European Journal of Case Reports in Internal Medicine

Acute Hydrocephalus Associated with *Streptococcus anginosus* Meningitis

Masako Sakurai[1], Takashi Watari[2,3], Itaru Nakamura[4], Kazunari Azuma[1], Hiroshi Homma[1]

[1] Department of Emergency and Critical Care Medicine, Tokyo Medical University, Tokyo, Japan

[2] General Medicine Center, Shimane University, Shimane, Japan

[3] Division of Hospital Medicine, University of Michigan Health System, Ann Arbor, MI, USA

[4] Department of Infection Prevention and Control, Tokyo Medical University, Tokyo, Japan

Doi: 10.12890/2022_003653 - European Journal of Case Reports in Internal Medicine - © EFIM 2022

Received: 21/10/2022
Accepted: 03/11/2022
Published: 22/11/2022

How to cite this article: Sakurai M, Watari T, Nakamura I, Azuma K. Hiroshi H. Acute hydrocephalus associated with Streptococcus anginosus meningitis. *EJCRIM* 2022;**9**: doi:10.12890/2022_003653

Conflicts of Interests: The authors declare there are no competing interests.
Ethical approval: The national and local guidelines do not require permission to publish case reports.
Patient consent: Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

ABSTRACT

Introduction: Meningitis-related acute hydrocephalus is rare, challenging to diagnose, and has a high mortality rate.
Case description: Here we describe the case of a 76-year-old patient diagnosed with bacterial meningitis who developed acute hydrocephalus and subsequently died.
Discussion: Although meningitis-related acute hydrocephalus is usually non-occlusive, occlusive hydrocephalus may also occur. Moreover, worsening hydrocephalus despite cerebrospinal fluid drainage should prompt a diagnosis of obstructive hydrocephalus. In such conditions, potential management strategies include ventriculoperitoneal shunt and endoscopic third ventriculostomy.
Conclusion: In patients with meningitis-related hydrocephalus, worsening despite appropriate antibiotic administration, treatment may be complicated by ventriculitis and obstructive hydrocephalus, which can be fatal. If intracranial pressure is not medically controlled, bilateral decompression craniectomy should be considered as a potential management strategy.

LEARNING POINTS

- The extreme rarity of obstructive hydrocephalus caused by bacterial meningitis can lead to delayed diagnosis and treatment.
- Ventriculoperitoneal shunt and endoscopic third ventriculostomy are the indicated management strategies for early diagnosis of obstructive hydrocephalus.
- Bilateral decompression craniectomy may be an option in such cases.

KEYWORDS

Acute hydrocephalus, bacterial meningitis, non-obstructive hydrocephalus, obstructive hydrocephalus, septic shock, *Streptococcus anginosus*

BACKGROUND

Acute hydrocephalus is a rare complication of meningitis associated with *Escherichia coli* and *Streptococcus pneumoniae* [1–4]. Meningitis-related acute hydrocephalus has a high mortality rate, ranging between 50% and 60% [4, 5]. We report a case of hydrocephalus associated with bacterial meningitis caused by *Streptococcus anginosus*.

European Journal of Case Reports in Internal Medicine

CASE DESCRIPTION

A 76-year-old man experienced loss of consciousness at his home and was transported to our emergency medical centre via ambulance. His medical history revealed diabetes, and his family reported seeing him 2 days previously and finding him well.

Investigations

On arrival, the patient's Glasgow Coma Score was 6 (E1V1M4). Pupils were unequal in size (3.0 and 2.0 mm) and non-reactive to light. The gaze was conjugate and deviated to the right. Heart rate was 90 beats/min, blood pressure was 160/85 mmHg, and SpO_2 was 100% on oxygen delivered at 10 l/min via a mask with a reservoir bag. Respiratory rate was 20 breaths/min, and body temperature was 35.5°C. Laboratory investigations showed a white blood cell count of 26,700/µl, CRP 38.6 mg/l, procalcitonin 19.09 ng/ml, blood urea nitrogen 101.3 mg/dl, creatinine 7.41 mg/dl and potassium 5.6 mEq/l. The results of arterial blood gas analysis indicated pH 7.384, partial pressure of oxygen 178.6 mmHg, partial pressure of carbon dioxide 32.7 mmHg, bicarbonate ion concentration 19.7 mmol/l, base excess −5.50 mmol/l, lactate levels 1.6 mmol/l, and blood glucose concentration 288 mg/dl. Opening pressure during lumbar puncture was 300 mmH_2O. Cerebrospinal fluid examination showed xanthochromia and white turbidity; the cell count and protein concentration were 3,616/mm^3 and 300 mg/dl, respectively. Head computed tomography (CT) showed enlargement of the lateral ventricles, including the inferior temporal horns (*Fig. 1*). The Evans index was 0.384. Extracranial CT findings included abscess formation in the right infratemporal fossa.

