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Letter to the Editor

Stenotrophomonas maltophilia brain abscesses after implantation of motor cortex stimulator

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ABSTRACT

We describe the first case of a patient with brain abscesses caused by *Stenotrophomonas maltophilia* as a complication after motor cortex stimulator implantation. Brain abscesses pose a challenge in diagnosis and treatment, because microbiological diagnosis is not always achieved, antibiotic drugs may not penetrate well into the CNS and some bacteria have resistances to typical empirical antibiotic drugs. In this case diagnosis was only made after removal of the stimulator and a long term treatment with antibiotic drugs was necessary. As neurostimulation devices become more common, formerly rare bacteria may become a more common complication. Bacteria with biofilm properties and a problematic resistance spectrum like *Stenotrophomonas maltophilia* should be included in the differential diagnosis, because they will not respond to the typical empirical treatment.

Dear Editor,

Brain abscesses are central nervous system (CNS) infections that pose a challenge in diagnosis and treatment and should be a differential diagnosis in patients developing signs of CNS infection after neurosurgical intervention. Abscess sampling and microbiological diagnosis are imperative to steer treatment but bacterial diagnosis is only achieved in about 80% of patients [1]. Typical causative bacteria are streptococci, enterobacteriaceae, bacteroides spp. and staphylococci [1]. Calculated antibiotic treatment takes these bacteria into account. Uncommon bacteria may present a particular treatment challenge, especially when microbiological sampling is impeded by the limited accessibility of the neurostimulation device. Here we report on a patient who developed brain abscesses with *Stenotrophomonas maltophilia* after a cortical pacemaker for pain treatment was implanted.

The 74 year old man was admitted to our hospital with a worsening of his right sided hemiparesis. He had a long past medical history, pertinent was a left thalamic ischemia nine years prior and a subsequent development of a thalamic pain syndrome, which was refractory to medical treatment alone. Six months prior to the admission at our hospital, a motor cortex stimulator had been implanted subdurally over his left motor cortex (Medtronic Prime Advanced SureScan, electrodes 2× Surescan 3587A, 2× Surescan 37,083–60; Medtronic, Dublin, Ireland, last stimulation settings: 2.0 V, 450 µs stimulus length and 40 Hz frequency). These stimulators are used in the treatment of medically refractory pain syndromes [2] and are typically implanted epidurally, which was not possible in his case because of anatomical constraints. On the day after surgery he had his first right-sided clonic seizures. The seizures were thought to be procedure related, as they had started even before the full activation of the stimulator. The stimulation, once activated, was not judged as a factor for seizure precipitation. While he had received the stimulator at a different hospital, he was followed up at our hospital because of the proximity to his residence. Over the course of the next six months, despite antiepileptic drug treatment, his seizures did not subside at a frequency of 1–2/month and his preexisting mild post-stroke hemiparesis worsened from 4+/5 to 3–4/5. With magnetic resonance imaging (MRI) not possible due to the stimulator, repeated computed tomographies (CTs) were limited in their diagnostic value for the metal artifacts from the stimulator (Fig. 1A). Systemic inflammatory markers were not elevated, he never had fever and no meningeal signs. His thalamic pain syndrome had markedly improved with a 50–60% pain reduction.

After admission to our hospital, a CT scan showed a white matter hypodensity (Fig. 1B). Cerebrospinal fluid (CSF) analysis showed 7 cells/µl, elevated CSF protein (90 mg/dl) and normal CSF glucose. Over the next days he developed a status of clonic seizures of the right side, mainly of the face, the neck and the arm, causing dysphagia and eventually aspiration pneumonia. He was admitted to our neurological ICU and had to be intubated. A contrast enhanced CT now demonstrated a round, contrast enhancing structure (Fig. 1C), raising the suspicion of a cerebral abscess. Calculated antibiotic treatment for the initial pneumonia with piperacillin/tazobactam was adapted to meropenem and vancomycin. With the suspicion of intracerebral abscesses, the stimulator was removed after thorough discussion, because it meant the end of the successful treatment of his pain syndrome. Intraoperatively, a purulent infection was found subdurally with involvement of the adjacent cortical tissue and microbiological sampling was performed. Now, an MRI was possible and revealed several disseminated abscesses within the left hemisphere on the day following surgery, especially in the subcentral tissue (Fig. 1D). On the same day, *Stenotrophomonas maltophilia* was isolated from the samples. Antibiotic treatment was adapted to intravenous moxifloxacin and trimethoprim/sulfmethoxazole, the only two available antibiotics this strain was susceptible to. After eight weeks a first abscess size reduction was documented (Fig. 1E). Additionally, only then could the seizures be controlled. 64 days after initiating moxifloxacin and trimethoprim/sulfmethoxazole, the therapy could be stopped after repeated follow-up MRIs. The patient was seizure free, could be weaned of the ventilator and was transferred to a rehabilitation hospital, where he further improved and was able to stand, walk with a walker and had no speech impairment. His thalamic pain was less than before in the short term follow up.

