ornithine, lysine, and ribose and therefore was not B. suis. Because of the high indices with alanine, asparagine, and glutamate and a low index with ribose, it was identified as B. melitensis (table 1). Agglutination only with monospecific antiserum to B. abortus identified the isolate as B. melitensis biovar 2 [2].

To our knowledge, there are no reports of B. suis isolation from the Arabian peninsula, and pig farming is forbidden by Muslims; however, B. melitensis biovar 2 has been recovered in the Middle East. B. suis biovar 3 and B. melitensis biovar 2 have similar dye resistance characteristics and are agglutinated only by monospecific antiserum to B. abortus [2]. Up to now, they have been distinguished by the resistance of B. melitensis to lysis by Fi phage at RTD and to lysis by Tb phage at RTD × 10,000. Most strains of B. melitensis are lysed only by Berkeley and Izatnagar phages [2], although some strains from the Middle East and Europe are lysed by Wb phage [3] However, the oxidative utilization pattern of isolate 92/72 showed that it was a variant of B. melitensis biovar 2. We are unaware of any previous report of a strain of B. melitensis that was sensitive to Fi and Tb phages. This identification was unlikely to be due to phage variation, as the phage batches were regularly checked against type strains to ensure specificity.

Bryan Worsley, Stewart Goodwin, Keith Jahans, and Camille Atallah
Department of Medical Microbiology, Faculty of Medicine and Health Sciences, United Arab Emirates University, and Department of Surgery, Tawam Hospital, Al Ain, United Arab Emirates; and FAO/WHO Collaborating Centre for Reference and Research on Brucellosis, Central Veterinary Laboratory, New Haw, Weybridge, Surrey, United Kingdom

References

Liver Abscess Due to Streptococcus sanguis

We report a case of liver abscess due to Streptococcus sanguis. We believe that gastric mucosal biopsy was the most likely source of infection.

A 39-year-old male was admitted to the hospital for investigation of a liver lesion. Two weeks before presentation, he had developed a fever while visiting relatives in another state and was admitted to a hospital there. Abnormal results of liver function tests prompted the performance of abdominal CT, and an 8-cm lesion was found in the right lobe of the liver. Treatment with intravenous ceftriaxone resulted in defervescence, and he was discharged with instructions to take oral cefuroxime; he was scheduled to undergo a liver biopsy as an outpatient. He returned home and was admitted to this hospital for the biopsy and for parenteral antibiotic therapy.

Findings on physical examination were normal. Apart from an elevated serum alkaline phosphatase level (193 U/L; normal range, 36–128 U/L), results of routine biochemistry and liver function tests were normal. An abdominal CT scan revealed an 8-cm lesion in the anterior portion of the right lobe, with multiple hypoechoic areas (figure 1). Ultrasonographically guided aspiration was done, and 60 mL of brown sanguineous fluid was obtained from two of the hypoechoic areas. Inflammatory cells were seen on microscopy, and culture of the aspirate yielded pure growth of S. sanguis. Cytology did not reveal any evidence of malignancy. Serologic tests for evidence of amebic or echinococcal infection were negative. A barium enema study showed diverticulosis of the sigmoid colon.

Two months before this admission, gastroesophageal reflux disease had been diagnosed. Upper gastrointestinal endoscopy with gastric and esophageal mucosal biopsy was done, followed by studies of pH and motility. The patient was taking omeprazole. He had no history suggestive of diverticulitis, and he had no

Figure 1. Abdominal CT scan showing an 8-cm hepatic lesion with multiple hypoechoic areas that was caused by Streptococcus sanguis infection.
risk factors for HIV infection. Treatment with intravenous penicillin G (2.5 million units every 4 hours) plus oral metronidazole (500 mg every 8 hours) for 1 week resulted in a reduction in the size of the abscess. Domiciliary treatment with intravenous ampicillin (2 g every 8 hours) and oral metronidazole (500 mg every 8 hours) was continued for an additional 3 weeks.

A follow-up ultrasonographic examination showed that the hypoechoic lesions within the liver had completely resolved, and the patient remained afibrile.

