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A case of meningitis due to *Achromobacter xylosoxidans denitrificans* 60 years after a cranial trauma

Authors' Contribution:

- A Study Design
- B Data Collection
- C Statistical Analysis
- **D** Data Interpretation
- **E** Manuscript Preparation
- F Literature Search
- **G** Funds Collection

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Summary

Background:

Achromobacter xy losoxidans (AX) is a non-fermentative aerobic Gram-negative bacillus. It is an opportunistic pathogen and the causative agent of various infections. We report an original case of late posttraumatic meningitis due to AX denitrificans.

Case Report:

An 83-year-old man was hospitalized for acute headache, nausea and vomiting. The emergency brain computer tomography (CT) scan did not reveal any anomaly. In his medical history, there was an auditory injury due to a cranial trauma incurred in a skiing accident 60 years earlier. Cytobiochemical analysis of the cerebrospinal fluid (CSF) revealed increased levels of neutrophils and proteins. The CSF bacterial culture was positive: the Gram stain showed a Gram-negative bacillus, oxidase + and catalase +, and the biochemical pattern using the API 20 NE strip revealed *AX dentrificans*. Late posttraumatic meningitis on a possible osteomeningeal breach was diagnosed even though the breach was not confirmed because the patient declined a second brain CT scan. The patient was successfully treated with meropenem.

Conclusions:

This report demonstrates the importance of searching for unusual or atypical organisms when the clinician encounters meningitis in a particular context, as well as the importance of adequate follow-up of craniofacial traumas.

key words:

Achromobacter xylosoxidans denitrificans • elderly subject • late posttraumatic meningitis

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BACKGROUND

Achromobacter xylosoxidans (AX) is a non-fermentative aerobic Gram-negative bacillus, first described by Yabuuchi and colleagues [1]. It has been isolated from the normal flora of the ear and gastrointestinal tract in humans and from various hospital and environmental water sources [2]. Achromobacter xylosoxidans is an opportunistic pathogen and the causative agent of various, mainly nosocomial, infections [3,4]; indeed, in their analysis of 54 cases, Gómez-Cerezo et al found that 52 (96%) episodes were cases of nosocomial bacteremia [4]. The literature also describes cases of non-nosocomial infections due to AX, mainly in frail patients, such as respiratory tract infection in cystic fibrosis patients [5]. Whether the AX infection is nosocomial or not, it usually occurs in immunocompromised patients, such as patients with neoplasms, diabetes mellitus, advanced HIV infection, chronic renal failure treated with hemodialysis, chronic obstructive pulmonary disease, liver cirrhosis, or cystic fibrosis [4,6]. Achromobacter xylosoxidans is frequently misidentified by routine laboratory tests, thus seriously compromising control measures related to epidemiology studies, and is often highly resistant to various commonly-used antibiotics [7]. We report an original case of late posttraumatic meningitis due to AX denitrificans in an elderly man.

CASE REPORT

An 83-year-old man was hospitalized for acute headache with nausea and vomiting. The emergency brain computer tomography (CT) scan performed at admission did not reveal any anomaly. His medical history consisted of prostatic adenocarcinoma successfully treated by total prostatectomy and hormone therapy, atrial fibrillation, epilepsia, and hypercholesterolemia. He had been treated with captopril, hydrochlorothiazide, atorvastatin, fluindione, leuprorelin and topiramate. During a detailed interview, the patient reported auditory injury due to a cranial trauma incurred in a skiing accident 60 years earlier. Clinical examination found general weakness, nausea, morning vomiting and temporal headache. His spouse reported that the patient had suffered from paroxysmal otorrhea, but was unable to provide details (how long, appearance). There was no fever. Apart from sleepiness, there were no neurological signs and the rest of the examination was normal. Biological screening showed an increase in C-reactive protein at 50.5 mg/L (normal <3.2), fibrinogen at 4.9 g/L (normal 2-4), erythrocyte sedimentation rate at 26 mm/h, and neutrophils at 16 200 cells/mm³ (normal 1800–7500). Hepatic and pancreatic enzymes were not elevated.

