PAPERS AND SHORT REPORTS

Microbiology of pyogenic liver abscess

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Abstract

Sixteen patients with pyogenic liver abscesses were studied over 10 years to discover the causative organisms of the condition. Pus was subjected to Gram-negative smear or gas-liquid chromatography to detect volatile acids characteristic of anaerobes and then cultured. All isolates were identified by conventional methods and tested for sensitivity to appropriate antimicrobial agents. Bacteria were grown from the liver abscesses in all 16 patients. Streptococcus milleri Lancefield group F was the commonest organism isolated from the pyogenic liver abscesses, being found in 13 patients.

If Strep milleri is isolated care should be taken not to mistake it for an anaerobe, and finding the organism in the blood should alert the clinician to the possible presence of a liver abscess.

Introduction

Pyogenic liver abscess is uncommon: Rubin *et al*[1] found an incidence of 0·016% among hospital admissions in their series, and Perera *et al*[2] quoted a figure of 0·03% among medical and surgical admissions. No one hospital, and certainly no individual clinician, is therefore likely to have extensive experience of this condition. Since the first comprehensive study, published by Ochsner *et al* in 1938,[3] there have been many case reports and reviews,[1 4-8] in which the presenting signs and symptoms and the appropriate investigations have been discussed in detail. The need for early and adequate surgical drainage has been emphasised, though the efficacy of percutaneous drainage is currently being assessed.[9]

We present our experience of the causative organisms in this condition, based on a prospective study of 16 patients with pyogenic liver abscesses seen at St Thomas's Hospital between 1970 and 1980.

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Patients

During 1970-80, 16 patients (11 men aged 30-74 years (mean 54·5), and five women aged 50-68 years (mean 57·4)) were shown by needle aspiration or at operation to have one or more macroscopic collections of pus in the liver. Amoebic abscesses were not included in this series.

Six patients gave a history of abdominal operations from six weeks to four years before: two for perforated duodenal ulcer, one cholecystectomy, one Polya gastrectomy, one appendicectomy, and one ileal resection for Crohn's disease. Two further patients had cholelithiasis, one diverticular disease, and in one a small-bowel fistula was found at operation. In the remaining six patients no suggestive underlying cause was identified. Fifteen of our 16 patients survived, the single death occurring in a man with severe diabetes who also developed a subphrenic abscess.

Microbiological methods

Pus in a universal container was submitted to the laboratory in all cases, usually within minutes of aspiration. A smear was Gram stained and, for cases presenting from 1975 onwards, gas-liquid chromatography performed directly on the pus to detect the volatile fatty acids characteristic of anaerobes. The specimens were cultured on Columbia agar (Oxoid CM331) with 10% defibrinated horse blood aerobically (latterly in 5-10% CO_2) and anaerobically for five days. From 1975 a special anaerobic medium was also used: brain-heart infusion agar (Oxoid CM375) containing 0·05% cysteine hydrochloride, 1% vitamin K haemin solution, 0·5% yeast extract, 100 mg neomycin/l or 10 mg nalidixic acid/l, 7·5% whole defibrinated horse blood, and 2·5% lysed (by freezing and thawing) defibrinated horse blood. All isolates were identified by conventional methods and tested for sensitivity to appropriate antimicrobials.

Results

Bacteria were grown from the liver aspirate in all 16 cases. In 10 patients *Streptococcus milleri* was the only organism isolated. In a further three patients *Strep milleri* was present with one other organism (*Fusobacterium necrophorum*, *Bacteroides fragilis*, and *Peptococcus sp*, respectively). *Strep milleri* was thus present, alone or

in mixed growth, in 13 of our patients. *Escherichia coli* was found in only two patients, in mixed growth in both cases, with *Peptostreptococcus anaerobius* and *F nucleatum* in one and with *B fragilis* in the other. One patient had a pure growth of *Strep pneumoniae* type 15. In 12 of the 13 patients from whom *Strep milleri* was recovered the organism carried the Lancefield F antigen, and in the remaining one the Lancefield group was not A, C, F, or G. Gas-liquid chromatography was performed directly on the pus in 11 cases. In five volatile fatty acids characteristic of anaerobes (predominantly butyric acid) were detected in addition to lactic, acetic, and propionic acid. Anaerobes were recovered on culture from four of these five specimens and were almost certainly present but not grown in the remaining specimen, since Gram-negative fusiform bacteria were seen in the smear. *Strep milleri* alone was recovered from the six specimens of pus which did not produce anaerobic traces on gas-liquid chromatography.

