Compressive Myelopathy Due to Nocardiosis from Dermal Lesion


Abstract

Nocardiosis refers to locally invasive or disseminated infection associated with *Nocardia* species. Most infections enter through respiratory tract and then disseminate systemically. Rarely primary nocardial infection of the skin of the back may spread to contiguous structures including vertebrae. A 30 years male presented with indolent skin lesion in the upper dorsal region of the back for one year following an accident and subsequently developed features of spinal cord compression and parenchymal involvement of lung. The rarity of such type of spread in an immuno-competent individual has been highlighted.

**INTRODUCTION**

Nocardiosis refers to locally invasive or disseminated infection associated with *Nocardia* species, aerobic actinomycetes that cause several characteristic syndromes. The aerobic actinomycetes are a large and diverse group of Gram positive bacteria that appear on microscopy as branching filamentous cells. *Nocardia* species are usually found in soil, but may be aerosolized. Most infections enter through respiratory tract, and then disseminate systemically. Rarely skin infection occurs primarily following traumatic abrasion. Skin is a common site of metastatic infection also. Primary nocardial infection of the skin of the back may spread to contiguous structures including deeper tissues such as vertebra. Nocardia rarely cause epidural abscess with or without concurrent vertebral osteomyelitis. Extradural granuloma formation with spinal cord compression has also been reported.

We are presenting a case of 30 years male individual who had an indolent skin ulcer in the upper dorsal region of the back for a period of one year following a railway accident and subsequently developed features of spinal cord compression.

**CASE REPORT**

A 30 years nondiabetic, nonalcoholic, male individual, cultivator by profession sustained in injury to the back following a fall from the train in August 1994 without any immediate sequelae. After two months, he noticed a swelling on the upper dorsal region of back. The swelling was firm and burst after two months producing multiple sinuses with scanty serous discharge. He had few relapses and remissions in next five months. He developed three episodes of haemoptysis from mid-1996 to January, 1997. At the same period he experienced weight loss of 6 kg over a period of one year. He was put on antituberculous drugs (ATD) empirically and there was marked well being with improvement of skin lesion and weight gain. While on ATD, he developed tingling, numbness, heaviness and weakness...
of both the lower limbs followed by sphincteric disturbance in the form of hesitancy. He developed flexor spasm and observed girdle sensation in the lower part of the chest wall. His symptoms were gradually increasing in severity to the extent that he became bed bound by December 2000. He was subsequently hospitalized. Physical examination revealed temperature - 97.5°F, pulse - 80/min., respiration - 20/min., BP - 120/70 mm of Hg. Patient was malnourished and had mild pallor and bilateral firm, mobile, non-tender axillary lymphadenopathy. He had a diffuse scarred skin lesion with multiple sinuses on the middle part of the upper back (Fig. 1). Neurological examination revealed normal higher function and cranial nerves, spastic paraplegia with disuse wasting of anterior and lateral compartmental muscles, exaggerated deep tendon reflexes, bilateral extensor plantar response, absent cremasteric and abdominal reflexes, sensory level at D5 and retention of urine. Gait and stance could not be tested.

Investigation revealed haemoglobin level varying from 7.5 gm% to 11.5 gm% with normal total and differential count and normal platelet count. ESR varied from 138 to 148 mm in the first hour. Blood biochemistry showed FBS - 76 mg% per dl, creatinine 0.9 mg/dl and normal LFT. Chest X-ray done on 13.11.2000 showed scarry shadows in the right upper zone and repeat chest X-rays on 26.7.2000 showed haziness in both apices with non-specific pneumonitic changes. X-ray of dorsolumbar spine was normal. Serology from VDRL, HIV-1 and HIV-2 using ELISA Tirdot test for antibodies of HIV-1 and HIV-2 were non-reactive. Mantoux test done on two occasions using 5TU showed negative response. MRI of dorsal spine done on 13.12.2000 showed involvement of D2 to D6 vertebrae characterized by hyperintensity in T2 WI and hypointensity in T1 WI. Large epidural mass, lobulated pre- and para-spinal mass and cord compression was seen at D2 to D6 level with focal areas of hyperintensity (Figs. 2 and 3). Sputum for AFB done on five consecutive days was found to be negative. Culture of sputum showed growth of *Streptococcus viridans*, and no growth of *Mycobacterium tuberculosis* was observed. Skin material smear showed plenty of pus cells and necrotic debris and no microorganism. Cover slip preparation failed to show any ray fungus. Pus for KOH preparation showed no fungal element. No growth of pathogen on pus culture was noticed.

The patient underwent appropriate surgical debridement on 22nd of December 2000 in combination with antimicrobial therapy (cefotaxime and amikacin) for two weeks. Following the therapy the skin lesion improved.

