American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2018; 19: 329-334
DOI: 10.12659/AJCR.908000

Received: 2017.11.10
Accepted: 2018.01.14
Published: 2018.03.22

Pyogenic Ventriculitis and Meningitis Caused by *Streptococcus Acidominimus* in Humans: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 49
Final Diagnosis: Pyogenic ventriculitis and meningitis caused by *Streptococcus acidominimus*
Symptoms: Confusion • fever • headache
Medication: —
Clinical Procedure: Antibiotics
Specialty: Infectious Diseases

Objective: Rare disease

Background: *Streptococcus acidominimus*, which belongs to the viridans streptococci group, is rarely considered pathogenic in humans. However, over the past 10 years, this bacterium has been reported to cause serious infections in humans, particularly among the critically ill. This article is the first case report of pyogenic ventriculitis (PV) and meningitis caused by *S. acidominimus* in North America.

Case Report: A 49-year-old Asian male presented to the emergency department with complaints of a headache, fever greater than 37.8°C (100°F) and confusion, of approximately 3 days duration. He was unable to speak coherently or follow approximately half of the given commands. He appeared ill; an intracranial infection was suspected. Magnetic resonance imaging of the brain showed: 1) infected proteinaceous material and pus-like material throughout the cerebral sulci and in the occipital horns of both lateral ventricles, 2) ependymal signal abnormality of the posterolateral margin of the occipital horn of the left lateral ventricle, and 3) early hydrocephalus suggestive of ventriculitis and meningitis. The blood and cerebrospinal fluid cultures were positive for *S. acidominimus*. The patient improved with minimal deficits after 6 weeks of IV ceftriaxone without requiring a neurosurgical intervention, such as an intraventricular drain or neuroendoscopic surgery.

Conclusions: PV and meningitis caused by *S. acidominimus* are rare but potentially fatal intracranial infections. Therefore, despite the risk of generalizing, our case report suggests that PV and meningitis caused by *S. acidominimus* can be effectively treated with a prompt and prolonged course of IV ceftriaxone without neurosurgical intervention.

MeSH Keywords: Brain Diseases • Cerebral Ventriculitis • Diffusion Magnetic Resonance Imaging • Meningitis, Bacterial • Viridans Streptococci

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/908000

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Background

Streptococcus acidominimus, which belongs to the viridans streptococci group, is commonly found in food and bovine udders and is rarely considered pathogenic in humans [1]. However, Wu et al. [2] summarized previous case reports of highly invasive diseases, including meningitis, brain abscess, Gradenigo syndrome/otitis media, endocarditis, empyema, acute cholecystitis, sepsis, and massive ascites, caused by *S. acidominimus* in humans. In addition, a previous case report of meningitis due to *S. acidominimus* was reported by Akaike et al. in Japan. The Spanish medical literature reported another case of meningitis due to *S. acidominimus* in 1990 [3]. Although Li et al. [4] reported one case of nosocomial *S. acidominimus* meningitis/ventriculitis in China, the authors failed to discern ventriculitis from meningitis. Interestingly, no case reports of ventriculitis due to *S. acidominimus* have been published. We report the first case of ventriculitis and meningitis due to *S. acidominimus* in North America.

Case Report

A 49-year-old Asian male presented to the emergency department with a headache, fever greater than 37.8°C (100°F) and confusion, of approximately 3 days duration. He had immigrated to the USA from Laos in 2004. His past medical history was significant for essential hypertension, hyperuricemia/gout, and recently diagnosed type 2 diabetes mellitus with a glycosylated hemoglobin A1c level of 8.3 within the 4 months prior to his admission. He was non-compliant with the diabetic diet and oral hypoglycemic agents recommended. He reported no prior earache or loss of hearing.

