CASE REPORT

Tuberculosis of the cavum revealed by acute facial pain

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SUMMARY

An 85-year-old woman presented for assessment of recurring episodes of intense hemifacial pain, mimicking trigeminal neuralgia, associated with tinnitus. A necrotic tumour of the cavum with compression of the left Eustachian tube and skull-base invasion was discovered on brain MRI. Although the tumour was initially thought to be malignant, the histopathological findings on the biopsy were compatible with tuberculosis, later confirmed by the cultures. F-18-fluorodeoxyglucose positron emission tomography (PET)/CT showed an intense signal of the cavum, cervical and mediastinal lymph nodes, and also of two small nodules of the apex of each lung. Currently, after 9 months of combined antituberculosis antibiotics, the initial lesion has almost disappeared from both PET scan and MRI. This case highlights the importance of systematically screening for tuberculosis in the assessment of nasopharyngeal tumours.

BACKGROUND

Primary tuberculosis of the nasopharynx is an exceptional condition. However, various clinical presentations have been described, especially in developing countries, such as sore throat, running nose, progressive deafness of one ear or otalgia.^{1 2} On imaging, the lesions can easily be mistaken for malignancies.

CASE PRESENTATION

An 85-year-old woman of north African origin presented to the short stay geriatric ward, for the assessment of paroxysmal episodes of intense left-sided facial pain. Notably, she had a history of insulin-requiring type 2 diabetes, chronic anxiety and hypertension. She never suffered migraine. She had no history of tuberculosis and recalled no such case in her family or in her circle of relatives. She was born in Algeria and moved to France in the 1960s. She had received a BCG vaccine during her childhood.

The first symptoms occurred in February 2013, with a unique episode of spontaneous intense pain following the territory of the maxillary nerve (V2). The pain affected the left sinus and cheek, and also irradiated behind the eye, with a feeling of stabbing and burning. According to the patient, this first episode lasted approximately 3 h and resolved completely. In September 2013, however, several relapses, lasting from 1 to 4 h, led to her hospitalisation. At that time, she also described mild hearing loss and tinnitus in her left ear. On examination, she had no local hyperaemia of the skin, no sweating, no otorrhea and no signs of facial palsy.

The neurological examination was normal, especially regarding the other cranial nerves. She had no fever, but felt chronic tiredness, and a mild loss of weight was noted (2 kg in a few months). There was no cough, dyspnoea or pulmonary sputum.

INVESTIGATIONS

MRI revealed a necrotic tumour of the cavum with compression of the left Eustachian tube and skull-base invasion (figure 1), initially thought to be cancerous.

The tumour appeared as submucosal on rhinofibroscope. There was no laryngeal lesion. Otoloscopy revealed serous mucous otitis, explained by the compression of the Eustachian tube. A biopsy of the tumour was performed for histological analysis. The results were in favour of a mycobacterial infection, with cellular architecture in the shape of an epithelioid and gigantocellular granuloma, but without caseous necrosis. There was no acidoalcohol-resistant bacillus on direct examination, but the PCR, and later the cultures, confirmed the diagnosis of infection by mycobacterium tuberculosis.

F-18-fluorodeoxyglucose (FDG) positron emission tomography (PET)/CT (figures 2 and 3, panel C) imaging showed an intense FDG uptake of the cavum, cervical and mediastinal lymph nodes, and also of two small nodules of the apex of each lung. These nodules were visible on CT scan but not on the initial chest X-ray (figure 3, panels A and B). This was in favour of concomitant pulmonary infection, even though the patient was asymptomatic.

A pulmonary fibroscopy with bronchoalveolar lavage was also performed, with negative results both on direct examination and cultures.

DIFFERENTIAL DIAGNOSIS

Regarding the facial pain, the patient had no history of essential trigeminal neuralgia. The painful paroxysms could last up to 4 h and there was no trigger zone. This was, therefore, more in favour of secondary rather than essential neuralgia, which was corroborated by the findings on MRI. Moreover, the symptoms rapidly and completely disappeared under antituberculosis therapy.

On the basis of the initial MRI (figure 1) and F-18 FDG PET/CT (figure 2, panel A), a neoplasm of the cavum was the primary hypothesis. After the pathology results, however, tuberculosis became the most likely diagnosis. However, nasopharynx malignancies have previously been described to be potentially associated with tuberculosis.³ The

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Figure 1 Coronal (A) and sagittal (B) T1-weighted MRI showing a necrotic tumour in the cavum, with peripheral gadolinium enhancement (arrows), compression of the left Eustachian tube and skull-base invasion.

disappearance of the necrotic tumour under antituberculosis therapy allowed excluding an underlying cancer.

Finally, we were unable to assess on potential complication of meningitis, as the patient was reluctant to undergo a lumbar puncture. Given that she had no neurological sign other than the facial pain, this hypothesis remains unlikely.

TREATMENT

As soon as the bacteriological samplings were sent to the laboratory—without waiting for results of the cultures—the patient was placed under four associated antituberculosis antibiotics: rifampicin, isoniazid, pyrazinamide and ethambutol. At 2 months, as the evolution was favourable, and as multisusceptible mycobacterium tuberculosis grew in cultures, pyrazinamide and ethambutol were withdrawn. The treatment was well tolerated. The antimycobacterial therapy was stopped at month 9, as the clinical evolution and imaging were favourable.

