CASE REPORT

Lumbar Vertebral Osteomyelitis with Mycotic Abdominal Aortic Aneurysm Caused by Highly Penicillin-Resistant
Streptococcus pneumoniae

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We present a case of vertebral osteomyelitis with an adjacent abdominal aortic mycotic aneurysm caused by a highly penicillin-resistant Streptococcus pneumoniae strain. The occurrence of all three phenomena in a single patient has not been previously described. This presentation offers the opportunity to reflect on the increasing incidence of S. pneumoniae as a resistant pathogen, the treatment of highly penicillin-resistant S. pneumoniae, and the etiologic agents of both vertebral osteomyelitis and mycotic aneurysm.

CASE REPORT

A 52-year-old woman with no significant medical history presented to Thomas Jefferson University Hospital in March of 1999. She complained of low, central back pain without radiation. The pain had begun after a fall approximately 2 months before admission. The pain was persistent despite the use of over-the-counter nonsteroidal anti-inflammatory agents and a muscle relaxant. The pain was associated with a poor appetite, loss of 20 pounds, and subjective fevers. The patient denied chills or night sweats. She also denied current or past headache, stiff neck, rhinorrhea, sore throat, otalgia, or cough. There was no chest pain, shortness of breath, or dyspnea on exertion. There was no abdominal pain, nausea, vomiting, or flank pain.

In the week prior to admission, the back pain had intensified and was now associated with lower-extremity weakness, urinary urgency, paresthesias, and constipation. She was admitted for evaluation of symptoms consistent with spinal cord compression.

Physical exam revealed a temperature of 98.5°F, a pulse rate of 100 beats per min, 18 respirations per min, and a blood pressure of 150/80 mm Hg. The heart rate was regular, without a murmur, rub, or gallop. The lungs were clear without rales, rhonchi, wheezes, or crackles. The abdomen was soft and non-tender without organomegaly. The upper extremities were neurologically normal. The lower extremities had bilateral tenderness without organomegaly.

A computed tomographic scan of the abdomen demonstrated a 5-cm juxtarenal abdominal aortic aneurysm located directly superior to an area of extensive bony destruction of the vertebral body at the L3-to-L4 level. In the abdomen, the aorta, iliacs, and inferior vena cava. Dilation of the aorta was also demonstrated and was felt to be consistent with a mycotic aneurysm. Severe tricompartment stenosis was shown at the L3-to-L4 and L4-to-L5 levels.

A magnetic resonance image of the spine (Fig. 1) was consistent with discitis and osteomyelitis at L3 to L4, with prevertebral and psoas microabscess formation present. Infection was found to extend into the paraspinal space, consistent with discitis and osteomyelitis. There was obliteration of the fat plane surrounding the psoas muscle, as well as the fat plane posterior to the aorta, iliacs, and inferior vena cava. Dilation of the aorta was also demonstrated and was felt to be consistent with a mycotic aneurysm.

A computed tomographic scan of the abdomen demonstrated a 5-cm juxtarenal abdominal aortic aneurysm located directly superior to an area of extensive bony destruction of the end plates of L3 and L4, with surrounding inflammatory changes of the psoas muscle.

After imaging and evaluation by neurosurgeons, the Infectious Diseases Service was consulted. The differential diagnosis included bacterial and mycobacterial infection, as well as malignancy. The bacteria considered likely were Staphylococcus aureus and Salmonella species. Decadron was initiated for the spinal cord compression. Blood cultures were drawn, and em-

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sensitivities of the organism were as follows: penicillin resistant cocci which were identified as *Streptococcus pneumoniae* after admission. Despite aggressive resuscitative measures, she expired 48 h later, the patient was found to be unresponsive and pulseless. A vascular surgery consultation was requested. Shortly thereafter, the patient was noted to have new abdominal pain and urgent surgery and implantable hardware and devices, gram-negative organisms, beta-hemolytic streptococci, and enterococci. Rarer pathogens include fungi (usually in an immunocompromised host) and tubercle bacilli. *S. pneumoniae* has been occasionally associated with sickle cell disease but is otherwise rarely reported as a cause of vertebral osteomyelitis.

Recent case reports have brought attention to *S. pneumoniae* as an important pathogen in vertebral osteomyelitis (4, 13, 17, 24). In general, increasing resistance to penicillins and cephalosporins has been well documented (6, 15) although resistance to antibiotics is not clearly associated with any increase in mortality (21). Accordingly, antibiotic resistance has also been recognized in vertebral osteomyelitis (3, 8, 12).

Our patient did not have any antecedent history that would suggest an initial focus of pneumococcal infection. The case presented did involve a mycotic aneurysm which involved the abdominal aorta adjacent to the infected lumbar vertebrae. A rupture of the aneurysm caused her death. Whether the initial process was a vertebral osteomyelitis which eroded the abdominal aorta or a mycotic aneurysm which eroded posteriorly to cause vertebral osteomyelitis is not known.

The term mycotic aneurysm was originated by Osler in the Gulstonian lectures of 1885 to designate the pathophysiology involved in the formation of arterial dilation due to septic degradation of the arterial wall. The mushroom shape of the aneurysm led to the term “mycotic,” which has since been applied primarily to fungal infections but still carries its original designation for intravascular infections. The bacteria were believed to originate from infected heart valves during an episode of infective endocarditis and embolize to peripheral arteries. Most infected abdominal aneurysms arise either from direct extension of an adjacent infectious process or by microbial arteritis, in the absence of known infective endocarditis (23).

Mycotic aortic aneurysm is a recognized complication in patients who have lumbar vertebral osteomyelitis (22). The bacteriology of mycotic aneurysms includes *S. aureus* and salmonellae, but *S. pneumoniae* has also been reported, albeit infrequently (5, 11, 14). A penicillin-resistant pneumococcus has been reported in only one case of a mycotic aneurysm (1).

The treatment of infections caused by highly penicillin-re-
sistant *S. pneumoniae* remains controversial. Serious infections such as bacteremias and meningitis are difficult to treat because of poor drug penetration into the central nervous system and high burdens of organisms. Various regimens have been tested, including high-dose penicillin, chloramphenicol, carbapenems, high-dose cephalosporins, quinolones, vancomycin, and rifampin (10). There is no uniformly recommended antibiotic or antibiotic combination, and research is needed to determine optimal therapy (9, 16).

While unusual, outbreaks of pneumococcal disease have occurred and a recent report highlights an outbreak of multiresistant pneumococcal pneumonia (20). Prevention of pneumococcal infection with multivalent polysaccharide vaccine is felt to be effective, underutilized, and perhaps one of the last lines of defense against seriously invasive disease caused by an organism with rapidly expanding resistance (19).

The case presented highlights many important points about several processes. We believe this is the only reported case to involve highly penicillin-resistant *S. pneumoniae* and vertebral osteomyelitis with an adjacent mycotic aneurysm. It also provides an opportunity to emphasize the necessity of a coordinated effort between the clinician and the microbiology laboratory to develop a reasonable therapeutic plan.

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**REFERENCES**