Clinical findings

The patient appeared ill but well-nourished. He was febrile, with a temperature of 38.2°C (100.8°F), heart rate of 115 beats per minute, respiratory rate of 30 breaths per minute, and blood pressure of 141/92 mm Hg in the left upper extremity in the supine position. His point of care testing for glucose at bedside was 396 mg/dL (22 mmol/L). He was unable to speak coherently or follow approximately half of the given commands. He was easily distracted and had dry mucus membranes. Nuchal rigidity was increased. A funduscopic examination showed no papilledema. Babinski reflex showed plantar extension bilaterally. The cardiorespiratory, gastrointestinal, musculoskeletal, and skin examinations were unremarkable.

Diagnostic assessment

Laboratory testing

Blood cultures were performed, and the patient underwent lumbar puncture prior to the administration of antibiotics. His cerebrospinal fluid (CSF) protein level was elevated at 527 mg/dL (5.27 g/L), his glucose level was low at 14 mg/dL (0.78 mmol/L), his RBC count was 208 cells/µL, and his WBC count was 35 418 cells/µL.

Imaging

Fluid-attenuated inversion recovery (FLAIR) magnetic resonance imaging (MRI) and diffusion-weighted MRI (DW MRI) on brain (Figure 1A, 1B, respectively) showed high signal intensity throughout the cerebral sulci and in the occipital horns of both lateral ventricles. Moreover, the axial apparent diffusion coefficient (ADC) image (Figure 1C), which is a companion image to DW MRI, showed that the signal in the lateral ventricle was mostly bright white; however, small, somewhat triangular and crescent-shaped areas were observed in the dependent posterior aspects of the lateral ventricle/occipital horn, where the fluid had a dark appearance (Figure 1C, white arrows). These dark areas corresponded to the high signal areas on the DW MRI, indicating infected proteinaceous material/pus. Both blood and infected proteinaceous material/pus have high signals in DW MRI. However, we believed that the high signal represented infected proteinaceous material/pus and not blood because a) the susceptibility weighted (SW) brain MRI (Figure 1D) showed that the material in the occipital horns (dependent) had a high signal that was not typical of blood/hemorrhage, which is usually black/lowest intensity on an SW MRI; b) the non-contrast computed tomography (CT) head (Figure 1E) did not show acute hemorrhage/blood in the ventricle or in the cerebral sulci. Moreover, ependymal signal abnormality of the posterolateral margin of the occipital horn of the left lateral ventricle and early hydrocephalus were observed.

To summarize, 1) increased signal abnormality on the DW MRI in the occipital/dependent horn of the cerebral ventricles with corresponding dark material was seen on the axial ADC companion image (Figure 1C) and was indicative of PV [5–7], and 2) the increased signal in the sulci on the FLAIR MRI (Figure 1A) indicated proteinaceous material in the CSF typically seen with meningitis. Although a post-contrast MRI was not performed upon initial presentation, this did not limit the diagnosis of ventriculitis.

Other studies

An electroencephalogram (EEG) showed no epileptiform activity, and the MRI of the lumbar spine, chest x-ray, and transthoracic echocardiography (TTE) were negative for infection.

