Evaluation of Minor procedures used in the management of pilonidal sinus

ESSAY

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By

Mohamed Hussein Riad Zedan

M.B.B.Ch.

Supervisors

Prof. Dr. AYMAN ABDALLA ABD RABU

Professor of General Surgery
Faculty of Medicine - Ain Shams University

Prof.Dr. MAHMOUD AHMED ELSHAFEY

Professor of General Surgery
Faculty of Medicine - Ain Shams University

Dr. MOHAMED EZZAT AMAN ELSERAFY

Lecturer of General Surgery
Faculty of Medicine - Ain Shams University

Faculty of Medcine

Ain Shams University

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الطبيب/محمد حسين رياض زيدان
بكالوريوس الطب والجراحة

المشرفون

أ.د.أيمن عبد الله عبد ربه
أستاذ الجراحة العامة
كلية طب عين شمس- جامعة عين شمس

أ.د. محمود أحمد الشافعي
أستاذ الجراحة العامة
كلية طب عين شمس- جامعة عين شمس

د. محمد عزت أمان الصيرفى
مدرس الجراحة العامة
كلية طب عين شمس - جامعة عين شمس

كلية الطب
جامعة عين شمس
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INTRODUCTION

Pilonidal disease has been source of debate for many years. **Hodges** in **1880** who described pilonidal disease considered it to be acquired. However, the disease is suggested to be of an embryonic origin **(karulf & perry, 1998).**

In **1833, Herbert Mayo** described a cyst that contained hair just below the coccyx**.** The condition was originally reported by Anderson (1847); in a letter to the editor of the Boston Medical Surgical Journal. **Hodges** in **1880** coined the name "pilonidal" from the Latin words *pilus,* which means hair, and *nidus,* which means nest. Pilonidal disease consists of a spectrum of entities ranging from asymptomatic hair containing cysts and sinuses to a large abscess in the sacrococcygeal area **(Chintapatla et al**., **2003).**

The controversies surrounding the origin of pilonidal disease first came to light during World War II when soldiers were treated for pilonidal disease. The disease was the leading cause of nontraumatic sick days taken by United States soldiers. Most soldiers had recovery times of approximately 100 days **(Chintapatla et al**., **2003).**

It is a common problem in adulthood. In England in 2000-2001, a total of more than 10000 admissions were recorded for pilonidal disease. Pilonidal sinus occurs in the cleavage between

the buttocks (natal cleft) and can cause discomfort, embarrassment and absence from work in addition to recurrence following surgery and the need for frequent and time-consuming wound care **(Coates, 2002).**

Pilonidal sinuses present from the age of puberty to about 40 years of age, but rarely in older people .Three quarters of patients are men, predominantly in their 20s.About 1% of men in the age group at risk develop a pilonidal sinus .Affected women tend to be younger, which is consistent with their earlier onset of puberty. Patients are usually ,but not invariably ,dark and hairy,and are often obese .Pilonidal sinues are less common among Africans and Asians owing to different hair characteristics and growth patterns. Half of the affected patients present as emergencies with an acute pilonidal abscess; the remainder have fluctuating discomfort and chronic infection with afoul smelling discharge. Examination reveals the characteristic opening in the natal cleft ,through which a tuft of hair maybe seen emerging. (**DJ JONES, 1992)**

Many different approaches have been described ranging from a conservation treatment to an extensive surgical excision for pilonidal diseases. But none proved successful in eliminating the complications attendant to such procedures, like delayed wound healing, infection and the rate of recurrence **(Da Silva, 2000)**.

There are several surgical treatments proposed and there is no consensus as to the best technique to be employed. In acute cases, abscess aspiration or drainage with or without curettage are performed. For chronic disease, there are several therapeutic options, such as curettage, excision with no primary suture (open technique), excision with primary suture (closed technique) and marsupialization. Recently, excision associated with flap techniques (fasciocutaneous V-Y flaps, Z-plasty, rhomboid flap and gluteus maximum muscle myocutaneous flap) are found to improve the results. However one technique can give variable results from one centre to another due to variable application of minute details in such technique **(Chintapatla et al**., **2003)**.

