematogenous dissemination¹⁵⁾. Spinal cord compression of thoracic vertebrae due to actinomycosis are very rare and usually are confused with metastatic involvement of the spine^{4,7,9,13,22)}. If treated in time the prognosis for actinomycosis is good, but the condition can be difficult to diagnose.

Case Report

A 56-year-old woman was admitted to the hospital reporting a 2-week history of upper back pain and progressive weakness in both legs. Neurologic examination was remarkable for loss of pinprick and temperature sensation on the left side of her body below approximately T2 and downward. She had 1/5 power in the right leg and 2/5 power in the left leg with

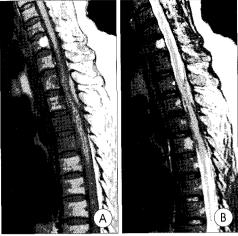
hyporeflexia in both legs. The patient had urinary incontinence and loss of anal tone, but she had no fever or other systemic complaints. The result of laboratory tests were negative except for an elevated erythrocyte sedimentation rate. Plain radiographs of the spine revealed no abnormality. Magnetic resonance imaging demonstrated involvement of the T3 and T4 vertebral bodies. Intervertebral discs were spared. An epidural infiltration extended from T1 to T5. The mass was slightly hyperintense in comparison with the spinal cord and showed enhancement on gadolinium administration. In the axial section of the spine, the epidural lesion located left and posterior space. No remarkable changes were found within the spinal cord (Fig. 1).

The results of repeated microbiological studies of blood, throat, urine, stool, and surgical specimens as well as specific antibody titration studies did not suggest any infectious disease. Computed tomography, MR imaging, and ultrasonographic screening of the thorax, and abdomen along with echocardiography studies also failed to demonstrate any underlying disease. And the test for immunodeficiencies revealed no abnormal results.

The patient underwent surgery via the posterior approach to the T1-T4 level for diagnosis and decompression of the affected spinal cord because of the progressive neurological

[•] Received : April 19, 2006 • Accepted : June 2, 2006

Address for reprints: In-Soo Kim, M.D., Department of Neurosurgery, Dongsan Medical Center, Keimyung University School of Medicine, 194 Dongsan-dong, Jung-gu, Daegu 700-712, Korea Tel: +82-53-250-7065, Fax: +82-53-250-7356, E-mail: neurokim@dsmc.or.kr





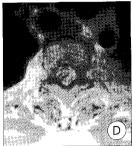




Fig. 1. Sagittal T1 (A) and T2 (B)—weighted images show a large ill—defined low signal intensity mass like lesion at the T1—T5 level, and also visualize decreased vertebral fatty marrow signal intensity at the T3,4 body of vertebrae on T1—weighted image. Axial T2 (C) and T1 (D)—weighted images clearly visualize the extradural located mass lesion and show a paravertebral soft tissue mass, and show right anterolateral displacement of the thoracic spinal cord. On Gadolinium enhanced axial T1—weighted image (E) shows diffuse enhancement of the mass with multiple non—enhancing portions.

deficit. After laminectomy from T1 to T4 level, yellowish pus with granule was apparent. Multiple biopsies were obtained for histopathological and microbiological studies from laminas, and an epidural mass.

A histopathological examination revealed a tissue reaction composed of acute inflammatory cells that penetrated adjacent structures. Multiple microabscess formation was observed, in which each formation contained bacterial colonies consisting of radiating filaments capped by eosinophilic hyaline material when stained with hematoxylin and eosin. Periodic acid-Schiff and methenamine silver stain testing were also performed, and the diagnosis of actinomycosis was confirmed (Fig. 2).

The patient was given intravenously administered ampicillin 8 gram daily for 6 weeks during her hospital stay, and a regimen

of oral amoxicillin (500mg/8 hours) was advised for the next 6 months as an outpatient. After 8 months, symptoms of her neurological disability slight improved, 2/5 power in the right leg and 4/5 power in the left leg, but she remained dependent in her wheelchair.

Discussion

A ctinomycosis is relatively a rare disease. It is a chronic suppurative infection characterized by peripheral spread to contiguous tissues without limitation by anatomical barriers. Actinomyces species are found in the normal flora of the oral cavity, so that the disease is more frequently seen in patients with poor mouth hygiene, and gastrointestinal and pelvic tracts

but not freely in nature. The species have low pathogenicity and may cause endogenous infection after trauma with or without immune suppression. Dental infection, tooth extraction, head trauma and sinus surgery can also predispose a patient to actinomycosis of the central nervous system^{2.6,12,13,19)}.