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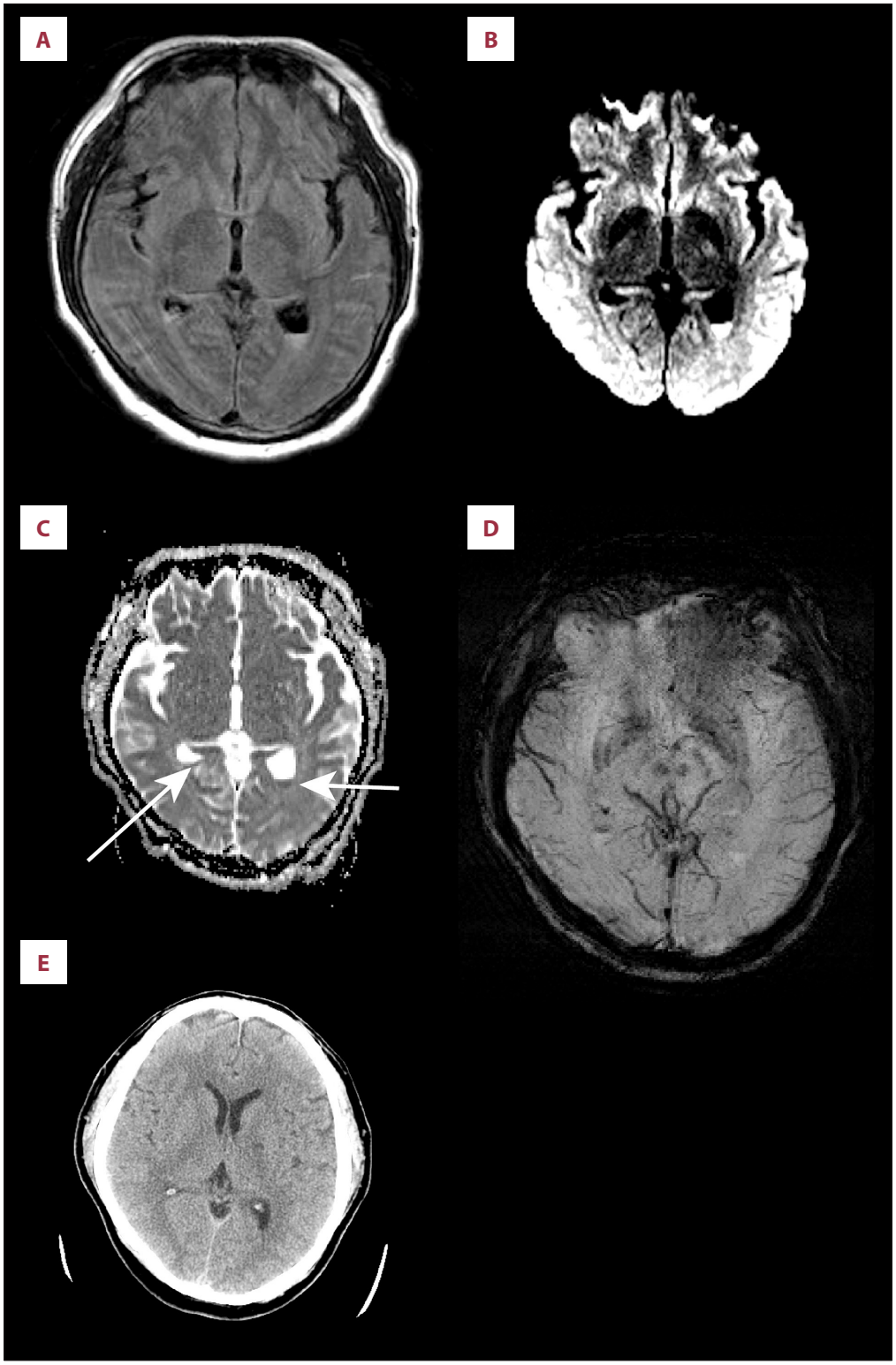

Figure 1. Brain magnetic resonance imaging (MRI) findings on the day of hospital admission. (**A**) Fluid-attenuated inversion recovery (FLAIR). (**B**) Diffusion-weighted MRI (DW MRI). (**C**) Axial apparent diffusion coefficient (ADC) image (a companion image to Figure 1B); the white arrows show the somewhat triangular and crescent-shaped dark areas representing infected proteinaceous material/pus. (**D**) The susceptibility-weighted (SW) MRI. (**E**) Non-contrast computed tomography (CT) head image showing the absence of acute hemorrhage/blood.

