Mini-review

The role of anaerobic bacteria in cutaneous and soft tissue abscesses and infected cysts

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Abstract

This review presents the aerobic and anaerobic microbiological aspects and management of cutaneous and soft tissue abscesses, paronychia, anorectal, pilonidal, and perirectal abscesses, infected epidermal cysts, hidradenitis suppurativa, and pustular acne lesions. These infections often occur in different body sites or in areas that have been compromised or injured by foreign body, trauma, ischemia, malignancy or surgery. In addition to group A beta-hemolytic streptococci and Staphylococcus aureus, the indigenous aerobic and anaerobic cutaneous and mucous membranes local microflora usually is responsible for these generally polymicrobial infections. These infections may occasionally lead to serious potentially life-threatening local and systemic complications. The infections can progress rapidly and early recognition and proper medical and surgical management is the cornerstone of therapy.

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Cutaneous and soft tissue abscesses and infected cysts infections are among the most common infections, and may sometimes lead to serious local and systemic complications. These infections can be potentially life-threatening infections that may have rapid progress. Their early recognition and proper medical and surgical management is therefore of primary importance.

In addition to group A beta-hemolytic streptococci (GABHS) and Staphylococcus aureus, the indigenous aerobic and anaerobic cutaneous and mucous membranes local microflora usually is often responsible for polymicrobial infections. Anaerobic infections of the skin and soft tissue frequently occur in areas of the body that have been compromised or injured by foreign body, trauma, ischemia, malignancy or surgery. Because the indigenous local microflora usually is responsible for these infections, anatomic sites that are subject to fecal or oral contamination are particularly at risk (Fig. 1).

This review summarizes the aerobic and anaerobic microbiological aspects and management of cutaneous and soft tissue abscesses, paronychia, anorectal abscesses, pilonidal abscess, perirectal abscess, infected epidermal cysts, hidradenitis suppurativa, and pustular acne lesions.

1. Cutaneous abscesses

Subcutaneous and cutaneous abscesses can be caused by polymicrobial aerobic and anaerobic bacteria. Although the primary management of these infections is usually through surgical drainage, knowledge of their microbiology allows institution of empiric antimicrobial therapy prior to the availability of culture results.

1.1. Microbiology

The commonest organisms involved in skin and soft tissue infections are S. aureus and GABHS [1]. They frequently cause impetigo, furunculosis, cellulitis, and wound infections [2]. Recently, many of the S. aureus isolates are methicillin resistant. Gram-negative enteric bacteria (i.e. Enterobacter spp., and Escherichia coli) are also recovered occasionally.

The predominant anaerobes are Gram-positive cocci, Gram-negative bacilli (including Bacteroides fragilis group
and, *Prevotella* and *Porphyromonas* spp.), and *Fusobacterium* spp. [1–3]. Anaerobes predominate in infections of the vulvovaginal, buttocks, perirectal, finger, and head areas. Aerobic bacteria are prevalent in the neck, hand, leg, and trunk areas. Many of these infections are polymicrobial. *S. aureus*, the most prevalent aerobe, is found whenever abscesses originate from skin surface. It is, however, recovered less often from the buttocks, perirectal, and vulvovaginal areas. The infections at these latter sites generally originate from adjacent mucous membranes rather than skin. Among Gram-negative aerobes, *Enterobacter* spp. are recovered mostly from the trunk and legs, while *E. coli* is recovered mainly from the vulvovaginal, buttocks, and perirectal areas. *Peptostreptococcus* spp. which are normal skin inhabitants and part of the endogenous gastrointestinal flora [4], are also recovered from infections at all sites. *B. fragilis* group, which predominate in the feces, are cultured most frequently from abscesses of the perirectal area. Pigmented *Prevotella* and *Porphyromonas* spp., which occurs in stools as well as in the oral cavity [2,4], are isolated from infections proximal to these sites and from the head and neck. Most strains of *B. fragilis* group and many of *Prevotella Porphyromonas* and *Fusobacterium* spp. are resistant to penicillins. Beta-lactamase-producing bacteria (BLPB) are recovered in about half of the abscesses.

1.3. Diagnosis

Redness, tenderness, heat, and swelling are characteristic of skin and subcutaneous tissues infections. Associating lymphangitis is characterized by the presence of reddish streaks extending proximally and associated with tender enlargement of regional lymph nodes. Systemic symptoms can be mild, and include fever, and malaise. Fluctuation in the abscess indicates that it is ready for drainage. Laboratory findings include leukocytosis, rapid sedimentation rate, and often positive blood cultures. Some organisms can cause bacteremia more frequently, and manipulation including surgical incision of the abscess may be followed by transient bacteremia.