Usually, actinomycosis involves cervicofacial, thoracic, abdominopelvic regions and several other organs, including skin, brain, pericardium and extremities. Spinal involvement, as in this case, has been demonstrated only in a few cases^{7,10,11,17)} and is mostly the result of contiguous spread of a primary paraveretebral focus, such as a retropharygeal abscess, mediastinal suppuration or perforation of ap-

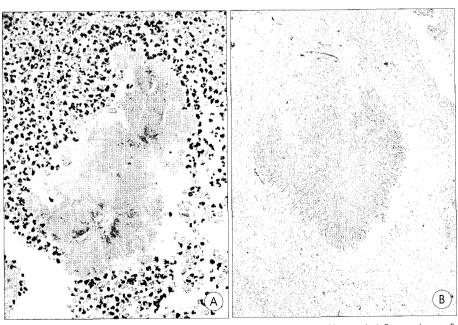


Fig. 2. Histopathologic findings showing typical sulfur granules surrounded by acute inflammatory cells (A) (H&E X 200). Sulfur granules are composed of branching filaments with peripheral palisading clubs (B) (Silver stain X 200).

pendix^{1,22)}. The clinical picture of spinal actinomycosis is vague but back pain is common, usually of a dull and constant nature and often with periods of exacervation. Occasionally the pain is of a definite nerve or root type²²⁾. Often there is remarkable stiffness of the involved vertebral segments⁷⁾.

The infection is thought to spread to the spine by direct extension. It usually involves the adjacent pedicles, transverse processes and heads of the neighbouring ribs. Spinal cord compression and associated paraplegia or tetraplegia, mostly due to an epidural abscess, was described only in a few cases^{9,11,16}. Radiographic findings of spinal actinomycosis show usually saw-toothed borders and honeycombed architecture of vertebral bodies, due to rarefaction surrounded by sclerotic bone, but vertebral collapse is unusual^{5,7,22)}. However, the CT or MRI findings are not specific and may show epidural abscess. Unlike tuberculosis and brucellosis of the spine, the intervertebral disc spaces are usually spared¹⁷⁾ and it is only rarely involved²²⁾.

Diagnosis of spinal actinomycosis is difficult and is often delayed until the stage of the disease advances. It must be confirmed histologically and microbiologically. Diagnosis of actinomycosis can be made by demonstration of typical sulfur granules in infected material, as is seen on routine hematoxylin and eosin staining or on direct examination. In the present case, histopathological studies demonstrated inflammatory granulation tissue and microabscess formation driven by the gram-positive sulfur-containing filamentous bacteria. Actinomyces are morphologically indistinguishable from Nocardia species when using a gram stain that shows aerobic growth and partial acid-fastness¹⁸⁾. As a predictive feature, when stained with hematoxylin and eosin, Actinomyces filaments become basophilic and terminate in eosinophilic clubs but the Nocardia species do not³⁾. Undoubtedly, accurate diagnosis of actinomycosis should be based on bacteriological isolation of the microorganism; however, this is not always possible, and these histopathological differences become more important in this circumstance. Differential diagnosis includes nocardiosis, tuberculosis, nonspecific osteomyelitis, other fungal infections, and malignancy of a metastatic origin. In association with osteomyelitis, the involvement of more than two vertebral bodies and the intervertebral disc space is uncommon. Metastatic lesions usually cause the collapse of vertebral bodies, leaving preserved intervertebral disc space, and are frequently limited to the collapsed vertebrae. Spinal tuberculosis invariably involves vertebral bodies and intervertebral disc spaces, together with relative preservation of the posterior bony elements, and frequently results in compression fractures. Coexistence of destructive and proliferative bony changes with collapsed disc space rather than the affected vertebral bodies, is the characteristic finding of spinal brucellosis²⁰⁾.

Treatment of spinal actinomycosis is based on long-term

antibiotic therapy and surgical drainage or decompression of the spinal cord, if necessary. In some cases, surgical treatment with osteosynthesis was necessary²¹⁾. Actinomycosis should be treated with an intravenous administration of high dose of penicillin G for 6 to 8 weeks and should be continued for at least an additional 4 to 6 months by orally administering effective antibiotics to prevent recurrence of the disease. If penicillin fails, or in case of penicillin allergy, sulfonamides or other antibiotics such as chloramphenicol, clindamycin, erythromycin, cephalosporins, and tetracyclines are effective^{8,14,15)}.