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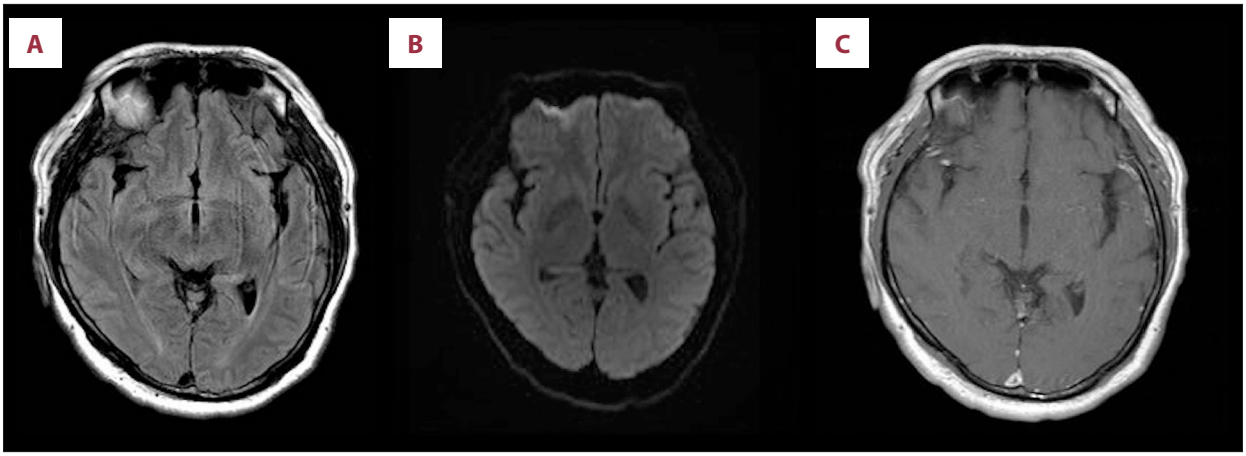

Figure 2. Brain magnetic resonance imaging (MRI) findings after 4 weeks of IV ceftriaxone. (**A**) Fluid-attenuated inversion recovery (FLAIR). (**B**) Diffusion-weighted images (DWI). (**C**) Post-contrast, 18 mL of gadopentetate dimeglumine (Magnevist).

