Rare Association of Meningoencephalitis and Brain Abscess with *Staphylococcus pettenkoferi* Infection: Case Report and Literature Review

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Abstract

Meningitis is inflammation of the pia and arachnoid membrane surrounding the brain and spinal cord. Brain abscesses are suppurative infections that develop in the parenchyma tissue of the brain. Both these diseases can be of external origin such as trauma and surgical intervention. They can be of internal origin due to complications from infections. *Staphylococcus pettenkoferi* is not generally associated with these pathologies but we present such a rare association in a young man who presented with symptoms that led to a diagnosis of diffuse brain edema, which was relieved with emergency decompressive surgery. Cultures of peripheral blood and brain tissue yielded *S. pettenkoferi* and he was further diagnosed with meningitis and treated for the same. However, the patient developed serious complications and passed away within a week. We also discuss the reports in the literature that describe cases associated with this rare pathogen.

Keywords: *Staphylococcus pettenkoferi*; Meningitis; Brain Abscess; Turkey.

Introduction

The genus *Staphylococcus* consists of more than forty species and subspecies that are ubiquitous in nature and can colonize or infect a wide variety of animals. In addition to coagulase positive *Staphylococcus aureus subsp. aureus*, coagulase negative staphylococci (CoNS) are among the most frequently isolated bacterial species clinically. CoNS can settle in foreign bodies such as intravenous catheters, prosthetic heart valves and joint prostheses as nosocomial pathogens and cause infection in patients.[1] They may also infect premature babies, patients with malignant diseases, patients receiving chemotherapy, and organ transplant receipients.[2,3] Two strains of CoNS of human origin have been isolated and identified as *Staphylococcus pettenkoferi*,[4,5] but this name has not yet become popular in literature. Worldwide, CoNS is among the most common organisms causing nosocomial bacteremia.[6] *S. pettenkoferi* has been accepted as a new member of CoNS.

Case Report

A 33-year-old Turkish man was admitted to the emergency service in a poor general condition. The patient complained of chills, nausea and vomiting from the morning. Afterwards, he developed unconsciousness along with involuntary swaying movements in his arms and legs. Clinical examination revealed fever: 37.4 C; Glasgow Coma Scale score: 11; oxygen saturation: 96%; heart rate: 100/min; respiratory rate: 32/min; and blood pressure: 150/90 mm/Hg. There was no visible rash on the body.

During neurosurgery consultation, the patient was unconscious, pupils were dilated, indirect light reflex (ILR) was absent in both eyes, and the response to pain stimulus was localized more to the left upper extremity. There was movement in the right upper extremity and deep tendon reflexes (DTR) were +/+. Nuchal rigidity was not detected. Laboratory results: white blood cell (WBC): 23 cells/mm^3 (reference range: 450–1100); C-reactive protein (CRP): 102 mg/L (5–10); procalcitonin: 20 ng/mL (0–5); and the erythrocyte sedimentation rate (ESR): 96 mm/h (0–20). There were no pathological findings in other biochemical tests. Anti-hepatitis C (HCV) and anti-human immunodeficiency virus (HIV) tests yielded negative results.

The patient's blood was sent for culture. Meanwhile emergency brain computed tomography (CT) scan and brain diffusion magnetic resonance imaging (MRI) revealed diffuse cerebral edema. Edema was evident in the apparent diffusion coefficient (ADC) sequence in MRI due to the absence of bleeding and no covering lesions. These indications led the neurosurgery team to arrive at a preliminary diagnosis of central nervous system (CNS) infection. The infectious diseases team was consulted in view of the patient's suspicious history of seizures. They recommended intravenous (IV) levetiracetam 1500 mg initially, followed by a maintenance dosage of 2 × 500 mg infusion (inf.). Also recommended was empirical administration of broad-spectrum antibiotics: meropenem 3 g twice daily, vancomycin 2 g once daily, and acyclovir 800 mg thrice daily.