Conclusion

The clinical and radiological findings of actinomycosis L closely resemble metastatic tumors and other infectious processes. Diagnosis may be difficult due to a general lack of familiarity with the disease and the fastidious nature of the organism in culture. If properly diagnosed, it is almost always curable.

References

- 1. Alday R, Lopez-Ferro MO, Fernandez-Guerrero M, Ruiz-Barnes P: Spinal intrathecal empyema due to Actinomyces israelii. Acta Neurochir 101:
- 2. Brown JR: Human Actinomycosis. A study of 181 subjects. Hum Pathol 4:319-330, 1973
- 3. Chen-Wei H: Actinomycosis of the brain: Case report. J Neurosurg **63**: 131-133, 1985
- 4. Cope VZ: Actinomycosis of bone with special reference to infection of the vertebral column. J Bone Joint Surg 33B: 205-214, 1951
- 5. Crank RN, Sundaram M, Shields JB: Case report 197. Cervical actinomycosis with spinal involvement. Skeletal Radiol 8: 164-167, 1982
- 6. Eftekhar B, Ketabchi E, Ghodsi M, Ahmadi A: Cervical epidural actinomycosis. J Neurosurg (Suppl 1)95: 132-134, 2001
- 7. Ernst J, Ratjen E: Actinomycosis of the spine: Report of two cases. Acta Orthop Scand 42: 35-44, 1971
- 8. Garrod LP: The sensitivity of Actinomyces israelii to antibiotics. Br Med Ir 1: 1263-1264, 1952
- 9. Kannangara DW, Tanaka T, Thadepalli H: Spinal epidural abscess due
- to Actinomyces israelii. **Neurology 31**: 202-204, 1981 10. Kaplan AH, Weber DJ, Oddone EZ, Perfect JR: Infection due to Actinobacillus actinomycetem comitans: 15 cases and review. Rev Infect Dis 11: 46-63, 1989
- 11. Kim KY, Cho KH, Ahn YM, Kim SH, Ahn YH, Yoon SH, et al: A Surgical Treatment of the Cervicothoracic Spinal Actinomycosis with Spinal Cord Compression. J Korean Neurosurg Soc 27: 381-386, 1998
- 12. Kirsch WM, Stears JC: Actinomycotic osteomyelitis of the skull and cpidural space. Case report. **J Neurosurg 33**: 347-351, 1970
 13. Lane T, Goings S, Fraser DW, Ries K, Pettrozzi J, Abrutyn E: Disse-
- minated actinomycosis with spinal cord compression; report of two cases. Neurology 29: 890-893, 1979
- 14. Lerner PI: Susceptibility of pathogenic actinomycetes to antimicrobial compounds. Antimicrob Agents Chemother 5: 302-309, 1974
- 15. Lerner PI: Actinomyces and Arachnia species, in Mandell GL, Douglas RG Jr, Bennett JE (eds): Principles and Practice of Infectious Disease, ed 2. New York: John Wiley & Sons Inc., 1985, pp1427-1433
- 16. Muller PG: Actinomycosis as a cause of spinal cord compression: a case report and review. Paraplegia 27: 390-393, 1989
- 17. Oruckaptan HH, Senmevsim O, Soylemezoglu F, Ozgen T: Cervical actinomycosis causing spinal cord compression and multisegmental root failure: case report and review of the literature. Neurosurgery 43:937-940, 1998

- Smego RA: Actinomycosis of the central nervous system. Rev Infec Dis 9:855-865, 1987
 Sundaram C, Purohit AK, Prasad VS, Meena AK, Reddy JJ, Murthy
- JM : Cranial and intracranial actinomycosis. Clin Neuropathol 23 : 173-177, 2004
- 20. Tekkok IH, Berker M, Ozcan OE, Ozgen T, Akalin E: Brucellosis of
- the spine. Neurosurgery 33: 838-844, 1993
 21. Voisin L, Vittecoq O, Mejjad O, Krzanowska C, Defives T, Cambon-Michot C, et al: Spinal abscess and spondylitis due to actinomycosis. Spine 23: 487-490, 1998
 22. Young WB: Actinomycosis with involvement of the vertebral column. Case report and review of the literature. Clin Radiol 11: 175-182, 1960