At operation, the extensive lesion consisting of multiple pockets of granuloma with fibrosis was in direct continuity from skin to the extradural space through the subcutaneous tissue and muscle. Histology from surgically debrided tissue (Fig. 4) from dorsal spine showed fibrofatty tissue heavily infiltrated with inflammatory cells and multiple microabscesses and granule of *Nocardia braziliensis* in the centre. Gram stain showed Gram positive beaded filamentous organism confirmed *Nocardia*. Thereafter we treated him with cotrimoxazole and minocycline.

The patient’s neurological status gradually improved over next one year to the extent that he could walk without support but continued to have sphincteric problems and he remained catheterized. In March 2002 he had another bout of massive haemoptysis and was admitted in Chest Department. A CT scan of thorax revealed a small area of infiltration in the right
upper lobe adjacent to the para-spinal soft tissue (Fig. 5). Brochoalveolar lavage did not reveal *Mycobacterium* or pathogenic fungi including *Nocardia*. His blood examination showed total leucocyte count of 7000/cumm with lymphocyte 27% of WBC, absolute CD4 count of 567, absolute CD8 count of 378, and CD4/CD8 ratio of 1.5. The patient received blood transfusion and third generation cephalosporin and amikacin for two weeks at the chest ward. When we saw him in the month of July 2002 he did not have any pulmonary symptoms and his neurological status were same as before.

**DISCUSSION**

Our patient had infection by *Nocardia braziliensis* in epidural space with secondary spinal cord compression. On the first look it appears that the infecting organism entered through the respiratory tract. This is evidenced by the features of systemic involvement in the form of generalized weakness, significant anaemia, weight loss, and persistent high ESR. The patient also had repeated bouts of haemoptysis and skiagram of chest revealed nonspecific scarring. In a study, Kreuger *et al* have shown that 20-30% of patients of pulmonary nocardiosis have signs of CNS involvement. However, looking the case in totality, it appeared that skin was the portal of entry for the infecting organism. There was a strong temporal correlation between the site of skin abasion and subsequent development of skin lesion. It is said that spread from a primary pulmonary focus is presumed when nocardial infection is found in any organ other than skin. Therefore, the presence of skin lesion in our patient suggests skin to be the probable site of entry. Moreover, the MRI revealed features of involvement of skin, subcutaneous tissue, muscle, and adjoining vertebrae. The operative finding also suggested contiguous spread of infection from skin and CT scan of thorax demonstrated pulmonary lesion in direct relation to the paraspinal soft tissue structures.

Nocardial infections of the CNS have also been reported following trauma, apparently by direct implantation of the infecting agent. Although primary skin infection may spread by extension to contiguous structures and regional lymph nodes, systemic spread from such foci is extremely rare. Cell mediated immunity of the individual plays an important role for nocardial infection to spread and become disseminated. Superficial nocardiosis after minor skin trauma is not necessarily associated with compromised cell mediated immunity, but it may progress to disseminated disease in that setting. Our patient did not have deficiency of cell mediated immunity was evidenced by negative ELISA for antibodies against HIV-1 and 2 viruses, normal CD4 and CD8 cell count. Nevertheless he developed contiguous spread of infection from skin to paravertebral soft tissue, vertebrae, and spinal cord, and adjacent lung parenchyma. Such a spread in an immunocompetent individual is a rare phenomenon. However, the infection in our patient probably did not spread haematogenously as no distant structures were involved. Haematogenous route is an important pathway for nocardial infection to extra-pulmonary sites. About one-fifth of patient with disseminated disease present only with extra-pulmonary disease, which probably spread haematogenously from an inapparent or healed pulmonary focus.

Laboratory studies, other than bacteriology are of little aid in diagnosis. The radiographic pattern of pulmonary nocardiosis is not diagnostic. The abnormality that might be present on chest X-ray is quite nonspecific and might be
indistinguishable from other pathology of lung such as tuberculosis, abscess, bacterial pneumonia, and malignancy.  

Because the initial immune response to *Nocardia* is pyogenic, skin lesion may be treated as staphylococcal in origin or may be self-limited. This may be one of the causes of delay in diagnosis in our patient. Our patient received a course of cotrimoxazole and antitubercular therapy and he showed improvement with both of these treatments. The improvement following cotrimoxazole is due to susceptibility of *Nocardia* to it and the improvement following antitubercular drugs might be due to inclusion of rifampicin in ATD regime that has antibacterial activity against *Nocardia*.

*Nocardia* is often difficult to recover from clinical material. In order to increase the likelihood of identifying patients infected with *Nocardia* a high index of suspicion must be maintained. In our patient, smear of the exudates of the skin lesion did not reveal any *Nocardia*, but the tissue from space occupying lesion of the dorsal spine showed Gram-positive beaded filamentous organism.

Unsatisfactory improvement of neurological status in our patient might be due to delayed surgery when permanent damage to spinal cord has already occurred.

**References**


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**Announcement**

The office bearers of the **Association of Physicians of India, Kota Division Chapter**, for the year 2003.

Chairman : GS Taterh

Vice Chairman : A Jain

Hon. Secretary : AR Pathan

Hon. Treasurer : Deepti Sharma

Sd/-

GS Taterh

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