Recent reports have implicated *Streptococcus milleri* as a frequent cause of pyogenic liver abscesses [1]. Other etiologic organisms that have been reported include *Escherichia coli*, *Pseudomonas* species, *Staphylococcus aureus*, anaerobes, and *Mycobacterium tuberculosis* [2, 3]. *S. sanguis* is a rare cause of liver abscesses, and to our knowledge, this infection has been reported only once before [4]. *S. sanguis* is a normal inhabitant of the mouth and has caused endocarditis and prosthetic joint infections [5].

Liver abscess has been reported as a rare complication of injection sclerotherapy for esophageal varices. There have also been isolated case reports of the occurrence of liver abscesses following duodenal biopsy in an immunosuppressed individual [2] and after vasoconstrictor injection of a bleeding duodenal ulcer in another report [3]. For our patient, the occurrence of the abscess within 3 months after endoscopy suggests that the gastric or esophageal mucosal biopsy was the predisposing event that led to its development secondary to *S. sanguis* bacteremia. The patient had no history suggestive of diverticulitis (which can also be a predisposing factor), and *S. sanguis* is not normally found in the colon.

Mucosal biopsy may rarely lead to serious metastatic infections, even in otherwise healthy, immunocompetent adults. Because *S. sanguis* is a commensal in the mouth, it can rarely cause pyogenic liver abscesses. No guidelines exist for the use of prophylactic antibiotics after endoscopic mucosal biopsy, except in the presence of valvular heart disease. We do not propose that prophylactic antibiotics be used routinely following mucosal biopsy. However, in view of the increasing reliance on mucosal biopsies to diagnose *Helicobacter pylori* disease, the incidence of liver abscesses complicating these procedures may also increase.

**Sushil George, Ashok Wadhwa, Karl Mersich, and C. Richard Magnussen**

*Department of Medicine, St Mary’s Hospital, and the University of Rochester School of Medicine and Dentistry, Rochester, New York*

**References**


Obstruction of the Left Main Coronary Ostium Due to an Aortic Vegetation: Survival After Early Surgery

Coronary embolization of vegetations is a frequent complication of infective endocarditis [1], but occlusion of a coronary ostium due to an aortic valve vegetation is a rare and life-threatening event. Death occurred in all nine previously reported cases in which the coronary ostium was occluded by an aortic valve vegetation [2–8]. We report the case of a patient who had an obstruction of the left main coronary artery that was due to an aortic valve vegetation and who survived after undergoing surgery. We describe, to our knowledge, the first case in which a patient who had an occlusion of a coronary ostium due to an aortic valve vegetation survived.

A previously healthy 43-year-old man was admitted to our hospital in January 1990 for lumbar pain, fever, and chills that occurred a few weeks after he had received a barium enema for evaluation of chronic abdominal pain. Physical examination revealed lumbar stiffness and a systolic and diastolic murmur at the aortic area. An electrocardiogram (ECG) showed sinus tachycardia. A transthoracic echocardiogram revealed an aortic vegetation with moderate left ventricular dilatation. Blood cultures yielded *Streptococcus bovis*.

Treatment with iv amoxicillin (2 g six times daily) and im gentamicin (200 mg daily) was initiated. Complete bed rest was ordered because of the lumbar pain, and thus an anticoagulant (7,500 units of subcutaneous heparin three times daily) was prescribed. Abdominal echography and colonoscopy did not reveal colonic disease. Despite the fact that the results of a blood culture were negative for *S. bovis* 2 weeks after antibiotic therapy was initiated and that biological parameters of inflammation decreased, a transesophageal echocardiogram showed that the mobile aortic vegetation had increased in size (10 mm × 15 mm), that the posterior aortic valve (figure 1) was perforated, and that there was grade 3/4 aortic regurgitation.

Therapy with oral captopril (12.5 mg twice daily) was initiated. One week later, dyspnea was noted while the patient was resting in bed. A chest roentgenogram revealed severe pulmonary congestion. An ECG showed decreased amplitude of R waves, slight ST segment elevation and inverted T waves in the anteroseptal territory, and ST segment depression and inverted T waves in the