PAPERS AND ORIGINALS

Anaerobic axillary abscess

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Summary and conclusions

Fifty-two patients with axillary abscesses were seen during two years. *Staphylococcus aureus* was isolated from 34, anaerobic bacteria from 12, and skin flora from five; in one case the pus was sterile. Seven patients with hidradenitis suppurativa had recurrent infection with abscess formation, which was bilateral in three. Anaerobes were isolated in five of these cases and skin flora alone in two.

Anaerobes are secondary invaders in this condition, and histological examination shows that the primary abnormality is obstruction of pilosebaceous follicles and apocrine glands, associated with keratin plugging of the follicles. Chemotherapy offers little hope of cure, although metronidazole removes the offensive smell of the discharge. Radical surgery is usually indicated.

Introduction

Axillary abscesses may account for 16% of cutaneous abscesses, and although *Staphylococcus aureus* has long been considered to be the commonest organism implicated, evidence exists that anaerobes are also important¹; this is particularly so in hidradenitis suppurativa, a chronic infection of the apocrine glands.^{2,3} The isolation of anaerobes in our laboratory from a patient with hidradenitis suppurativa led us to review all cases of axillary abscess treated at this hospital over the past two years, and we report our findings.

Methods

Pus from each axillary abscess was submitted to the laboratory, either in a sterile container or on a swab. A Gram-stained film was made initially from each specimen, and if sufficient material was obtained gas-liquid chromatography was used to detect volatile fatty acids in those specimens that were not obviously staphylococcal.⁴

All specimens were cultured on Columbia agar (Oxoid CM331) with 10% defibrinated horse blood and incubated aerobically and anaerobically in an atmosphere of 10% carbon dioxide in nitrogen in an anaerobic jar. Some specimens were also cultured anaerobically on brain-heart infusion agar (Oxoid CM375) containing 0.05% cysteine hydrochloride, 1% vitamin K-haemin solution, 0.5% yeast extract, 100 mg neomycin/l, 7.5% whole defibrinated horse blood, and 2.5% lysed (by freezing and thawing) defibrinated horse blood. Anaerobic isolates were fully identified by conventional methods, and their susceptibility to penicillin, tetracycline, and metronidazole was assessed by disc testing.

Results

During two years 52 axillary abscesses were drained, 15 in men and 37 in women. Right-sided abscesses were more common in women (ratio 4:1), but the sex distribution for left-sided abscesses was equal. Bilateral disease was present in only four patients, of whom three had hidradenitis suppurativa. The mean age at presentation was 29 years.

Anaerobic bacteria were isolated from 12 (23%) of the 52 patients (six men and six women). The table shows the identity of these organisms and their susceptibility to penicillin, tetracycline, and metronidazole; they included a wide variety of anaerobes, *Peptococcus* spp, *Bacteroides melaninogenicus*, and *B. corrodens* being most frequently isolated. *B. fragilis* was present in only two cases. In only three of the 12 anaerobic abscesses were aerobic bacteria isolated: these included *Streptococcus milleri*, *Proteus mirabilis*, and skin flora. *Staph aureus* was the sole pathogen in 34 patients (65%), and 31 of these organisms were resistant to penicillin (91%). Aerobic skin flora was isolated from five further abscesses, and in one case *Proteus mirabilis* was also present. Many pus cells were seen on the Gram-stained smear from a further patient but no bacteria were isolated.

Only three of the 34 patients in whom *Staph aureus* was isolated had had previous axillary abscesses, whereas five of the 12 patients with anaerobic infections (all with axillary hidradenitis suppurativa) had had recurrent abscesses, often with a chronic discharge, for between two and 10 years. One of these five patients (case 6) had a coexisting anaerobic perianal abscess, from which *Bacteroides* spp (not *B. fragilis*, but not further identified) was isolated. Another patient (case 12) with bilateral hidradenitis suppurativa had a co-

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Results of culture and gas-liquid chromatography (GLC) of pus obtained from 12 anaerobic axillary abscesses

