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Bacterial Colonization and Histopathological Changes in Foreskin Tissue of Circumcised Children

Haqi Ismael Jasim albadry , Harith Ahmed Mustafa
University of Samarra, College of Education, Department of Biology
*Correspondence email: Haqi.asmaeil@gmail.com

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ABSTRACT
Background: The penile foreskin is a retractable fold of skin covering the glans and is rich in microbial flora. Circumcision, one of the most common pediatric procedures performed for cultural, religious, or medical reasons (such as pathological phimosis), removes this tissue and may alter local microbial colonization and infection risk. Post-operative wound infections remain a concern, with both Gram-positive and Gram-negative bacteria implicated **This study aims** to identify the bacterial species colonizing the foreskin of young children and to examine the histopathological effects of these microbes on foreskin tissue. Methods: Foreskin samples were collected from 49 boys (aged 1 day to 12 years) undergoing circumcision. Prior to excision, a sterile swab of the inner foreskin was taken and cultured on nutrient media to isolate bacteria. Isolates were identified using biochemical tests (e.g. catalase) and confirmed with an automated VITEK© system. Foreskin tissue specimens were fixed in 10% formalin, processed and embedded in paraffin, sectioned at 6 µm, and stained with hematoxylin and eosin (H&E) for histological examination. **Results:** Bacterial growth was obtained from 40/49 (81.6%) swabs. The most common isolates were Staphylococcus aureus (33%) and Escherichia coli (30%), followed by Klebsiella pneumoniae (15%), Pseudomonas aeruginosa (12%), and Proteus mirabilis (10%). Microscopic examination of the foreskin tissue revealed no significant age-related differences in baseline skin structure. However, samples with bacterial colonization showed notable inflammatory and degenerative changes. There was subepidermal inflammatory cell infiltration, vascular congestion, hemorrhage, and focal necrosis of the stratified squamous epithelium, along with fragmentation of the keratin layer and collagen fibers in the dermis. E. coli in particular was associated with intense dermal inflammation, edema, and accumulation of neutrophils, whereas other aerobes (e.g. S. aureus, P. mirabilis, K. pneumoniae) tended to cause milder superficial changes. These findings indicate that bacteria play a central role in post-circumcision tissue damage and inflammation. Conclusion: The foreskin of children is frequently colonized by potentially pathogenic bacteria, notably S. aureus and E. coli. Circumcision samples demonstrated histopathological evidence of inflammation and tissue injury associated with these microbes. Awareness of the common organisms and their effects on wound tissue can inform preventative strategies and prompt treatment to reduce post-circumcision infectious complications.

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1. INTRODUCTION

The foreskin (prepuce) is a loose, retractable fold of skin and mucous membrane that covers the glans penis and forms an essential part of the male external genitalia (1) (2). It is more than a simple skin flap; many consider the foreskin to provide protection to the glans and contribute to penile sensitivity. Anatomically, the inner surface of the foreskin is lined by variably keratinized stratified squamous epithelium, similar to other frictional mucosal surfaces such as the oral and vaginal mucosa (6). The foreskin's epithelium is continuous with the external penile skin and the mucosa of the glans, creating a unique environment where microorganisms can reside. Usually, the foreskin is regarded as just a redundant piece of skin (1) (2) or a simple fold of skin and mucosa (3); however, its removal via circumcision can potentially eliminate certain health risks associated with foreskin pathology and infections (1).

Circumcision is among the most common pediatric surgical procedures worldwide. It is often performed for religious or cultural reasons, but it also serves therapeutic purposes – for example, to treat pathological phimosis or recurrent balanoposthitis (23). Typically, only a small amount of skin is removed during neonatal circumcision (4). Despite its frequency and generally low risk, post-circumcision complications such as infection can occur. In fact, surgical site infection rates after circumcision have been reported in some studies to range from 70% to 90%, depending on factors such as the definition of infection, use of prophylactic antibiotics, and sample size (3) (4). Wound infections can significantly impede healing and may lead to adverse outcomes. Both Gram-positive and Gram-negative bacteria present on the skin can colonize the circumcision wound and cause sepsis of the site, which in severe cases may become life-threatening (5).

