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*Case Report*

# Intracranial infection caused by *Mycoplasma hominis* after neurosurgical operation: an easily overlooked but serious condition

Ying Liu[1], Jiong Tang[2], Yirong Wang[1], Fenfang Cui[1], Yan Yang[1]

*[1] Department of Pharmacy, The Third People's Hospital of Chengdu, Affiliated Hospital of Southwest Jiaotong University, Chengdu, China*
*[2] Department of Pharmacy, Chengdu Seventh People's Hospital, Chengdu, China*

## Abstract

Introduction: *Mycoplasma hominis* (*M. hominis*) is a commensal that mainly colonizes in the microflora of the genitourinary tracts and is associated with urogenital tract infections. There are reports of central nervous system (CNS) infections in neonates caused by *M. hominis*. Nevertheless, *M. hominis* CNS infections in non-neonatal patients are extremely rare. Herein, we have reported a case of a man who suffered from intracranial infection secondary to *M. hominis* after neurosurgical operation. Additionally, we reviewed the relevant published literature to raise awareness on such infections and highlight the importance of proper treatments.

Case presentation: A 68-year-old man underwent emergence craniotomy for intracerebellar hemorrhage. He presented with a moderate fever unresponsive to piperacillin-tazobactam on the seventh day after the surgery. His body temperature continued to increase, and he presented with signs of CNS infection. The antimicrobial therapy was switched to meropenem and vancomycin. No obvious reduction in the body temperature was observed. The cerebrospinal fluid (CSF) obtained previously revealed tiny point colonies which were morphologically consistent with *M. hominis* and subsequently confirmed by metagenomic next-generation sequencing (mNGS). Thus, *M. hominis* induced intracranial infection was diagnosed, and a combination therapy with moxifloxacin and minocycline was implemented. Fortunately, the patient's body temperature decreased to normal range after effective antibiotic therapy.

Conclusions: Based on the lesson of our case and a thorough review of published literature, the possibility of *M. hominis* induced CNS infections after neurosurgical intervention should not be ignored, especially when there is no response to standard antimicrobial therapy.

**Key words:** *Mycoplasma hominis;* CNS infections; neurosurgical operations; case report; review.

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## Introduction

*Mycoplasma hominis* (*M. hominis*) is a commensal which mainly colonizes in the microflora of the genitourinary tracts, and has a limited distribution in the oral cavity and respiratory tract mucosa of healthy individuals [1]. It can be an opportunistic pathogen associated with urogenital tract infections, such as cervicitis, urinary tract infection, pelvic inflammatory disease and post-abortal fever [2], but extra-urogenital infections secondary to *M. hominis* have rarely been reported. Several cases of central nervous system (CNS) infections due to *M. hominis* have been described in premature neonates, which may be associated with either intrauterine infection or vertical transmission during birth [3–5]. However, *M. hominis* induced CNS infections among non-neonatal patients are exceedingly rare.

It is challenging to identify *M. hominis* caused CNS infections in time. Firstly, *M. hominis* is an atypical pathogen which lacks a rigid cell wall that prevents detection by Gram staining and clear observation by routine light microscopy. Besides, the lack of cell wall makes *M. hominis* innately resistant to antibiotics inhibiting the synthesis of cell wall [6]. Moreover, the growth of *M. hominis* requires special selective media and conditions, as well as prolonged incubation time; and they may be overlooked on standard culture media because of their small colony sizes and slow growth rate [7]. In addition, the primary pathogens causing CNS infections are generally Gram-negative and Gram-positive bacteria [8,9]. *M. hominis* may sometimes be considered as contamination other than pathogenic bacteria; and this results in a high rate of missed diagnosis. Therefore, early diagnosis and prompt treatment of CNS infections due to *M. hominis* are difficult to some extent.

