

Table. Results of deletion typing for *Mycobacterium tuberculosis* and *M. bovis* in goats, Nigeria*

Region of difference	<i>M. tuberculosis</i>	<i>M. bovis</i>
RD1	+	+
RD4	+	–
RD9	+	–
RD12	+	–

*+, present; –, absent.

with advanced TB may occur, given the endemic nature of TB in humans in Nigeria (10). TB cases caused by EAI strains have been found in humans in southwestern Nigeria (4; S.I. Cadmus et al., unpub. data), a finding that supports zoonotic transmission of this organism from humans to goats. However, different lineages of *M. tuberculosis* may vary in host range, and EAI genotype strains may be adapted to human and animal hosts. Conversely, human-to-animal transmission of *M. tuberculosis* has been reported in Nigeria relative to infection in cattle (3,4). Thus, confirmation of TB in goats supports the possibility of risk for TB transmission between humans and animals in Nigeria.

This study should be interpreted in the context of its limitations. Because the sources of the animals were unknown, we could not determine whether the organisms were imported from a neighboring country (3). In addition, we lacked information on the breed and condition of the animals. However, we have identified *M. tuberculosis* and TB in goats in Nigeria. Additional studies of other slaughterhouses in Nigeria are needed to confirm our results.

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Streptococcus suis Meningitis, Hawaii

To the Editor: *Streptococcus suis* is a swine pathogen and zoonotic agent responsible for septicemia and meningitis (1). *S. suis* is in emergence in some Asian countries. Indeed, this pathogen has been described as the most and second-most common cause of adult meningitis in Vietnam and Thailand, respectively (2,3). Moreover, during an outbreak in People's Republic of China in 2005, 39 of 215 patients died from *S. suis* diseases (4). On the other hand, only 2 human *S. suis* cases have been reported in the United States (5,6). Here, we describe a first case of human *S. suis* meningitis in Hawaii.

The patient, a 34-year-old Tongan male with no medical history who worked as a coconut tree trimmer, was singing in his church choir when he developed an acute-onset, global headache and emesis. Upon hospital admission, he described a week of

antecedent nonspecific symptoms for which he had taken nonsteroidal anti-inflammatory drugs without relief.

On examination, he was afebrile, tired-appearing but alert, and with stable vital signs. He presented mild meningismus and photophobia; no rash was observed. Blood tests showed 27,600 leukocytes/ μ L with 65% neutrophils; 168,000 platelets/ μ L; hemoglobin 17.3 g/dL; and creatinine 1.4 mg/dL. A computed tomography scan of the head was read as showing substantial motion artifact and a possible cerebral mass. Nuclear magnetic resonance imaging (MRI) of the head showed no mass, but T2-weighted images (postgadolinium) suggested both increased grey/white matter contrast consistent with diffuse cortical edema, and vascular congestion/inflammation of the sulci.

Cerebrospinal fluid (CSF) obtained from a lumbar puncture had 2,770 leukocytes/ μ L with 94% neutrophils; glucose 30 mg/dL; and protein 230 mg/L. A Gram stain of the CSF showed numerous gram-positive cocci, mostly in pairs and short chains (Figure). Empiric intravenous therapy with dexamethasone, vancomycin, and ceftriaxone was administered for possible pneumococcal meningitis.

Blood cultures grew a *Streptococcus* species, later identified by 16S rRNA gene sequencing as being *S. suis*, sensitive to penicillin, vancomycin, and ceftriaxone. The isolate was assigned to serotype 2 by the coagglutination test (7) and shown by Western blot to produce sulysin, extracellular protein factor and muramidase-released protein, which are virulence markers often associated with highly virulent strains of Eurasian, but not North American, origin (1,8). A strain of this phenotype was responsible for a previous US *S. suis* meningitis case, but the patient had been infected in the Philippines (5; unpub. data).

Upon identification of the *S. suis* isolate, the patient was questioned

about swine contact. He described slaughtering by hand several noncommercially raised pigs over the preceding several weeks for a church-related luau. The patient did not recall any clear incident of mucosal exposure to pig blood or secretions. The exact route of *S. suis* infection for humans is not known. However, most cases have been linked to accidental inoculation through skin injuries (1). The patient did not wear gloves, masks, or any other protective equipment during the prolonged process of butchering the pigs, and his exposure to pig blood, skin, and internal organs was extensive. He sustained multiple small cuts on his hands during butchering. No other church members who participated in preparing pigs for the luau became ill.

The patient was treated with ceftriaxone and a 4-day course of dexamethasone. His headache and meningismus improved progressively, and he was discharged after 6 days to complete a 2-week course of intravenous ceftriaxone. However, 1 day after discharge, the patient complained of headaches

and mild-to-moderate bilateral hearing loss. He was readmitted; a repeat lumbar puncture showed resolving CSF pleocytosis, and an MRI showed that his prior radiographic findings had normalized. The symptoms, attributed to residual meningeal/cerebral edema, resolved quickly after the reintroduction of steroids. Audiometric testing suggested mild sensorineural hearing loss in the right ear. The patient completed the remainder of his intravenous ceftriaxone course and was discharged on a 2-week course of amoxicillin and oral steroids.

