Bacteriology of Inflamed and Uninflamed Epidermal Inclusion Cysts

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Objective: To determine whether inflamed and uninflamed epidermoid cysts differ in the number and/or type of bacteria inhabiting them.

Design: A controlled study. We obtained aerobic and anaerobic bacterial culture specimens from 25 inflamed and 25 uninflamed epidermoid cysts.

Setting: A university medical center.

Patients: Nonimmunocompromised adults without recent systemic use of antibiotics.

Results: The 2 groups did not differ significantly with respect to number of bacterial isolates, “no growth” cultures, and aerobic, anaerobic, or potential pathogens cultured.

Conclusions: The microbiological milieu of inflamed epidermoid cysts is similar to that of uninflamed cysts. Possible mechanisms for inflammation are discussed.

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The epidermal inclusion cyst is a common acquired skin cyst. When such cysts become inflamed they are often referred to as infected and treated by incision and drainage and often by administering systemic antibiotics. The presupposition that infection plays a significant role in the inflammatory process has never been studied in a controlled manner. Only 2 studies have addressed this issue in any manner. In 1977 Leppard et al1 cultured 11 uninflamed epidermoid cysts and found that 73% grew common skin commensals, including Staphylococcus epidermidis, anaerobic gram-positive cocci, and Corynebacterium acnes. In 1980 Brook2 cultured 231 “epidermal cyst abscesses,” 192 of which yielded bacterial growth. One hundred eight of the 192 grew anaerobic organisms (predominantly Peptostreptococcus species and Bacteroides species) and 135 of 192 grew aerobic organisms (predominantly Staphylococcus aureus). Twenty-three patients in this study had previous antimicrobial therapy. Brook stressed the importance of anaerobic bacteria and S. aureus in the infected cysts and recommended surgical drainage as the treatment of choice with the addition of antimicrobials such as cefoxitin or a combination agent of imipenem-cilastatin sodium in selected cases. Valentine3 points out that this was not a controlled study yet concedes that S. aureus likely played a role in some of the infected cysts.

Our study was undertaken to better define the microbiological milieu of the inflamed and uninflamed epidermal inclusion cyst.

RESULTS

Twenty-five inflamed and 25 uninflamed cysts were cultured. Patients ranged in age from 21 to 78 years. The mean age of patients with inflamed cysts was 49 years (age range, 21-78 years) and the mean age for those with uninflamed cysts was 46 years (age range, 22-74 years). The percentage of women was 40% in the inflamed and 32% in the uninflamed cyst groups. The location of the cysts was similar in both groups (Table 1).

Each group did not differ significantly with respect to number of isolates, average number of isolates per patient, no growth cultures, aerobic cultures, anaerobic cultures, normal flora, or potential pathogens (Table 2 and Figures 1 through 3). The majority of positive cultures were quantitated at 2+ or 3+. The positive cultures from the inflamed cysts were quantified as follows: 1+ growth (n=6), 2+ (n=11), 3+ (n=12), and 4+ (n=3). The positive cultures from the uninflamed cysts were quantified as fol-
PATIENTS AND METHODS

Patients were recruited from the outpatient dermatology service at the University of Texas Medical Branch at Galveston. All patients were 18 years or older with an epidermal inclusion cyst that was being removed for therapeutic or cosmetic reasons. Any patient who had received antibiotics within 1 month of their procedure or who was immunosuppressed was excluded.

An inflamed cyst was defined as a cyst known to present for months to years that subsequently developed fluctuance and erythema and contained a localized collection of purulent material in addition to keratin debris on incision. An uninflamed cyst was defined as an intradermal nodule without any evidence of inflammation that yielded a cheesy keratinous material.

The overlying skin was prepared with povidone iodine or chlorhexidine gluconate, then isopropyl alcohol; 1% lidocaine was administered locally. Cysts were incised with a sterile surgical blade and then a sterile swab was inserted into the cyst. One swab was reinserted into the standard aerobic transport media (Culterette II Collection and Transport System, Becton Dickinson & Co, Cockeysville, Md) and the other into anaerobic transport media.

Specimens were processed routinely by the University of Texas Medical Branch microbiology laboratory and plated within 1 hour after arrival onto the following media: sheep blood agar, chocolate agar, colistin-nalidixic acid agar, MacConkey blood agar with heme and vitamin K, blood agar with kanamycin sulfate, and vancomycin and thioglycolate broth (the latter 3 are anaerobic media). Plates were incubated at 35°C in 5% to 10% carbon dioxide. An anaerobic chamber was used to incubate the plates for anaerobic growth. Microorganisms were identified using the standard methods of Gram stain, aerotolerance, and Rapid ANA (antinuclear antibody) System (Innovative Diagnostic Systems LP, Norcross, Ga) II. The Manual of Clinical Microbiology was used as a reference. No identification of species was performed for coagulase-negative staphylococci, diphtheroids, enterococci, viridans streptococci, or Bacillus species.