Differential diagnosis

A diagnosis of non-occlusive hydrocephalus due to bacterial meningitis was suggested by: the loss of consciousness; elevated white blood cell count, CRP and procalcitonin; elevated cell count and protein levels on cerebrospinal fluid examination; and CT findings.

Treatment

The patient was intubated and placed on a mechanical ventilator. Continuous renal replacement therapy was initiated for the acute kidney injury. After blood and cerebrospinal fluid cultures were obtained, meropenem, vancomycin, acyclovir and ampicillin were initiated.

Outcome and follow-up

On the second day of patient illness, his blood pressure dropped and septic shock was suspected. Consequently, vasopressors were initiated. On the third day, *S. anginosus* was detected in blood and cerebrospinal fluid cultures. Treatment was shifted from meropenem and ampicillin to ceftriaxone, based on antibiotic sensitivity results. The isolated bacterial culture from the blood was positive in BACTEC aerobic and anaerobic bottles (BD, Franklin Lakes, NJ, USA). The isolates were identified by the rapid ID 32 STREP system (bioMérieux Japan Ltd, Tokyo, Japan) with an identification rate of 93.8%. The minimum inhibitory concentration (MIC) of the antibiotics was determined using the MicroScan WalkAwayPlus system (Siemens, Tokyo, Japan) by the broth microdilution method, and the MIC of penicillin G, cefotaxime, meropenem and vancomycin at µg/ml doses of <0.03, <0.12, <0.12 and 1.0, respectively. On Day 3, an incision was made in the area of the right inferior cranial fossa abscess; however, no drainage was performed. On Day 6, fluoroscopy-guided puncture of the right infratemporal fossa abscess was performed, and pus was obtained. Head CT showed worsening hydrocephalus with sediment in the trigone of the lateral ventricles, consistent with ventricular inflammation (*Fig. 2*). An inadequate treatment response was considered, and so a therapeutic and diagnostic lumbar puncture was performed. Cerebrospinal fluid drainage did not improve the patient's level of consciousness. However, fluid examination showed a lower cell count and improvement in inflammation. Therefore, we decided that continuous cerebrospinal fluid drainage was unnecessary. A brain MRI scan was scheduled but could not be performed due to lack of an appointment. On Day 10, the patient's pulse rate and blood pressure decreased, and pupillary light response disappeared. Head CT was repeated and showed aggravation of hydrocephalus (*Fig. 3*). Consequently, an emergency external ventricular drainage was performed. The lateral ventricular pressure was remarkably high (600 mmH2O). The patient's neurological status did not improve, and the brainstem reflexes disappeared despite continuous cerebrospinal fluid drainage. On Day 12, repeat head CT showed a properly placed ventricular catheter, narrowed lateral ventricles, and a blurred boundary between the cerebral cortex and white matter *(Fig. 4)*. However, the patient died the following day.

DISCUSSION

The incidence of acute hydrocephalus in bacterial meningitis is approximately 3%. Causative bacteria include *E. coli*, pneumococci, meningococci and *Listeria monocytogenes* [1–4]. Mortality is high in patients with bacterial meningitis-related hydrocephalus caused by cerebral herniation [4, 5]. Hydrocephalus in bacterial meningitis is usually non-obstructive and caused by impaired cerebrospinal fluid absorption through the arachnoid villi [1, 4, 6, 7]. However, obstructive hydrocephalus may occur if the inflammatory exudate obstructs the foramina of Luschka and Magendie; obstruction typically occurs after 2 weeks, when neutrophils begin to degenerate and fibroblasts proliferate in the exudate [6].

European Journal
of Case Reports in
Internal Medicine

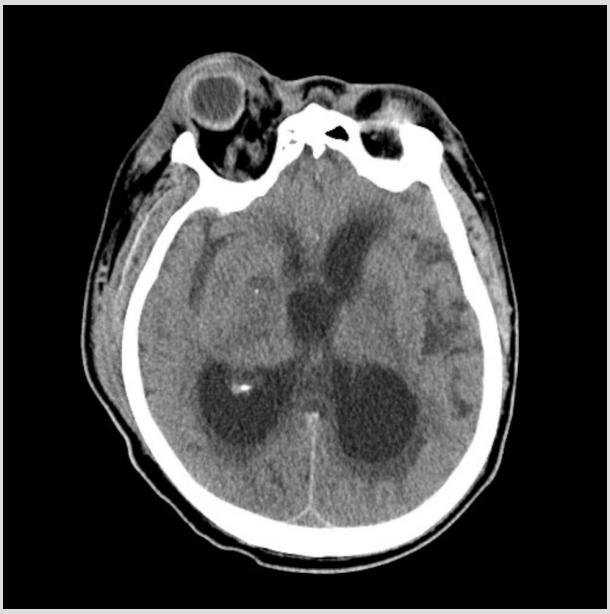

Figure 1. Head computed tomography on patient arrival showing enlargement of the lateral ventricles, including the inferior temporal horns