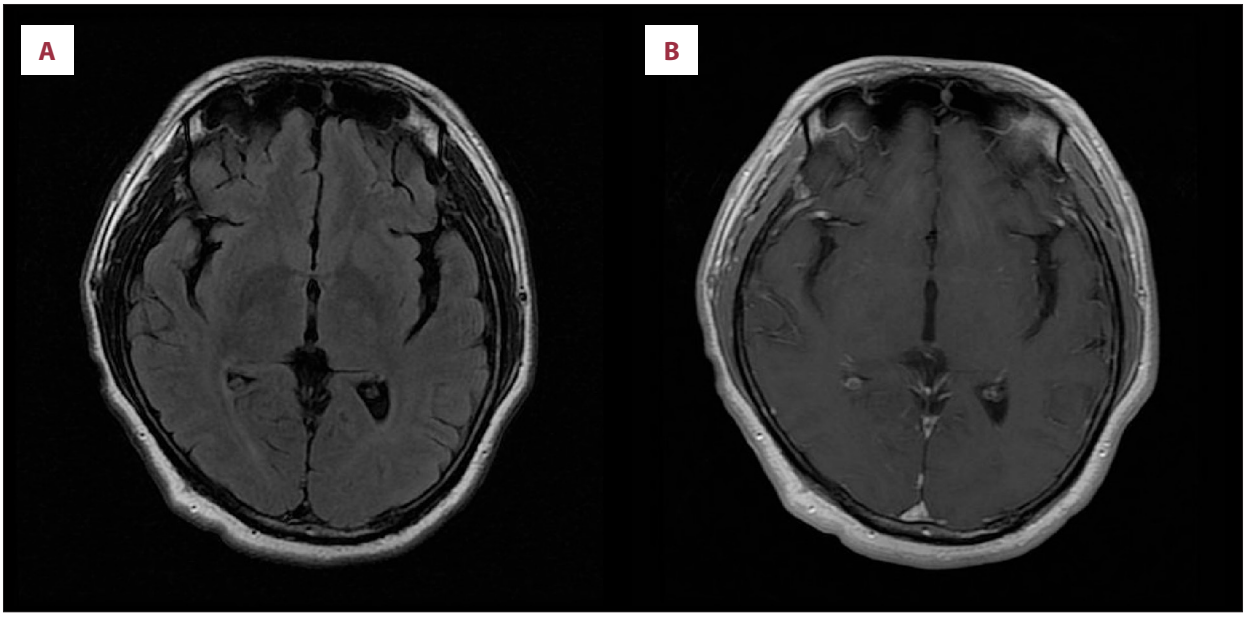

Figure 3. Brain magnetic resonance imaging (MRI) findings after 6 weeks of IV ceftriaxone. (**A**) Fluid-attenuated inversion recovery (FLAIR). (**B**) Post-contrast, 9 mL of gadobutrol (Gadovist).

Timeline and therapeutic intervention

The patient was initially treated with IV ceftriaxone (2 g every 12 hours), vancomycin, dexamethasone, and acyclovir. The blood and CSF cultures were positive for gram-positive cocci, which was identified as *S. acidominimus* within 24 hours (with testing performed at the Mayo Clinic Laboratories, Rochester Main Campus, Rochester, MN, USA). Fortunately, the *S. acidominimus* was sensitive to ceftriaxone, cefepime, penicillin, meropenem, and vancomycin but resistant to erythromycin. Therefore, vancomycin, acyclovir, and dexamethasone were discontinued after day 1 of hospitalization.

The patient showed dramatic improvement in mentation, with the ability to speak and follow given commands after 24 hours. He was discharged from the hospital within 10 days with a peripherally inserted central catheter (PICC) to allow him to complete the remaining treatment at home.

Follow-up and outcomes

The patient was treated with IV ceftriaxone (2 g every 12 hours) for 6 weeks. A registered nurse administered this medication at the patient's home to ensure 100% compliance. A repeat brain MRI performed a) at 4 weeks (Figure 2C, post-contrast) and b) at 6 weeks ((Figure 3A FLAIR and (Figure 3B) post-contrast) after initial presentation showed remarkable resolution

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Table 1. Summary of cases of pyogenic intracranial infections caused by *Streptococcus acidominimus*.

Year	Country	Age (years)	Gender	Location of infection	Underlying medical conditions	Sample	CSF pleocytosis	Antibiotic treatment (weeks)	Neurosurgical intervention	Outcome	Reference
1988	Japan	41	M	Meningitis	None	CSF	Yes	4 weeks	No	Survived with deafness	[1]
2007	USA	60	M	Brain abscess	None	Pus drainage	Not performed	6 weeks	No	Survived without deficits	[15]
2017	USA	49	M	Pyogenic ventriculitis, meningitis	Diabetes	CSF, blood culture	Yes	6 weeks	No	Survived with residual dizziness, sensorineural deafness and tinnitus	Present study

M – male; CSF – cerebral spinal fluid.

of the pus-like material in the occipital horns of the lateral ventricles. Corresponding improvements were observed in a repeat CSF analysis performed at 4 weeks after initial presentation, including a decrease in the WBC count to 43 cells/μL from 35 418 cells/μL, a decrease in the protein level to 66 mg/dL (0.66 g/L) from 527 mg/dL (5.27 g/L), and negative gram stain and culture results. Clinically, the patient recovered with residual dizziness/imbalance, sensorineural deafness, and bilateral non-pulsatile tinnitus.

Discussion

Pyogenic ventriculitis (PV) is a rare, severe, and debilitating intracranial infection due to inflammation of the ventricular ependymal lining and is associated with pus in the ventricular system [8]. This infection can lead to hydrocephalus and death if not promptly recognized and treated. PV is synonymous with pyogenic intraventricular empyema (PIE), pyogenic ependymitis, and pyocephalus [9]. Other common bacterial intracranial infections include brain abscess, meningitis, cerebritis, and subdural/epidural empyema [10].

The clinical manifestations of primary PV are non-specific, and therefore DW MRI of the brain is considered the most sensitive tool for the early diagnosis of PV [7,11]. Indeed, DW MRI of the brain in our case showed bright intensity representing pus in the dependent/occipital horn of the lateral ventricle, which showed notable improvement after 4 weeks of IV antibiotic treatment, as reflected in Figures 1–3.