The lumbar puncture, performed because of the clinical features and inflammatory syndrome, showed cloudy but non-purulent cerebrospinal fluid (CSF). Cytobiochemical analysis of the CSF revealed an increase in levels of neutrophils, to more than 10000 cells/mm³, and proteins at 5.61 g/L (normal 0.12–0.6). After 1 night of incubation in an atmosphere of 10% carbon dioxide, the CSF bacterial culture was positive on blood agar and chocolate agar plates (approximately 15 colonies each). The bacterium was identified using standard methods: the Gram stain showed a Gram-negative bacillus, oxidase + and catalase +, and the biochemical pattern using the API 20 NE strip (bioMerieux,

France) revealed AX dentrificans. Unfortunately, the otorrhea was not observed during the hospitalisation of the patient and analysis of the discharge was therefore not possible. Late posttraumatic meningitis due to AX denitrificans on a possible osteomeningeal breach in the context of auditory injury after a skiing accident 60 years earlier was diagnosed, even though the breach was not confirmed because the patient declined a second brain CT scan. Antibiotherapy with meropenem at 2 g/8 hours was immediately started for a period of 15 days. After this treatment, the general weakness, nausea, morning vomiting, temporal headache, sleepiness, and the inflammatory syndrome vanished, with normalization of the cytobiochemical analyses and bacteriological culture of CSF. The patient received anti-meningococcal, anti-pneumococcal and anti-haemophilus influenzae vaccinations before returning home as an autonomous elderly individual.

DISCUSSION

Achromobacter xylosoxidans has been isolated from CSF, as well as from blood, respiratory secretions, skin, wounds, peritoneal fluid, ear discharge, bones, joints, endocardium, and central venous catheters [8]. However, only a few cases of meningitis (clinical features) related to AX have been published previously [9]. These cases of AX meningitis described in the literature were mainly nosocomial (catheter) [10] or reported in newborns or young children [11–14], immunocompromised subjects [15] or patients with an obvious lesion (extensive burns) [16]. None of these patients was elderly (age >75 years). To our knowledge, our report is the first case of meningitis due to AX denitrificans in an elderly man with no severe comorbidities.

The discovery of *AX denitrificans* in the CSF culture of this patient was not due to contamination during the lumbar puncture. This has been demonstrated by the microbiological laboratory of our hospital in a series of blood culture contaminations [17]. This non-contamination of the CSF is supported by the large number of colonies (15 colonies). In addition, the antibiogram showed that the strain had the same antibiotic resistance pattern as the reference strain, named ATCC15173, studied in a previous publication of the microbiological laboratory of our hospital [18].

Another original aspect of our report is the fact that the meningitis occurred on a possible osteomeningeal breach subsequent to a cranial trauma 60 years ago. Though the literature contains a few case reports of meningitis on a breach several years after the trauma, the delay between the cranial trauma and the occurrence of the meningitis was far less than 60 years in these observations [19,20]. Tissue atrophy and the subsequent changes in brain compliance with aging may have played a role in the reopening of the breach several years after the cranial trauma. Nonetheless, *AX denitrificans*, which is an environmental bacterium, could have entered via the auditory canal, reached a persistent osteomeningeal breach several years after the cranial trauma, and subsequently caused the meningitis.

Among the organisms that cause posttraumatic meningitis, *Streptococcus pneumonia* is the most frequent [19,20]. Acute rhinorrhea occurs in 50% of cases of posttraumatic meningitis [21]. CSF otorrhea can also be seen, as in our report [22].

Without treatment, posttraumatic meningitis could cause severe neurological complications and death; hence the interest in preventing them by actively searching for an osteomeningeal breach after cranial trauma. As for *AX* infections, all sites considered, the mortality rate ranges from 3% to 80% [8].

The management of meningitis following an osteomeningeal breach is based on antibiotherapy (trimethoprim-sulfamethoxazole, antipseudomonal penicillins, ceftazidime, cefoperazone, β -lactam/ β -lactamase-inhibitor combinations or carbapenems) [23] and surgery performed to prevent recurrences. In our report, the patient was treated with appropriate antibiotherapy (meropenem); but because he was opposed to surgery, he declined a second brain CT scan (the only brain CT scan performed at the admission did not reveal the osteomeningeal breach).

CONCLUSIONS

This report demonstrates the importance of searching for unusual or atypical organisms when meningitis occurs in a particular context, as well as the importance of adequate follow-up of craniofacial traumas. In addition, we emphasize the need for detailed questioning of the patient and/or his relatives.

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