Clinical Case Reports

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CASE REPORT

Spontaneous *Escherichia coli* bacterial meningitis mimicking heatstroke in an adult

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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

Key Clinical Message

Bacterial meningitis can be difficult to recognize: the meningeal triad symptoms are not always present and the alteration in mental status can mimic heatstroke. *Escherichia coli* meningitis is a serious disease whose early diagnosis, appropriate antibiotic treatment, and hemodynamic management are essential.

Keywords

Bacterial meningitis, *Escherichia coli*, gram-negative bacilli, heatstroke.

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, $3.8 \times 10^4/\mu L$; 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, $2.4 \times 10^4/\mu L$). 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 (/mm^3)	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.

Table 1. The patient's laboratory data findings on admission.

Arterial blood gas analysis (O_2 10 L/min)		Complete blood cell count		Biochemistry	
pH	7.573	WBC	$2.2 \times 10^3/\mu L$	TP	6.5 g/dL
pCO_2	14.4 mmHg	Hgb	15.3 g/dL	Albumin	3.2 g/dL
pO_2	171 mmHg	Hct	46.9%	AST	54 U/L
HCO_3	13.3 mmo/L	PLT	$13.2 \times 10^4/\mu L$	ALT	19 U/L
Base excess	−5.6 mmol/L			T-Bil	3.1 mg/dL
Lactate	56 mg/dL	Coagulation		CK	154 U/L
		PT	96%	Glucose	131 mg/dL
		INR	1.03	BUN	17 mg/dL
		APTT	32.9 sec	Creatinine	1.37 mg/dL
		Fibrinogen	619 mg/dL	Na	131 mmol/L
		FDP	34 μg/mL	K	3.9 mmol/L
		D-dimer	9.1 μg/mL	Cl	98 mmol/L
				CRP	13.5 mg/dL

$PaCO_2$, carbon dioxide partial pressure arterial; PaO_2, oxygen partial pressure arterial; HCO_3, bicarbonate; WBC, white blood cell count; Hct, hematocrit; Hgb, hemoglobin; PLT, platelet count; PT, prothrombin time, INR, international normalized ratio; APTT, activated partial thromboplastin time; FDP, fibrin and fibrinogen degradation products; TP, total protein; ALT, alanine transaminase; AST, aspartate transaminase; T-Bil, total bilirubin; CK, creatine kinase; BUN, blood urea nitrogen; K, potassium; Na, sodium; Cl, Chloride, CRP, C-reactive protein.

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, a potentially life-threatening disorder caused by an extreme elevation in body temperature. Heatstroke is characterized by an elevated core body temperature of 40°C or above and central nervous system disturbances that result in delirium, convulsions, or coma. Although heatstroke is commonly seen in environments characterized by hot, humid weather, there are no diagnostic tests specific for heatstroke. Immediate external and internal cooling and support of organ system function (stabilizing the airway, breathing, and circulation) are the two essential therapeutic objectives in patients with heatstroke [7]. 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.