ables of age, height, weight, and body surface area. The independent variables were assessed in a univariate linear regression model. Data are presented as mean ± SD. The significance of the results was determined at P < .05.

Of the 125 trainees, 86 subjects (all male) completed the study; 53 were white, 19 were African American, 10 were Hispanic, and 4 were Asian. The mean age (±SD) was 21 ± 3 years, the mean ±SD height was 177 ± 8.3 cm, the mean ±SD weight was 76 ± 10.8 kg, and the mean ±SD body surface area was 1.93 ± 0.07 m².

Significant serum levels of penicillin (mean ± SD, 0.072 ± 0.108 μg/mL) were detected 1 day after all 86 serum samples were obtained. Penicillin was detectable in only 34 (39.5%) of 86 samples after 7 days (mean ± SD level, 0.010 ± 0.010 μg/mL) and in only 3 (3.4%) of 86 samples obtained after 14 days (mean level, 0.016 μg/mL). Penicillin was not detected in any of the 86 samples obtained 21 and 28 days after the injection. Age, height, weight, and body surface area were not significantly related to penicillin concentration determined at 1 and 7 days after injection.

Serum penicillin levels in the study subjects at 1 day after injection are similar to those in a previous report [4]. This accord suggests that the integrity of the serum specimens was maintained throughout collection, storage, transport, and testing, and it supports the validity of the penicillin assay. The cause of the decrease in serum penicillin levels after the first week after our study subjects were injected is unknown.

We conclude that serum levels of penicillin that inhibit GABHS in basic trainees in the army persist for only 1–2 weeks after administration of 1.2 million units of PGB. Despite these observations, there has been no evidence that GABHS infection is not controlled when this agent in this dosage is given to basic trainees in the military early in the training cycle [2]. Our findings, if confirmed, may be important in devising strategies to allow continued control of these infections in basic trainees in the military.

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Escherichia vulneris as a Cause of Intravenous Catheter—Related Bacteremia

In 1982, Brenner et al. [1] applied the designation of Escherichia vulneris to organisms in the Enterobacteriaceae family that were formerly known as Alha group 1, Enteric group 1, or API group 2. The DNA of these organisms was shown to be highly related, thus indicating they were a distinct species. Biochemical reactions that differentiated E. vulneris from other Escherichia species included lack of indole production and ornithine decarboxylase production. Unlike other Enterobacteriaceae, E. vulneris was methyl red positive and did not utilize citrate [2].

Most isolates of E. vulneris have been recovered from wounds (vulneris [Latin]: of a wound). Pien et al. [3] described a series of 12 patients who had E. vulneris isolated from soft tissues. The isolates did not produce infection in mice, and the authors questioned whether the organism was a pathogen that merited treatment in humans. The following case report demonstrates that E. vulneris can cause iv catheter—related bacteremia.

A 62-year-old woman with poor nutritional status because of Crohn's disease presented with rigors and a fever (temperature, to 102.3°F); she was receiving total parenteral nutrition via a peripherally inserted central venous catheter (PICC). On physical examination, the PICC insertion site was free of erythema, discharge, and tenderness. The remainder of the examination was unremarkable. Blood cultures yielded enteric gram-negative bacilli, and she was admitted to the hospital. Treatment with intravenous cefazidime (1 g t.i.d.) and nafcillin (1 g every four hours) was started. The PICC was removed, and semiquantitative culture of the catheter tip also yielded 30 colonies of the same organism [4].

The organism was presumptively identified as E. vulneris by the Vitek GNI automated system (bioMérieux Vitek, Hazelwood, MO). Conventional biochemical tests resulted in definitive identification of the organism as E. vulneris. It did not produce indole, citrate hydrolyse, or ornithine decarboxylase, and it did not ferment sorbitol; it produced lysine decarboxylase and orthonitrophenyl-β-D-galactopyranoside, and it fermented arabinose. The organism was motile [5]. Antibiotic testing based on breakpoint susceptibilities with use of the Vitek system revealed that the organism was susceptible to first-generation cephalosporins.

The patient was afebrile during her entire hospitalization. Her oral intake of food improved; she did not require placement of a new central venous catheter for total parenteral nutrition. Treatment was switched

Reprints or correspondence: Dr. Anne C. Spaulding, Division of Infectious Diseases and Immunology, Department of Medicine, University of Massachusetts Medical Center, 55 Lake Avenue North, Worcester, Massachusetts 01655. Clinical Infectious Diseases 1996;22:728–9 © 1996 by The University of Chicago. All rights reserved. 1058-4838/96/22204-0003/02.00
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 meningismus. 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 on 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 meningitis.

References


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