Herein, we report a case of a 68-year-old man who suffered from intracranial infection caused by *M. hominis* after neurosurgical intervention for intracerebral hemorrhage, and was successfully treated

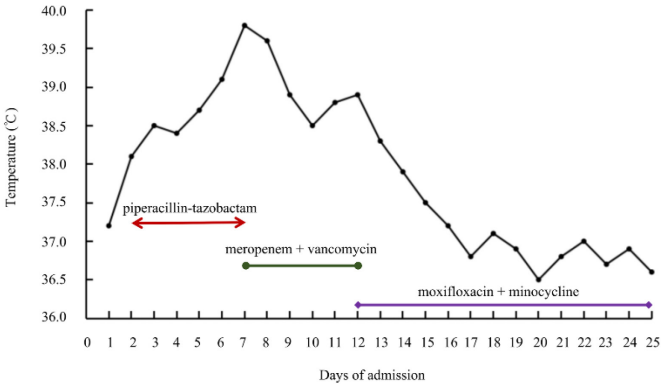
by moxifloxacin and minocycline. Additionally, we review the relevant English literature to elucidate the clinical manifestations, diagnostic methods and treatment approaches of *M. hominis* associated CNS infections, to increase the understanding of these easily overlooked infections and highlight the importance of early recognition and prompt treatment. We present this article in accordance with the CAse Report (CARE) reporting checklist (Supplementary Figure 1).

## Case presentation

A 68-year-old man with a history of hypertension and hyperlipemia developed a sudden onset of severe headache and nausea, followed by disturbance of consciousness. He was immediately sent to the local hospital on 18 December 2023. An urgent cerebral computed tomography (CT) scan indicated an intracerebellar hemorrhage rupturing into the ventricular system. He received emergence craniotomy and evacuation of intracranial hematoma, with an external ventricular drain (EVD) insertion on the same day. The patient received endotracheal intubation and urethral catheterization after surgery. However, an obstruction of the EVD tube was observed 2 days after the operation, and the new cerebral CT scan indicated an enlargement of the lateral ventricle and the drain insertion location was surrounded by extensive edema. As a result, the patient underwent a second debridement, and a new EVD was inserted for drainage.

The patient was then transferred to our hospital, on 23 December 2023, for further treatment. The patient had a Glasgow coma scale score of 6, and he was maintained on urethral catheterization and endotracheal intubation on admission. The patient presented with a moderate fever (38.1 ℃) on the second day after admission (Figure 1). Laboratory examination indicated that the white blood cell (WBC) count was 11.2 × $10^9$/L (normal range 4–10 × $10^9$/L), and the percentage of neutrophil count (Neut%) was 81.5% (normal range 40–75%). Cultures of peripheral blood, sputum, urine, and cerebrospinal fluid (CSF) were performed to exclude the underlying cause of fever. Meanwhile, piperacillin-tazobactam (4.5 g q8h) was commenced as empirical treatment for infection. Examination of the reddish CSF from the EVD tube indicated a slight

**Figure 1.** Change in the patient's body temperature and antibacterial regimens during hospitalization.




increase of protein and WBC (Table 1), which was regarded as a consequence of intracerebellar hemorrhage. On the fourth day after admission; the results of blood culture, urine culture and CSF culture were negative for bacteria and fungi; and the sputum culture of the patient indicated *Klebsiella pneumoniae*, which was sensitive to piperacillin-tazobactam based on antimicrobial drug sensitivity test; thus, the antibiotic therapy remained unchanged. However, the temperature of the patient continued to increase and spiked to 39.8 ℃ on the seventh day after admission (Figure 1). Moreover, the patient presented with neck stiffness at this time, and the CSF examination was performed again. The biochemical test of CSF indicated a sharp elevation of WBC and protein, and a decrease of glucose and chloride (Table 1). However, Gram staining and subsequent culture of CSF revealed only numerous polymorphonuclear leukocytes with no visible microorganisms. Intracranial infection was highly suspected in consideration of the patient's clinical manifestation and laboratory test results. Therefore, the antimicrobial therapy was switched to meropenem (1 g q8h) and vancomycin (1 g q12h). The patient's temperature had decreased slightly after 5 days of treatment, but the body temperature still fluctuated between 38.5 ℃ and 39.0 ℃ (Figure 1). The CSF obtained on the following day, which was the seventh day of admission (29 December 2023), revealed tiny point colonies after incubating for 6 days on blood agar culture medium; and this was

