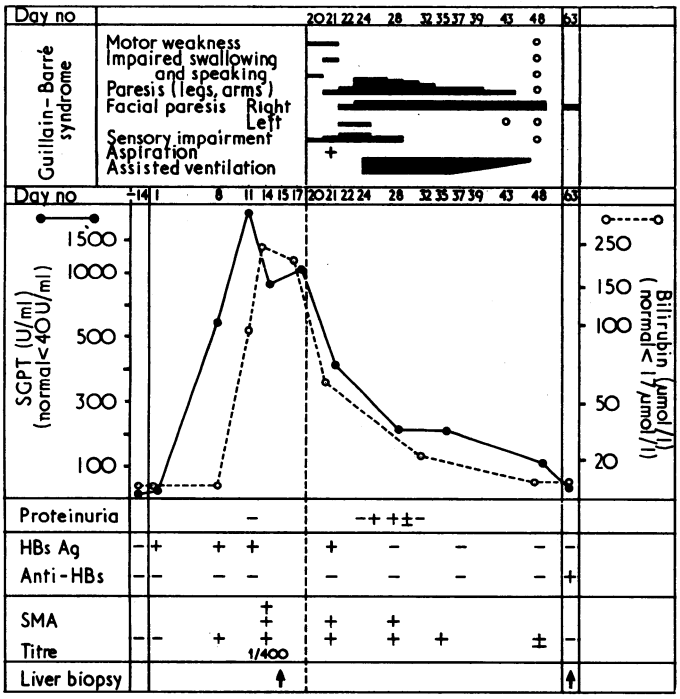
1 June (day 1) her serum, which was being checked routinely every two weeks, contained HBsAg, subtype adw, and the transaminases had risen slightly, but were still within normal limits. Nausea, vomiting, and upper abdominal discomfort then developed and on day 11 she was admitted to hospital. By then she had no complaints. There was no history of arthralgia, exanthema, skin eruptions, or fever, nor of intolerance to fat or tobacco. She did not use any medication. Her father had had a polyneuropathy complicating diphtheria in 1930. Physical examination showed jaundice and a tender, 1-cm palpable, liver. The spleen was not palpable and there were no spider nevi or liver palms. The highest values of serum alanine transaminase (SGPT) were recorded on day 11, and of serum bilirubin and smooth muscle antibody (SMA) on day 14. A liver biopsy performed on day 15 showed features of acute hepatitis, including extensive confluent necrosis of hepatocytes and portoportal bridging. Immunofluorescence for HBsAg was negative. Treatment consisted of absolute bedrest.



Course of acute hepatitis B and Guillain-Barré syndrome.

On day 20 neurological symptoms developed (see figure). Spinal fluid protein was raised (1·8 g/l); cell count, glucose, and chloride were normal; and HBsAg was not demonstrable. Respiratory musculature became affected: vital capacity measured 1200, 950, and 600 ml on days 22, 23, and 24 respectively. Tracheotomy was performed on day 24 and assisted ventilation started. From day 28 the patient improved neurologically and on day 48 assisted ventilation was discontinued. By then neurological functions were normal, with the exception of a right-sided facial paresis, which only subsided after five months. On day 28 HBsAg conversion, accompanied by proteinuria, became fully apparent. At day 63 she had become negative for SMA and positive for serum anti-HBs, while bilirubin and transaminases had become normal. A repeat biospy showed slight prominence of Kupffer cells and small periportal connective tissue strands. After mobilisation at day 67 the liver function remained normal and recovery was uneventful.

Attempts to show laboratory evidence of several viral infections associated with GBs all failed. Apart from SMA no autoantibodies (including anti-myelin antibody[4]) were present.

Comment

Although GBS may be elicited by viral infections of different origin, it is rare in AVH and we are not aware of documented cases of GBS after acute HBsAg-positive hepatitis. Our patient developed GBS 20 days after the first, preclinical, manifestation of hepatitis B. The condition was completely reversible.

The transient proteinuria concurring with the conversion to HBsAg-seronegativity may have been due to deposition of HBsAg/anti-HBs immune complexes in glomeruli, while participation of neural antigens in complex formation[5] seems less likely.

Whether in AVH the unusually high SMA titre heralded the development of GBS, a syndrome in which autoimmunity may play a part, remains to be established.

Dr J R H Brentjens critically revised the manuscript. The HBsAg determinations and subtyping were performed by Dr B Houwen.