There is substantial evidence on wound healing pathophysiology indicating that damaged blood vessels in an injury can hinder the delivery of immune factors (antibodies and immune cells) to the tissue, thereby creating a favorable environment for microbial growth (8). Even though a fresh surgical or burn wound is initially sterile, it can rapidly become colonized by opportunistic bacteria if proper hygiene and antisepsis are not maintained (8). Organisms that commonly colonize skin or the periurethral area – such as Staphylococcus aureus, Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, and Proteus mirabilis – are frequently implicated in wound infections (8). These pathogens can cause local tissue damage, delay wound healing, and occasionally lead to systemic infection.

Untreated chronic inflammation or infection of the foreskin (balanoposthitis) can have serious consequences. It may result in scarring and pathologic phimosis, meatal stenosis, urethral stricture, or even progression to squamous cell carcinoma of the penis in rare cases (7) (19). Balanitis and balanoposthitis are especially common in young uncircumcised boys under five years of age, particularly those with phimosis (21). Nonetheless, these infections can also occur in infancy; understanding their etiology in the context of circumcised infants is important for early management.

Objective: The primary aim of this study is to identify the bacterial species that colonize or infect the foreskin in pediatric patients and to evaluate the histopathological changes in the foreskin tissue associated with these bacteria. By determining the most common microbes and their tissue effects, we hope to better understand the infectious complications of the foreskin and the benefits of circumcision in preventing recurrent balanoposthitis and related pathological changes.

2. MATERIALS AND METHODS

Sample Collection

This study was conducted in the microbiology laboratory of the College of Education, University of Samarra (Salah al-Din, Iraq) from August 1, 2024 to December 10, 2024. A total of 49 foreskin samples were collected from male children ranging in age from 1 day old to 12 years old. These children were undergoing circumcision in local clinics in Samarra. Before surgical removal of the foreskin, a sterile cotton swab was used to take a swab from the inner surface of the foreskin (the mucosal side that lies against the glans). The swab was immediately cultured by inoculation onto nutrient agar media and incubated at 37°C for 24 hours to promote bacterial growth. Subsequent subcultures were made on selective and differential culture media as necessary to obtain pure isolates.

Bacterial Identification

Isolated bacteria were initially characterized by colony morphology and standard biochemical tests. For Gram-positive cocci, a catalase test was performed to differentiate staphylococci (catalase-positive) from streptococci (catalase-negative). All bacterial isolates were then identified to the species level using the VITEK automated identification system (bioMérieux, France). This system uses biochemical and enzymatic profiling for precise identification. The procedure involved preparing a bacterial suspension of the isolate in 0.45% saline (adjusted to the recommended turbidity), inoculating the appropriate VITEK ID cards, and incubating in the VITEK analyzer, which provided organism identification based on its database. Identification results from the VITEK were cross-checked with the biochemical test results for consistency.

No antibiotic susceptibility testing was performed as part of this study; the focus was on identification of the pathogens present. All culture work and identification followed standard microbiological protocols and quality controls (14).

Histological Slide Preparation

Immediately after circumcision, each foreskin tissue specimen was fixed in 10% neutral buffered formalin for at least 24 hours. The tissues were then processed for histology following standard paraffin-embedding techniques (9). Briefly, fixed tissues were washed in water, then dehydrated through a graded series of ethanol (70%, 80%, 90%, and 100% alcohol). After dehydration, tissues were cleared in xylene and then infiltrated with molten paraffin wax at ~60°C. The paraffin-infiltrated tissues were embedded in paraffin blocks, which were then sectioned at 5–6 µm thickness using a rotary microtome. The sections were mounted on glass slides and stained with hematoxylin and eosin (H&E) (9). After staining, slides were coverslipped with DPX mounting medium.

Histopathological Examination

Prepared slides were examined under a light microscope by a pathologist without prior knowledge of the specific microbial findings for each case. Histological evaluation focused on the condition of the epidermis and dermis of the foreskin, looking for inflammatory changes, degenerative changes (such as necrosis or degeneration of cells), and any other pathological alterations. Particular attention was given to the presence of inflammatory cell infiltrates (e.g., neutrophils, lymphocytes, macrophages), the state of the keratin layer, evidence of tissue necrosis, edema, hemorrhage, and fibrosis. Representative photomicrographs were captured from the slides showing key findings. Images were taken at various magnifications (primarily ×10 and ×40 objectives) using a digital microscope camera. These images were later labeled to highlight specific histological features corresponding to the presence of bacteria. Figure numbers were assigned to each distinct image, and each labeled feature in the images was denoted by a letter (A, B, C, etc.) in the figure captions.