Statistical analysis was performed to test for the differences in the proportions of isolated organisms across the 2 groups using the test

\[ Z = \frac{\hat{p}_1 - \hat{p}_2}{\sqrt{\hat{p}(1-\hat{p})\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}} \]

where \( z \) is distributed approximately and \( \hat{p} \) is an estimate of the common parameter defined as the sum of the specific organism count from both groups divided by the sum of the total number of isolates from both groups.

Coagulase-negative staphylococcus was the most frequently cultured organism in both groups (16 of 25 inflamed cysts and 13 of 22 uninflamed cysts of the aerobic positive cultures). Staphylococcus aureus was infrequently seen, being identified in 2 inflamed cysts and 1 uninflamed cyst. Among the 14 positive anaerobic cultures in each group of 25 cysts, Peptostreptococcus was the most commonly identified genus, isolated from 7 specimens in both the inflamed and uninflamed cyst groups.
H. L. Mencken (1880-1956) said, “For every human problem, there is a neat, simple solution; and it is always wrong.” A logical hypothesis for the pathogenesis of inflammation in the epidermoid cyst is that pathogenic organisms are present and responsible for the clinical change in the cyst; in other words, the cyst becomes infected. This explanation has been widely accepted but never tested in a controlled study. In our study, no significant difference was found between the microbiological features of inflamed and uninflamed epidermal inclusion cysts nor was there evidence that the organisms that were cultured had contributed to the inflammation. We were unable to discern any pattern to the types of organisms or the quantities cultured from the inflamed and uninflamed cysts.

Why, then, do epidermoid cysts become inflamed? If bacteria are not responsible for the inflammation, what is? Although this problem was not addressed in our study, we hypothesize that rupture of the cyst wall with resultant extrusion of its contents into the dermis contributes to the inflammation. Takematsu et al5 have studied the extrusion of its contents into the dermis. Also unanswered is why the cyst ruptures in the first place. Is it induced by trauma or is there some other initiating event? We did not study the conditions within the cyst, for example the pH or pO2. Does this affect any bacteria that might be present?

The mainstay of therapy of the inflamed cyst has been incision and drainage, with or without the use of systemic antibiotics. Brook2 recommends drainage and use of broad-spectrum systemic antibiotics to cover for both staphylococcus and anaerobic organisms in selected cases. Ho and McLean7 recommend that “a fluctuant, probably infected cyst should be incised, drained, and cultured; if there is no improvement, antibiotic treatment should be started.” Young and Mathes8 recommend incision and drainage alone for an “acutely infected cyst.” Would the inflamed cyst resolve without any treatment? If the organism is not significantly contributing to the inflammation, then would use of antibiotics with known anti-inflammatory properties, such as erythromycin or tetracycline, alter the clinical course? It would be interesting to perform a 4-armed controlled study on the natural course of the inflamed epidermoid cyst with and without incision and drainage and with or without the use of systemic antibiotics.

In summary, we found no apparent difference in the microbiological milieu of inflamed and uninflamed epidermoid cysts. We hope this study generates a renewed interest in critically examining the thoughts and practices regarding inflamed epidermoid cysts. We believe questions regarding the pathogenesis and appropriate treatment should be addressed in additional controlled studies.

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REFERENCES


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without associated monoclonal gammopathy. We report a case of scleredema in a patient with diabetes mellitus that responded to electron beam therapy after treatment with topical, intraleisional, and systemic corticosteroids had failed. Two years after receiving electron beam therapy, the patient remains free of disease. This treatment should be considered as a therapeutic option in patients with persistent scleredema and associated systemic complications.

REFERENCES


Submissions

Clinicians, local and regional societies, residents, and fellows are invited to submit cases of challenges in management and therapeutics to this section. Cases should follow the established pattern. Submit 4 double-spaced copies of the manuscript with right margins nonjustified and 4 sets of the illustrations. Photomicrographs and illustrations must be clear and submitted as positive color transparencies (35-mm slides) or black-and-white prints. Do not submit color prints unless accompanied by original transparencies. Material should be accompanied by the required copyright transfer statement, as noted in “Instructions for Authors.” Material for this section should be submitted to George J. Hruza, MD, Cutaneous Surgery Center, Suite 16411, 1 Barnes Hospital Plaza, St Louis, MO 63110. Reprints are not available.

Correction

Error in Expansion. In the article titled “Bacteriology of Inflamed and Uninflamed Epidermal Inclusion Cysts” published in the January issue of the ARCHIVES (1998;134:49-51), ANA was mistakenly expanded as “antinuclear antibody” on page 50. Actually, it should have been RapID ANA System.