Blood cultures were performed on 11 patients and were positive in seven, all but one of these growing *Strep milleri*. *E coli* was recovered from the remaining patient. In each case the same organism was cultured from the subsequent liver aspirate.

Discussion

Strep milleri Lancefield group F was the commonest organism isolated from pyogenic liver abscesses in this prospective study. Bacteria isolated from oral lesions by Guthof in 1956[10] and later from dental root canals by Ottens and Winkler[11] were both classified as *Strep milleri* by Colman and Williams.[12] Probably the organism known in the USA as *Peptostrep intermedius* or latterly *Strep intermedius* is identical. Parker and Ball, in an extensive investigation of streptococci isolated from human infections, found *Strep milleri* to comprise nearly one-third of all streptococci from patients with pyogenic infections, including 13 of 16 brain abscesses.[13]

Though often initially isolated anaerobically and readily mistaken for an anaerobic organism, *Strep milleri* is neither a true anaerobe nor a microaerophile. It will grow well only in a carbon dioxide enriched atmosphere. It is always resistant to metronidazole, and this together with the distinctive caramel odour so characteristic of strains bearing the Lancefield F antigen provide the most useful guide to its identification in the laboratory. Differentiation of *Strep milleri* from true anaerobic streptococci has important therapeutic implications: both types of streptococci are invariably sensitive to penicillin but only the genuine anaerobic streptococci are sensitive to metronidazole.

There have been numerous reports of the microbes involved in pyogenic liver abscess, but no consistent pattern has yet emerged. This can be attributed partly to the difficulties in culturing fastidious organisms, particularly anaerobes, but also to taxonomic difficulties with the streptococci. Early this century anaerobes were recognised as important in the pathogenesis of liver abscesses both in man[14] and in animals.[15] Yet in 1938 Ochsner *et al*[3] reported that 30% of abscesses were caused by *E coli* and that "streptococci," not further identified, were also common. At the time, and for the next three decades, anaerobic cultures were either neglected or not performed assiduously. "Sterile" abscesses comprised up to 50% of cases in one series,[16] and the frequency with which *E coli* was found was even higher than in Ochsner's patients.[5 17] In 1960 Sherman and Robbins[18] emphasised the role of other Gram-negative organisms, and in 1964 Block *et al*[19] noted the apparent increasing proportion of Gram-negative infections in the postantibiotic era.

Patterson *et al* in 1967 renewed interest in anaerobes when they reviewed 13 liver abscesses caused by anaerobic and microaerophilic streptococci[20] and suggested that anaerobic cocci might be more common in human infections than was generally recognised, primarily because adequate anaerobic culture techniques were not often used. In 1972 Sabbaj *et al*[4] voiced the same opinion, reporting 25 anaerobic infections in 47 patients with liver abscesses. Twenty of the 50 organisms isolated were "anaerobic or microaerophilic streptococci or cocci." In 1975 Bateman *et al*[21] reported three patients with pyogenic liver abscesses caused by *Strep milleri*, two of these being Lancefield group F, and this was shortly followed by a report of two further cases.[22]

Although the first four abscesses in our series were diagnosed at a time when exacting anaerobic techniques were not in routine use, the subsequent cases were specifically investigated for anaerobes. The results obtained from gas-liquid chromatography suggest that anaerobes, most probably fusobacteria, were present but not grown in only one case. It is worth noting that the recovery of fusobacteria and the more fastidious slow-growing anaerobes is particularly difficult in the presence of large numbers of *Strep milleri*. In such cases careful examination of a Gram-stained smear together with gas-liquid chromatography may be the most useful indication of their presence.

Probably many of the "anaerobic and microaerophilic streptococci" reported by Patterson *et al*,[20] Sabbaj *et al*,[4] and more recently Satiani and Davidson[7] and Silver *et al*[8] were, in fact, *Strep milleri*. Recent reviews, however, have emphasised the importance of coliforms and anaerobes. Verlenden and Frey in 1980[23] stated "The most likely pathogens include enteric Gram-negative rods of which *E coli* is the most common, anaerobes and *Staphylococcus aureus*," and this has been the opinion of other authors.[1 24 25] Perera *et al*[2] commented "this review emphasises, again, the importance of anaerobes as causative agents in pyogenic liver abscess," but among their 16 patients were two infections with "peptostreptococci," one "microaerophilic streptococcus," one *Strep milleri* in mixed growth with *B fragilis*, and three cases where the responsible organism was not isolated. Careful laboratory technique should result in the recovery of fastidious organisms, including anaerobes, both from pus and from blood cultures facilitate the differentiation of *Strep milleri* from true anaerobic streptococci, and confirm our findings that it is the commonest infecting organism in pyogenic liver abscess.