On the second day, the patient underwent contrast-enhanced cranial MRI which revealed widespread cerebral edema and tonsillar herniation. He was intubated and emergency decompressive surgery performed. After bilateral decompressive hemicraniectomy, bilateral star-shaped opening of the dura, the necrotic and pus-filled cerebrum was washed [Figure 1]. The necrotic pus tissue was observed to have organized with abscess formation. The brain tissue was taken for culture. The bones were embedded anteriorly, each on its own side. Anti-edema treatment at the maximum level was initiated.

Inotropik support was also started as the patient had developed low blood pressure. The antibiotic doses were regulated as advised by nephrology and gastroenterology specialists in view of disorders in liver function and kidney function. In the follow-ups, the control MRI reported as follows: "In the sections passing through the supratentorial level, cortical widespread signal increase and diffusion restriction are observed in these areas. The sulci are noticeably erased." These findings suggested meningoencephalitis. Due to high urea, creatinine, and electrolyte imbalance in the patient's current biochemistry, daily dialysis was initiated.

The patient's brain tissue and peripheral blood culture revealed catalase positive growth and *Staphylococcal* spp. was confirmed by gram staining. Polymerase chain reaction (PCR) was performed to detect the presence of methicillin resistance and to distinguish the strain from *S. aureus*. Neither nuc nor mecA gene was detected, confirming the pathogen to be a coagulase-negative methicillin-susceptible *Staphylococcus* species. Growth on matrix assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-ToF MS) resulted in identification of *S. pettenkoferi*. It was inoculated on blood agar (BA) and sensitivity was investigated [Figure 2 and Table 1]. The general condition of the patient, who was being administered methicillin in addition to other treatment, deteriorated further, despite a regression in C-reactive protein. On the seventh day of follow up, he suffered a cardiac arrest and could not be revived despite cardiopulmonary resuscitation for 50 minutes.

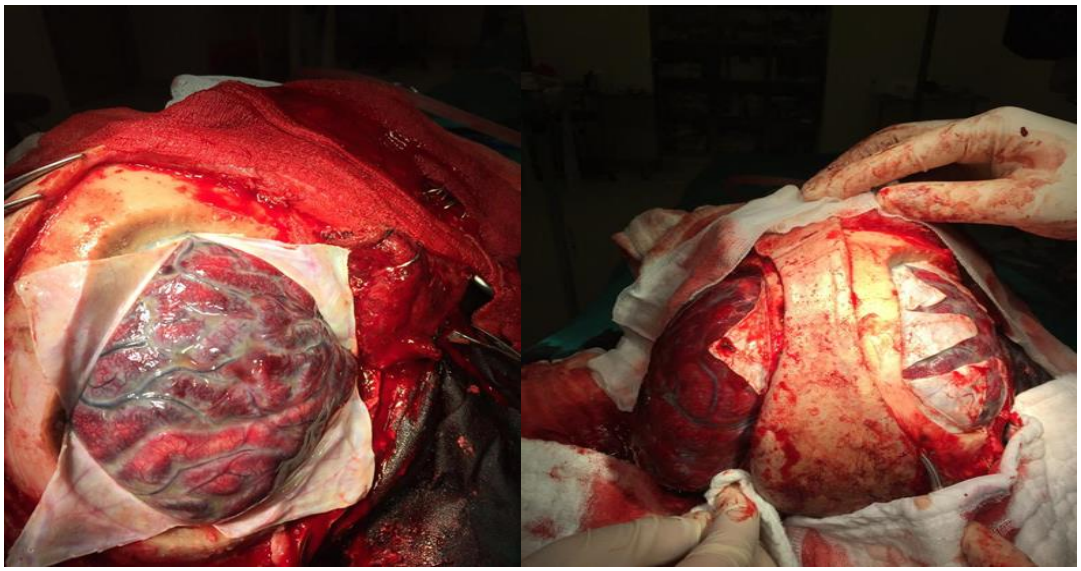

Figure 1: Intraoperative images. (a) The right hemisphere after decompression (the first to be decompressed). Following the classic question mark incision, the fronto-parieto-temporal bone window opens, and the right parietal lobe covered with hemorrhage and pus is visible under the star-shaped dura mater. (b) The left hemisphere is seen decompressed with a larger window, and similar hemorrhage and pus are observed.