**Table 1.** Biochemical and culture results of the patient's cerebrospinal fluid (CSF) during hospitalization.

| Day of admission | WBC (× $10^6$/L) | Protein (mg/L) | Glucose (mmol/L) | Chloride (mmol/L) | Culture |
|---|---|---|---|---|---|
| Day 2 | 57 | 492.9 | 5.71 | 133.8 | Negative |
| Day 7 | 1423 | 4299.5 | 1.64 | 113.9 | *M. hominis* |
| Day 12 | 1122 | 3757.7 | 2.06 | 105.8 | *M. hominis* |
| Day 18 | 365 | 865.1 | 2.72 | 123.3 | Negative |
| Day 25 | 42 | 355.2 | 3.23 | 126.1 | Negative |

WBC: white blood cell count; M. hominis: Mycoplasma hominis.

morphologically consistent with *M. hominis*. A repeat lumbar puncture was conducted on the same day, and 5 mL of the CSF was collected for pathogen identification by metagenomic next-generation sequencing (mNGS). The result indicated 1,664 sequence reads corresponding to *M. hominis* (Table 2), suggesting that it was the potential pathogenic organism. The presence of *Escherichia coli*, *Cutibacterium acnes*, *Staphylococcus aureus*, and *Staphylococcus epidermidis* were also detected at a low level, and were more likely to be contamination (Table 2). Clinical laboratory tests were conducted and reported by the Department of Clinical Laboratory Medicine of the Third People's Hospital of Chengdu. mNGS was performed and reported by Huayin Medical Laboratory in Chengdu, China.

The patient's medical history, clinical presentation, physical signs, and the results of CSF culture and mNGS, indicated that *M. hominis* caused the intracranial infection, and a combination therapy with moxifloxacin (0.4 g qd) and minocycline (100 mg q12h) was implemented. The temperature of the patient decreased to normal range on the fifth day of effective antibiotic therapy. His CSF became sterile and the profile of CSF normalized after a total duration of 14 days (Table 1). The patient remained apyretic and was subsequently discharged from the hospital. After 1 month of follow-up, the patient was still under treatment for residual neurological dysfunction at the rehabilitation unit in the local hospital, and was without any symptoms of CNS infection.

## Discussion

*M. hominis* is a commensal which primarily colonizes in the genitourinary system, and rarely distributes in the upper respiratory tracts of healthy individuals [1]. It is generally responsible for urogenital tract infections, such as pelvic inflammatory illnesses, postpartum fever, and vaginosis [2,10]. There have been reports of CNS infections caused by *M. hominis* in neonates, especially those born prematurely [3–5], but *M. hominis* rarely induces CNS infections among non-neonatal patients. To the best of our knowledge, a total of 26 cases of *M. hominis* induced CNS infections in non-neonatal patients have been reported in English literature since 1950, up to the end of July 2024; and Supplementary Table 1 provides an overview of these cases, along with the present case. Generally, Gram-negative and Gram-positive bacteria are the primary pathogens causing CNS infections [8,9]. Therefore, *M. hominis* may not be empirically considered as the pathogenic bacteria at the initial stage of suspicion of CNS infections, without microbiological evidence. Additionally, the clinical manifestations and biochemical test results of *M. hominis* associated CNS infections lack specificity. It is difficult to distinguish them from CNS infections caused by bacteria or viruses. In our case, the patient presented with persistent high fever, and initially we empirically considered the pathogen as bacteria other than *M. hominis*, which resulted in a delay of diagnosis. Therefore, the actual incidence of CNS infections caused by *M. hominis* may be largely underestimated.

