INTRODUCTION

Brain abscess is one of the most serious infections of the central nervous system. It consists of a focal purulent collection of the brain parenchyma.\(^2,15\) Despite notable improvement in outcome since the introduction of more powerful diagnostic methods and targeted antibiotics, currently the mortality rate is as high as 10%.\(^2,5,12,15\) The most common source of infection is the contiguous otorhinogenic area, while a dental origin is rather rare. Dissemination of microorganisms from the oral cavity microbiota is thought to be the underlying cause of infection in <10% of brain abscesses.\(^4,5,16,19\) Moazzam \textit{et al.} have recently published the largest review of orally originated intracranial infections, a total of 60 cases.\(^12\) The most common pathogens found were \textit{Streptococcus viridans} (55%) and \textit{Fusobacterium} spp. (20%). Conversely, \textit{Parvimonas micra} (Pm) was involved in <5% of these cases.\(^12\)
*Pm*, formerly known as *Peptostreptococcus micros* and *Micromonas micros*, is a Gram-positive anaerobic coccus that is normally found in the human flora of the oral cavity and gastrointestinal tract. This bacterium has generally been associated with polymicrobial infections of the oral cavity, whereas infections outside this area are rare, particularly in healthy people. Most such infections involve the spine (45%), followed by the joints, heart valves, and pleura. Brain abscesses caused by *Pm* are extremely rare. Through a systematic PubMed literature review using the search terms “brain abscess” combined with “odontogenic” or “dental” or “anaerobic” or “*Pm*” or “*P. micros*” or “*M. micros*” and by studying the reference lists of the articles collected, we have found only one similar case to ours in this report. Our patient, as well as one reported 10 years ago by Kwon et al., had a solitary brain abscess in which orally originated *Pm* was identified as the lone causative microorganism. We discuss the diagnostic and treatment strategies of these two cases in addition to those of nine patients with either multiple brain abscesses due to *Pm* monomicrobial infection or polymicrobial brain abscess including *Pm*. Odontogenic brain abscesses caused by *Pm* are life-threatening unless correctly managed by a multidisciplinary team including neurosurgeons, infectious-disease specialists, and maxillofacial surgeons.

**CASE DESCRIPTION**

A 62-year-old male presented to the hospital with a 1-week history of headache and an episode of transitory paresthesia in the left upper limb followed by a 2-min lapse in awareness and a claw-like left hand position highly suggestive of an atypical absence seizure. He had no history of fever, malignancy, diabetes mellitus, or corticosteroid use. Laboratory results revealed a normal white blood cell count and a moderately elevated C-reactive protein (63.6 mg/L—normal levels are below 10 mg/L—). The patient was alert and presented neither neurological deficits nor signs of meningeal irritation. An urgent brain computed tomography (CT) scan demonstrated an expansive process in the right parietal lobe [Figure 1a]. For better characterization a magnetic resonance imaging (MRI) was also performed, revealing a cystic cortico-subcortical lesion [Figure 1b]. Gadolinium injection demonstrated an irregular rim enhancement of the lesion that spread to the subarachnoid space. Restricted diffusion-weighted imaging MRI showing a bright signal within the lesion (restriction of water diffusion), low apparent diffusion coefficient value within the lesion fluid. (B4) MR spectroscopy showing a high lipid level (lip) and decreased N-acetylaspartate, (B5) gadolinium-enhanced T1-weighted MRI showing a low-intensity lesion with rim enhancement which extended to the subarachnoid space (arrow).

On day 3, the patient underwent craniotomy with neurophysiological monitoring given the proximity of the lesion to the motor cortex. After opening the dura mater, a swollen brain was found [Figure 2]. A catheter was first used to drain purulent fluid and reduce brain swelling. Then, the adjacent cortex was opened to wash the cavity with saline, antibiotics (vancomycin, and gentamicin), and hydrogen peroxide. Several tissue samples from the cavity were also taken for pathological study, which revealed acute inflammatory changes but no malignant cells. The blood cultures were negative. Likewise, Gram staining of the abscess fluid showed no bacteria. The presence of *Pm* was confirmed through DNA sequencing of the abscess material.

**Figure 1:** Preoperative neuroradiological studies. (A) Computed tomography scan showing an isodense 3-cm-parietal mass with surrounding digitiform edema, (B) preoperative magnetic resonance imaging (MRI). (B1) T2-weighted MRI showing a hyperintense lesion with significant perilesional edema, (B2) diffusion-weighted imaging MRI showing a bright signal within the lesion (restriction of water diffusion), (B3) low apparent diffusion coefficient value within the lesion fluid. (B4) MR spectroscopy showing a high lipid level (lip) and decreased N-acetylaspartate, (B5) gadolinium-enhanced T1-weighted MRI showing a low-intensity lesion with rim enhancement which extended to the subarachnoid space (arrow).

