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Case Report

Biliary Peritonitis Following the Removal of a T-tube

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Introduction

The removal of a T-tube following exploration of a common bile duct (CBD) rarely results in complications. However, we wish to report a case of biliary peritonitis following the removal of a latex rubber T-tube.

Case Report

An otherwise fit 28 year old female presented with a four year history of biliary colic. Cholecystectomy was carried out through an upper right paramedian incision. The per-operative cholangiogram showed a single small calculus at the lower end of the CBD. The stone was removed with some difficulty through a longitudinal choledochotomy. A 16FG latex "Kehr T-tube" was inserted and the CBD closed with interrupted chromic catgut taking care not to include the tube in the suture. The long limb of the tube was brought out to the right of the incision.

The patient made an uneventful post operative recovery and a T-tube cholangiogram at 10 days showed free flow into the duodenum and no evidence of stones. The T-tube was removed without difficulty, but immediately the patient began to complain of deep right upper quadrant pain. Over the next 12 hours the pain spread to the right iliac fossa and then became generalised. On examination the patient was pyrexial (38.0) with a tachycardia (100) and demonstrated generalised tenderness, guarding and rebound.

At emergency laparotomy 450 ml of lightly blood stained bile was removed from the peritoneal cavity. The leak appeared to be from the site of the choledochotomy rather than any other part of the well formed T-tube track. Peritoneal lavage was performed and suction drains placed in the vicinity of the leak and in the right paracolic gutter.

The patient subsequently made an uncomplicated recovery.

Discussion

Three other cases of biliary peritonitis following the removal of latex rubber T-tubes have been reported[1,2] although the problem is certainly commoner than this would suggest. Other materials such as polyvinyl chloride and silicon rubber have been found unsuitable because removal tends to be followed by leakage[3,4].

We wish to make two observations about this case. Firstly, although the use of large T-tubes facilitates the subsequent removal of any retained stones using a steerable catheter inserted down the track, it seems likely that the large size of the tube in relation to the duct used in this case was the main cause of the subsequent leak.

Secondly, treatment of this complication is contentious. Conservative management has been used but may predispose to the development of subphrenic abscess[3]. Reinsertion of the T-tube at emergency laparotomy is an alternative treatment which has given satisfactory results[3], but this may prove technically difficult if dense adhesions have formed. Laparotomy and adequate drainage from the region of the leak proved easy and successful in this case, and we think this procedure should be considered first when treating biliary peritonitis which occurs following the removal of a T-tube.

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Hospital Acquired Native Valve Endocarditis Caused by *Acinetobacter calcoaceticus* and Treated with Imipenem/Cilastin

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SUMMARY: A case of hospital acquired endocarditis due to *Acinetobacter calcoaceticus* in a severely burned patient is presented. Both aortic and mitral native valves were affected and the organism was resistant to most antimicrobial agents.

Introduction

Acinetobacter calcoaceticus, a Gram-negative non-fermentative aerobic cocco-bacillus, is a frequent and often persistant coloniser of the hands of hospital personnel and of the skin and urine of in-patients[1]. It is isolated from burn wounds[2] and infrequently as a culture contaminant from blood cultures[3] and may cause a wide variety of infections[4,5].

Acinetobacter is an uncommon pathogen, usually affecting the seriously ill and compromised patient, causing 1.4% of hospital acquired infections[4].

Few well documented cases of endocarditis due to *A. calcoaceticus* have been reported[5–8], and, to our knowledge none from the UK. We present a case of hospital acquired *Acinetobacter* endocarditis in an extensively burned patient.

Case Report

In a bomb explosion, a 27 year old soldier sustained 40% burns involving limbs and trunk, and injuries to his left leg which required below knee amputation. During his first 12 weeks in hospital he developed continual pyrexia, hypoproteinaemia, lost 30 kgs and underwent seven procedures for excision of slough and skin grafting. Cloxacillin and ampicillin were started on admission and continued for 25 days. Netilmicin and piperacillin were given from days 22 to 28 for clinical septicaemia but with negative blood cultures.

