A rare cause of unilateral hearing loss

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A 54 year old white male first presented in 1978 to the medical department of the hospital with a six month history of shortness of breath and productive cough. His symptoms became progressively worse and necessitated two hospital admissions. He had vitiligo for 14 years and hereditary ichthyosis. He was a non-smoker and he worked in a chemical factory with lacquers and thinners.

Examination revealed both inspiratory and expiratory wheeze and a few crepitations at the right lung base. Abdominal examination showed a palpable spleen of about 8 cm and a palpable liver of about 6 cm below the costal margin. Investigation confirmed angiotensin converting enzyme of 72 IU (normal range 10–65). The chest radiograph is shown in fig 1 and the respiratory function showed a mixed obstructive and restrictive picture. Bronchoscopy revealed hyperaemic mucosa which was irregular with white “cobblestoning” and there was narrowing of both bronchi compatible with endobronchial sarcoidosis. The Kveim test was positive and lymph node biopsy confirmed sarcoidosis. He had maintenance doses of steroids varying between 5 mg and 25 mg daily for 18 years until remission of symptoms about two years before.

He was referred to the ear, nose, and throat department in November 1997 with deafness in the left ear which followed an ear infection. Examination of the ears showed a dull tympanic membrane on the left while the right side was normal. Pure tone audiometry showed mild bilateral high tone sensorineural hearing loss and an additional conductive loss in the left ear. The tympanogram showed type B tracing in the left ear. He had a left myringotomy and grommet insertion. Examination of the postnasal space was normal on visual inspection and palpation. A random biopsy was performed and tissue from the left side of the postnasal space revealed hyperplastic lymphoid tissue in which there were some active germinal centres. There were several discrete epithelioid granuloma containing Schaumann bodies including some Langhans type multinucleated giant cells. There was no caseous necrosis or special giant cells. Special stains failed to reveal any fungi or acid fast bacilli. There was no evi-

Figure 1  Chest radiograph showing bilateral hilar and widespread nodular shadows throughout both lung fields with a normal heart size.

Figure 2  This low power view of the postnasal lymphoid tissue shows the epithelial surface on the far left. Deep to this is a reactive lymphoid follicle and to the right and slightly below this follicle there is non-caseating epithelioid granuloma containing a darkly staining Schaumann body (haematoxylin and eosin × 40).

Figure 3  This medium power view shows in more detail the non-caseating granuloma and the Schaumann body. Such granulomas are typical of sarcoidosis and several such lesions were present within the biopsy (haematoxylin and eosin × 160).
dence of malignancy and the histological finding was in keeping with sarcoidosis (figs 2 and 3).

Questions
(1) What is the differential diagnosis?

(2) What does the chest radiograph show?

(3) What is the cause of deafness in the left ear?

(4) What are the otolaryngological manifestations of sarcoidosis?

A difficult psychiatric patient

Max J Henderson

A 74 year old man was admitted to a psychiatric ward at the request of his community psychiatric nurse. He arrived unaccompanied by either his nurse or his family. The history in the community psychiatric nurse’s letter was that the patient’s family had been concerned for the past week as he had taken to his bed. They had needed to wash and feed him. He slept a lot. They had alerted the nurse who, having seen the patient at home, arranged for an urgent admission.

From the old medical notes it was clear the patient had a long psychiatric history dating back 40 years. His initial diagnosis was obsessive-compulsive disorder but the majority of his admissions had been for agitated depression. He had taken at least one overdose in the past. There was a documented history of alcohol abuse, but it was not clear if this was still an issue. More recently he had developed idiopathic Parkinson’s disease. Cognitive impairment had been noted on his last admission: computed tomography had showed cerebral atrophy and some small infarcts and he had been started on aspirin. His medication which accompanied him on admission also included paroxetine, lithium, and co-beneldopa (Madopar).

Very little history was available from the patient, who needed to be roused from sleep. He said he felt “terrible” but could not elaborate. He denied any pain. He admitted being sleepy. No clear psychotic features were noted but it was questioned whether or not the patient understood.

Examination of the patient’s cardiovascular and respiratory systems was unremarkable as was that of the abdomen. Neurological examination revealed normal cranial nerves. Parkinsonian features were clearly demonstrated with rest tremor and cogwheel rigidity worse on the left. The patient could walk but needed assistance and conclusions about his gait were not drawn. Reflexes were normal and symmetrical—plantars were both downgoing.

Initial results showed a normal haemoglobin, a slightly raised white cell count \(12.3 \times 10^9/l\), normal urea, creatinine, and electrolytes, normal glucose on BM finger prick testing and normal urine dipstick.

Questions
(1) What is the diagnosis in this patient and what would you also consider?

(2) What particular risk factors for this condition were present in this patient?

(3) How is this condition normally managed?