**Figure 2:** Intraoperative photographs. (A) The patient was placed in the lateral position, (B) following dura opening, a swollen brain was found with a whitish subarachnoid area (arrow), (C) a catheter was inserted at the point where the abscess capsule was closest to the cortex. Brownish liquid pus was aspirated for mass decompression and later microbiological study. (D) Surgical view following removal of the whitish subarachnoid area and cavity washing.
fluid exhibited no organisms, and routine aerobic/anaerobic cultures revealed no bacterial growth. Therefore, abscess samples were sent to another hospital for the amplification of bacterial 16S ribosomal RNA (rRNA) with polymerase chain reaction. When Pm was demonstrated as the causative agent of the infection, vancomycin was discontinued.

The patient was also assessed by maxillofacial surgeons who detected severe periodontitis with infected mandibular cysts. This condition was considered the primary source of brain infection, and 1 week after craniotomy the patient underwent extraction of the lower teeth and curettage of mandibular cysts. Unfortunately, the material of the extracted teeth was not sent for microbiological studies. The patient was discharged in excellent conditions after 3 weeks of intravenous antibiotics, followed by 1 week of oral metronidazole and moxifloxacin. Oral antibiotics were maintained for 5 additional weeks. Follow-up MRIs demonstrated favorable progress without any radiological suspicion of recurrence [Figure 3].

**DISCUSSION**

The seriousness of brain abscesses requires prompt joint action by neurosurgeons and infectious-disease specialists. Merely focusing on the focal suppurative process of the brain parenchyma is not enough for a successful treatment. It is paramount to identify and eradicate the primary source of infection and the specific causative microorganism. In the case presented, Pm isolated in the brain abscess was assumed to have an oral origin based on the evident signs of dental infection. Abscess location in the parietal lobe supports that hematogenous dissemination is the major pathophysiological mechanism of brain abscess of odontogenic origin.[12] Our case highlights the importance of careful clinical and radiological examination of the maxillofacial area whenever other infective sources are not detected before defining as unknown the origin of intracerebral abscesses.[9] When obvious signs of dental disease such as periodontitis or caries are lacking, a history of dental procedures, particularly tooth extraction performed 1–4 weeks before the onset of intracranial infection, should also be considered.[5,7,9] Even poor oral hygiene alone has proven enough to lead to brain abscesses.[6]

After a thorough literature review, we found only ten previous cases of brain abscesses in which Pm, an organism typically found in the human oral flora, was isolated [Table 1]. Among the cases with a known source of infection, a dental origin was reported for all but one case with esophageal pleural fistula.[1,8,10,13] It is remarkable that all the patients in this series were in good general health, except for one with an esophageal carcinoma. In agreement with a recent systemic review of infections caused by Pm, this case series of brain abscesses sustains that most Pm infections occur in a polymicrobial environment.[3] Nevertheless, in our patient and three others in this series, Pm was the only isolated microorganism [Table 1]. It is worth mentioning that standard bacterial culturing was ineffective to detect Pm in these four cases, and the authors had to use gene amplification of bacterial samples and sequencing technologies to identify the pathogen.[8,10,18] Our report supports the diagnostic value of 16S rRNA to detect Pm in patients with culture-negative brain abscesses.[8,10,21] The application of matrix-assisted laser desorption/ionization-time-of-flight mass spectrometry has also proven useful to detect Pm.[7] The challenging identification of this strict anaerobe, particularly when antibiotic therapy was started before collecting the specimen, might have contributed to the infrequent reporting of brain abscesses caused by Pm.[9,12-14]

**Figure 3:** Postoperative magnetic resonance imaging (MRI) studies. Follow-up T2-weighted and gadolinium-enhanced T1-weighted MRI demonstrated progressive brain edema reduction, shrinkage of the residual cavity, and disappearance of contrast enhancement areas.
### Table 1: Summary of demographic, clinical, bacteriological, and treatment features of the patients with brain abscesses caused by *Parvimonas micra*.