All procedures were conducted in accordance with ethical standards. The use of de-identified foreskin specimens from routine circumcisions for research was approved by the local institutional review board, and verbal informed consent was obtained from the parents or guardians of the children.

3. RESULTS AND DISCUSSION

Bacterial Isolation and Frequency

Out of the 49 foreskin swab samples cultured, 40 (82%) yielded bacterial growth, whereas 9 samples (18%) showed no bacterial growth. A variety of bacteria were isolated from the positive cultures. The distribution of organisms is summarized as follows: Staphylococcus aureus was the most frequently isolated pathogen, found in 33% of culture-positive samples (13/40). The next most common isolate was Escherichia coli, present in 30% of positive samples. Klebsiella pneumoniae accounted for 15% of isolates, Pseudomonas aeruginosa for 12%, and Proteus mirabilis for 10%. No other significant bacterial species were identified beyond these five.

In many cases, the foreskin swabs grew mixed flora; however, the analysis focused on these predominant pathogenic or opportunistic bacteria. All S. aureus isolates were coagulase-positive, confirming them as Staphylococcus aureus (and not coagulase-negative staphylococci). The E. coli and Klebsiella isolates were lactose-fermenting Gram-negative rods distinguishable on MacConkey agar, whereas P. aeruginosa produced characteristic pigment on cetrimide agar and P. mirabilis exhibited swarming on blood agar. The high prevalence of S. aureus and Gram-negative enteric bacteria (E. coli, Klebsiella) highlights the mixture of skin and fecal flora that can colonize the foreskin, especially in diapered infants and young boys.

Histopathological Findings

Microscopic examination of H&E-stained foreskin sections revealed that the baseline histological structure of the foreskin skin was similar across the age range of the children in the study. The epidermis of the inner foreskin is a stratified squamous epithelium that showed variable keratinization. In the absence of infection, the epithelium was unremarkable, with a thin keratin layer, and the dermis contained connective tissue with blood vessels and sparse inflammatory cells, as expected for normal foreskin histology.

However, inflammation and tissue changes were noted in many specimens, especially those from which bacteria were isolated. Common histopathological changes observed included:

- Inflammatory cell infiltration: Many foreskin tissues showed infiltration of inflammatory cells, particularly neutrophils and macrophages, in the subepithelial (subdermal) region and sometimes extending into the epidermis. This was often associated with the presence of bacteria such as S. aureus and E. coli. In some sections, clusters of neutrophils (microabscesses) were seen within the epidermis, indicating pustular dermatitis of the foreskin.
- Epithelial degeneration and necrosis: Degenerative changes in the stratified squamous epithelium were evident in areas colonized by bacteria. Focal necrosis of the epidermis was observed as pallor or loss of nuclear detail in keratinocytes, sometimes with outright necrotic debris on the surface. The keratin layer (stratum corneum) in affected areas appeared thinned and fragmented.
- Keratin layer fragmentation: The normally continuous keratin layer was often fragmented or sloughed in infected specimens. This appeared as discontinuous, peeling keratin on the epithelial surface. Such fragmentation was noted particularly in cases with Proteus or Staphylococcus involvement, possibly due to bacterial keratinases or inflammatory exudates disrupting the keratin.
- Dermal edema and collagen fiber disruption: The dermis in many samples showed edema (manifested by pale staining and separation of collagen bundles) and fragmentation of collagen fibers. Collagen bundles that are usually organized appeared shortened and disintegrated in foci where inflammation was intense. This suggests tissue damage and incipient fibrosis due to inflammation.
- Vascular congestion and hemorrhage: Numerous sections showed blood vessels in the dermis that were dilated and engorged with red blood cells (congestion). In some cases, particularly those associated with E. coli, there was evidence of acute hemorrhage in the dermis, with extravasation of red blood cells into the interstitium. Capillaries near the epithelial surface were either packed with red cells or, conversely, some capillaries were empty (possibly due to postmortem artifact or local shunting of blood to other vessels).
- Subepidermal abscess formation: A few of the infected foreskin tissues (notably those with S. aureus, a known pus-forming organism) demonstrated small abscesses just beneath the epithelium, with collections of neutrophils and necrotic debris.

