Brucella or Staphylococcus. Triple antibiotic intravenous treatment with amoxicillin, 1 g, and chloramphenicol, 1 g every four hours, and metronidazole, 100 mg bid, was started, and the patient was scheduled for urgent surgery. Unfortunately, 24 hours after admission, he sustained a fatal cardiac arrest; there was electromechanical dissociation of the heart, suggestive of a possible ruptured aorta. Blood culture was positive for B melitensis; the agglutination titer level was 1:10,240, with an IgG titer of 1:1,250 (Table 2), all diagnostic of Brucella endocarditis.

**DISCUSSION**

Our previous study suggested that a combination of medical therapy and surgery is necessary for the successful eradication of Brucella infective endocarditis. In the active stage of infective endocarditis, annular, or myocardial abscesses are associated with high mortality; aggressive surgical intervention may be lifesaving in these patients.

The first of these three patients had Brucella aortic endocarditis with vegetation on the aortic valve. The endocarditis had recurred nine months after a period of eight weeks of antibiotic therapy alone, with abscess formation and a fistula connecting the aorta to the right ventricular outflow. This illustrates and supports our previous conclusion that the Brucella organism is slowly destructive and difficult to eradicate with medical therapy alone.

Some important clinical clues may suggest deep tissue invasion: the aortic valve is usually involved, and the bacteria are virulent; in these three cases, the aortic valve was involved, and the organism was B melitensis. The electrocardiographic changes of conduction defects can be a valuable clue to myocardial invasion; however, in these three cases, there was no evidence of conduction defect. The onset of pericarditis, with effusion, may indicate an annular abscess rupturing into the pericardial space; 2-D echocardiography revealed a small pericardial effusion in our second patient.

Gross abscesses of myocardium or their consequent aneurysms are apparently more frequent in Brucella endocarditis than in endocarditis caused by any other bacteria; they are apparently more frequent in endocarditis involving the semilunar valves than the atriocentricval valves. Abscesses occurred in 40 percent of fatal cases with Brucella endocarditis; 58 percent of those were due to the organism Brucella abortus.

This report of the management of three patients with Brucella endocarditis and aortic root abscess strongly supports the conclusion from our previous study: medical therapy alone does not seem to be sufficient to eradicate an organism with such a destructive character, with a tendency toward abscess formation. Early diagnosis of Brucella endocarditis, or its complications, clinically, serologically, and by echocardiography, with CT scan if required, is the first essential. Early surgical intervention, combined with triple antibiotic therapy with an aminoglycoside and tetracycline, together with either rifampicin or sulphonamethoxazole, then provides the best chance of preventing abscess formation and reduces the risk of mortality. The duration of medical therapy after early surgical intervention is still controversial, but continuation for three months may be advisable in those cases with aortic root abscess formation.

**ADDENDUM**

Since this manuscript was submitted, a fourth case of Brucella endocarditis with aortic root abscess, in a man aged 28 years, has been treated successfully by the same combination of medical therapy and surgical replacement of the aortic valve.

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**Ear Involvement in the Yellow Nail Syndrome**

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Recognized features of the yellow nail syndrome include yellow nails, lymphedema, and pleural effusions. We report a patient with the additional feature of keratosis obturans, which may be a manifestation of this syndrome in the external ear. (Chest 1990; 98:1534-35)

The yellow nail syndrome was first described in 1964 with 13 cases of lymphedema associated with nail dystrophy. Pleural effusions, bronchiectasis, and sinusitis have also been reported. Hard wax impaction in the external auditory meatus, thought to represent keratosis obturans, has been associated with the yellow nail syndrome in one case report. We describe a patient in whom all these features and keratosis obturans coexist.

**CASE REPORT**

A 40-year-old woman first presented in 1955 with nasal blockage and sinusitis. At the age of 55 years, a diagnosis of the yellow nail syndrome was made on the basis of chronic nail changes and persistent mild edema of her face and ankles.

At age 64 years, she was referred to the Oster Chest Unit, Oxford, England, because of a two-year history of productive cough and recurrent chest infections. A clinical diagnosis of bronchiectasis was made. She had gross nail deformity with yellow discoloration.

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were moderate and effusions. Spirometry showed FEV of 1.7 and FVC of 2.7 L (BTPS). She had been a regular heavy smoker.

Bilateral conductive deafness led to a diagnosis of keratosis oburans of the external auditory meatus. This has been removed at regular intervals since with hearing improvement each time.