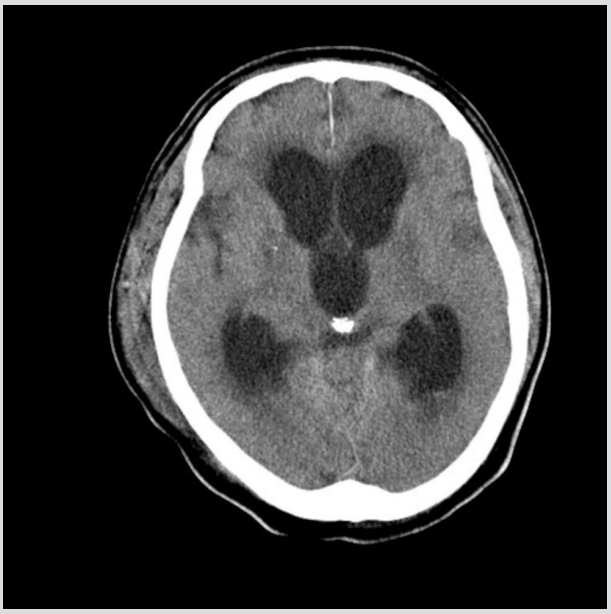

Figure 2. Day 6 of illness, worsening of acute hydrocephalus

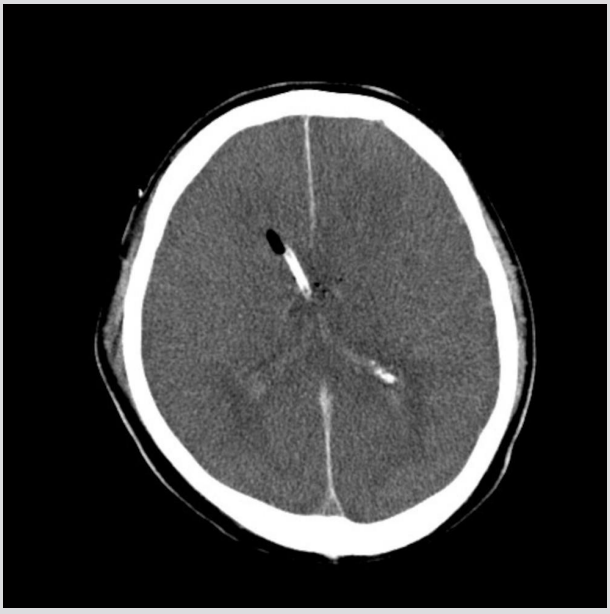

Figure 3. Day 10 of illness, further worsening of acute hydrocephalus

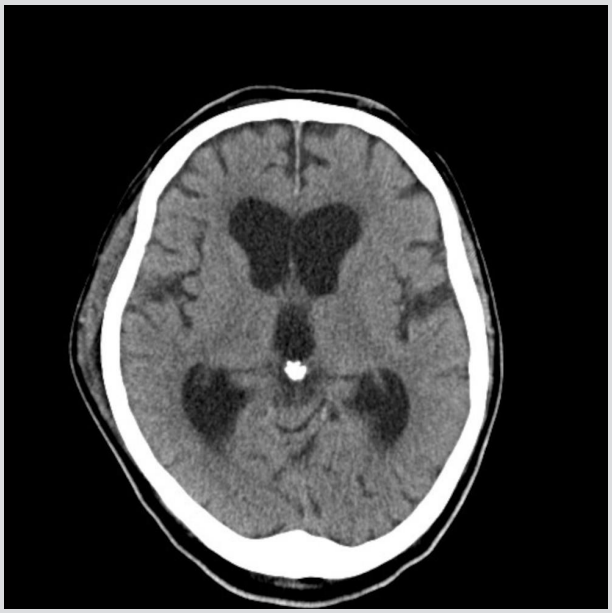

Figure 4. Computed tomography on Day 12 of illness showing blurring of the cortical–white matter junction in the cerebral hemispheres

S. anginosus was detected in the patient's blood and cerebrospinal fluid cultures. Moreover, there have been reports of invasive pyogenic infections caused by *S. anginosus*. Furthermore, patients who developed ventriculitis and multiple abscesses subsequent to meningitis caused by *S. anginosus* later died. However, to our knowledge, there have been no published case reports on bacterial meningitis complicated by hydrocephalus [8–10]. Hence, it remains unclear whether bacterial meningitis caused by *S. anginosus* predisposes to acute hydrocephalus. In this case, no other infections were found besides the right infratemporal fossa abscess infection. Culture from the abscess detected Bacteroides, which was not thought to be related to the patient's bacterial meningitis.