Notably, there was no significant difference in the type of tissue reaction across different age groups – i.e., infants versus older children showed similar patterns of inflammation when infected. Instead, the differences in histopathology correlated more with the type of bacteria present. For instance, tissues with heavy Escherichia coli growth tended to have more marked neutrophilic infiltration, edema, and even small hemorrhages, suggesting a vigorous acute inflammatory response. On the other hand, tissues associated with primarily

Proteus mirabilis or Klebsiella pneumoniae (facultative aerobes) showed somewhat milder surface epithelial damage, though still with some focal keratin layer disruption and inflammatory infiltrates in the dermis.

Representative Histological Images

To illustrate the above findings, representative photomicrographs from the foreskin tissue sections are presented in Figures 1–7 with corresponding descriptions of the labeled features:

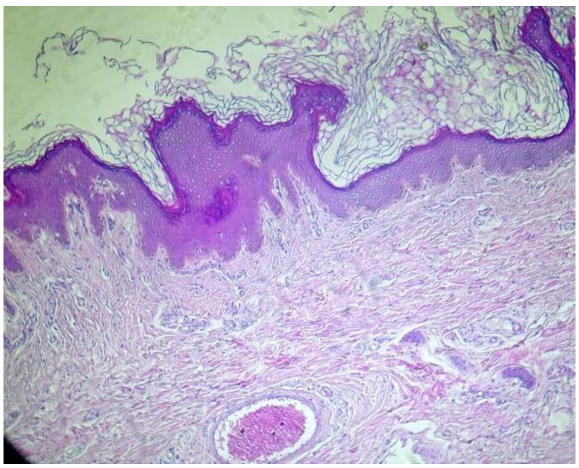

Figure 1: Histological section of a child's foreskin tissue (H&E stain, ×10). The inner foreskin epithelium is thrown into multiple folds. (A) Dark-staining granular layer of the epidermis. (B) Fragmented keratin filaments on the epithelial surface. (C) Bundles of collagen fibers in the dermis. (D) Dilated blood vessel in the dermis engorged with blood (congestion).

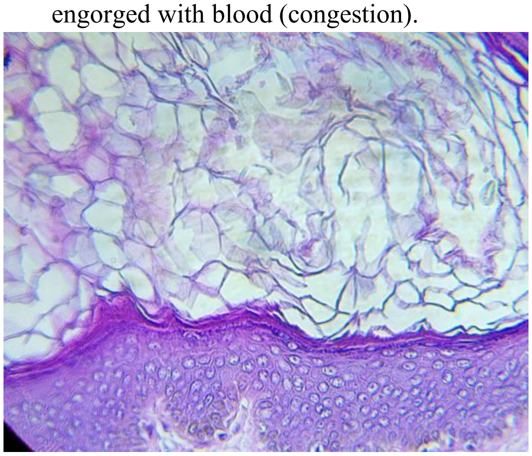

Figure 2: Higher magnification of foreskin epidermis (H&E, ×40). **(A)** Epithelial cells with pale-staining nuclei, indicating some degenerative changes. **(B)** Keratinized granular cells at the surface of the epithelium. **(C)** Network of broken/fragmented keratin fibers (surface debris). **(D)** Cluster of macrophages accumulated just beneath the epidermis.

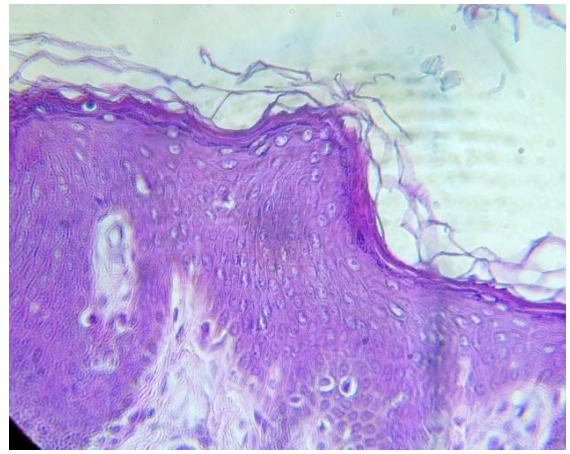

Figure 3: Foreskin epithelium with underlying inflammation (H&E, ×40). **(A)** Collections of macrophages beneath the epidermis (subepidermal). **(B)** Degeneration of basal epithelial cells at the epidermal-dermal junction (vacuolar change in basal layer). **(C)** Fragmented keratin strands peeling from the surface. **(D)** Area of epithelial cell necrosis (loss of nuclei in the keratinocytes).

