Thoracic actinomycosis presenting as a brachial plexus syndrome

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Thoracic actinomycosis is a well described entity. The clinical spectrum is broad, ranging from the asymptomatic patient with an abnormality on chest radiography to the chronically ill patient with fever, weight loss, and skin lesions from sinus tracts draining a lung abscess. Here we report a more unusual presentation, a patient with a chest wall lesion and right handed weakness found to be secondary to a brachial plexus lesion induced by actinomycosis.

Case report

A 37 year old right handed truck driver was well until four months before his admission to hospital, when he noted a small painless blister on his right anterior chest wall. One month later the blister broke and a 1 × 2 cm non-healing persistent ulcer formed, which drained scant amounts of clear fluid. Two weeks before admission he noted weakness in his right hand, particularly affecting his grip strength. He consulted his physician who noted the chest lesion and performed a biopsy. During the next two weeks weakness of the right hand increased dramatically so that he was unable to grip a steering wheel and a second small blister appeared on the right side of his neck. At this point he was seen at the John Cochrane Veterans Administration Medical Center and admitted for further evaluation. He denied fatigue, fever, weight loss, chronic cough, or sputum production. There was no history of exposure to tuberculosis. He denied any right arm or shoulder trauma. He had had severe carious disease with multiple tooth abscesses, and, 10 months before admission, all his teeth had been extracted.

On admission he was alert, thin, and appeared well. He was not feverish, and all heart rate and blood pressure readings were normal. He was edentulous, and his oropharnyx was clear of lesions. There was a 2×1 cm draining lesion on the right side of the neck, 8 cm above the clavicle. The surrounding skin was indurated with the suggestion of a cluster like mass underneath it. A second lesion, consisting of a 3×3 cm granulating tissue mass, draining sero sanguinous fluid was located on the anterior chest wall 5 cm above the right nipple. A probe showed a track in the centre of the lesion extending 2 cm into the chest wall. There were no abnormalities on auscultation or percussion of the chest. Examination of the heart and

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percussion of the chest. Examination of the heart and

abdomen showed no abnormality. There was considerable weakness in the right hand, including an inability to flex or extend the hand against resistance or to abduct fingers in extension. There was weakness of abduction, adduction, and opposition of the thumb and weakness in both radial and ulnar extension. Grip strength was negligible. There was no weakness in the more proximal arm muscles and no sensory loss. No other neurological abnormalities were seen.

The haemoglobin concentration was 10·1 g/dl and the white cell count 12.6 × 10° with a normal differential count. The erythrocyte sedimentation rate was 35 mm in first hour. All other blood studies were within normal limits. A chest radiograph showed consolidation in the right apex with paratracheal lymphadenopathy. Computed tomography of the neck and chest with contrast showed an irregular soft tissue density in the apex and anterior segment of the right upper lobe. Small air bubbles could be seen within the mass, and there was thickening of the adjacent pleura. Sections through the C₈-T₂ vertebral regions showed obliteration of the normal fat planes between the scalene muscles suggestive of inflammation in this region. Review of histological appearances of material obtained from the biopsy of the chest wall lesion showed numerous sulphur granules composed of non-acid-fast, thin branching, filamentous organisms with clubbed endings consistent with actinomyces. Anaerobic cultures from the chest lesion did not grow the organism despite prolonged incubation. Based on the biopsy findings thoracic actinomycosis was diagnosed and treatment with intravenous benzyl penicillin (three million units every four hours) begun. After seven days of treatment improvement in the skin lesions and in the strength of the patient's right hand was obvious. By the 20th day of treatment the right neck lesion had healed and the chest lesion was half of its original size. The right hand grip strength improved to 30 pounds per square inch having been immeasurable at the start of treatment. A repeat chest radiograph on the 30th day of penicillin treatment showed almost complete resolution of the upper lobe infiltrate. After 40 days of parenteral penicillin he was discharged home with plans to treat him with oral penicillin for another six months. Six months later his chest wall lesion had healed and the chest radiograph showed no remaining upper lobe infiltrate.

Discussion

The differential diagnosis in our patient included neoplasm (both primary lung and metastatic), fungal infection, anaerobic lung abscess, and tuberculosis. The history of

carious disease and recent dental work, the skin lesions, and the draining sinus tracts in the chest and neck suggested the diagnosis of thoracic actinomycosis. The biopsy confirmed the diagnosis. The extent of his disease was impressive, with the right upper lobe, the anterior chest wall, and the soft tissue of the right neck affected according to the physical findings and the radiographic studies. The neurological findings were consistent with a right brachial plexus lesion affecting the lower cervical and first thoracic roots. Whether the lesion involved direct nerve invasion or compression of the plexus secondary to inflammation was not clear. The rapid and complete clinical response to antibiotic treatment, however, suggests the second explanation.

Since the original description in 1882 by Ponfick' over 300 reports of cases of thoracic actinomycosis have been published.²⁻⁷ Most of the early series comprised patients with histories of chronic fever, weight loss, skin lesions, the development of draining sinus tracts, and the eventual discovery of underlying pulmonary lesion.²⁻⁴ Later series emphasised less distinctive presentations of thoracic actinomycosis such as haemoptysis, empyema, a mass on chest radiography, or chest pain.⁵⁻⁷ Rarer reported presentations include brain abscess, pericarditis, and abdominal pain secondary to lower thoracic intercostal nerve invasion.³ To our knowledge, this is the first reported case of a

brachial plexus lesion associated with thoracic actinomycosis. Our patient shows that thoracic actinomycosis may present in a variety of ways and that prolonged medical treatment can be curative, even with extensive disease.

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References

- 1 Ponfick E. Die Actinomyclose des Menschen. Berlin: Hirschwald. 1882.
- 2 Cope Z. Actinomycosis. London: Oxford University Press, 1938.
- 3 Bates M, Cruickshank G. Thoracic actinomycosis. *Thorax* 1957; 12:99-124.
- 4 Cutler EC, Gross RE. Actinomycosis of the lung and pleura. American Review of Tuberculosis 1940;41:358-62.
- 5 McQuarrie DG, Hall WH. Actinomycosis of the lung and chest wall. Surgery 1968; 64:905-11.
- 6 Brown JR. Human actinomycosis: a study of 181 subjects. Hum Pathol 1973;4:319-30.
- 7 Slade PR, Slusser BV, Southgate J. Thoracic actinomycosis. Thorax 1973; 28:73-85.