ately resistant (10) or highly resistant (18) to penicillin. Of the 28 penicillin-resistant strains, two (7.1%) had high-level resistance to ceftriaxone, and 11 (39.3%) were intermediately resistant to ceftriaxone. Twenty (71.4%) of the 28 strains had high-level resistance to erythromycin, and one additional strain was intermediately resistant to erythromycin.

Three fluoroquinolones, ciprofloxacin, levofloxacin, and trovafloxacin, were used for susceptibility testing with Etest methodology. The MIC$_{90}$ values of these agents were 1.5, 1.5, and 0.19 µg/mL, respectively, and the ranges of MICs were 0.38 to 1.5, 0.75 to 1.5, and 0.094 to 0.19 µg/mL, respectively.

Statistical differences were demonstrated for penicillin susceptibility on the basis of the specimen source of the pneumococci; blood isolates were more susceptible to penicillin than were either respiratory ($P = .0085$) or ear ($P = .0110$) isolates. There was no statistical difference in penicillin susceptibility between ear and respiratory isolates.

A decline in penicillin susceptibility among $S$. pneumoniae strains isolated at our center was first seen in 1993. Similar to results of surveys of susceptibility testing at other medical centers [3], our findings indicate that pneumococcal strains that are resistant to penicillin are often resistant to other classes of antibiotics and so deserve to be referred to as multidrug resistant.

Newer fluoroquinolones have an expanded spectrum of antibacterial activity that includes multidrug-resistant $S$. pneumoniae and have been used for the empirical treatment of community-acquired pneumonia [1]. Trovafloxacin was the most active fluoroquinolone tested in the present investigation. The MIC$_{90}$ of trovafloxacin was ~10-fold lower than that of levofloxacin, the other newer fluoroquinolone examined in this study, and ciprofloxacin, an older fluoroquinolone.

 Significant differences were seen in penicillin susceptibility between blood isolates recovered in the present study and respiratory and ear isolates. Previous work from other centers [4–7] supports our findings. Bédos and colleagues [4] acknowledged that the invasive serogroups 1, 3, 4, 5, 7, 11, 15, and 18 rarely carry antibiotic-resistant phenotypes. They theorized that this occurrence could explain the differences in penicillin susceptibility seen between invasive and noninvasive strains.

**Spinal Epidural Abscess After Tattooing**

Acute spinal epidural abscess (SEA) is encountered infrequently and accounts for 0.2–1.2 cases per 10,000 admissions to tertiary care hospitals [1]. Although it is one of the causes of back pain, the diagnosis is usually not considered initially unless the presentation is classical [2]. We report a case of SEA caused by a tattoo that became infected.

In August 1997 a 25-year-old female presented with a history of bilateral leg weakness and paresthesias below the umbilicus for 1 day and midback pain for 1 week. She was unable to urinate for 3 hours before admission. She denied bowel incontinence, fever, chills, or trauma to the back. She had been tattooed on her left buttock 2 weeks prior to admission. One week later, she noticed irritation and drainage from the tattoo.

At physical examination, lower extremity weakness was noted as well as serous drainage and pustular lesions over the tattoo. Her medical history included tonsilllectomy, left knee arthroscopy in 1988 and 1991, and recurrent ear infections. She had received treatment with phenytoin and fenfluramine from November 1996 to July 1997. She denied any intravenous drug use. The erythrocyte sedimentation rate was 70 mm/h, and the WBC count was $14.3 \times 10^9/L$ with 16% band forms. Liver function tests and electrolyte and calcium levels were normal.

Progressive thoracic myelopathy at the T10 spinal level was diagnosed. A gadolinium-enhanced MRI scan of her thoracolumbar spine revealed a posterior epidural abscess at T9 through T10 with spinal cord compression (figure 1). She was seronegative for HIV and hepatitis B surface antigen, and hepatitis B surface antibody was nonreactive. A transthoracic echocardiogram showed mild aortic incompetence without vegetations.

She underwent emergent decompression laminectomies at spinal levels T8 through T10 to drain the SEA. She was treated with dexamethasone (10 mg) before surgery. Cultures of specimens

**References**

from the tattoo and epidural abscess yielded *Staphylococcus aureus* (oxacillin-susceptible, β-lactamase-positive); isolates from both sites had similar susceptibilities. She was then treated with intravenous nafcillin (12 g/d) continuously for 4 weeks followed by oral cephalexin (4 g/d) for 4 weeks. The subsequent clinical course was uneventful except for left subclavian vein thrombosis at the site of the central venous catheter. Her neurological deficits had completely resolved by January 1998.

Tattooing can transmit viruses like hepatitis B virus and cause local and systemic bacterial infections [3]. Currently, localized infections probably occur with improper wound care [4]. To our knowledge, this is the first case of SEA attributed to tattooing. Although the prevalence of tattoos among intravenous drug users is higher than that among other groups [5] and SEAs have been described in these individuals, this patient denied intravenous drug use.

Most SEAs are caused by *S. aureus* [2], although gram-negative organisms are frequently isolated [6]. Approximately three-fourths of the cases have an identifiable source [7]. MRI is highly sensitive for diagnosis [8] and is also used for subsequent follow-up. Accepted care includes surgical drainage and antibiotics [9], although medical management is used for selected patients [7].

Rare as they are, SEAs are frequently the subject of litigation. Maslen et al. [8] reported that of 16 patients with SEAs three had considered and one was pursuing litigation against physicians. In this case, the patient is pursuing litigation against the tattooing agency. This case demonstrates the following: serious bacterial infections may complicate cosmetic tattooing, early imaging plays a crucial role in the appropriate management of SEA, and a careful history and physical examination can usually identify the source of infection.

Ashish Chowfin, Anil Potti, Anil Paul, and Paul Carson

Department of Medicine, Division of Infectious Diseases, University of North Dakota School of Medicine and Health Sciences and MeritCare Health System, Fargo, North Dakota

References