CASE REPORT

Spontaneous Escherichia coli bacterial meningitis mimicking heatstroke in an adult

Kenichiro Ishida 1, Mitsuhiro Noborio 1, Masayuki Nakamura 2, Yohei Ieki 1, Taku Sogabe 1 & Daikai Sadamitsu 1

1Department of Acute Medicine & Critical Care Medical Center, Osaka National Hospital, National Hospital Organization, 2-1-14 Hoenzaka Chuo-ku, Osaka 540-0006, Japan
2Staff Training Division, Osaka National Hospital, National Hospital Organization, 2-1-14 Hoenzaka Chuo-ku, Osaka 540-0006, Japan

Correspondence
Kenichiro Ishida, Department of Acute Medicine & Critical Care Medical Center, National Hospital Organization, Osaka National Hospital, 2-1-14 Hoenzaka Chuo-ku, Osaka 540-0006, Japan. Tel: +81-6-6942-1331; Fax: +81-6-6943-6467; E-mail: kenichiro1224@gmail.com

Funding Information
No sources of funding were declared for this study.

Received: 15 October 2015; Revised: 14 November 2015; Accepted: 20 January 2016

Clinical Case Reports 2016; 4(4): 323–326
doi: 10.1002/ccr3.509

Introduction

Escherichia coli (E. coli) is a frequent pathogen in pediatric bacterial meningitis [1], but is rare in adult forms of the disease [2–4]. In general, acute bacterial meningitis is diagnosed based on clinical signs and cerebrospinal fluid (CSF) analysis. The differential diagnosis includes several diseases that must be ruled out; however, when the symptoms of acute bacterial meningitis are atypical, a delay in treatment can easily result in a serious outcome. Here, we describe the case of a patient with spontaneous E. coli meningitis that developed secondary to a urinary tract infection.

Case Presentation

The patient was a 64-year-old male. He had no history of diabetes mellitus or neurosurgical operations. On a hot, humid day in summer, he was driving his car, the air conditioning system of which was broken. He felt fatigued and stopped to buy a soft drink at a convenience store, where he suddenly went into convulsions. When the emergency medical service arrived, he was unconscious; his tympanic membrane temperature was 42°C. On admission, he was febrile (bladder temperature, 40.5°C), and had a blood pressure (BP) of 88/56 mmHg and a heart rate (HR) of 165 beats per min (bpm). His level of consciousness, as measured using the Glasgow Coma Scale, was E2V2M5, and he had suffered a generalized tonic–clonic seizure. His pupils were of normal size and reacted to light. Neck rigidity was not present and both eardrums were intact. Although his seizure was treated with intravenous diazepam, endotracheal intubation with mechanical ventilation was required for continued loss of consciousness. His initial laboratory data revealed impaired liver function (aspartate transaminase, 54 U/L; alanine transaminase, 19 U/L; total bilirubin, 3.1 mg/dL). Both his serum creatinine level and his blood urea nitrogen were elevated (1.37 mg/dL and 17 mg/dL, respectively), indicating the beginning of renal failure. His blood sugar was normal. Serum C-reactive protein (CRP) was 13.5 mg/dL. Arterial blood gas analysis showed mixed
metabolic and respiratory acid–base disturbances (Table 1). Computed tomography of his head did not reveal any pathology. As these findings and the events leading to his collapse suggested heatstroke, rapid fluid resuscitation and cooling were started. His core body temperature was maintained at ~37°C and he was admitted to the intensive care unit (ICU) for further treatment. Because his hemodynamic instability (BP, 89/52 mmHg; HR, 118 bpm) persisted despite rapid fluid resuscitation, his blood pressure was maintained using an inotrope and a vasopressor.