| No. | Author, year [Reference] | Age, sex | Source of infection | Predisposing factors | Symptoms | Consciousness impairment | Surgical drainage | Isolated bacteria | Method of bacterial identification | Antibiotic therapy (route, duration) | Outcome (Follow-up) |
|-----|--------------------------|----------|---------------------|----------------------|----------|--------------------------|------------------|------------------|--------------------------|--------------------------|------------------|
| 1.  | Murdoch et al., 1988 [14] | 41 M     | Not found           | NA                   | Headache, vomiting  | No        | Yes                     | Abscess cultures         | Peptostreptococcus micros, Bacteroides ureolyticus, Fusobacterium, Streptococcus milleri | NA                       | NA               |
| 2.  | 6 M                      | Not found| NA                   | NA                   | Vomiting            | No        | Yes                     | Abscess cultures         | Peptostreptococcus micros, Streptococcus milleri | NA                       | NA               |
| 3.  | 32 F                     | NA       | NA                   | NA                   | Headache            | No        | Yes                     | Abscess cultures         | Peptostreptococcus micros, Streptococcus milleri | NA                       | NA               |
| 4.  | Mueller et al., 2009 [13] | 50 M     | Tooth extraction    | None                 | Weakness            | Yes (GCS 13/15)| Yes                     | Abscess cultures         | Fusobacterium nucleatum, Micromonas micros, Actinomyces meieri | Ceftriaxone, Rifampicin (IV, NA) | NA               |
| 5.  | 66 F                     | Not found| Esophageal carcinoma| Fever, headache, meningism | Yes (GCS 11/15) | Yes         | Abscess cultures         | Ceftriaxone, Rifampicin (IV, NA) | Fusobacterium nucleatum, Micromonas micros, Streptococcus oralis, Actinomyces turicensis | NA                       | NA               |
| 6.  | Kwon et al., 2009 [10]   | 49 F     | Periodontitis       | None                 | Fever, headache     | No        | Yes                     | 16 S rRNA (brain abscess) | Parvimonas micra | Ceftriaxone, Isemapicin, Metronidazole (IV, 4 wk)+Ceftriaxone (IV, 3 wk) | Good (2 m)               |
| 7.  | Vishwanath et al., 2016 [23] | 30 M | NA                   | None                 | Headache            | Yes (drowsiness) | Yes (stereotactic aspiration) | Abscess cultures         | Streptococcus spp, Fusobacterium nucleatum, Parvimonas micra | Vancomycin, Metronidazole, Amikacin (IV, NA) | Good (hospital stay) |
| 8.  | Akashi et al., 2017 [1]   | 68 M     | Periodontitis       | None                 | Fever, weakness     | No        | Yes (stereotactic aspiration) | Abscess cultures         | Streptococcus constellatus, Fusobacterium nucleatum, Parvimonas micra | Ampicillin and Metronidazole (IV, 6 wk) | Good (1.5 m) |

(Contd...)
Optimal treatment of intracranial *Pm* infections remains uncertain.\(^{[3,12]}\) Our patient, as well as all cases with a solitary brain abscess, underwent prompt surgical drainage \([\text{Table 1}]\). Nevertheless, antibiotic therapy alone might be a safe and suitable option for patients with small lesions, particularly when they are multiple and located deep within the brain.\(^{[17,18]}\) There are even more doubts regarding the most appropriate antibiotic regimen. Penicillin, amoxicillin (±clavulanic acid), piperacillin (±tazobactam), cefoxitin, ceftriaxone, imipenem, meropenem, ciprofloxacin, clindamycin, and metronidazole have all been found effective for treating *Pm*.\(^{[9,11]}\) Despite some *Pm* strains being resistant to metronidazole,\(^{[22]}\) antibiotic therapy for most *Pm* brain abscess cases involved the simultaneous use of ceftriaxone and metronidazole \([\text{Table 1}]\). All cases had a favorable outcome except a patient who discontinued antibiotic treatment after 2 weeks due to financial difficulties.\(^{[8]}\) Nonetheless, the simultaneous use of more than one antibiotic precludes ascertaining which one played the most determinant role in the favorable response. Finally, the greatest difference among the cases reported is the duration of the antibiotic therapy, which ranged from 6 to 12 weeks \([\text{Table 1}]\). Bearing in mind that successful patient outcome depends on long-term antibiotic treatment, its final duration should be individually based on strict clinical and neuroimaging follow-ups.

**CONCLUSIONS**

We report the successful diagnosis and treatment of a solitary brain abscess caused by *Pm* in a patient with severe periodontal infection. This case highlights the potential risk of untreated dental infections, which may lead to life-threatening brain abscesses even in healthy patients. This report also supports that 16S rRNA analysis is a valuable technique to detect *Pm* in cases with culture-negative brain abscesses.

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**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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