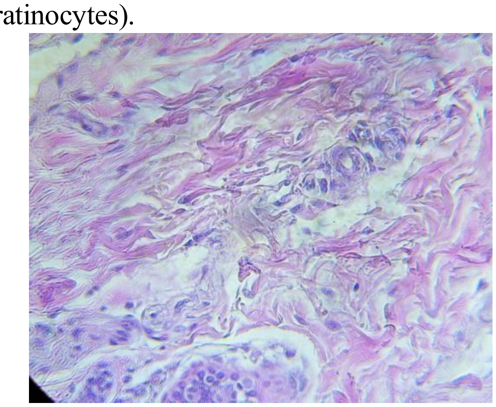

Figure 4: Section of the foreskin dermis showing connective tissue changes (H&E, ×40). **(A)** Short, fragmented bundles of collagen fibers in the dermis indicating structural breakdown of connective tissue. **(B)** Inflammatory infiltrate of white blood cells (leukocytes) dispersed in the dermal tissue. **(C)** Dermal macrophage cells phagocytosing debris.

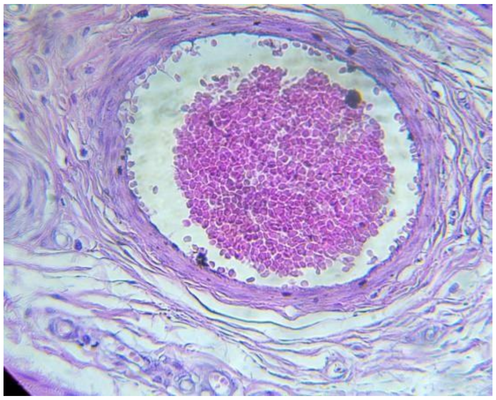

Figure 5: Foreskin dermis with vascular changes (H&E, ×40). **(A)** A blood vessel in the dermis engorged with red blood cells (marked congestion). **(B)** Noticeable dissociation and breakdown of collagen fibers surrounding the vessel. **(C)** Inflammatory white blood cells (leukocytes) present in the vicinity of the vessel.

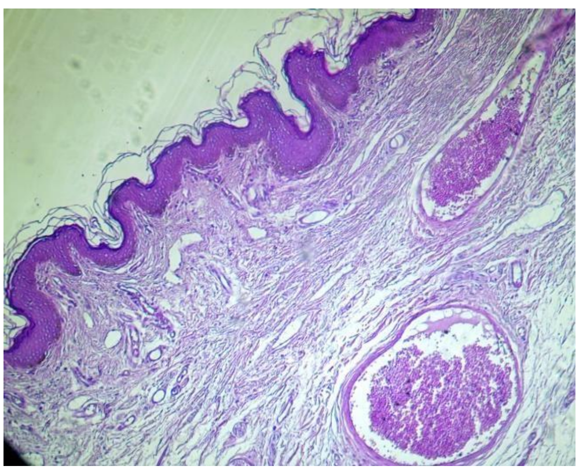

Figure 6: Low magnification view of an inflamed foreskin (H&E, ×10). The epidermis is undulating with multiple folds. **(A)** Folded epidermis with overlying keratin strands. **(B)** Clumped keratin debris on the surface of the epidermal folds. **(C)** Fragmented collagen fiber bundles in the dermis beneath the epithelium. **(D)** Dilated dermal blood vessels filled with blood (hyperemic vessels). **(E)** Small capillaries in the dermis that are empty (devoid of blood), adjacent to congested vessels.