Case No	Age	Sex	Fatty acids in pus on GLC*	Obligate anaerobes	Aerobes
1	60	F	ND	<i>Bacteroides</i> spp <i>B. corrodens</i> <i>Peptococcus asaccharolyticus</i> <i>B. melaninogenicus</i> <i>B. ovatus</i> †† <i>B. fragilis</i> † <i>B. bivius</i> <i>Peptostreptococcus</i> spp <i>Bacteroides</i> spp <i>B. oralis</i> † <i>Fusobacterium nucleatum</i> <i>Peptococcus</i> spp	
2	23	F	ND	<i>B. corrodens</i> <i>B. melaninogenicus</i> <i>P. asaccharolyticus</i> † <i>Peptostreptococcus anaerobius</i> <i>P. asaccharolyticus</i> <i>P. prevotii</i> †	
3	37	M	Butyric Isovaleric	<i>Peptostreptococcus</i> spp <i>Bacteroides</i> spp <i>B. oralis</i> † <i>Fusobacterium nucleatum</i> <i>Peptococcus</i> spp	
4	44	F	Isobutyric Butyric Isovaleric	<i>B. corrodens</i> <i>B. melaninogenicus</i> <i>P. asaccharolyticus</i> † <i>Peptostreptococcus anaerobius</i> <i>P. asaccharolyticus</i> <i>P. prevotii</i> †	
5	22	M	Butyric		<i>Proteus mirabilis</i>
6	35	M	ND		Skin flora
7	31	M	ND	<i>Peptostreptococcus</i> spp <i>B. melaninogenicus</i> <i>P. prevotii</i> <i>Peptococcus</i> spp	
8	19	F	ND	<i>B. melaninogenicus</i> <i>Eubacterium alactolyticum</i> <i>Bacteroides</i> spp <i>B. corrodens</i> <i>B. melaninogenicus</i> <i>B. corrodens</i> <i>Peptococcus</i> spp <i>B. ovatus</i> †† <i>Peptococcus</i> spp <i>E. alactolyticum</i> <i>Fusobacterium</i> spp <i>B. melaninogenicus</i> † <i>B. melaninogenicus</i> <i>Peptostreptococcus anaerobius</i> <i>F. nucleatum</i> <i>B. corrodens</i> <i>Bacteroides</i> spp <i>B. fragilis</i> †† <i>Peptococcus</i> spp <i>Eubacterium</i> spp	
9	47	F	Isobutyric Butyric Isovaleric Caproic		
10	51	M	Isobutyric Butyric Isovaleric		<i>Streptococcus milleri</i>
11	27	M	Isobutyric Butyric Isovaleric		
12	23	F	ND		

*Excluding acetic, lactic, and propionic acids, which were produced in all cases.

†Penicillin resistant.

††Tetracycline resistant.

ND = Not done.

existing pilonidal abscess, which had preceded the onset of the axillary infection by several months; seven different anaerobes were isolated from the pilonidal abscess, all of which were also cultured from the axillary specimen. Two of the 34 patients from whom only skin flora was recovered on culture also had axillary hidradenitis suppurativa.

Axillary tissue was examined histologically in four patients from whom anaerobes were grown. Three of these patients, who had clinical hidradenitis suppurativa, showed keratin plugging of the pilosebaceous follicles (fig 1), while the fourth had a small keratin-filled "epidermal cyst" possibly representing a further manifestation of pilonidal

FIG 1—Pilosebaceous follicle plugged by laminated keratin. Haematoxylin and eosin $\times 40$ (original magnification).

occlusion. Prominent apocrine sweat glands were distended by retained secretions and lined by a flattened epithelium (fig 2). All types of skin appendage were surrounded by a mild lymphocytic infiltrate, and in three cases underlying abscesses were surrounded by chronic inflammatory granulation tissue containing hair fragments and foreign-body giant cells.

FIG 2—Axillary sweat glands, some of which are dilated. Haematoxylin and eosin $\times 40$ (original magnification).

Discussion

Axillary abscesses are usually assumed to be staphylococcal, and staphylococci have also long been considered to be the main pathogens in hidradenitis suppurativa.⁵⁻⁷ Thirty years ago, however, American workers reported isolating anaerobes from two patients with axillary hidradenitis,^{2,3} and in the UK Greely⁸ suggested that an anaerobic organism might be the "chief basic offender" in this condition characterised by "a peculiar putrid, odious, foul smelling, actually stinking, sickening, and nauseating type of purulent discharge." Our experience has confirmed these predictions and shown that anaerobes, not staphylococci, are the pathogens in hidradenitis suppurativa and are also responsible for an appreciable number of all axillary abscesses.

We isolated anaerobes from about 25% of our cases of axillary abscess, in only 25% of which were aerobes also isolated. This contrasts greatly with our experience of anaerobic infection associated with the gastrointestinal tract, where aerobes, often coliforms and streptococci, are invariably isolated with anaerobes. Multiple anaerobic species were cultured from nine of the 12 axillary abscesses, with up to nine different organisms being obtained from a single specimen, whereas Meislin *et al*¹ reported a single anaerobic species "on average" in their cases. Most probably the better the specimen and the greater the laboratory expertise the greater will be the number of anaerobic species isolated.