The relevance of these findings and conclusions is twofold: firstly, it is of therapeutic importance that this organism, if isolated, should not be mistaken for an anaerobe, especially since it is resistant to metronidazole. Secondly, the finding of *Strep milleri* in the blood of an ill, febrile patient should alert the clinician to the possible presence of a liver abscess and initiate appropriate investigations for its detection.

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(*Accepted 15 July 1981*)

Ingrowing toenails: an evaluation of two treatments

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Abstract

Most of the procedures used for treating ingrowing toenails cause considerable discomfort and have high failure rates. This study evaluated two methods of treatment: (a) a simple procedure, and (b) angular phenolisation. Patients were seen in a special toenail clinic and were assessed for severity and duration of symptoms. Those with no permanent deformity of the nail fold and with only minor infection were treated by the simple procedure. The nail was nicked and torn down to expose the infected nail fold. The patients were then taught to clean the area, spray it with povidone-iodine dry powder, and pack the nail fold with a twist of cotton-wool. Patients with recurrent or severe ingrowing toenails were treated by angular phenolisation. One hundred patients were treated by the simple procedure and 61 of these had had no recurrence after six months. A total of 280 phenolisations were carried out over 18 months and 272 were successful.

The treatments described are simple, effective, and well tolerated and should be considered as alternatives to traditional treatment.

Introduction

The ingrowing toenail is largely a disease of civilised society. Those who wear no shoes or who make their own do not appear to suffer from the condition. The increasing use of man-made fibres in socks and of plastic materials in shoes adds to the problem, as does the cost of footwear in general. Toes need a soft flexible toebox to the shoe (except where there is risk of injury) and adequate ventilation.

The current methods available for treating ingrowing toenails are generally unsatisfactory.[1] Cutting a notch in the centre of the nail is traditional but largely ineffective. Ointments and creams induce maceration. Silver nitrate reduces a granuloma but does not remove the cause of the underlying infection and it renders the nail black, opaque, and friable, making future treatment difficult. Simple avulsion has been adequately condemned and should not be used as a method of treatment. Zadik's operation and wedge resection require time and a fair degree of skill; aseptic techniques are necessary and tissue planes are crossed. Patients undergoing these procedures experience postoperative pain and need to spend time off work to recover. The recurrence rate after these procedures is from 16% to 28%. A recent development has been the introduction of the plastic gutter, but each kit costs £2·30, and a failure rate of 44% has been shown.[2]

This study was designed to investigate the technical and administrative problems associated with the management of ingrowing toenails and to evaluate two methods of treatment: (*a*) a simple procedure which could be used by general practitioners, chiropodists, and school nurses; and (*b*), for more severe cases, angular phenolisation.

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Patients and methods

Most patients attending the accident and emergency department of this hospital (resident catchment population 518 000) with toenail problems were referred to a special toenail clinic. Patients were seen at five-minute intervals during the first two hours of the clinic session for assessment, simple treatment, or review. During the second two-hour session up to six patients were treated under ring block at 15-minute intervals. These arrangements proved sufficient to cope with new patients without creating a waiting list. Clerical work was dealt with by the accident and emergency department clerks in the normal course of their duties, and one state-enrolled nurse employed for two and a half hours was sufficient for laying up, dressing, and clearing away.

Two methods of treatment were used.

SIMPLE TREATMENT PROCEDURE

Patients who had been experiencing symptoms for one month or less without permanent deformity of the nail fold and with only mild infection were treated by the simple procedure.

Firstly, debris was removed with 1% Savlon solution. The nail was then nicked using 12 cm (4½ inch) nail pliers (fig 1 (*a*)) and torn down (fig 1 (*b*)) to expose the infected nail fold. The nail usually tears true, and because the nail is already undermined by infection the pain caused is less than that caused by a ring-block injection. The patients were instructed and shown how to clean the area twice daily with 1% Savlon solution and to spray the area with povidone-iodine dry powder spray (Disadine DP; Stuart Pharmaceuticals). They were shown how to pack the nail fold firmly with a twist of cotton-wool (fig 1 (*c*)) to prevent the fold collapsing inwards under shoe pressure until the nail grew out. They were also given advice on footwear and hygiene and were advised to abstain from physical education and field sports for one month. Finally, the patients were told to return for assessment after two to four weeks. If patients were then free from pain they were