He was again admitted 2 days after completing treatment, with disabling dizziness. On exam he showed new torsional nystagmus, more pronounced with left lateral gaze, consistent with a right peripheral vestibulopathy. An MRI of the head was again normal. Oral dexamethasone promptly resolved his vestibulopathy, and the patient was discharged on a slow steroid taper. After a month, dexamethasone was discontinued. The patient has been asymptomatic since, and his hearing loss has resolved fully.

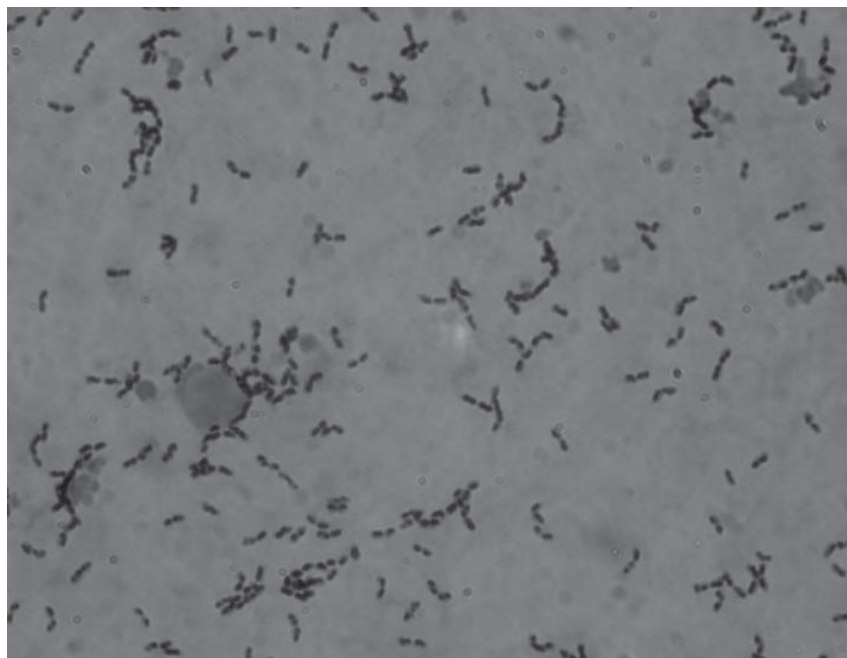


Figure. Gram-positive cocci, mostly in pairs and short chains, found in cerebrospinal fluid from a 34-year-old man with *Streptococcus suis* meningitis. The sample was not centrifuged before staining. Original magnification $\times 1,000$.

The role of steroids in treating patients with *S. suis* infection remains unclear, although this case illustrates that the inflammation associated with this infection can be profound and can require prolonged steroid therapy. Since at least 2 cases of relapse have been reported after 2 and 4 weeks of treatment (1), prolonged therapy should be considered for infections caused by this pathogen. Hearing loss from *S. suis* meningitis occurs frequently and can be irreversible (1). Hawaii's swine industry is characterized by small herds and a high degree of concentration (9). This case of human *S. suis* meningitis in Hawaii emphasizes the need for these data to be generated and made available. Indeed, this bacterium is increasingly recognized as a significant zoonotic agent in Asia; although it remains a relatively rare cause of human infection elsewhere, persons in close occupational contact with pigs or pork products are at higher risk than others (1). Increasing awareness of this disease is expected to help counter human *S. suis* infections.

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Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have 1 Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

**Chorioamnionitis
and Neonatal
Sepsis from
Community-
associated MRSA**

To the Editor: Chorioamnionitis is a common cause of maternal and neonatal illness and death (1), but chorioamnionitis attributed to *Staphylococcus aureus*, including methicillin-resistant *S. aureus* (MRSA), is reported infrequently (2–5). In the context of the rising incidence of community-associated MRSA (CA-MRSA) infections (6), we report an apparent case of CA-MRSA chorioamnionitis.

The patient, a 31-year-old woman with polycystic ovary syndrome and hypothyroidism, had 1 prior pregnancy but no viable offspring. After a clomiphene-assisted conception, routine ultrasound at 21 weeks' gestation identified a shortened cervix (5 mm). The patient declined amniocentesis for cerclage and was treated with pelvic rest and vaginal progesterone. Five days later, she arrived at the emergency department with foul-smelling vaginal discharge. At this time, the patient was afebrile and hemodynamically stable, had no abdominal pain, and had a leukocyte count of 9.5×10^3 cells/mm³.

Premature rupture of membranes was diagnosed, and the patient was admitted and administered intravenous ampicillin and azithromycin. Nine days into treatment, at 23 weeks' gestation, 210 hours after membrane rupture, a 415-g live-born girl was delivered spontaneously in footling breech with Apgar scores of 1 (1 min) and 5 (5 min). During admission, the mother was never febrile and did not complain of abdominal tenderness or chills. The highest leukocyte count was 12.4×10^3 cells/mm³. The mother was discharged the day after delivery without further complications. At 6-week follow-up, she remained well, with no signs of infection.