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Pasteurella multocida septicaemia associated with chronic liver disease

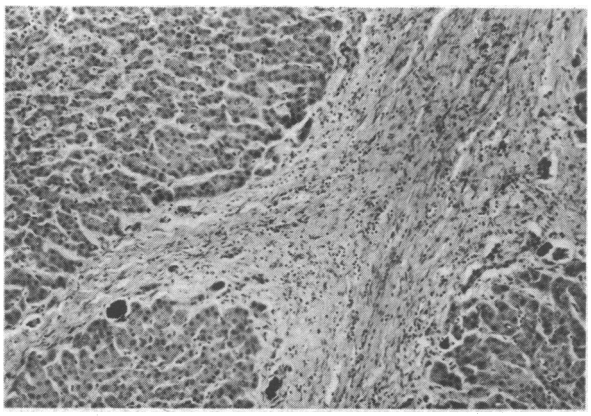
In the case of the patient with chronic liver disease and terminal *Pasteurella multocida* septicaemia reported here the organism was possibly acquired from a dog, though there was no history of an antecedent bite.

Case report

The patient, an 18-year-old man, had had recurrent bronchitis in infancy and at 22 months had been admitted to hospital with bronchopneumonia and hepatomegaly. A liver biopsy had shown biliary fibrosis and bile ductular proliferation with no evidence of cirrhosis. The result of a sweat test and duodenal trypsin, serum copper, and caeruloplasmin levels were normal. In February 1974 he was referred to the Professorial Medical Unit, Southampton. The liver and spleen were grossly enlarged and the results of liver function tests were: total bilirubin 102 μmol/l (6 mg/100 ml), alkaline phosphatase 41 KAU/100 ml, total plasma protein 56 g/l, plasma albumin 30 g/l. Tests for autoimmunity were negative, HB antigen and alpha-fetoprotein were not detected, and the alpha-1-antitrypsin level was normal. A clinical diagnosis of biliary cirrhosis secondary to partial biliary atresia was made.

In November 1974 he was admitted with increasing jaundice, abdominal pain, and vomiting. He was deeply jaundiced (total bilirubin more than 342 μmol/l (20 mg/100 ml)), feverish, and hypotensive. Blood cultures grew *P. multocida* sensitive to ampicillin. He was treated with ampicillin and a standard regimen for hepatic failure. Serum agglutination tests for *Yersinia pseudotuberculosis* and *Y. enterocolitica* were negative. His temperature fell to normal but his general condition deteriorated and he died two weeks after admission.

At necropsy the liver was found to be small and finely nodular. The common bile duct was patent and the intrahepatic biliary system consisted partly of a cavity, 8 cm by 4 cm, filled with a mass of fused pigment stones and inspissated bile. On sectioning the liver there were two discrete nodules, which were shown histologically to be hepatocellular carcinomas; the remainder of the liver showed secondary biliary cirrhosis (see fig). A mass of



Area of secondary biliary cirrhosis and cholestasis. (Haematoxylin and eosin × 60.)

omentum, which on section contained locules of pus, was attached to the anterior border of the right lobe of the liver. Culture of the pus grew. *P multocida*. Other necropsy findings included evidence of portal hypertension with oesophageal varices and a spleen weighing 1020 g. The organism isolated by blood culture and from the pus in the abscesses gave the characteristic cultural appearances and odour of *P. multocida*. It was a short, slender, slightly ovoid, Gram-negative bacillus which was non-motile at both 22°C and 37°C. Glucose, mannitol, sucrose, and trehalose were fermented without gas production. The indole test was positive. There was no fermentation of maltose, dulcitol, lactose, salicin, sorbitol, xylose, or dextrin. The O-nitrophenyl-β-D-galactopyranoside test was negative, as was the test for urease production. The organism was sensitive to penicillin, tetracycline, erythromycin, and streptomycin by the disc method. Tube dilution tests showed that it was sensitive to 0·5 μg/ml of penicillin G and to 0·25 μg/ml of ampicillin.

Comment

There have been occasional reports of *P. multicoda* septicaemia in patients with cirrhosis.[1-5] In several of the cases there was a history of a preceding animal bite. In one the same strain of *P. multocida* was isolated from the patient and from a cat that had bitten the patient.[1] In another case *P. multocida* was cultured from abscesses in a cirrhotic liver.[4] In the present case the patient gave no history of an animal bite. His family kept a dog, however, and further tests on the *P. multocida* isolated showed that it belonged to Frederiksen biotype 6 (dog type). Therefore he may have acquired the organism from the dog.

We thank Professor D H Wright for information about the necropsy and Dr N S Mair, Director, Public Health Laboratory, Leicester, for biotyping the *P. multocida* and arranging the Yersinia agglutination tests.