**Table 2.** Results of mNGS analysis of the patient's CSF.

| Species | Number of sequences reads |
|---|---|
| *Mycoplasma hominis* | 1664 |
| *Escherichia coli* | 13 |
| *Cutibacterium acnes* | 41 |
| *Staphylococcus aureus* | 46 |
| *Staphylococcus epidermidis* | 34 |

mNGS: metagenomic next-generation sequencing; CSF: cerebrospinal fluid.

In most cases reported previously (Supplementary Table 1), the patients had predisposing host factors such as prior head traumas, neurosurgical interventions, endotracheal intubation, or urinary catheterization. Therefore, it was speculated that the main routes of *M. hominis* induced CNS infections can be classified as: direct contamination during head traumas, contamination during neurosurgical interventions, and seeding of intracranial sites secondary to transient bacteremia caused by endotracheal intubation or genitourinary manipulation [11]. Generally, *M. hominis* is typically unable to infiltrate beyond the mucosae of the genitourinary or respiratory tracts [12]. However, when the mucosae are damaged by instrument manipulation, trauma, or neurosurgery; the surface proteins of *M. hominis* promote cell adhesion, and *M. hominis* can spread to other sites of the body via bloodstream and contribute to infections [13,14]. In the present case, the patient received craniotomy and EVD insertion prior to CNS infection, and underwent urinary catheterization and endotracheal intubation during hospitalization, which may have been the high-risk factors that contributed to *M. hominis* induced CNS infection of this patient. However, it is difficult to confirm the exact sources of the patient's CNS infection.

Definite diagnosis of *M. hominis* induced CNS infections is difficult to some extent. Firstly, *M. hominis* is an atypical organism which lacks a rigid cell wall, and it cannot be stained with Gram's stain and observed under the light microscope. Secondly, unlike *Mycoplasma pneumoniae* or *Ureaplasma urealyticum*, serological detection is not a common test for *M.*

*hominis* [7,15]. A positive culture of *M. hominis* is the golden standard for identification, but it is challenging. As shown in Supplementary Table 1, a few previous cases reported that *M. hominis* isolates had been identified by conventional culture, but it turned out to be time-consuming [16–20]. In the present case, only tiny point colonies were observed after incubating CSF for 6 days, which induced a delay in etiological confirmation and initiation of appropriate therapy. Thus, specific diagnostic methods are required for rapid identification of *M. hominis*.

Only one of the previously reported cases (Supplementary Table 1) were diagnosed by mNGS13, as was done in the present case. mNGS directly detects the whole genomes of clinical samples, and unbiasedly identifies unknown pathogens by comparing and analyzing with the known microbial sequence databases [21]. mNGS shows a higher pathogen detection rate than traditional microbial cultures (95.0% vs. 60.0%) [22], and is gradually becoming a promising tool for pathogen-specific diagnosis. However, the cost involved in mNGS for pathogen detection is relatively high, and the results obtained through this technology is prone to contamination with environmental species [23]. In most of the previously reported cases (Supplementary Table 1), the definitive diagnoses of *M. hominis* were made by 16S ribosomal RNA (rRNA) sequencing [24]. The conserved region of 16S rRNA is the main gene sequence used for the construction of primers [25], and 16S rRNA sequencing by real-time reverse transcription polymerase chain reaction (RT-PCR) has been widely used to identify unknown pathogens. In general, the identification of species was performed by comparing the obtained sequences with known sequences in the GenBank database using specific software tools, such as BLAST [26]. Recently, matrix-assisted light desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS) was applied for the rapid identification of *M. hominis* from culture-positive isolates, as long as the software libraries included the spectrum [27]. This method is supposed to be time-saving, and has high-specificity [23,28]. The identification methods of *M. hominis* in clinical practice may be complementary. Several cases have reported that the identifications of *M. hominis* failed by MALDI-TOF MS, even when the spectra of this species was represented in the database [11,29]; however, the definite diagnosis of *M. hominis* were made by 16S rRNA sequencing and traditional culture. There are also case reports that demonstrate that the detection of *M. hominis* was successfully made by MALDI-TOF MS, but the result of 16S rRNA sequencing was negative [30].