OUTCOME AND FOLLOW-UP

The facial pain symptoms rapidly disappeared after starting the combined antibiotics. After 9 months of follow-up and treatment, only the mild left tinnitus persisted. The MRI performed at the end of the treatment showed that the necrotic tumour had been washed out. F-18 FDG PET/CT performed at month 9 also showed significant improvement, with a total disappearance of the F18 uptake (figure 2, panel C). No relapse occurred during the year of follow-up after treatment discontinuation. The patient is doing well and experiences only very mild recurrent serous otitis.

Figure 2 F-18-fluorodeoxyglucose positron emission tomography/CT performed before (A), 2 months after (B), and at the end of antituberculosis therapy (C), showing a high level of F-18 uptake by the tumour, which progressively decreased and disappeared with the antituberculosis therapy (the level of F-18 uptake was determined by the SUV_{max} , as indicated).

Figure 3 (A) Chest X-ray showing only mild perihilar congestion. (B) Axial chest CT scan showing two small nodules in the apex of each lung and (C) F-18-fluorodeoxyglucose positron emission tomography/CT revealing a hyperintense signal of two pulmonary nodules and mediastinal lymph nodes.

DISCUSSION

Nasopharyngeal and/or cavum tuberculosis is an exceptional condition, even in endemic countries.^{2–5} Among the extrapulmonary localisations of tuberculosis, upper airway is involved in <2% of cases. As for the nasopharynx, the estimation lies around 0.1% only.^{2–6} However, the number of case reports has recently been increasing, probably due to improved diagnostic techniques.⁷ This strongly suggests that this entity could be underdiagnosed, especially in countries where mycobacterial infections are less frequent.

Mechanisms for primary nasopharyngeal infections remain uncertain. The nasopharyngeal involvement could occur due to direct mucosal infection after inhalation of the bacilli or because of reactivation of dormant acid-fast bacilli in the adenoids.⁸ In this case, the infection could well have been secondary to a primary pulmonary infection that had gone unnoticed.

Various clinical presentations can be seen. There could be a female predominance.¹ Most frequently, patients present with a sore throat, running nose, nasal obstruction, postnasal drip, epistaxis or chronic cough.² Progressive deafness of one ear, otalgia or tinnitus have also been described.^{1–9} The diagnosis can often be made through the assessment of a cervical lymph node.^{1–10} Patients can sometimes be asymptomatic¹¹ or present with atypical symptoms such as isolated snoring,¹² maxillary sinusitis¹³ or even diplopia.⁸ To the best of our knowledge, this diagnosis had never previously been considered as a potential differential diagnosis for trigeminal neuralgia.

Essential and secondary (or atypical) trigeminal neuralgias differ both by their clinical presentation and causes. Essential neuralgia is typically described as unilateral, recurrent, sudden and intense ‘flashes’ of facial pain with a feeling of stabbing, burning or ‘electric shock’, lasting a few seconds and often triggered by the stimulation of a well-recognised zone on the face.¹⁴ Secondary neuralgia mostly leads to a dull, continuous pain with intense and more prolonged paroxysms of several hours.^{14–15} The possible causes of trigeminal neuralgia are numerous: vascular, inflammatory, neoplastic, malformative or post-traumatic.^{14–15} Any lesion on the pathway of the fifth cranial nerve, from the sensory nuclei up to the skin, can be responsible for secondary neuralgia.¹⁵ In this case, regarding the size of the initial lesion and skull-base invasion, it is likely that the facial pain was caused by a compression of the trigeminal nerve or branches.

The diagnosis of extrapulmonary localisations of tuberculosis can be difficult. Infections of the upper airway are usually seen in conjunction with primary pulmonary tuberculosis, but the pulmonary symptoms can be very subtle, or even absent.² Therefore, such localisations can easily be confused with malignancies.¹ Co-existence of tuberculosis with a confirmed malignancy is also possible.³

MRI commonly shows a mass or a diffuse mucosal thickening of intermediate signal intensity on T1 and T2-weighted

sequences, with moderate contrast enhancement on T1 images.¹⁶ The radiological aspect can also mask an angiofibroma.⁵ The diagnosis cannot be affirmed on imaging alone and requires a biopsy for histopathological examination.

The risk of relapse after treatment is estimated at around 1%, due to the emergence of multiresistant bacilli.⁴ Treatment compliance is the cornerstone of clinical success, but the duration of the antimycobacterial therapy is unknown in such localisation. In our case, the evolution was favourable after 9 months. The decision to stop the antimycobacterial treatment was facilitated by the disappearance of the tumour on MRI and F-18 FDG PET/CT. Six to nine months of therapy is probably enough to obtain a cure in most cases. More and more, F-18 FDG PET/CT is used in monitoring the response to antimycobacterial therapy in patients with extrapulmonary tuberculosis.^{17 18}

Although this was a complex and rarely encountered case, it is important for clinical practice to raise awareness on this misleading condition, and to systematically screen for tuberculosis in the assessment of nasopharyngeal and/or cavum tumours.

Learning points

- ▶ Nasopharyngeal and/or cavum tuberculosis is an exceptional condition, and is probably underdiagnosed.
- ▶ Nasopharyngeal and/or cavum tuberculosis can be revealed by secondary trigeminal neuralgia with facial pain, hearing loss and tinnitus due to Eustachian tube compression and skull-base invasion.
- ▶ Tuberculosis of the cavum with skull-base invasion can easily be mistaken for malignancies. MRI is insufficient to make the distinction.
- ▶ Monitoring the therapeutic response with F-18-fluorodeoxyglucose positron emission tomography/CT could be useful in patients with nasopharyngeal and/or cavum tuberculosis.

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