Pus or material recovered by aspiration or incision should be Gram stained and cultured for both aerobic and anaerobic bacteria as well as fungi. Radiological studies may reveal localized collections of pus when free gas is present or when abnormal tissue density is observed. Ultrasound, computed tomography (CT), angiography, and radionuclide scans may be helpful [5].

1.4. Management

Surgical drainage is the treatment of choice. Although antimicrobials may prevent suppuration if given early or prevent spread of an existing abscess, they are not an adequate substitute for surgical evacuation. Heat application can relieve the pain and facilitate suppuration and liquefaction.

Some antimicrobials can be partially inactivated by pus and by low pH (aminoglycosides and quinolones). The activity of some antibiotics that are effective against multiplying organisms (i.e. beta-lactams) is impaired by the failure of bacteria to multiply in pus. Furthermore, phagocytosis is reduced in the abscess cavity. Because of the combination of these factors, many abscesses are resistant to antimicrobial therapy.

Because anaerobic bacteria frequently are associated with cutaneous abscesses, especially in areas adjacent to mucosal membranes, their presence should be anticipated if antimicrobial therapy is given. Appropriate antimicrobials include clindamycin, metronidazole, cefoxitin, a
carbapenem (e.g. imipenem, meropenem, ertapenem), or a combination of a beta-lactamase inhibitor (i.e. clavulanic acid, tazobactam) and a penicillin (i.e. amoxicillin, ticarcillin, piperacillin). If staphylococcal infection is suspected, or when no initial clue for etiology is available, a penicillinase-resistant penicillin (e.g. oxacillin) is given. Macrolides or vancomycin can be administered to penicillin allergic individuals, and an aminoglycoside, or quinolone, or a fourth generation cephalosporin (i.e., cefazidime, cefepime) can be given when Gram-negative aerobic bacilli are suspected. Recently, there has been an increase in the recovery of methicillin resistant *S. aureus* (MRSA). Patients with serious infections where *S. aureus* is suspected should therefore be initially given agents active against MRSA until susceptibility results are available. These include vancomycin, daptomycin, linezolid, quinupristin/dalfopristin and tigecycline.

1.5. Complications

Locally or systemically spread of the infection may occur. Local spread generally follows the path of least resistance along fascial planes. Lymphatic spread may lead to lymphangitis, lymphadenitis, or bubo. Involvement of veins may lead to infective thrombophlebitis, bacteremia, septic embolization, and systemic dissemination.

2. Paronychia

Paronychia is an inflammation of the structure surrounding the nails. Whether acute or chronic, paronychia results from a breakdown of the protective barrier between the nail and the nail fold. The introduction of organisms into the moist nail crevice results in the bacterial or fungal (yeast or mold) colonization of the area. It is common in housewives, cleaners, nurses, children who suck their fingers, or those who often have their hands in water [6]. Paronychia occurs more common in women than in men, with a female-to-male ratio of 3:1.

2.1. Microbiology and pathogenesis

The bacteriology of paronychia is polymicrobial due to aerobic and anaerobic bacteria in about three fourth of the cases. The predominant aerobic organisms are *S. aureus*, *Streptococcus* spp., *Eikenella corrodens*, GABHS, *Klebsiella pneumoniae*, *Proteus* spp., *P. aeruginosa*, and *Prevotella* spp. The predominant anaerobic bacteria are Gram-negative bacilli of oral origin (*Prevotella* and *Porphyromonas*), *Fusobacterium*, and *Peptostreptococcus* spp. BLPB are present in about half of the patients [7].

The anaerobic organisms isolated are generally part of normal oropharyngeal flora that are self-inoculated through nail biting and finger sucking from the patient’s own mouth flora onto the finger. This phenomenon is parallel to the acquisition of infection following human bites and clenched fist injuries. In studies that applied methodology for cultivation of both aerobic and anaerobic organisms in such infections, anaerobic organisms were recovered from about half of the patients studied [8].

Acute paronychia usually results from a trauma, which breaks down the physical barrier between the nail bed and the nail; this disruption allows the introduction of pathogens. Activities, such as nail biting, finger sucking, manicuring, or artificial nail placement can also induce such trauma.