Generally, bacteria are the primary pathogens that cause CNS infections [8,9], and the manifestations of *M. hominis* induced CNS infections lack specificity. Thus, in clinical practice, clinicians may preferentially consider the diagnosis of bacteria associated CNS infections, and antimicrobial agents targeting bacteria may be initiated empirically. However, as mentioned above, *M. hominis* lacks cell wall, and it is intrinsically resistant to antimicrobial agents that interfere with the synthesis of cell wall, such as β-lactam and glycopeptide antibiotics, which are the cornerstone of first-line treatment of CNS infections [31,32]. Currently, there is no standard treatment criteria for *M. hominis* induced CNS infections. In most of the previously reported cases (Supplementary Table 1), fluoroquinolones which inhibit the replication of DNA and tetracyclines that affect the synthesis of protein constituted the mainstay treatment of CNS infections caused by *M. hominis* [6,7,29,33–38]. Fluoroquinolones are lipophilic agents with a molecular mass of around 300 Da and low binding to plasma proteins [39]. Among them, the fourth-generation fluoroquinolones such as moxifloxacin and gatifloxacin show significantly high CSF penetration, and the concentrations in CSF can reach greater than 1 μg/mL [33,40]. Similarly, tetracyclines (e.g., doxycycline and minocycline) have been described as agents that can achieve desirable CSF penetration [39]. Even an in vitro study indicated that *M. hominis* was resistant to tetracyclines at increasing frequency [41], and tetracyclines showed excellent efficacy in treating CNS infections caused by *M. hominis* [7,36,37,42,43]. In addition, a few cases reported that *M. hominis* associated CNS infections were successfully treated by clindamycin, chloramphenicol, or azithromycin in combination with fluoroquinolones or tetracyclines [11,19,33,44–46].

In the present case, antibacterial therapy consisting of meropenem (1 g q8h) and vancomycin (1 g q12h) was initiated when intracranial infection was suspected. However, the empirical treatment showed poor efficacy during this period. At this time, the culture of CSF showed tiny point colonies after incubating for 6 days, which was morphologically consistent with *M. hominis*, and this was subsequently confirmed by mNGS. Therefore, after the etiological confirmation of *M. hominis*, a combination treatment of moxifloxacin (0.4 g qd) and minocycline (100 mg q12h) was initiated, which was supposed to exert a synergistic antibacterial effect. Fortunately, the patient's body temperature decreased to the normal range on the fifth day of

effective treatment, and his CSF became sterile after a total treatment duration of 14 days (Table 1). It is worth noting that in overwhelming majority of previously reported cases and the present case, the key to successful treatment of *M. hominis* associated CNS infections was not only the appropriate antimicrobial therapies, but also prompt surgical debridement and drainage. This emphasizes the importance of surgical interventions, because the antibiotics used in some previous cases were regarded to have inadequate penetration in the blood brain barrier, making it difficult to quantify the contribution of those antibiotics to the resolution of CNS infection [16,17]. Additionally, there have been cases of patients who were cured effectively by surgical drainage and debridement alone [27,47], suggesting that the successful treatment response was due to the surgical intervention. This highlights the importance of optimal therapeutic strategy in combination with timely surgical interventions for the management of *M. hominis* induced CNS infections.

## Conclusions

Although *M. hominis* induced CNS infections in non-neonatal patients are rare, and the diagnosis of such infections are challenging to some extent, clinicians should be aware of the possibility of *M. hominis* as a pathogen of CNS infections, especially when the patients are not responding to regular broad-spectrum antibiotics. Additionally, the importance of timely antibacterial treatments in combination with proper surgical interventions is emphasized in the management of *M. hominis* induced CNS infections.

