

Tuberculosis of the nasopharynx misdiagnosed as Wegener's granulomatosis

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Even when tuberculosis was a common disease in the United Kingdom, tuberculosis of the nasal mucous membranes was considered rare. This case report describes a patient with tuberculosis affecting the nasal mucous membranes and nasopharynx who was initially misdiagnosed as suffering from Wegener's granulomatosis. Inappropriate treatment with immunosuppressive drugs permitted the development of miliary tuberculosis.

Case report

A 44 year old caucasian female office worker presented with a three month history of nasal obstruction, catarrh, and left sided deafness. There was no relevant past medical history, although her paternal grandmother died of tuberculosis. She had not previously had a tuberculin test or received BCG vaccination, and denied any recent contact with tuberculosis. Physical examination revealed granular inflammation of the nasal mucosa and nasopharynx, and there was a fluid level behind the left tympanic membrane. A chest radiograph was normal.

The haemoglobin concentration was 0.11 g/l, the white blood cell count $5.5 \times 10^9/l$ with a normal differential count, and the erythrocyte sedimentation rate (ESR) 27 mm in one hour. Blood urea and plasma electrolytes, results of liver function tests, and a protein electrophoretic strip were all normal. Serological tests for syphilis gave negative results and a complement fixation test showed no evidence of recent herpes simplex infection. A throat swab was negative for bacterial pathogens.

A provisional diagnosis of Wegener's granulomatosis was made and the patient underwent biopsy of the postnasal space and had a grommet inserted into the left tympanic membrane. Microscopic examination of the biopsy material showed granulation tissue with collections of pale histiocytes but no diagnostic features. Wegener's granulomatosis was still considered the most likely diagnosis and the patient was treated with azathioprine 150 mg daily and prednisolone 40 mg daily. One month later the patient's condition was unchanged, the diagnosis was reconsidered, and a further biopsy of the nasopharynx was undertaken. Histological examination showed nasopharyngeal tissue with a heavy lymphocytic infiltrate and containing giant cell histiocytic granulomas. There was no evidence of vasculitis and stains for acid fast bacilli were negative. Again the histological appearances were thought to support the diagnosis of

Wegener's granulomatosis and the immunosuppressive drugs were continued.

Four months later the patient was readmitted to hospital with drenching night sweats and a cough productive of scanty yellow sputum. On physical examination she was unwell with a pyrexia of 38°C. She had a tachycardia and tachypnoea and again had severe granular inflammation of the nasal mucosa and nasopharynx. There were no focal signs on auscultation of the chest. Fundoscopic examination of the eyes showed multiple small foci of choroidoretinal atrophy suggestive of individual tubercles. A chest radiograph showed bilateral miliary shadowing. The haemoglobin concentration was 0.099 g/l, the white blood cell count $2.2 \times 10^9/l$ (72% neutrophils, 20% lymphocytes), and the ESR 17 mm in one hour. Sputum examination was negative for pathogens, including acid fast bacilli. Fibreoptic bronchoscopy was performed and patchy inflammation was noted throughout the bronchial tree, which contained small amounts of mucopus. Bronchial washings were positive for acid fast bacilli (the Ziehl Neelsen stain being used) and mucosal biopsy specimens showed granulomatous inflammation and occasional acid fast bacilli. *Mycobacterium tuberculosis* was subsequently cultured from these specimens.

The patient was treated with isoniazid, rifampicin, and pyrazinamide and began to feel better within 48 hours. One month later she was back to normal health and the chest radiographic appearances were again normal. The inflammation of her nasal passages and nasopharynx had completely resolved, although she has been left with a chronic perforation of the left tympanic membrane and conductive deafness of the left ear.

In view of the unusual nature of this case we re-examined the original biopsy material from the postnasal space and took further sections from the tissue blocks. When stained by the Ziehl Neelsen method one section showed a tiny fragment of necrotic tissue with large numbers of acid fast bacilli.

Discussion

We believe that the demonstration of acid fast bacilli and giant cell histiocytic granulomas in a biopsy sample from this patient's postnasal space, together with the complete resolution of nasal inflammation after antituberculous chemotherapy, suggests that the correct diagnosis in this patient was tuberculosis of the nasopharynx. The incorrect diagnosis of Wegener's granulomatosis lead to inappropriate treatment with immunosuppressive drugs, thus permitting the development of miliary tuberculosis.

Tuberculosis of the nasal mucous membranes and naso-

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pharynx is a very rare condition and we are not aware of any recent reports on the subject. In 1931 Havens¹ reported five cases of primary tuberculosis of the nasal mucous membranes seen at the Mayo Clinic and was able to review 15 other cases cited by American authors from 1893 onwards. Of these 20 patients, 16 were women and four men. The mean age was 46 years (range 15–73) and the most common mode of presentation was nasal obstruction secondary to granular masses in the septum or nasal turbinates. In his book *Tuberculosis of the Upper Respiratory Tracts* Ormerod reviewed several cases of tuberculosis of the nasal passages from the European literature.² He too found a preponderance of female patients and reported that in most the disease occurred primarily in the nose, with no evidence of tuberculosis in the lungs.

We feel that the case we describe is directly comparable with those cited by Havens and Ormerod, although our patient appears to have had more advanced disease that affected the whole of the nasopharynx with obstruction of the eustachian tube. It is not possible to distinguish between primary and postprimary tuberculosis in this patient as her tuberculin sensitivity on presentation is not known and her

initial chest radiograph showed no evidence of old tuberculosis.

Our patient therefore differs from most patients with upper respiratory tract tuberculosis, in whom the infection usually occurs as a result of expectorating heavily infected sputum from extensive pulmonary disease.³

This case emphasises that unusual forms of tuberculosis still occur and that the diagnosis should always be considered in cases where the histological appearances suggest granulomatous disease.

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References

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