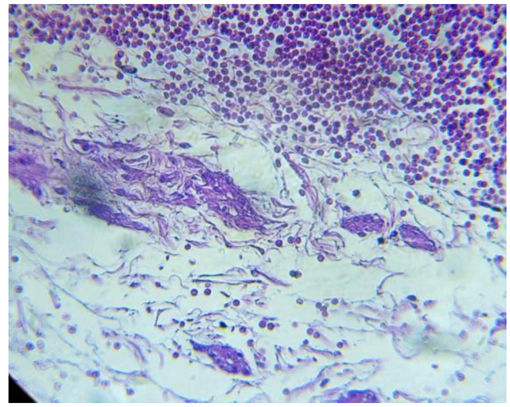

Figure 7: High-power view of foreskin dermis with acute inflammation (H&E, ×40). **(A)** Area of acute hemorrhage (collection of extravasated red blood cells) in the dermis. **(B)** Numerous inflammatory white blood cells (neutrophils) scattered in the dermal tissue. **(C)** Pronounced fragmentation of collagen fibers due to tissue necrosis. **(D)** Aggregates of macrophages (histiocytes) cleaning up debris. **(E)** Capillaries engorged with blood within the inflamed area.

In this study, *Staphylococcus aureus* emerged as the most common bacterial species isolated from the foreskins of circumcised children, comprising about one-third of the positive cultures. This finding is in line with prior reports that *S. aureus* is a leading cause of post-surgical wound infections(5) and is frequently part of the normal skin flora of patients (especially colonizing the nostrils, skin, and perineum) and medical staff(5)(6). The high prevalence of *S. aureus* in our samples may be explained by its ubiquitous presence on skin and its opportunistic nature—circumcision creates a wound that can be readily infected by skin commensals. Contamination of the circumcision site can be endogenous (from the child's own skin flora) or exogenous (from the environment or caregiver's hands). In surgical settings, *S. aureus* is indeed recognized as a major infectious agent, even in operating theaters, being the most common cause of surgical site infections in various procedures(6).

Escherichia coli was the second most prevalent organism (30%) in our study. *E. coli* is not a typical skin inhabitant but is a common gut bacterium; its presence likely reflects fecal contamination, especially in infants and young toddlers who wear diapers. This organism is also a known cause of balanoposthitis and urinary tract infections in boys. The relatively high rate of *E. coli* isolation suggests that careful perineal hygiene is important even after circumcision, as the procedure itself does not eliminate the risk of colonization by enteric bacteria.

The recovery of *Klebsiella pneumoniae*, *Proteus mirabilis*, and *Pseudomonas aeruginosa* in a subset of samples (accounting collectively for ~37% of isolates) underscores that a variety of Gram-negative rods can colonize the foreskin. *Klebsiella* and *Proteus* are also enteric bacteria often linked to diaper area infections, while *P. aeruginosa* is an environmental organism known for colonizing moist areas and having a predilection for causing infections in compromised tissues. A study by Kingston and colleagues(20), which compared infections in circumcised versus uncircumcised males, similarly found that these types of organisms can be involved in balanitis or wound infections. In our findings, Gram-negative bacteria in total slightly outnumbered Gram-positive (*S. aureus*) isolates, which aligns with some reports that Gram-negative pathogens are increasingly common causes of post-circumcision infections(7)(20). The robust nature of *P. aeruginosa*, for instance, allows it to survive even in disinfectant solutions and on hospital surfaces, making it a formidable opportunist in any wound(7(. *Proteus* species are known for their urease activity and association with diaper rash and ammonia odor, which could contribute to inflammation of the foreskin region prior to circumcision.

It is notable that *Staphylococcus aureus*—despite being Gram-positive—was the top pathogen. This can be attributed to its role as a common colonizer and its virulence factors that facilitate wound infection(5). *S. aureus* produces enzymes and toxins (such as coagulase, hemolysins, and leukocidins) that can damage tissue and evade the immune system(5). Additionally, many individuals carry *S. aureus* asymptomatically, so it can readily contaminate a surgical site from the patient's skin or the hands of caregivers if aseptic technique falters.