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### Consent for publication

Written informed consent was obtained from the patient and his family for the publication of any data included in this report.

### Authors contributions

YL, literature analysis, writing—original draft; JT, literature analysis, tables and figures; YW, literature analysis; FC, literature analysis; YY, writing—original draft, writing—critical evaluation and editing. All the authors made fundamental contributions to this manuscript and approved the submitted version.

### Corresponding author

Yan Yang, PhD.
Department of Pharmacy, The Third People's Hospital of Chengdu, Affiliated Hospital of Southwest Jiaotong University,
No.82, Qinglong Street, Qingyang District, Chengdu, 610014, China.
Tel: 86-18200359139
Fax: 86-61318506
Email: yanyang1947@swjtu.edu.cn

### Conflict of interests

No conflict of interests is declared.

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## Annex – Supplementary Items

**Supplementary Figure 1.** CARE checklist of information to include when writing a case report.

CARE case report guidelines

**CARE Checklist of information to include when writing a case report**



| Topic | Item | Checklist item description | Reported on Line |
|---|---|---|---|
| Title | 1 | The diagnosis or intervention of primary focus followed by the words "case report" | Title |
| Key Words | 2 | 2 to 5 key words that identify diagnoses or interventions in this case report, including "case report" | Key words |
| Abstract (no references) | 3a | Introduction: What is unique about this case and what does it add to the scientific literature? | Introduction |
| | 3b | Main symptoms and/or important clinical findings | Abstract |
| | 3c | The main diagnoses, therapeutic interventions, and outcomes | Abstract |
| | 3d | Conclusion—What is the main "take-away" lesson(s) from this case? | Abstract |
| Introduction | 4 | One or two paragraphs summarizing why this case is unique (**may include references**) | Introduction paragraph 1-2 |
| Patient Information | 5a | De-identified patient specific information. | Case presentation paragraph 1 |
| | 5b | Primary concerns and symptoms of the patient. | Case presentation paragraph 2-3 |
| | 5c | Medical, family, and psycho-social history including relevant genetic information | Case presentation paragraph 1 |
| | 5d | Relevant past interventions with outcomes | Case presentation paragraph 2 |
| Clinical Findings | 6 | Describe significant physical examination (PE) and important clinical findings. | Case presentation paragraph 2 |
| Timeline | 7 | Historical and current information from this episode of care organized as a timeline | Case presentation paragraph 2-3 |
| Diagnostic Assessment | 8a | Diagnostic testing (such as PE, laboratory testing, imaging, surveys). | Case presentation paragraph 2 |
| | 8b | Diagnostic challenges (such as access to testing, financial, or cultural) | Case presentation paragraph 2 |
| | 8c | Diagnosis (including other diagnoses considered) | Case presentation paragraph 3 |
| | 8d | Prognosis (such as staging in oncology) where applicable | Case presentation paragraph 3 |
| Therapeutic Intervention | 9a | Types of therapeutic intervention (such as pharmacologic, surgical, preventive, self-care) | Case presentation paragraph 3 |
| | 9b | Administration of therapeutic intervention (such as dosage, strength, duration) | Case presentation paragraph 2 |
| | 9c | Changes in therapeutic intervention (with rationale) | Case presentation paragraph 3 |
| Follow-up and Outcomes | 10a | Clinician and patient-assessed outcomes (if available) | Case presentation paragraph 3 |
| | 10b | Important follow-up diagnostic and other test results | Case presentation paragraph 3 |
| | 10c | Intervention adherence and tolerability (How was this assessed?) | Case presentation paragraph 3 |
| | 10d | Adverse and unanticipated events | No |
| Discussion | 11a | A scientific discussion of the strengths AND limitations associated with this case report | Discussion |
| | 11b | Discussion of the relevant medical literature **with references**. | Discussion |
| | 11c | The scientific rationale for any conclusions (including assessment of possible causes) | Discussion |
| | 11d | The primary "take-away" lessons of this case report (without references) in a one paragraph conclusion | Conclusion |
| Patient Perspective | 12 | The patient should share their perspective in one to two paragraphs on the treatment(s) they received | No |
| Informed Consent | 13 | Did the patient give informed consent? Please provide if requested | **Yes** ☑ **No** ☐ |

