to cefazolin on the basis of the results of susceptibility testing, and she completed a total of 14 days of iv antibiotic therapy. On follow-up, the patient was asymptomatic; blood cultures were negative.

The source of this patient’s symptomatic E. vulneris bacteremia was apparently her PICC, since the organism was recovered from both blood and catheter-tip cultures, clinical improvement occurred following removal of the catheter, and an alternative source of infection was not identified [4]. We are unaware of other reports of catheter infection associated with this organism. Our experience runs contrary to that of Pien et al. who did not believe that E. vulneris was a pathogen [3].

A few other convincing reports of E. vulneris infection appear in the literature. E. vulneris has been implicated as a cause of osteomyelitis [6], wound infection [7], and urosepsis [8]. Our report adds to the growing evidence that E. vulneris is a true pathogen. To our knowledge, this is the first report of iv catheter–related bacteremia due to this organism. Although there was a favorable outcome after antibiotic treatment, whether catheter removal alone would have cleared the patient’s infection remains speculative.

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Aseptic Meningitis Secondary to Carbamazepine Therapy

Drug-induced aseptic meningitis has been reported in patients receiving nonsteroidal antiinflammatory drugs, trimethoprim-sulfamethoxazole, and azathioprine [1]. To date, only three cases of aseptic meningitis associated with the administration of carbamazepine have been reported in the United States [2–4]. We report the fourth case of aseptic meningitis in a patient with trigeminal neuralgia who was treated with carbamazepine, and we review three previously published cases.

A 41-year-old female was admitted to an outside hospital because of a 3-day history of fever, chills, stiff neck, photophobia, myalgia, and diffuse maculopapular rash. She had been diagnosed with trigeminal neuralgia 6 months before admission and was receiving therapy with carbamazepine (600 mg/d). One month after the diagnosis was made, she stopped taking the medication after her symptoms resolved. Ten days before admission, she again started taking carbamazepine (dose, 300 mg/d) because of right-sided facial pain.

On admission she was febrile (temperature, 39.0°C), and physical examination was remarkable only for a diffuse erythematous rash and meningisms. Findings of her initial lumbar puncture showed a WBC count of 25/mm³ (75% neutrophils, 14% monocytes, and 11% lymphocytes), a protein level of 53 mg/dL, and a glucose level of 44 mg/dL. Therapy with iv ceftriaxone was initiated. On her third hospital day, she complained of worsening right-sided facial pain, and her dose of carbamazepine was increased to 600 mg/d. By the fifth hospital day, the results of all cultures of CSF were negative. A latex agglutination test for bacterial antigens in the CSF was negative, as were serum titers for Epstein-Barr virus, cytomegalovirus, echovirus, coxsackievirus, and Mycoplasma species. Antinuclear antibodies were not detected. Therapy with ceftriaxone was discontinued. An MRI scan of her head did not reveal any abnormalities.

Her headaches and fever persisted, so a second lumbar puncture was performed on the sixth hospital day; examination of the CSF obtained showed a WBC count of 61/mm³ (63% neutrophils, 26% lymphocytes, and 9% monocytes), a protein level of 41 mg/dL, and a glucose level of 42 mg/dL. She received therapy with ampicillin, doxycycline, and clarithromycin. Her maculopapular rash persisted. On the seventh hospital day, carbamazepine therapy was discontinued. On the eighth day she continued to have persistent meningeal signs and fevers (temperature to 39.4°C) and was transferred to our institution for further evaluation.

On transfer to our hospital, a diffuse maculopapular rash was noted over the patient’s face, chest, trunk, and extremities; nuchal rigidity was also observed. Physical examination did not reveal any other abnormalities. Her peripheral WBC count was 8,200/ mm³ with 83% neutrophils. Her liver enzymes were elevated (aspartate aminotransferase level, 46 U/L; alanine aminotransferase level, 133 U/L; and lactate dehydrogenase level, 293 U/L). Treatment with antibiotics was discontinued. Within 24 hours the patient became afebrile. Her maculopapular rash resolved completely, and nuchal rigidity was no longer noted. The patient was discharged from the hospital 48 hours later, and her symptoms did not recur. She refused to undergo a repeated lumbar puncture or to be rechallenged with carbamazepine. One year later, she remained free of symptoms.