European Journal of Case Reports in Internal Medicine

Old age is a risk factor for the development of hydrocephalus in the acute phase of bacterial meningitis [5]. Persistent intracranial hypertension in patients with bacterial meningitis with acute hydrocephalus may improve after external ventricular drainage [5]. On Day 6, the patient underwent a lumbar puncture due to worsening hydrocephalus but was uneventful. In retrospect, based on the patient's advanced age, ineffectiveness of the multiple lumbar punctures, and presence of deposits in the lateral ventricular triangular pyramids on head CT, occluded hydrocephalus should have been the diagnosis considered, instead of non-occluded hydrocephalus. Decompression craniectomy may be an option when medical intracranial pressure treatment fails to improve intracranial hypertension [11, 12].

CONCLUSION

In patients with meningitis-related hydrocephalus, worsening despite appropriate antibiotic administration, treatment may be complicated by ventriculitis and obstructive hydrocephalus, which can be fatal. If intracranial pressure is not medically controlled, bilateral decompression craniectomy should be considered as a potential management strategy.

REFERENCES

1. Bodilsen J, Schønheyder HC, Nielsen H. Hydrocephalus is a rare outcome in community-acquired bacterial meningitis in adults: a retrospective analysis. *BMC Infect Dis* 2013;**13**:321. https://doi.org/10.1186/1471-2334-13-321
2. Brouwer MC, Van De Beek D. Management of bacterial central nervous system infections. *Handb Clin Neurol* 2017;**140**:349–364. https://doi.org/10.1016/B978-0-444-63600-3.00019-2
3. Durand ML, Calderwood SB, Weber DJ, Miller SI, Southwick FS, Caviness Jr VS, et al. Acute bacterial meningitis in adults. *N Engl J Med* 1993;**328**:21–28. https://www.nejm.org/doi/full/10.1056/NEJM199301073280104
4. Kasanmoentalib ES, Brouwer MC, van der Ende A, van de Beek D. Hydrocephalus in adults with community-acquired bacterial meningitis. *Neurology* 2010;**75**:918–923. https://doi.org/10.1212/wnl.0b013e3181f11e10
5. Wang KW, Chang WN, Chang HW, Wang HC, Lu CH. Clinical relevance of hydrocephalus in bacterial meningitis in adults. *Surg Neurol* 2005;**64**:61–66. https://doi.org/10.1016/j.surneu.2004.10.009
6. Mactier H, Galea P, McWilliam R. Acute obstructive hydrocephalus complicating bacterial meningitis in childhood. *BMJ* 1998;**3168**:1887–1889. https://doi.org/10.1136/bmj.316.7148.1887
7. Settle C, Wilcox MH. Acute obstructive hydrocephalus complicating bacterial meningitis. *BMJ* 1999;**318**:123. https://doi.org/10.1136/bmj.318.7176.123b
8. Fazili T, Riddell S, Kiska D, Endy T, Giurgea L, Sharngoe C, et al. Streptococcus anginosus group bacterial infections. *Am J Med Sci* 2017;**354**:257–261. https://doi.org/10.1016/j.amjms.2017.05.011
9. Suzuki H, Hase R, Otsuka Y, Hosokawa N. Bloodstream infections caused by Streptococcus anginosus group bacteria: a retrospective analysis of 78 cases at a Japanese tertiary hospital. *J Infect Chemother* 2016;**22**:456–460. https://doi.org/10.1016/j.jiac.2016.03.017
10. Lee SB, Jones LK, Giannini C. Brainstem infarcts as an early manifestation of Streptococcus anginosus meningitis. *Neurocrit Care* 2005;**3**:157–160. https://doi.org/10.1385/ncc:3:2:157
11. Baussart B, Cheisson G, Compain M, Leblanc PE, Tadie M, Benhamou D, et al. Multimodal cerebral monitoring and decompressive surgery for the treatment of severe bacterial meningitis with increased intracranial pressure. *Acta Anaesthesiol Scand* 2006;**50**:762–765. https://doi.org/10.1111/j.1399-6576.2006.01038.x
12. Perin A, Nascimben E, Longatti P. Decompressive craniectomy in a case of intractable intracranial hypertension due to pneumococcal meningitis. *Acta Neurochir (Wien)* 2008;**150**:837–842. https://doi.org/10.1007/s00701-008-1596-8