**Supplementary Table 1.** Summary of the reported cases of CNS infections secondary to M. hominis in non-neonatal patients (1950 to July 2024).

| No. | Year | Age / Gender | Underlying conditions | Diagnosis | Clinical manifestation | Diagnostic methods | SI | Antibiotic therapy after diagnosis | Treatment course | Outcome | Ref |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1950 | 20/M | trauma, surgery | brain abscess | fever, headache, stiff neck | C | Yes | streptomycin | 7 days | cured | [16] |
| 2 | 1981 | 29/M | trauma, craniotomy, urinary tract catheterization | brain abscess | fever | C | Yes | erythromycin | 2 weeks | cured | [17] |
| 3 | 1988 | 11/F | medulloblastoma, radiation therapy, chemotherapy | CNS infection | fever | C | Yes | methacycline | 3 weeks | died | [18] |
| 4 | 1990 | 76/M | subarachnoid hemorrhage, ventriculostomy, intubation, urinary tract catheterization | meningitis | fever, unresponsive | C | No | no antibiotic therapy at the request of his family | NA | died | [19] |
| 5 | 1995 | 20/M | trauma, surgery, orbital abscess, urinary tract catheterization | brain abscess | fever, coma | C | Yes | Initial with metronidazole, cefotaxime and doxycycline parenterally, then changed to clindamycin orally. | 4 weeks | cured | [20] |
| 6 | 1997 | 22/F | vaginal delivery, intracranial hematoma, craniotomy | brain abscess | left-side weakness and numbness, fever | IFT+IBT | Yes | NA | NA | cured | [47] |
| 7 | 1997 | 18/F | trauma, intraventricular bleed, ventriculostomy | meningitis | fever, altered mental status | C | Yes | Initial with doxycycline, ciprofloxacin and erythromycin, then erythromycin was discontinued and chloramphenicol was added. | NA | cured | [45] |
| 8 | 2003 | 17/F | trauma, vaginal delivery, subdural hematoma, craniotomy | brain abscess | headache, fever, photophobia, nausea | C+PCR | Yes | doxycycline + clindamycin | 3 weeks | cured | [44] |
| 9 | 2003 | 40/F | cavernous angioma, craniotomy, prolonged episode of ulvar irritation and ulceration | brain abscess | headache, left facial nerve palsy, slurred speech, nausea | C+16S | Yes | ciprofloxacin + metronidazole | 6 weeks | cured | [24] |
| 10 | 2006 | 40/M | head trauma, cystoscopy, urinary tract catheterization | brain abscess | hematuria, retention of urine, confused | PCR | Yes | tetracycline | NA | cured | [38] |
| 11 | 2008 | 48/M | colloid cyst, obstructive hydrocephalus, surgery | meningitis cranial osteomyelitis | fever, confusion | C + 16S | Yes | Gatifloxacin + clindamycin (Gatifloxacin was ceased after two weeks of therapy and clindamycin was changed to the oral formulation) | 3 months | cured | [33] |
| 12 | 2008 | 17/F | trauma, craniotomy | meningitis | fever | C | Yes | gatifloxacin, moxifloxacin (After one month intravenous gatifloxaci was changed to oral moxifloxacin) | 6 weeks | cured | [33] |
| 13 | 2011 | 41/F | hematoma, surgery, uterus curettage | brain abscess | obnubilation, coma | 16S | Yes | doxycycline | 12 weeks | cured | [36] |
| 14 | 2012 | 48/F | subarachnoid hemorrhage, craniotomy | meningitis | fever | 16S | Yes | moxifloxacin | 2 weeks | cured | [34] |
| 15 | 2012 | 40/M | trauma, craniotomy | brain abscess | fever | C + 16S | Yes | doxycycline | 16 days | cured | [37] |
| 16 | 2014 | 43/M | trauma, surgery, intubation | brain abscess | fever, delirium tremens | MALDI-TOF MS + 16S | Yes | levofloxacin + doxycycline | NA | cured | [30] |