On day 1 of his hospitalization, the patient’s serum CRP was elevated (16 mg/dL) and a coagulation abnormality (platelets, 3.8 × 10^9/L; fibrin and fibrinogen degeneration products, 92 μg/mL; D-dimer, 20 ng/mL) was noted as well. Urinalysis showed 6–8 pus cells, the presence of gram-negative bacteria, and 0–1 red blood cells per high-power field. Septic shock with disseminated intravascular coagulation (DIC) was considered, for which the patient was intravenously administered 1.5 g ampicillin/sulbactam every 8 h. On day 2 of his hospitalization, the patient’s serum CRP was markedly elevated (31 mg/dL) and his coagulation abnormality had worsened (platelets, 2.4 × 10^9/L). Urgent investigation of an occult bacterial infection other than a urinary tract infection was ordered, for which a lumbar puncture was performed, given the patient’s thrombocytopenia. CSF analysis showed an elevated cell count, increased protein, and a decreased glucose level (Table 2). Based on these results, he was diagnosed with acute bacterial meningitis. Treatment consisted of 2 g of meropenem administered intravenously every 8 h and replacing ampicillin/sulbactam therapy.

By the next day, our patient had improved in response to broad-spectrum antibiotic therapy. His urine and blood cultures, obtained on day 1 of hospitalization, showed the growth of *E. coli* susceptible to ampicillin, cefotaxime, and gentamicin. Accordingly, meropenem was de-escalated to cefotaxime. Despite a negative CSF culture, spontaneous *E. coli* meningitis secondary to a urinary tract infection was diagnosed based on the CSF results and positive urine and blood cultures. On day 13 of hospitalization, the patient was weaned from mechanical ventilation and extubated. He remained in the ICU for 15 days and was then transferred to a clinical ward. After 3 weeks, both his serum CRP and CSF findings had normalized (Table 2). At discharge, his neurological condition was normal.

### Discussion

This case raises two important issues. First, bacterial meningitis can be difficult to recognize: the meningeal

#### Table 2. Serial changes of the cerebrospinal fluid findings.

| Day 2 | Day 4 | Day 7 | Day 10 | Day 18 |
|-------|-------|-------|--------|--------|
| Cell (/mm³) | 732 | 265 | 11 | 12 | 28 |
| Mono (%) | 16 | 26 | 27 | 75 | 67 |
| Poly (%) | 84 | 74 | 73 | 25 | 33 |
| Cl (mEq/L) | 121 | 129 | 127 | 127 | 125 |
| Total protein (g/dL) | 169 | 42 | 32 | 30 | 27 |
| Glucose (mg/dL) | 37 | 46 | 52 | 66 | 53 |
| Serum glucose (mg/dL) | 94 | 84 | 89 | 93 | 89 |

Mono, mononuclear leukocyte; Poly, polymorphonuclear leukocyte; Cl, Chloride.
triad symptoms are not always present and the alteration in mental status can mimic heatstroke. Second, spontaneous E. coli meningitis can progress rapidly, even in an otherwise healthy person.

The classic features of acute bacterial meningitis are fever, nuchal rigidity, and a change in mental status, although not all patients will have all three features [5]. In general, acute bacterial meningitis is diagnosed based on clinical signs and CSF analysis. Our patient was diagnosed on day 2 of his hospitalization following CSF analysis. Previous studies have shown that low serum CRP levels distinguish heatstroke from infection of the central nervous system (CNS) [6]. Thus, in our patient, the high serum CRP level and hyperpyrexia on admission should have suggested bacterial meningitis. There were several reasons why we failed to initially diagnose acute bacterial meningitis. First, because our patient suffered from a generalized epileptic seizure, a detailed clinical history could not be taken. Second, he was admitted to our hospital on a hot, humid day in summer after driving his car, the air conditioning system of which was broken. Thus, his altered mental status with hyperpyrexia was consistent with heatstroke. Consequently, it must be distinguished, for example, from CNS infection.

Acute bacterial meningitis caused by gram-negative bacilli is rare in adults. It has been reported secondary to trauma or neurosurgery, but also as a spontaneous illness [8, 9]. E. coli is a frequent pathogen in pediatric bacterial meningitis [1] but is rare in adult forms of the disease [2–4]. E. coli may cause infections of the bloodstream, urinary tract, biliary system, and peritoneal space. Comorbidities such as diabetes, cirrhosis, and malignancy are risk factors for E. coli bacterial meningitis [10, 11]. Among the features of the disease are rapid progression and high mortality [11, 12]. Although the CSF culture was negative in our patient, E. coli was detected in his blood and urine cultures. Therefore, bacterial meningitis secondary to sepsis from a urinary tract infection was diagnosed, aided by additional information from the CSF findings. The consistently negative CSF cultures were probably due to the prior antibiotic treatment. In general, CSF cultures may be negative even when bacterial meningitis is diagnosed [5, 10].