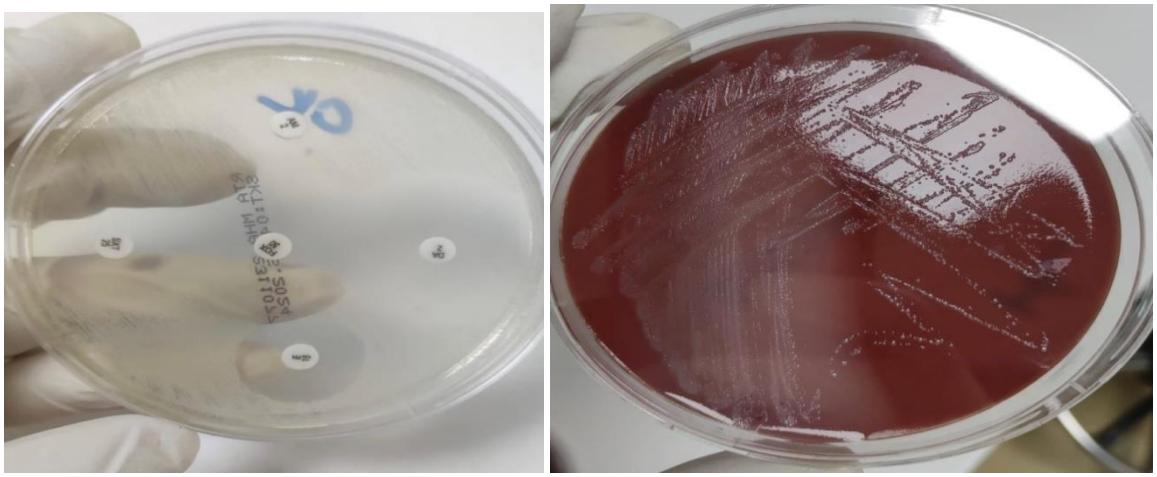

Figure 2: *Staphylococcus pettenkoferi* is detected in (a) blood agar, and (b) disc diffusion test.

Table 1: Susceptibility Test.

Clindamycin	S ≤ 0.25 mg/L
Erythromycin	S 0.5 mg/L
Methicillin/sefoksitin	S 2.0 mg/L
Trimethoprim-sulfamethoxazole	S ≤ 10.0 mg/L
Vancomycin	S 1.0 mg/L

Note. *S: Sensitivity.*

Discussion

There are limited reports of *S. pettenkoferi* infections in the literature. It may present variously as bacteremia, osteomyelitis, wound infection, or meningoencephalitis as in our patient. *S. pettenkoferi* was first isolated in

2002 in Germany by Trülzsch et al.[4] from the blood of a young woman with extrapulmonary tuberculosis, then from the wound of an elderly patient with leukemia and DM. In 2007, the same study group reported three more isolates of *S. pettenkoferi* from different patients.[5] One more case of osteomyelitis caused by this bacterium was reported in the same year.[7] Another study revealed the pathogenic role of *S. pettenkoferi* in a prosthetic joint infection case.[8]

From 2002 till 2024, very few cases of *S. pettenkoferi* infection were reported worldwide. In one of these cases,[9] the bacteria grew in the peripheral blood culture of an elderly woman who was admitted after falling unconscious at home. A few days earlier she had experienced symptoms consistent with vertigo. She had a history of hypertension, type 2 diabetes mellitus, psoriasis, dyslipidemia, and epilepsy. She also had a trunk rash that had been present for two weeks. Her initial treatment with vancomycin was switched to intravenous cloxacillin after two days, leading to her recovery. There are more case reports of elderly patients infected by this bacterium.[4,5,8,10] Also reported are cases of premature babies, children, and adolescents, two of whom (aged 9 and 15 years) had genetic diseases.[4,5,11] A 17-year-old patient from China exhibited dermatological manifestations.[12]