Chronic paronychia often occurs in individuals whose hands are repeatedly exposed to moisture or have prolonged and repeated contact with irritants or chemicals. Especially susceptible are housekeepers, dishwashers, bartenders, and swimmers.

2.2. Diagnosis

Acute paronychia is manifested by erythema, edema, fever, and tenderness. There is less erythema in chronic paronychia, with is characterized by a cushion like thickening of the paronychial tissue. The nail plates can be thickened and discolored, with pronounced transverse ridges.

This infection generally starts as a subcuticular or intracutaneous infection with local exudate which eventually spreads under the fingernail base. Infection may follow the nail margin or extend beneath the nail and suppurate. Rarely, it can penetrate deep into the finger, causing tendon necrosis, and osteomyelitis. The chronically infected nail eventually becomes distorted.

Bacterial culture for aerobic and anaerobic bacteria is indicated especially when the exudate is purulent. A microscopic examination in potassium hydroxide and culture for *Candida* and dermatophytes are also indicated. A large amount of budding yeast on potassium hydroxide examination suggests that *Candida* may be of etiologic significance. A positive culture for *Candida* in the absence of a positive potassium hydroxide examination and clinical signs suggestive of candidiasis may indicate that the organism is a nonpathogen.

2.3. Management

An acute infection is best treated with hot compresses or soaks of the affected finger 3–4 times per day and an appropriate systemic antibiotic. If the infection does not resolve or progresses to an abscess, it should be drained promptly. A purulent pocket should be opened cautiously with a scalpel. Infection extending along the tendon sheaths requires prompt surgical incision and drainage.

The initial treatment of chronic paronychia consists of the avoidance of inciting factors such as exposure to moist environments or skin irritants. Proper recovery is facilitated by keeping the affected lesion dry. Choice of footgear for infection of the toes may also be considered. Mild cases of chronic paronychia can be treated with warm soaks. Chronic paronychia caused by dermatophytes that are
The abscess is usually formed initially within the inter-
suppuration and abscess formation within the anal gland.
Infection of the static glandular secretions results in
arising mostly from the obstruction of anal crypts.

3.2. Pathogenesis

The infection is generally polymicrobial due to aerobic
and anaerobic bacteria. The predominant anaerobes are
Gram-negative bacilli (including B. fragilis group and
pigmented Prevotella and Porphyromonas spp.), Gram-
positive anaerobic cocci, Fusobacterium and Clostridium
spp. The predominant facultatives and aerobes are E. coli,
S. aureus, GABHS, P. aeruginosa, and Proteus morganii
[9,10].

3.3. Diagnosis

The clinical presentation correlates with the abscess
anatomical location. Patients with perianal abscesses often
complain of dull perianal discomfort and pruritus. The
pain often is exacerbated by movement and increased
perineal pressure from sitting or defecation. Physical
examination demonstrates a small, erythematous, well-
defined, fluctuant, subcutaneous mass near the anal orifice.

Ischiorectal abscesses typically present with systemic
fevers, chills, and severe perirectal pain and fullness
consistent with the more advanced nature of this process.
External signs are minimal and may include erythema,
induration, or fluctuance. On rectal examination, a
fluctuant indurated mass can be encountered.

Intersphincteric abscesses are sometimes difficult to
diagnose and present with rectal pain and localized
tenderness on examination. Suspicion of an intersphinc-
teric or supravaginal abscess may require confirmation by
CT scan, magnetic resonance imaging (MRI), or anal
ultrasonography.

3.4. Management

Drainage is the mainstay of therapy. The abscess should
be incised to prevent spread of the infection. Fistulous
tracts must be opened and excised. Gram stain and cultures
should be done. Administration of antimicrobials effective
against anaerobic bacteria and enteric Gram-negative rods
is generally essential especially in the presence of a systemic
inflammatory response, diabetes, or immunosuppression.
The antimicrobials effective against anaerobes include:
clindamycin, cefoxitin, chloramphenicol, or metronidazole.
An aminoglycoside, a quinolone or third-generation
cephalexins provide coverage for Gram-negative enteric
rods. Single-agent therapy with cefoxitin, a carbapenem or
the combination of a penicillin (such as ampicillin or
ticarcillin) and a beta-lactamase inhibitor (such as sulbac-
tam or clavulanic acid) may be adequate.

3.5. Complications

Complications include septicemia, anorectal fistulas,
anal gangrene, and abscess recurrence.
4. Pilonidal abscess

Pilonidal sinus is a cyst which is a small midline closure defect that can collect debris and subsequent become inflamed. When it communicates with the subarachnoid space it serves as a route of bacterial entry into the central nervous system. It occurs more common in males than in females.