Our patient had aseptic meningitis secondary to carbamazepine therapy. The temporal relationship of the ingestion of carbamazepine to the onset of signs and symptoms of meningitis and the rapid resolution of signs and symptoms once the drug was discontinued is evidence that carbamazepine was the most likely cause of our

References


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Table 1. Features of four cases of carbamazepine-induced aseptic meningitis (including the present report).

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Underlying disease</th>
<th>Dose of carbamazepine (duration of treatment)</th>
<th>Presenting symptoms</th>
<th>Hospital course</th>
<th>CSF findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 [2]</td>
<td>Trigeminal neuralgia</td>
<td>200 mg/d (2 d)</td>
<td>Fever, stiff neck, maculopapular rash, back pain, paresthesias</td>
<td>Symptoms resolved after drug was discontinued; patient was rechallenged 5 days later, and symptoms recurred; patient was discharged and did not take carbamazepine; symptoms did not recur</td>
<td>WBCs, 341/mm³ (97% PMNs); protein, 167 mg/dL; glucose, 77 mg/dL</td>
</tr>
<tr>
<td>2 [3]</td>
<td>Bipolar disorder</td>
<td>300 mg/d (2 mo)</td>
<td>Fever, maculopapular rash, confusion, myoclonus</td>
<td>Symptoms resolved 2 days after drug was discontinued; patient was rechallenged at time of discharge and was readmitted 3 days later with recurrent symptoms; patient was discharged without recurrence of symptoms</td>
<td>WBCs, 33/mm³ (85% lymphocytes); protein, 65 mg/dL; glucose, 161 mg/dL</td>
</tr>
<tr>
<td>3 [4]</td>
<td>Trigeminal neuralgia</td>
<td>400 mg/d (4 w)</td>
<td>Fever, maculopapular rash</td>
<td>Symptoms resolved after drug was discontinued; patient was discharged without recurrence of symptoms</td>
<td>WBCs, 56/mm³ (50% lymphocytes), protein, 77 mg/dL; normal glucose</td>
</tr>
<tr>
<td>4 [PR]</td>
<td>Trigeminal neuralgia</td>
<td>300 mg/d (10 d)</td>
<td>Fever, stiff neck, headache, photophobia, maculopapular rash</td>
<td>Symptoms worsened when dosage of carbamazepine was increased to 600 mg/d; drug was discontinued 4 days later, and symptoms resolved within 48 hours; patient was discharged without recurrence of symptoms</td>
<td>WBCs, 61/mm³ (75% PMNs); protein, 41 mg/dL; glucose, 42 mg/dL</td>
</tr>
</tbody>
</table>

NOTE. PMN = polymorphonuclear cell; PR = present report.

The three patients whose cases were previously reported presented with fevers and maculopapular rash, as did our patient (table 1) [2–4]; in all four cases, findings in CSF were consistent with aseptic meningitis. Three of these four patients were receiving carbamazepine therapy for trigeminal neuralgia. The symptoms of meningitis were present after the patients received doses of carbamazepine as low as 200 mg/d and resolved within 48 hours once the drug was withdrawn. All four patients received various antibiotic regimens and were hospitalized for as long as 10 days before the diagnosis of carbamazepine-induced aseptic meningitis was made.

As carbamazepine is increasingly being used to treat seizure disorders, psychiatric illnesses, and trigeminal neuralgia, carbamazepine-induced aseptic meningitis is likely to be seen more often. Use of carbamazepine should be considered in the differential diagnosis for patients who present with fevers and maculopapular rash and whose CSF findings are consistent with aseptic meningitis. By recognizing the possibility of carbamazepine-induced aseptic meningitis, we can prevent prolonged hospitalizations and excessive antibiotic use for patients with meningeval irritation secondary to carbamazepine therapy.

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References