In 1981, when she was 66 years old, bilateral pleural effusions were noted. These were tapped but they reaccumulated rapidly. Cytologic study of the pleural fluid showed no malignant cells, culture was negative, and the protein content was 5.4 g/dl. Other investigations were normal except for a raised erythrocyte sedimentation rate (ESR) at 69 mm/h. Spirometry in the presence of the effusions gave an FEV of 1.02 L and FVC of 1.69 L (57 percent and 66 percent predicted, respectively). Because of persistent dyspnea due to recurrent pleural effusions, she underwent successful tetracycline pleurodeses. Her condition remains stable three years later with bilateral basal pleural thickening but no obvious effusions. Most recent spirometry in 1989 gave an FEV of 1.5 L and FVC of 2.5 L (predicted, 1.7 L and 2.4 L).

DISCUSSION

The yellow nail syndrome has now been well characterized and consists of yellow nails, lymphedema, and pleural effusions. Bronchiectasis and sinusitis are often associated.

The etiology is unknown, but lymphangiography5 has shown very variable lymphatic hypoplasia. Fluid kinetics of the pleural effusions suggest that the accumulation is due to ineffective drainage rather than excess production of pleural fluid.8 Pleural effusions often occur late in life and are sometimes preceded by infection.

Moran and Larkworthy9 speculated that keratosis oburans was a manifestation of the yellow nail syndrome in the external auditory meatus. However, in an early description of the association between bronchiectasis and keratosis oburans,s a secretomotor reflex mediated through vagal channels in the auditory branch of the vagal nerve was postulated. An active cough reflex induced by stimulation of that nerve was cited as evidence; also, the bronchiectasis and the ear problems were usually ipsilateral. In patients without associated bronchiectasis, an underlying hyperemia and chronic inflammation have been observed in the external auditory meatus with keratosis oburans and consequent increased desquamation suggested as the mechanism for the keratin plug.

If keratosis oburans is a manifestation of the yellow nail syndrome, our patient represents the first report in which all the different features coexist. It is now 20 years since the diagnosis was made and she remains well, emphasizing the good prognosis in these patients.

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**Bilious Pleural Effusion following Liver Biopsy**

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Pleural effusions in patients with chronic liver disease are common and usually are of little consequence. Bilious pleural effusion can occur following percutaneous biopsy or cholangiography procedures if the pleura is traversed. This report emphasizes the role of biliary tract obstruction in the development of a bilious effusion and the importance of biliary tract decompression in treatment. We discuss the laboratory evidence supporting the diagnosis of bilious effusion and review the reported experience with this complication.

(Chest 1990; 98:1535-37)

Extravasation of bile into the thoracic cavity is an unusual cause of pleural effusion which tends to occur in a limited number of clinical settings. Bilious pleural effusion was initially described as a complication of echinococcal and amebic,1 pyogenic,4 and tuberculous subphrenic abscesses. It is a rare complication of abdominal trauma which involves the liver,2 and can also occur in acute duodenal perforation in the presence of a congenital defect in one of the hemidiaphragms.3 The extensive use of percutaneous transhepatic biliary tract decompression in recent years has been associated with numerous case reports of bilious pleural effusion complicating this procedure.** Herein we report a case of bilious pleural effusion complicating orthotopic liver transplantation and subsequent liver needle biopsy and percutaneous transhepatic cholangiography.

CASE REPORT

A 59-year-old man with hepatic failure secondary to chronic active hepatitis underwent liver transplantation. Postoperative liver function tests remained elevated though there was no biopsy evidence of rejection nor was there any evidence of biliary tract obstruction. Nineteen months later, he was referred back to the Mayo Clinic for evaluation of jaundice. At the time of admission, his prednisone dosage was 20 mg twice a day. Examination revealed a thin, icteric man with mild peripheral edema, but who was otherwise normal. Laboratory evaluation revealed a total bilirubin of 8.8 mg/dl; direct, 6.6 mg/dl; AST, 312 U/L; ALT, 101 U/L; and alkaline phosphatase, 1,628 U/L. On the day of admission, ultrasonography of the liver showed no evidence of intrahepatic or extrahepatic ductal ectasia and the portal vein and hepatic artery were patent by Doppler echocardiography. A transhepatic cholangiogram showed normal sized intrahepatic ducts, mild dilation of the native common bile duct (which contained some debris), but no evidence of obstruction. A percutaneous liver biopsy under ultrasonic guidance suggested rejection, and a steroid pulse was given. Due to a rising bilirubin and suspicion of biliary obstruction, endoscopic retrograde cholangiopancreatography (ERCP) was performed on the 4th hospital day. A common duct stone was removed endoscopically. The next day the patient complained of mild shortness of breath, and the chest radiograph showed a moderate-sized right pleural effusion. Thoracentesis yielded 1,100 ml of fluid, and the patient's dyspnea resolved. A post-thoracentesis radiograph showed

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