Observation

A 59-year-old immunocompetent patient with no significant previous medical history was admitted to hospital with fever and right hemihypoesthesia. Brain computed tomography (CT) and magnetic resonance imaging (MRI) revealed multiple supra- and infratentorial bilateral lesions supposed to be secondary lesions. A thoracic CT scan was performed and revealed opacities in the inferior and middle right lung lobes. In hemoculture tests, \textit{Pseudomonas aeruginosa} was found. Cerebrospinal fluid (CSF) analysis showed no abnormalities. Complex empirical treatment with ceftazidime, ciprofloxacin and corticosteroids as well as antituberculosis treatment were started, but led to no clinical improvement. A week later, the patient was admitted to the intensive care unit of our hospital with right hemiplegia, increased tendinous reflexes, anisocoria and impaired consciousness (Glasgow score of 9). After intubation, brain MRI was performed (Figs. 1–3).
What is your diagnosis?

**Figure 1** T1-weighted gradient-echo contrast-enhanced axial image.

**Figure 2** T2-weighted fast spin-echo axial image.

**Figure 3** A. Diffusion B1000-weighted axial image. B. Apparent diffusion coefficient (ADC) mapping.

What is your diagnosis?
The brain MRI shows bilateral multiple infra- and supratentorial lesions located in the brain stem, vermis, cerebellum, basal nucleus, thalamus and semiolateral region. The lesions tend to conglomerate and show ring-like peripheral enhancement. The central necrotic core of the lesions shows low T1-weighted and high T2-weighted heterogeneous signal intensity. The core is surrounded by a T2-weighted hypointense rim associated with a large area of T2-weighted hyperintense peripheral edema, responsible for the major mass effect with transtentorial and subfalcine herniation. The lesions show high signal intensity on diffusion-weighted imaging (DWI) with a restricted apparent diffusion coefficient (ADC).

Brain hydrogen-1 MR spectroscopy (1H-MRS) shows very low N-acetyl aspartate (NAA) concentration, increased choline content, and a short TE (echo-time) peak at 1.3–1.5 ppm that persists in a long TE with no J-coupling. This peak is not characteristic of lactates or lipids and may correspond to an alanine peak.

The appearance of the multiple confluent microlesions, as described above, together with the restricted ADC are characteristic of multiple abscesses, whereas the conglomerated multicellular pattern is evocative of Nocardia infection.

Bronchoscopy, performed after the MRI findings, confirmed the diagnosis of Nocardia abscessus infection, and treatment with co-trimoxazole, amikacin and imipenem was initiated. The patient’s clinical condition improved, which allowed him to leave the intensive care unit a month later. Control MRI showed regression of the lesions and of the peripheral edema. No clear etiology of the infection was identified; however, it was suspected that the infection might be due to Nocardia farcinica, which was confirmed after analysis of a lung biopsy sample. The microbiological analysis revealed the presence of Nocardia farcinica, a rare species, which is part of the family Actinomycetaceae. Nocardia farcinica is aerobic, variably acid-fast, Gram-positive bacteria found in soil and in decaying vegetables. Human infection occurs mainly through inhalation or direct inoculation via the skin. Although Nocardia farcinica is a rare species, it now accounts for 1 to 2% of brain abscesses [1,2]. Nocardiosis most commonly occurs in immunocompromised patients who are defective in cell-mediated immunity. Nevertheless, a few cases of the infection have also been reported in immunocompetent patients [3]. In two-thirds of cases of Nocardia brain abscess, other organs — most commonly, the lungs and skin — are affected as well [1]. Nocardiosis usually results in immunosuppression and how can you eliminate them?

The major differential diagnosis is pyogenic abscess, which can also present with restricted diffusion, a hypointense rim on T2-weighted imaging and rim enhancement [2]. However, a multilocular pattern is not commonly seen in pyogenic abscesses. The peak on 1H-MRS, presumably attributed by us to alanine, is also observed in cases of pyogenic abscess, but is not considered characteristic of such abscesses [5].

Another differential diagnosis is brain tuberculoma. This may also have a multiple conglomerate character with contrast ring enhancement; however, it usually presents with a high ADC and is associated with arachnoiditis. Neither of these characteristic features was observed in the patients here.

Multiple brain metastases can also show rim enhancement with perilesional edema, but they usually have a high ADC, which, in our case, was relatively low. Hemorrhagic metastases can result in restricted ADC. Indeed, there have even been a few reports of a hypointense rim on T2-weighted sequences in such metastases. It should be noted, however, that these rims were only partly enhanced [6].

Several cases of brain tumor misdiagnosis have also been reported [3]. The distinction between brain tumor and brain abscess is essential. Indeed, in the case of cerebral tumor, corticosteroids can help to reduce the cerebral inflammation and, consequently, the edema, whereas, in the case of abscess, corticosteroid treatment would result in its rapid expansion [3,7].

In our patient, 1H-MRS demonstrated decreased NAA and increased choline peaks. However, such a pattern is non-specific and may also be observed in cases of either pyogenic abscess or metastases.

In conclusion, our case study demonstrates that abscesses due to Nocardia spp infection may be seen in immunocompetent patients. Early treatment is mandatory to avoid a severe clinical outcome. To make an early diagnosis, the multilocular pattern of the brain abscesses should be considered as suggestive of Nocardia origin.

Conflicts of Interests

No potential conflicts of interests relevant to this article were reported.

References