4.1. Microbiology and pathogenesis

The infection is generally polymicrobial due to enteric facultative aerobic and anaerobic bacteria. Anaerobic isolates can outnumber aerobes at a ratio of 5:1 [11]. The predominant anaerobic organisms were Gram-negative bacilli (including \textit{B. fragilis} group and pigmented \textit{Prevotella} and \textit{Porphyromonas} spp.), Gram-positive anaerobic cocci, \textit{Fusobacterium} spp., and \textit{Clostridium} spp. The main aerobic organisms are \textit{E. coli}, \textit{Enterococcus} spp., \textit{Proteus} spp., and \textit{Pseudomonas} spp.

4.2. Diagnosis and management

Physical findings depend on the stage of disease. In the early stages a sinus tract or pit is present in the sacrococcygeal area which can progress to midline edema or abscess. Examination of the abscess may reveal tenderness, fluctuance, warmth, purulent discharge, induration and cellulitis. Fever and other systemic signs of infection are uncommon.

Surgical drainage is the therapy of choice. However, antimicrobial therapy is also needed. The antimicrobial choices are similar to the one for perirectal abscess.

4.3. Complications

Complications include recurrence, abscess formation, systemic infection, squamous cell and verrucous carcinoma.

5. Infected epidermal cysts

Epidermal cysts are closed sacs with a definite wall that result from proliferation of surface epidermal cells. Production of keratin and lack of communication with the surface are responsible for cyst formation. Epidermal cysts can become infected, and an abscess can develop.

5.1. Microbiology

The organisms causing most epidermal cyst infections are \textit{S. aureus}, \textit{GABHS}, \textit{E. coli} and aerobic and anaerobic bacteria that originate from the normal flora adjacent to the site of the cyst infection. Anaerobes are isolated in about half of the patients. The predominant anaerobic organisms are \textit{Peptostreptococcus} spp. and Gram-negative bacilli (including 12 pigmented \textit{Prevotella} and \textit{Porphyromonas} spp. and \textit{B. fragilis} group) [12].

\textit{S. aureus} is the predominant isolate in infections in the trunk and extremities, but anaerobes are frequently isolated in cyst abscesses in rectal, vulvovaginal, head, and scrotal areas.

5.2. Management

Surgical drainage is the treatment of choice for an epidermal cyst abscess. However, recurrences are frequent because the keratin producing lining of the cyst is not removed. Administration of systemic antimicrobials may be indicated in selected cases, especially in immunocompromised patients or in instances where local or systemic spread of the infection has occurred.

Antimicrobial treatment of mixed infections requires the administration of antimicrobials effective against both aerobic and anaerobic bacterial components of the infection. Antimicrobials that provide coverage for \textit{S. aureus} as well as the anaerobic bacteria include cefoxitin, clindamycin, a carbapenem, and the combination of beta-lactamase inhibitors and a penicillin. A combination of metronidazole and a beta-lactamase-resistant penicillin can be an alternative. Coverage against MRSA is discussed at the section on cutaneous abscesses.

6. Hidradenitis suppurativa

Hidradenitis suppurativa (HS) is a recurrent inflammation of the apocrine sweat glands, particularly those of the axilla, genital, and perianal areas. It can result in obstruction and rupture of the ducts and the development of a secondary infection. The lesions generally drain spontaneously, with formation of multiple sinus tracts and with hypertrophic scarring. Although not initially infected, the lesions frequently become secondarily infected. Often, patients with HS also are afflicted with acne, pilonidal cysts, and chronic scalp folliculitis; thus, which is termed follicular occlusion tetrad.

6.1. Microbiology and pathogenesis

The infection is generally polymicrobial due mainly to aerobic and anaerobic bacteria of skin and proximal mucous membranes origin. Anaerobic bacteria alone or in combination with aerobic organisms can be isolated from about two-thirds of patients. The most frequently aerobic bacteria are \textit{S. aureus} (isolated from about a third of cases), \textit{GABHS}, micro-aerophilic streptococci, and \textit{P. aeruginosa}. The predominant isolated anaerobes are \textit{Peptostreptococcus}, \textit{Prevotella}, \textit{Fusobacterium} and \textit{Bacteroides} spp. [13,14].