Although appropriate antibiotic treatment was delayed in our patient, his E. coli meningitis resolved without neurologic complications. This can, at least in part, be attributed to hemodynamic management with fluid resuscitation and the vasopressor therapy administered in the ICU. Our case emphasizes that E. coli meningitis is a serious disease whose early diagnosis, appropriate antibiotic treatment, and hemodynamic management are essential. A delay in its initial treatment often leads to a serious outcome.

**Conclusion**

We present a case of E. coli bacterial meningitis that developed in a healthy person. Bacterial meningitis can be difficult to recognize: the meningeal triad symptoms are not always present and the alteration in mental status can mimic heatstroke. E. coli meningitis is a rare disease, but its clinical course is serious. Early diagnosis, appropriate antibiotic treatment, and hemodynamic management are essential.

**Conflict of Interest**

None declared.

**References**

1. Shrestha, R. G., S. Tandukar, S. Ansari, A. Subedi, A. Shrestha, R. Poudel, et al. 2015. Bacterial meningitis in children under 15 years of age in Nepal. BMC Pediatr. 15:94 2nd ed. BioMed Central Ltd.
2. Sule, A. A., and D. Tai. 2007. Spontaneous Escherichia Coli meningitis in an adult. Crit. Care Shock 10:148–150.
3. Mofredj, A., J. M. Guerin, F. Leibinger, and R. Mamoudi. 2000. Spontaneous Escherichia coli meningitis in an adult. Scand. J. Infect. Dis. 32:699–700.
4. Abhilash K, G. S. 2013. A rare manifestation of Escherichia coli septicemia. Int. J. Res. Med. Sci. 1: 299–300.
5. Durand, M. L., S. B. Calderwood, D. J. Weber, S. I. Miller, F. S. Southwick, V. S. Jr Caviness, et al. 1993. Acute bacterial meningitis in adults – a review of 493 episodes. N. Engl. J. Med. 328:21–28.
6. Dahan, E., S. Dichtwald, E. Amar, P. Sorkine, and A. A. Weinbroum. 2013. Low plasma C-reactive protein level as an early diagnostic tool for heatstroke vs central nervous system-associated infection in the ED. Am. J. Emerg. Med. 31:1176–1180.
7. Bouchama, A., and J. P. Knochel. 2002. Heat stroke. N. Engl. J. Med. 346:1978–1988.
8. Yang, T.-M., C.-H. Lu, C.-R. Huang, H.-H. Tsai, N.-W. Tsai, P.-Y. Lee, et al. 2005. Clinical characteristics of adult Escherichia coli meningitis. Jpn. J. Infect. Dis. 58:168–170.
9. Mancebo, J., P. Domingo, L. Blanch, P. Coll, A. Net, and J. Nolla. 1986. Post-neurosurgical and spontaneous gram-
negative bacillary meningitis in adults. Scand. J. Infect. Dis. 18:533–538.
10. Domingo, P., V. Pomar, N. de Benito, and P. Coll. 2013. The spectrum of acute bacterial meningitis in elderly patients. BMC Infect. Dis. 13:108.
11. Pomar, V., N. Benito, J. López-Contreras, P. Coll, M. Gurguí, and P. Domingo. 2013. Spontaneous gram-negative bacillary meningitis in adult patients: characteristics and outcome. BMC Infect. Dis. 13:451.
12. Bouadma, L., F. Schortgen, R. Thomas, S. Wutke, F. Lellouche, B. Régnier, et al. 2006. Adults with spontaneous aerobic Gram-negative bacillary meningitis admitted to the intensive care unit. Clin. Microbiol. Infect. 12:287–290.