There appears to be no predisposition to *S. pettenkoferi* infections in terms of age and sex. In most cases, growth was observed in blood culture (at least two bottles / one set), and in one case, the growth was observed in bone biopsy. In the current case, bacterial growth was detected in brain tissue and blood cultures. Treatments were successful in most cases; while most patients who died had cancer, major surgery, or severe immunosuppressive conditions such as acquired immunodeficiency syndrome (AIDS), HCV, or cerebral toxoplasmosis. Most patients infected with *S. pettenkoferi* were also likely to have comorbidities such as pulmonary tuberculosis, extrapulmonary tuberculosis, diabetes mellitus, leukemia, prematurity, AIDS, senile immunosuppression, gastric cancer, lung cancer, chronic obstructive pulmonary disease (COPD), or hepatitis C.[8,9,13–16]

S. pettenkoferi infections may lead to bacteremia which subsequently may infect CNS. As the CNS infection spreads, it may lead to various complications such as skull base osteomyelitis, in turn causing cranial polyneuropathy.[14,17] The fact that our patient approached many different clinics before the current presentation may have given the infection the time to spread to the brain tissue, and the intra-surgical images [Figure 1] support this.

Our patient did not have any known comorbidity or blood-borne disease. During history taking, he revealed that he had a condition suggestive of alcohol dependency. Several studies in the literature, as well as the Centers for Disease Control and Prevention, have linked excessive alcohol consumption to the development of chronic diseases.[18] It is known that frequent and long-term alcohol use may suppress the immune system.[19] Our literature review has revealed three patients with possible history of alcohol or drug dependence.[16] In one case, *S. pettenkoferi* detected in multiple blood cultures of a patient with endocarditis led to consideration of this bacterium as the causative agent. It is not known whether his marijuana use facilitated bacterial growth through endocarditis or immune suppression.[20]

S. pettenkoferi do not usually cause infections in humans. For example, in a retrospective study conducted in the United States, its growth was detected in approximately 80 adult patients (> 18 years), three of whom were on immunosuppressive therapy, but the bacterium was not identified as the causative agent in any of them.[21]

Currently, *S. pettenkoferi* is recognized as a causative agent in cases where two or more biological cultures exhibited its growth in the absence of any other potential pathogen. Table 2 lists the demographic, clinical, treatment, and outcome data of patients in cases documented in the literature since 2002 where this bacterium was considered the causative agent.

Table 2: Demographic and clinical data of reported cases of *Staphylococcus pettenkoferi* infection since 2002.[4–13,15,16,20,22]

Author, year, country	Age/sex	Infection Pattern	Underlying Conditions	Clinical finding	Treatment	Outcome
Trülzsch et al.[4] (2002, Germany)	25/F	CA	Extrapulmonary tuberculosis	Bacteremia	?	Recovered
Song et al.[22] (2008, S. Korea)	76/M	CA	Leukemia, DM	Wound infection	Vancomycin	Recovered
Trülzsch et al.[4] (2002, Germany)	?	CA	?	Osteomyelitis	?	?
Mammina et al.[9,23] (2011, Italy)	49/M	NOCL	Post-traumatic hydrocephalus	Fever	Daptomycin, piperacillin-tazobactam	Died
Loïez et al.[7] (2007, France)	63/M	CA	Diabetes	Diabetic foot infection, osteomyelitis	Ampicillin, pristinamycin	Recovered
d'Azevedo et al.[9,24] (2010, Brazil)	56/?	CA	?	Fever	?	?
Morfin-Otero et al.[9,25] (2012, Mexico)	Premature baby	NOCL?	None	Fever	Ampicillin, amikacin	Recovered ?
Morfin-Otero, et al.[9,25] (2012, Mexico)	45/M	CA	AIDS, hepatitis C, cerebral toxoplasmosis	Fever	Clindamycin, trimethoprim-sulfamethoxazole, azithromycin, Amphotericin-b	Died
Hashi et al.[9] (2015, United Kingdom)	75/F	CA	HT, DM, psoriasis	Bacteremia	vancomycin	Recovered
Bilecen et al.[25] (2015, Turkey)	3 cases?	NOCL	?	Fever	?	?
Wutawunasheet al.[20] (2021, USA)	28/M	CA	Multiple Sclerosis, Used synthetic marijuana	Endocarditis	?	Recovered
Gisriel and Jacobs[8] (2022, USA)	81/F	CA	CAD, HT	PJI	Vancomycin, daptomycin, rifampin	Recovered
Wang et al.[12] (2023, China)	17/M	CA	None	Fever, Pustulosis	Linezolid	Recovered