The **histopathological analysis** of the foreskin tissues provides insight into how these bacteria affect the tissue at the microscopic level. Our observations indicated an acute inflammatory reaction in many samples with positive cultures. The presence of neutrophils, edema, and vascular congestion is typical of acute inflammation caused by bacterial infection(22). The stratified squamous epithelium of the inner foreskin in infected cases showed degeneration and focal necrosis. This finding is consistent with what is seen in other inflammatory conditions of the genital skin. For example, **Alyami et al. (2018)** examined foreskin specimens for suspected lichen sclerosus and frequently found evidence of chronic inflammation; in their series, most pediatric foreskin samples showed acute or chronic inflammatory changes, even when taken for a specific clinical indication(17). In our study, we similarly noted acute inflammatory features such as dermal infiltrates of inflammatory cells, subepidermal clefting, and fibrin deposition, which correspond with the description by Alyami et al. of pathological lesions in foreskin tissue. Notably, we documented congestion and hemorrhage alongside inflammatory infiltrates and epithelial necrosis, which parallels the "acute inflammation" noted by Alyami and colleagues(17).

The degenerative changes in the epithelium and the fragmentation of collagen in the dermis observed in our samples are akin to changes reported in balanitis xerotica obliterans (BXO, or lichen sclerosus et atrophicus of the penis). BXO is a chronic inflammatory condition; **Clouston et al. (2011)** described histological features of BXO such as an atrophic epidermis with loss of rete ridges, a zone of pallor (hydropic degeneration) in the basal layer, and an underlying band of chronic inflammatory cells(7). Our findings in acute post-circumcision inflammation share some similarities: we saw epidermal atrophy or erosion in focal areas and inflammatory cells beneath the epithelium. Additionally, another study of balanitis in children noted features like epidermal atrophy, basal cell vacuolar (hydropic) degeneration, and dermal inflammatory infiltrates(7). These changes were attributed to chronic inflammation in lichen sclerosus, but the fact that we see overlapping changes in acute infection suggests that persistent or repetitive acute balanoposthitis might lead to chronic inflammatory remodeling of the foreskin tissue if the foreskin were not removed.

Morris and Krieger (2017) have emphasized that bacteria are a major factor in causing penile inflammatory lesions and that circumcision has a preventative role against such conditions(21). They noted that aside from candida and dermatological conditions, bacterial species (especially *Streptococcus* spp.) are among the most common causes of infectious balanoposthitis(21). In their review, streptococci were identified as the second most common cause of balanitis after staphylococci, and they also listed other less common pathogens like *Haemophilus parainfluenzae*, *Klebsiella* spp., *Staphylococcus epidermidis*, *Enterococcus* spp., *Proteus* spp., *Morganella* spp., and *E. coli* as possible causative agents(21). While our study did not isolate *Streptococcus* species from the foreskin (possibly due to the aerobic culture conditions or their absence in our cohort), we did find several of the other organisms they listed (*Klebsiella, Proteus, E. coli*). This concordance underscores that the spectrum of bacteria we identified is clinically relevant and known to cause penile inflammation. *Streptococcus* might not have appeared in our cultures potentially because it often requires specific growth conditions or perhaps prophylactic antibiotics given prior to circumcision in some cases suppressed streptococcal growth.

Importantly, our observations suggest that even very young infants are not immune to foreskin colonization and inflammation. While balanoposthitis is classically described as occurring most commonly in preschool boys (2–5 years of age) who are uncircumcised(21), we found evidence of bacterial presence and tissue reaction in infants as young as 1–2 months old. This indicates that infection of the foreskin can begin in early infancy, likely facilitated by diapers (warm, moist environment that promotes bacterial growth) and possibly limited hygiene at that stage. Escala and Rickwood's classic study (1989) noted that balanitis affects about 4% of boys and is most common in those aged 2–5 years (often around the time of toilet training when smegma accumulation and poor hygiene may occur)(21). Our findings extend this knowledge by showing that significant microbial colonization can occur even earlier, and thus early circumcision (in the neonatal period) might preempt some of these issues by removing the foreskin before chronic colonization and recurrent inflammation set in.

Another point of discussion is the **clinical relevance** of these findings. The identification of *S. aureus* and *E. coli* as the predominant organisms suggests that antibiotic prophylaxis in circumcision might need to cover these bacteria in cases at high risk of infection, or at least that clinicians should be vigilant for infections caused by these organisms. Fortunately, most *S. aureus* isolates (especially community-acquired strains typical in this setting) would be sensitive to beta-lactamase resistant penicillins or first-generation cephalosporins, and *E. coli* and *Klebsiella* (if they cause wound infection) are often sensitive to gentamicin or trimethoprim-sulfamethoxazole, barring resistant strains. However, indiscriminate use of antibiotics is not recommended for every circumcision; rather, good surgical technique and hygiene are the mainstays of prevention. Our data simply highlight which organisms are most likely to appear if infection does occur.