The anaerobes isolated from the patients depend on the site of the infection and are part of the flora of the oropharynx (\textit{Prevotella} spp., \textit{Fusobacterium} spp., \textit{Peptostreptococcus} spp. and microaerophilic streptococci), gastrointestinal tract (\textit{Bacteroides} spp., \textit{Peptostreptococcus}...
spp.) [4] and skin (Peptostreptococcus spp.) and presumably reached the HS lesions from these sites.

6.2. Diagnosis

The primary lesions are reddish-purple nodules that gradually become fluctuant and drain. Irregular sinus tracts with repeated crops of lesions are formed, and reparative processes are only partially successful. The involved areas show a mixture of burrowing, draining tracts, and cicatricial scarring. In some cases, HI is associated with acne conglobata or dissecting cellulitis of the scalp, that is often associated with spondyloarthropathy.

The patient often presents with pain, multiple red, hard, raised nodules in areas where apocrine glands are concentrated. As suppuration progresses, surrounding cellulitis may emerge. Chronic recurrences result in palpable thick sinus tracts under the skin, which can turn into draining fistulas. In chronic condition the multiple nodules can coalesced and become surrounded by a fibrous reaction resulting in scarred and unsightly appearance of the area.

HI can present as a primary condition, but it may also be observed in association with: Crohn disease, irritable bowel syndrome, Down syndrome, Graves disease or Hashimoto thyroiditis, Sjögren syndrome, arthritis, and herpes simplex.

Culture of blood and any exudate and/or aspiration or drainage of larger nodules for aerobic and anaerobic bacteria should be obtained.

6.3. Management

Treatment of this condition is difficult and involves the administration of antimicrobial therapy, and moist heat locally to establish drainage in the initial phases of the infection. Large abscesses are surgically drained.

Gram’s stain results can guide the selection of empiric antimicrobial therapy. However, the final choice of agents should be determined by the isolation of specific organisms, aerobes and anaerobes, and the results of antimicrobial susceptibility testing.

Initial empiric antimicrobial therapy should be effective against S. aureus as well as potential aerobic and anaerobic pathogens. Antimicrobial agents active against S. aureus and anaerobic bacteria include clindamycin, a carbapenem, cefoxitin, and beta-lactamase inhibitor and penicillin combinations, and metronidazole with beta-lactamase-resistant penicillin. Cefoxitin and carbapenems also provide coverage against Enterobacteriaceae. However, agents active against Enterobacteriaceae (i.e., aminoglycosides, a quinolone, a fourth-generation cephalosporins) should be added when treating infections involving these bacteria. Coverage against MRSA is discussed at the section on cutaneous abscesses.

7. Pustular acne lesions

Acne vulgaris, a disorder of the pilosebaceous apparatus, is the most common skin disorder of the second and third decades of life.

7.1. Microbiology and pathogenesis

Bacterial factors are important in the pathogenesis of acne. Acne is believed to be associated and perhaps even caused by Propionibacterium acnes [15]. The improvement in acne patients treated with systemic antibiotics effective against P. acnes, as well as other organisms, support this concept.

The morphogenesis of acne lesions can be divided into two phases. The first one is non-inflammatory, during which keratin accumulates in affected follicles producing whiteheads (closed comedones), which have very small orifices, and blackheads (open comedones) which have distended orifices. The second phase is an inflammatory one during which a variety of inflamed lesions may develop from some of comedones.

P. acnes is known to be associated with the inflammatory process in acne lesions [15], Propionibacterium spp. possess immunostimulatory mechanisms such as complement activation, stimulation of lysosomal enzyme release from human neutrophils, and production of serum-independent neutrophil chemotactic factors [16]. Organisms other than P. acne may contribute to the inflammatory process. These include Peptostreptococi and anaerobic Gram-negative bacilli such as Porphyromonas and Prevotella spp. [17]. A recent study highlighted the polymicrobial nature of over two-thirds of culture positive pustular acne lesions and suggests the potential for pathogenic role of aerobic and anaerobic organisms other than P. acnes and Staphylococcus spp. in acne vulgaris [17].

7.2. Management

Antimicrobial therapy is a common adjuvant in the management of acne vulgaris. Topical or systemic antimicrobial agents effective against anaerobic bacteria including P. acnes (i.e. clindamycin, macrolides and tetracyclines) are beneficial. The empirical choice of antimicrobials may not always provide coverage for some of the resistant organisms that can be recovered from pustular acne lesions.Processing pustular specimens for aerobic and anaerobic bacteria can provide guidelines for adequate management of infected acne lesions.

References