Peptococci accounted for 10 (24%) of the 42 anaerobic isolates in our study, a proportion similar to that reported by Meislin *et al*,¹ and were thus most common. *B. melaninogenicus* and *B. corrodens* were also common (17% and 12% of isolates), whereas in the study of Meislin *et al* only 5% of isolates were *B. melaninogenicus* and *B. corrodens* was not found at all. In both series *B. fragilis* accounted for only 5% of isolates.

Interestingly, an earlier axillary swab in case 12 had grown only "scanty skin flora" on culture, but when swabs were taken by the microbiologist, delivered direct to the laboratory, and processed at once by a technician experienced in isolating anaerobes nine different anaerobic species were isolated. Possibly those specimens from which only skin flora or no

bacteria were isolated might also have yielded anaerobes had transport and laboratory conditions been optimal.

Wide surgical drainage is the established treatment for axillary abscesses, and its efficacy was clearly shown in this series as only three abscesses, caused by *Staph aureus*, recurred. Hidradenitis suppurativa presents a quite different clinical picture of multiple small abscesses with undermining and induration of the axillary skin and persistent foul discharge. Three of our seven patients underwent surgical drainage in the acute phase and were treated with metronidazole for two or three weeks. In two patients the infection did not recur, and the third was lost to follow-up. The patient in case 8 attended the casualty department almost weekly for 10 months with recurrent bilateral anaerobic abscesses after the birth of her second child. Three abscesses required repeated drainage, and she received metronidazole for six weeks with minimal effect and unacceptable nausea. She was finally cured by wide excision of the axillary skin. The patient in case 12 responded partially to metronidazole, the discharge lessening and its foul odour disappearing. Surgery was planned but not until drastic weight loss had been achieved. Two further patients underwent surgery with satisfactory results.

Hidradenitis suppurativa is traditionally believed to be a disease of apocrine sweat glands, although the keratin plugging that characterises this condition⁹ affects the pilosebaceous follicles rather than sweat ducts. In our cases the histological appearances suggested apocrine and pilosebaceous obstruction, and both may be important in promoting infection. We have recently emphasised the association of keratin plugging of

breast ducts with anaerobic infection in non-puerperal subareolar breast abscesses.⁴

Axillary abscesses are one of the commonest cutaneous infections and when associated with foul pus can be assumed to be anaerobic. Whether antimicrobial agents have an important role in managing most abscesses of this type, whatever the pathogen, is doubtful. Recurrent axillary sepsis and particularly hidradenitis suppurativa seem to suggest secondary infection with anaerobic bacteria of an underlying structural abnormality. While antimicrobials with specific activity against anaerobic bacteria may help in controlling acute exacerbations of hidradenitis suppurativa and certainly reduce the putrid odour of the discharge, patients with this condition usually require extensive surgical intervention to effect a permanent cure.

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Hospital outbreak of trimethoprim resistance in pathogenic coliform bacteria

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Summary and conclusions

In an investigation of outbreak of infection caused by coliform bacilli with plasmid-mediated trimethoprim (TMP) resistance many patients were found to be asymptomatic carriers of TMP-resistant coliform bacilli. Analysis of factors predisposing to rectal carriage of these organisms showed that the most important was previous treatment with co-trimoxazole, a sulphonamide, or ampicillin. The outbreak was controlled by a policy restricting the antibiotics given.

Geriatric units are an important source of hospital infection. When an outbreak occurs the logical sequence of steps to be taken is to monitor cases, identify the outbreaks, analyse the causative factors, plan corrective action jointly with laboratory staff, and monitor the outcome.

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Introduction

Co-trimoxazole, a combination of sulphamethoxazole and trimethoprim (TMP), was released for clinical use in 1969. Although strains of coliform bacteria resistant to TMP were found before the drug was introduced,¹⁻³ bacterial resistance to TMP has not generally increased.⁴ Initially, resistance to TMP was thought to be chromosomal, but in 1971 it was shown⁵ that some patients in St Pancras Hospital had infections due to coliform bacilli carrying R factors mediating TMP resistance. We describe an outbreak of plasmid-mediated TMP resistance in coliform bacilli isolated from patients in that hospital between 1972 and 1976 and the methods used to control it.

Methods

Identification of infection due to TMP-resistant bacilli—Specimens were cultured by standard techniques, and antimicrobial sensitivity tests were performed by Stokes's technique⁶ using discs containing 1.25 mg of TMP. Such tests were performed on organisms thought to be causing infections that might reasonably be treated with co-trimoxazole but not on organisms causing infections not usually so treated. Thus the recognition of TMP-resistant pathogens was incomplete.

Screening for asymptomatic carriage of TMP-resistant coliforms—All patients in a particular ward had a rectal swab taken using saline-moistened swabs. These were heavily inoculated without delay on to freshly poured plates of sensitivity test agar (DST, Oxoid) containing