From a histopathological standpoint, routine examination of circumcision specimens is sometimes debated(17). Our study provides a perspective that even in the absence of overt clinical balanitis, microscopic evidence of inflammation is common, which could suggest subclinical or low-grade infections were present. In cases where pathologists do examine foreskin tissue (for suspected pathology like lichen sclerosus), they should be aware that features such as inflammation, hemorrhage, and even early scarring can be due to infection by common bacteria and not only due to specific dermatoses. Gupta et al. (2009) have noted the importance of histopathology in diagnosing infectious diseases when microbiological cultures are inconclusive(22). In our context, while we did have cultures, the histology corroborated the presence and effect of organisms. In resource-limited settings, histology of a foreskin (which is a simple, low-cost procedure) could potentially reveal that an infection was present (through signs of acute inflammation) even if cultures were not done—thus indirectly pointing to an infectious etiology for a child's symptoms.

Overall, our findings reinforce the role of **circumcision in preventing recurrent foreskin infections**. By removing the foreskin, the warm, moist space where bacteria thrive is eliminated, and any existing infection is effectively excised. This likely explains why circumcision can be a definitive cure for chronic or recurrent balanoposthitis. The study also underscores the need for **proper hygiene** in uncircumcised boys: since organisms like *E. coli* are likely introduced from feces, diligent cleaning might reduce colonization and subsequent inflammation. For those who do develop balanoposthitis, our results suggest that empirical therapy could target *S. aureus* and Gram-negative rods.

One limitation of this study is the relatively small sample size and the fact that it was a single-center study. Additionally, we did not perform anaerobic cultures; certain anaerobes could also be present in the subpreputial space but would not have been detected with our methods. We did observe what was described as "anaerobic *E. coli*" effect in tissue (perhaps indicating *E. coli* thriving in poorly oxygenated tissue), but a dedicated anaerobic culture might have revealed organisms like *Bacteroides* or *Clostridium* if present. Another limitation is that we did not correlate each histological change with a specific organism in a rigorous way; our correlations are based on overall trends and knowledge of bacterial pathogenic mechanisms. Future studies could attempt to infect foreskin tissue *ex vivo* or in an animal model to directly observe which lesions each bacterium causes.

In conclusion, this research provides evidence that the **pediatric foreskin is frequently colonized by pathogenic bacteria** and that these bacteria can induce notable histopathological changes in the tissue. The **inflammatory damage** observed microscopically supports the clinical rationale for circumcision in cases of recurrent balanoposthitis or refractory infections. By removing the foreskin, one removes both the nidus for infection and the already damaged tissue, potentially preventing progression to more serious conditions. Our findings also highlight that clinicians should consider *S. aureus* and *E. coli* as prime suspects in any post-circumcision infection and manage accordingly. Moreover, even in asymptomatic boys, foreskin colonization is common, which suggests that education on hygiene and possibly early intervention could be beneficial.

CONCLUSION

This study identified the common bacterial species present on the foreskin of infants and young boys and documented the tissue changes associated with these microbes. We found that ***Staphylococcus aureus*** and ***Escherichia coli*** are the most prevalent colonizers of the pediatric foreskin, followed by ***Klebsiella pneumoniae***, ***Pseudomonas aeruginosa***, and ***Proteus mirabilis***. Histologically, foreskin tissues colonized by these bacteria exhibited acute inflammatory changes, including epithelial degeneration, inflammatory cell infiltration, and stromal disruption (edema, collagen breakdown, vascular congestion, and focal hemorrhage). These changes confirm that bacteria play a major role in causing foreskin tissue damage and inflammation.

Clinically, the results underscore the importance of maintaining hygiene in uncircumcised children and provide support for therapeutic circumcision in cases of recurrent infections. From a preventive medicine perspective, removing the foreskin eliminates the warm, moist environment conducive to bacterial growth, thereby significantly reducing the risk of balanoposthitis and its complications. The study's findings are particularly relevant in pediatric care and public health within communities where circumcision is commonly practiced or is being considered for health reasons.

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