PV is commonly caused by *Staphylococcus aureus*, *Neisseria meningitides*, *Enterococcus faecalis*, *Escherichia coli*, *Peptostreptococcus*, *S. intermedius*, *Listeria monocytogenes*, and methicillin-resistant *S. aureus* [12]. The underlying mechanisms include a ruptured brain abscess, meningitis extension from the ventricle, or a neurosurgical device or procedure [6]. No previous case report has described ventriculitis caused by *S. acidominimus*. This lack is surprising, because *S. acidominimus* belongs to the viridans streptococci group, which is known to cause infective endocarditis (*S. mutans*, *S. sanguinis*, *S. salivarius*, and *S. mitis*) and brain abscesses (*S. anginosus*, *S. milleri*, and *S. intermedius*). These bacteria enter the bloodstream (bacteremia) from the oropharynx, typically after dental surgery [13]. However, we did not find evidence of endocarditis, dental surgery/infections, pneumonia, ear infections, or a brain/spinal abscess as a source of infection in this patient. Therefore, the mechanism of *S. acidominimus* induced PV in this patient seemed to be a result of the hematogenous seeding of intracranial infection because both the blood and CSF cultures were positive for this bacterium. Bacteremia is probably not the only mechanism through which PV may occur, although reports of PV are scarce. Given that the brain MRI showed a remote fracture of the lamina papyracea (not shown), we postulate that *S. acidominimus* could have seeded via the nasopharyngeal route. Thereafter, the infection remained nascent until the patient became immunocompromised due to uncontrolled diabetes.

This case report has a few limitations. First, there was a mild motion artifact on the initial MRI (Figure 1), but this artifact was relatively minor and somewhat expected in a seriously ill patient. The image quality could not be improved. Second, a post-contrast MRI of the brain was not performed at the time of hospital admission. However, this lack did not limit our ability to diagnose ventriculitis. Additionally, we did not use literature concerning PV caused by other bacteria as a benchmark to classify the severity of PV, the duration of treatment, or the neurosurgical intervention because we were unaware of any guidelines for the surgical management of PV or dosing of IV

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antibiotics based on PV severity. As per the Micromedex© solution instructions, the maximum recommended daily dose of IV ceftriaxone is 4 gm for meningitis. No dose recommendation exists for PV. In fact, Wang et al. [14] treated a large group of patients who had PV (n=41) at a large medical center in China with neuroendoscopic surgery (NES) and did not find any cases of PV caused by *S. acidominimus*. Moreover, Wang et al. recommended the use of NES in cases with PV only in a large medical center, such as Kunming Medical Center. Therefore, we based our decision to treat our case of PV with 6 weeks of IV ceftriaxone due to a) the lack of consensus for surgical management of PV, b) the favorable clinical and radiological improvement with 6 weeks of IV ceftriaxone treatment, and c) the lack of NES expertise at our facility.

Conclusions

Although recognition of infections with *S. acidominimus* is increasing [2], this article is the first case report of PV and meningitis caused by *S. acidominimus* in North America, and occurred more than a quarter century after this infection was first reported in Japan, as shown in Table 1. Fortunately, despite reports of increasing resistance to antibiotics [2], *S. acidominimus* remains sensitive to ceftriaxone and other beta-lactam antibiotics. Indeed, as summarized in Table 1, patients with intracranial infections caused by *S. acidominimus* recover well (with minimal deficits) with prompt, appropriate, and prolonged IV antibiotic treatment and without neurosurgical interventions such as intraventricular drain placement or NES.

Acknowledgements

The author acknowledges the medical staff and management of Fairview-HealthEast St. John's Hospital for their support and cooperation. The author also thanks Dr. Blake Carlson, neuroradiologist, St. Paul Radiology, for his help in interpreting the imaging of the head. This manuscript was edited for English language usage by American Journal Experts (AJE).

Conflict of interest

None.

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