A. calcoaceticus was isolated in pure culture from a catheter specimen of urine on day 35, but disappeared spontaneously after subsequent change of catheter. Thereafter, his clinical condition and burn wounds improved and no antibiotics were given until day 76 when he received cephalexin for 12 days, followed by piperacillin from day 88 to 94 for purulent graft sites, from which two coliforms and *Pseudomonas aeruginosa* were isolated. On day 84 he underwent further skin grafting, and on day 87 insertion of a venous catheter. *A. calcoaceticus* was isolated from a single blood culture, taken on day 87, after 7 days incubation. During week 13 his condition deteriorated, with anaemia, persistant tachycardia and pyrexia, and a systolic murmur was heard for the first time. There was no additional evidence of endocarditis. One of two blood cultures taken on day 95 grew *A. calcoaceticus* after 5 days incubation; *Klebsiella* sp was isolated from his urine (day 96) and cephalexin was given again. His condition improved temporarily after blood transfusion, but eleven days later (day 107) he deteriorated suddenly with tachycardia, tachypnoea, toxaemia and confusion. Aortic systolic and diastolic murmers were present. Ampicillin and gentamicin were started. Within 2 days he had developed severe aortic regurgitation and congestive cardiac failure. The electrocardiogram showed atrio-ventricular conduction defects and echocardiograms indicated vegetations on the aortic and mitral valves.

A. calcoaceticus was isolated from blood cultures taken on days 107 and 109, and from his urine. At operation on day 111 the aortic valve was found to be destroyed, with large vegetations on the aortic root and septum. There were multiple abscess cavities around the annulus and two septal abscesses. The aortic valve was replaced with a prosthetic bovine pericardial valve, and vegetations on the anterior mitral leaflet removed and perforations repaired.

A heavy pure growth of *A. calcoaceticus* was obtained from the aortic valve vegetations. On the 10th day after operation he developed acute pulmonary oedema with worsening mitral valve regurgitation. Echocardiography showed a vegetation on the mitral valve; the next day this valve was replaced and a pure growth of *A. calcoaceticus* was obtained from the vegetation.

A variety of antibiotic combinations (see Fig 1) was given until detailed susceptibility tests were available when Imipenem plus cilastin (Merck, Sharp and Dohme Research Laboratories) was started on day 125 and continued for 21 days. He was apyrexial within 5 days of starting this agent but his cardiac function continued to deteriorate and permanent pacing was required. He developed irreversible left ventricular failure and died on day 155. At autopsy, the prosthetic aortic and mitral

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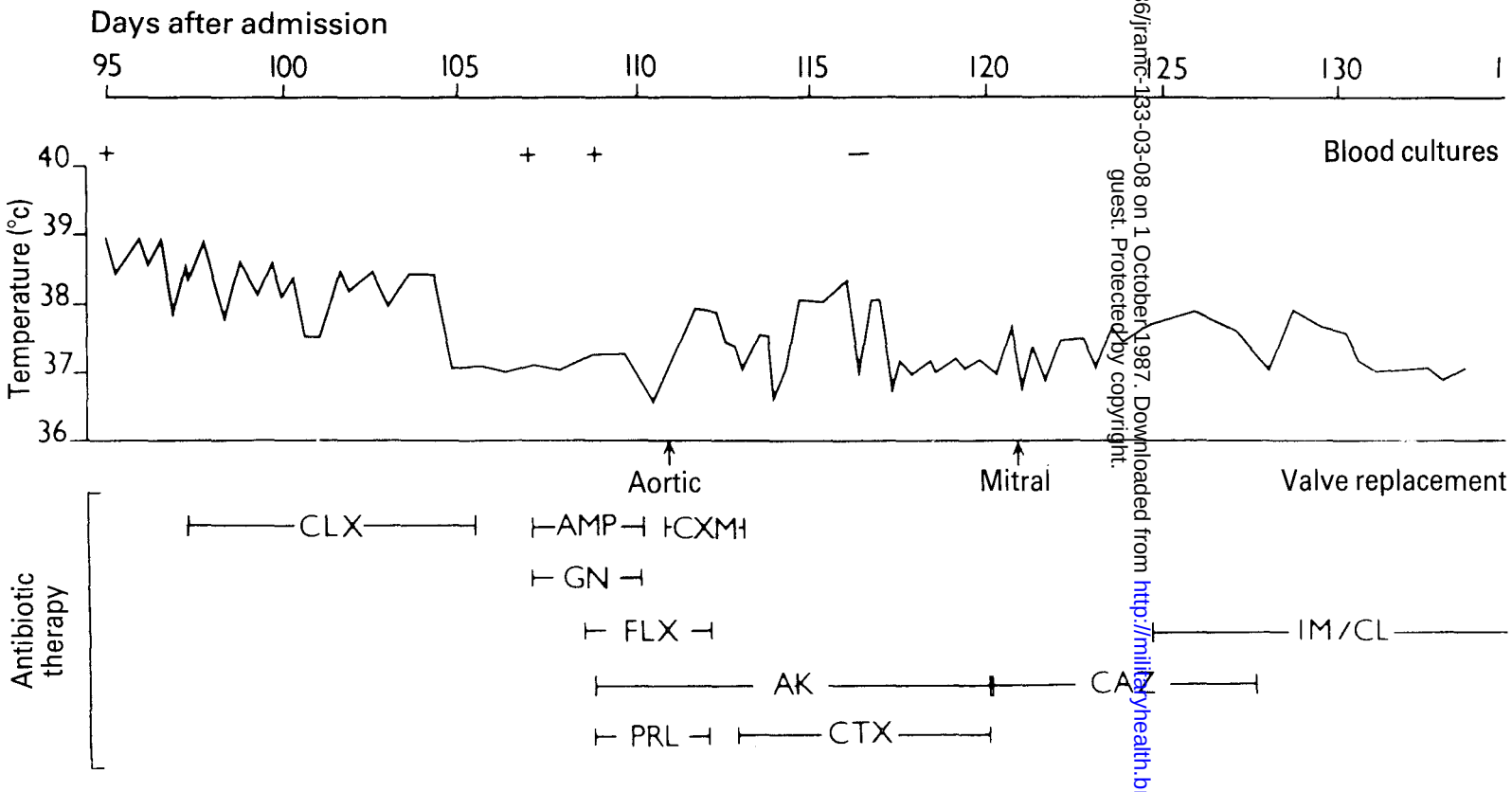