Strong et al.[13] (2021, USA)	73/M	CA	Immunosuppression, rituximab treatment	Extremity weakness, abdominal pain	Vancomycin	?
Tamura et al.[11] (2023, Japan)	9/F	NOCL?	Epileptic encephalopathy	Fever	Vancomycin	Recovered
Tamura et al.[11] (2023, Japan)	15/M	NOCL?	Chromosomal abnormality, epilepsy	Fever	Vancomycin	Recovered
Park et al[16]. (2015, S.Korea)	72/M	CA	DM, HT, Alcohol dependence	Septic shock	Ceftriaxone	?
Kang and Ryoo[14] (2019, S. Korea)	38/F	CA	Chemotherapy for gastric cancer	Fever	Methicillin	Recovered
Vecchia et al.[15] (2018, Italy)	88/F	CA	CAD, Alzheimer's, COPD	Fever	Tigecycline, rifampin	Recovered
Izaguirre-Anariba et al.[10] (2018, Italy)	79/F	CA	Small cell lung carcinoma	Fever	Meropenem, vancomycin, azithromycin	Died
Savini et al[15,26] (2016, USA)	86/M	NOCL	AAA surgery	Septic shock	Meropenem, clindamycin, etc.	Died

Note. *?: Not known / doubtful; NOCL: Nosocomial; CA: Community-acquired; DM: Diabetes mellitus; HT: Hypertension; OM: osteomyelitis; Bact: Bacteremia; ME: Meningoencephalitis; DFI: Diabetic foot infection; CAD: Coronary artery diseases; AAA: Abdominal aortic aneurysm; COPD: Chronic obstructive pulmonary disease.*

Early identification of the predisposing factors may provide an important warning whether brain abscess is likely to develop. Cerebrospinal fluid (CSF) collection is recommended in high-risk patients for early diagnosis and commencement of treatment of CNS infections. It is critical for clinicians to include this condition in the differential diagnosis.[27] In the current case, CSF was not taken due to diffuse brain edema and tonsillar herniation, but the agent was grown in direct peripheral blood culture and brain tissue culture. This was considered sufficient for initiating preemptive treatment.

Lately, the increasing use of MALDI-ToF in species determination is enabling easier detection of infectious bacterial strains. Therefore, we can expect increased reports of *S. pettenkoferi* infection in the future. In addition to surgical treatment, optimal medical management is vital for better prognosis of brain abscess cases.[28–32]

Conclusion

This case reveals *S. pettenkoferi*, a rarely encountered benign bacterium, in an uncharacteristic pathogenic role. Recognizing its role in bacteremia was a critical step in management. This case illustrates how certain predisposing factors can cause some microorganisms present as normal flora in the human body into becoming dangerous pathogens. When *S. pettenkoferi* is detected in culture, it may be appropriate to consider it as a potentially infectious agent rather than a contaminant and start preemptive treatment.

Disclosure

The authors declare no conflicts of interest. Written consent for publication was obtained from the patient's father.

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