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Insidious endocarditis caused by Chlamydia psittaci

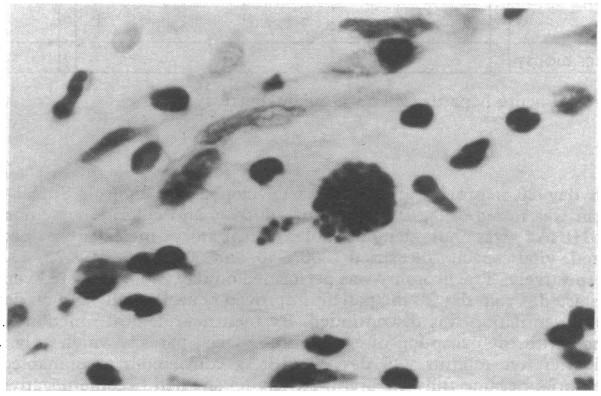
Published reports show that infective endocarditis due to *Chlamydia psittaci* may be clinically severe: two patients have died[1] and one has needed urgent aortic valve replacement.[2] The following case was more insidious, but immunofluorescence studies allowed precise definition of the infecting agent.

Case report

A previously well 48-year-old joiner became breathless on moderate exertion after two attacks of paroxysmal nocturnal dyspnoea in March 1974. There was no history of rheumatic fever, while his recent contact with birds had been limited to occasionally feeding pigeons in the garden. On examination his blood pressure was 140/80 mm Hg, and murmurs of mitral stenosis and regurgitation and of trivial aortic regurgitation were heard (these murmurs did not change during his subsequent illness). There was no evidence of infective endocarditis. The electrocardiogram showed atrial fibrillation, and a chest *x*-ray film enlargement of the heart, particularly in the area of the left atrium. Cardiac catheterisation showed mild pulmonary hypertension, while the indirect left atrial pressure was raised (25/10 mm Hg). Mitral valve calcification was seen on screening, and a mitral valve echogram showed thickened cusps. He was digitalised and advised to have mitral valve replacement.

On 5 July he was readmitted to hospital with a brachial artery embolus. Anticoagulant treatment was begun. His temperature was 37°-37·8° C on three occasions but he had no stigmata of infective endocarditis. Investigations showed: haemoglobin 9·8 g/dl, WBC $4·9 \times 10^9$/l, and ESR (Westergren) 70 mm in the first hour. The findings in the electrocardiogram and chest *x*-ray film were unchanged. Blood cultures (×13) were negative. Complement fixation titres to brucella and *Coxiella burnettii* were negative. Immunoglobulin study on 20 July showed that the IgM level was raised; IgG later became raised but both subsequently returned to normal after antibiotic treatment. On 19 July his spleen was found to be enlarged; infective endocarditis was suspected, the anticoagulants were stopped, and antibiotics were begun (benzylpenicillin 20 megaunits daily for six weeks and streptomycin 0·75 g twice daily for two weeks). Subsequently his haemoglobin level rose, the ESR fell, and splenomegaly regressed. On 26 July a psittacosis/lymphogranuloma venereum complement fixation titre of 1/320 was reported. On 16 October the titre was 1/16.

On 9 September at elective mitral valve replacement his mitral valve was found to be calcified and stenosed, with a fresh friable vegetation 1 cm in diameter overlying a necrotic area on the anterior cusp. The valve was replaced with a 29-mm Bjork-Shiley prosthesis. His postoperative recovery was uneventful. Histologically the left atrium and mitral valve showed chronic inflammation. Phloxine tartrazine stain showed numerous rounded brightly eosinophilic inclusions distending the mononuclear cells within the valve, vegetation, and pericardium. Machiavello stain showed similar but reddish inclusion bodies less than 1μ diameter (see figure)—thus characteristic of Levinthal-Coles-Lillie bodies, described in psittacosis.[3] Psittacosis-complement-fixation-positive control pigeon serum and rabbit antipigeon gammaglobulin were used to detect antigen from *Chl psittaci* by indirect immunofluorescence. Discrete areas of intracellular fluorescence, indicative of *Chl psittaci* antigen, were detected within the same cell type shown histochemically to contain Levinthal-Coles-Lillie bodies. Lymphogranuloma venereum cultures and trachoma smears failed to show intracellular fluorescence when stained in the same manner as the biopsy specimens.



Section of mitral valve stained by Machiavello's method. A large mononuclear cell is shown, packed with coccoid bodies less than 1μ diameter. These have the characteristics of Levinthal-Coles-Lillie bodies, typically found in infection with *Ch. psittaci* (×200.)

Discussion

Despite a dubious history of contact with birds (feeding pigeons in the garden), *Chl psittaci* was firmly established as the causative agent in this case on the basis of histological, serological, and immunofluorescence findings. Nevertheless, the clinical course was benign when compared with previous cases: the patient claimed to be asymptomatic when infective endocarditis was diagnosed and denied subjective improvement from antibiotic therapy, despite laboratory evidence that a focus of infection was being suppressed. Mitral valve replacement was not performed because of deterioration precipitated by infective endocarditis but because of his breathlessness on exertion, which had been static for six months.

This case shows that destructive valvular endocarditis due to *Chl psittaci* can proceed in the absence of subjective evidence of ill health and with a dubious history of bird contact shown only after close questioning.