| No. | Year | Age / Gender | Underlying conditions | Diagnosis | Clinical manifestation | Diagnostic methods | SI | Antibiotic therapy after diagnosis | Treatment course | Outcome | Ref |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 17 | 2014 | 17/M | trauma, spinal injuries, surgery, urinary tract catheterization | intramedullary spinal cord abscess | bicep and deltoid weakness, fever | C | Yes | Initial with vancomycin, moxifloxacin, and doxycycline, then changed to intravenous moxifloxacin and switched to oral moxifloxacin finally. | 6 months | cured | [13] |
| 18 | 2015 | 21/F | vaginal delivery, subdural empyema, surgery | spinal abscess | neck pain, nausea, vomiting, fever | C + 16S | Yes | moxifloxacin | 4 weeks | cured | [35] |
| 19 | 2016 | 39/M | trauma, subarachnoid hemorrhage, suprapubic catheterization, tracheal intubation | meningitis | fever, loss of consciousness | C + 16S + PCR | Yes | moxifloxacin | 15 days | died | [29] |
| 20 | 2016 | 71/M | intracerebral hemorrhage, craniotomy, endotracheal intubation, urinary tract catheterization | meningitis | fever, anepia, right-sided weakness | 16S | Yes | azithromycin + minocycline | 2 weeks | cured | [11] |
| 21 | 2016 | 28/F | decompressive laminectomy, urinary tract catheterization | epidural abscess meningitis | fever, headache, stiff neck | C + 16S | Yes | doxycycline | 4 weeks | cured | [7] |
| 22 | 2017 | 6/F | hydrocephalus, surgery | VPS infection meningitis | fever | C + 16S | Yes | clindamycin + ciprofloxacin | 6 weeks | cured | [46] |
| 23 | 2017 | 57/M | hematoma, arterio-venous malformation, craniectomy, urinary tract catheterization | brain abscess | fever | C + 16S | Yes | doxycycline | 8 weeks | cured | [42] |
| 24 | 2019 | 25/M | trauma, subdural hemorrhage, craniectomy, EVD, tracheostomy | ventriculitis | fever, coma | C + MALDI-TOF MS | Yes | ciprofloxacin | 2 weeks | cured | [6] |
| 25 | 2022 | 44/M | meningioma, craniectomy, intubation, urinary tract catheterization | meningitis | fever, neck rigidity | C + mNGS | Yes | moxifloxacin + doxycycline | 2 weeks | cured | [13] |
| 26 | 2023 | 52/M | trauma, craniectomy, tracheotomy, urinary tract catheterization | epidural empyema meningitis | fever | C+16S | Yes | minocycline | 4 weeks | cured | [43] |
| Present case | 2023 | 68/F | craniectomy, urinary tract catheterization, endotracheal intubation | intracranial infection | fever, neck stiffness | C + mNGS | Yes | moxifloxacin + minocycline | 2 weeks | cured | – |

C: culture; CNS: central nervous system; EVD: external ventricular drain; F: female; IBT: immunoblotting test; IFT: immunofluorescence test; M: male; MALDI-TOF MS: matrix-assisted light desorption/ionization time-of-flight mass spectrometry; mNGS: metagenomic next-generation sequencing; NA: not available; PCR: polymerase chain reaction; SI: surgical interventions; VPS: ventriculoperitoneal shunt; 16S: 16S rRNA sequencing.