Discussion

We describe the first case of brain abscesses caused by *Stenotrophomonas maltophilia*. In recent years, *S. maltophilia* has been increasingly reported as an infection especially on foreign material such as

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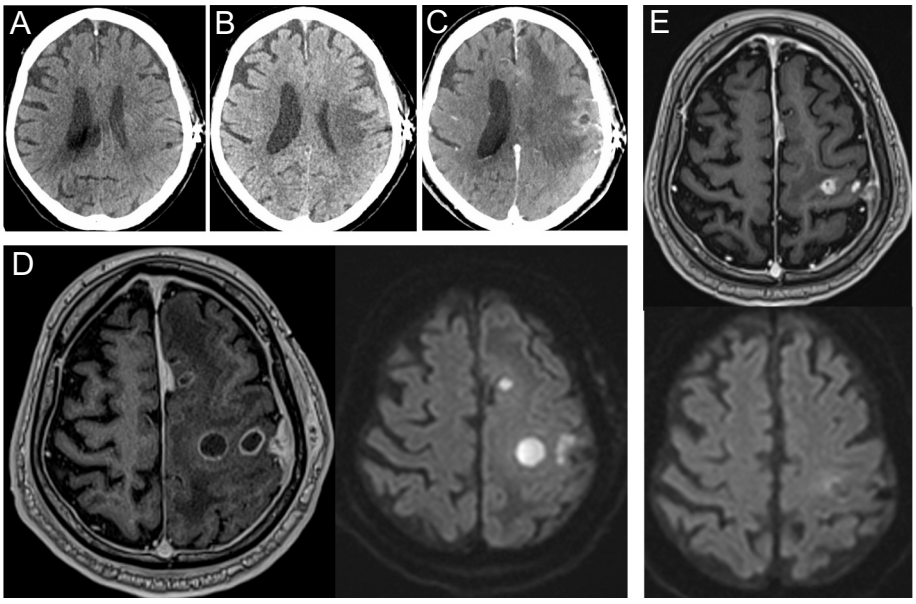

Fig. 1. Abscess imaging.

dialysis equipment [3] and cardiac pacemakers [4]. The adherence to foreign material is mediated by the biofilm production of *S. maltophilia* [4], and is of high importance when considering the increased use of neurostimulation devices for neurological and psychiatric indications. Between 1.2% and 9.4% of patients with intracranial stimulation devices develop infectious complications, which are mostly soft tissue infections, for example at the pectoral pouch of the device [5,6]. The stimulation devices may make diagnosis difficult in case artifacts obscure the relevant brain areas and when MRI, as in our patient, is not possible due to the stimulator. Additionally, the accessibility of the suspected lesion is limited for the intracranial part of the device and removing the neurostimulation device might spell the end to a successful treatment of the underlying condition. When the infection is located in the pectoral pouch for the device, microbiological sampling is considerably easier than intracranially.

When an abscess diagnosis is made or suspected, *Stenotrophomonas* should be an important differential diagnosis because of its biofilm abilities [4] and its problematic resistance spectrum, where the typical calculated antibiotic treatment for abscesses with meropenem and vancomycin/linezolid is ineffective and typically only moxifloxacin and trimethoprim/sulfmethoxazol and more rarely also ceftazidime and tigecycline are effective [7]. In closing, *Stenotrophomonas* and its limited antibiotic sensitivity should be considered as a differential diagnosis in patients with brain abscesses, especially in patients with foreign bodies like neurostimulation devices.

A) Cranial CT three months prior to admission: in sections not obscured by the artifacts from the more cranially positioned cortical stimulator, no clear pathology is visible. The stimulator leads are visible in the left subcutaneous tissue. B) Head CT upon admission: a white matter hypodensity is visible. C) Contrast enhanced CT on day 13: round contrast enhancing structures, suspicious of abscesses are documented. D) MRI on day 19: T1 contrast enhanced and diffusion-weighted imaging shows abscess structures in the left hemisphere. This axial section was obscured by metal artifacts from the stimulator on CT. E) MRI on day 64: Reduction of abscess size after long term treatment documented on T1 contrast enhanced and diffusion-weighted imaging.

Competing interests

None.

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