Key to antimicrobial therapy:
CLX: cephalexin, 2g/day oral: AMP: ampicillin, 2g/day IV;
GN: gentamicin, 240 mg/day IV; AK: amikacin, 1g/day IV;
FLX: flucloxacillin, 4 g/day IV; CXM: cefuroxime, 2.25 g/day IV;
PRL: piperacillin, 16 g/day IV; CTX cefotaxime, 4 g/day IV;
CAZ: ceftazidime, 6 g/day IV; IM/CL: imipenem-cilastin 2 g/day IV.

Fig 1. *A. calcoaceticus* endocarditis: clinical course and antimicrobial therapy.

valves and native pulmonary and tricuspid valves were intact, free from vegetations and sterile on culture.

Microbiology

All isolates of *A. calcoaceticus* exhibited similar antimicrobial susceptibilities as follows: Imipenem, Minimum Inhibitory Concentration (MIC) and Minimum Bactericidal Concentration (MBC) 0.25 mg/L; tobramycin, MIC 2 mg/L; amikacin, MIC 4mg/L; MBC 4–8 mg/L; gentamicin, MIC>128 mg/L; ampicillin, MIC>128 mg/L; piperacillin, MIC>256 mg/L; carbenicillin, MIC 2048 mg/L; cephalexin, MIC>128 mg/L; cefotaxime, MIC 16 mg/L; cefuroxime, MIC 32 mg/L; ceftazidime, MIC 4–8 mg/L and MBC 8mg/L and trimethoprim/sulphamethoxazole, MIC 6.4/128 mg/L.

Comment

Acute bacterial endocarditis arising from sepsis and bacteraemia is a recognised though uncommon complication of burns[9].

Acinetobacter endocarditis may involve both normal and diseased native valves, or prosthetic valves or the congenitally abnormal heart[5–8]. In our patient there was no evidence of pre-existing heart disease.

Acinetobacter bacteraemia most commonly results from colonised or infected intravenous catheters, but it may also occur with other infections including those in burns[2,5]. Antecedent broad spectrum antibiotics have invariably been given[2,5,6]. Before endocarditis developed *A. calcoaceticus* was isolated only from urine in our patient and early positive blood cultures (days 87 and 95) were considered not to represent bacteraemia. *Acinetobacter* was specifically sought, but was not isolated, from his burn wounds (although the sub-eschar space was not sampled), intravenous catheters and sputum. He had, however, been in hospital for 12 weeks, and undergone multiple surgical procedures to colonised wounds, repeated intravenous and urinary catheterisation, and had received prolonged and varied broad spectrum antibiotics. These factors, together with the compromised physiological and immunological functions following extensive burn injury, will predispose a patient to bacteraemia with organisms of low pathogenicity[2,5].

In common with previous reports[2,4,5], this strain of *A. calcoaceticus* was resistant to most antibiotics, including gentamicin and all cephalosporins. Imipenem, a carbapenem B-lactam with a very broad antimicrobial spectrum, was most active *in vitro* and appears to have eradicated the infection, but it is difficult to assess the role of any antibiotic when valve replacement is undertaken.

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