Healing usually takes about 2-10 weeks but can take much longer. Recurrence of the pilonidal sinus is not uncommon, so, is important to keep the area free from hairs and as clean as possible to reduce this risk over long periods. Once the wound has fully healed, the infection should not come back. If not, the risk of the pilonidal sinus recurring can be 10% - 25% **(Da Silva, 2000).**

AIM OF THE WORK

This review aims at the study of the different minor procedures described in literature for the management of pilonidal sinus and to evaluate the efficacy compared to the more sophisticated procedures.

SURGICAL ANATOMY

The skin over the sacrococcygeal region is resistant and thick. It is loose and movable over the convexity of sacrum but is bound down in the region of the anal crease. However this skin is thin in contrast with the thicker texture of skin of buttock proper. The natal cleft is maintained because the thin midline skin is attached to underlying ligamentous and aponeurotic fibers on the dorsum of sacrum and coccyx by dense, well defined and highly collagenous fascia. Caudally, this fascia is continuous with a dense mass at the tip of the coccyx, Laterally , each layer is continuous with the superficial fascia of the buttock by fibrous septa. Proximally , the fascia widens at the level of the posterior superior iliac spines to join the superficial fascia of the flanks **(Oliver & Middleditch, 1991).**

Blood supply:

The natal cleft has a poor blood supply derived mainly from medial twigs of posterior penetrating arteries from sacral foramina. The skin nearest the midline is supplied by numerous small and short branches arising from lumbar, lateral sacral and internal pudendal arteries. The lateral region is much better vascularized by the superior and inferior gluteal arteries **(Schoeller et al., 1997)**.

Nerve supply:

The skin overlying the gluteus maximus muscle has rich innervation by the superior clunial nerves from the dorsal branches of L3-L5, the median clunial nerves from the dorsal branches of S1-S3, the inferior clunial nerves from the pudendal nerve and the posterior cutaneous femoral nerve **(Schoeller et al., 1997)**.

EPIDEMIOLOGY

Age incidence:

The average age of onset is 21 years, but the mean age of hospital admission is 25 years. Few patients present for the first time under the age of 17 years, and it is exceedingly rare over the age of 45 years (**Kighley & Williams, 1999).**

Sex incidence:

All forms of pilonidal disease are reported to be more common in men than in women with a relative frequency ratio of between 2.2 and 4 to 1 . **(Sondenaa et al., 1995).**

Racial incidence:

Differences in incidence between races are also found. The incidence a highest among Caucasians and less among Africans and Asians. This probably results from differences in hair characteristics such as kinking, modulation, cuticular scale count and average curvature and from different daily rates of hair growth between races **(Jones, 1992).**

Karydakis in **1973,** has noted that between 1960 and 1971 the prevalence of pilonidal sinus at the time of enrollment into the Greek army rose from 4.9% to 14.8% He suggested that this increase might be due to a 3.2kg rise in the average body weight among Greek army recruits.

AETIOLOGY& PATHOGENESIS

The etiology of pilonidal disease has been source of debate for many years. Hodges (1880) who described pilonidal disease considered the lesions to be acquired. However, the diseaseis suggested to be of an embryonic origin **(karulf & perry, 1998).**

Three main theories were proposed:

- That it represented a vestigial sex gland, homologous with the preen gland of birds **(Stone, 1931).**
- That it originated from remnants of the medullary canal **(Kooistra, 1942).**
- That it evolved from dermal inclusions due to faulty coalescence of the median raphe (Frankowiak & Jackman, 1962).

If the congenital theory is true, then removal of all epithelial tracks to the sacral fascia would be curative. However, a high recurrence rate even with the removal of all tissues overlying the sacrum and coverage with rotation flaps derived away from the midline testifies for other factors to be important in the etiology of pilonidal **(Bascom, 1983).**

Congenital theories also could not account for the lack of intermediate stages between the congenital sinuses and tracks

noted in childhood and the pilonidal disease seen in adults. In contrast to acquired sinuses, congenital tracks are usually located more superiorly, over the lumbar area rather than the sacrum, do not contain hair, frequently communicate with the spinal canal and contain cuboidal epithelium rather than granulation tissue **(Goligher, 1984).**

In 1946 Patey and Scarff , reopened the debate of congenital versus acquired by noting that the interdigital pilonidal sinuses of barbers were pathologically identical to post anal pilonidal sinuses. Since then, there have been other reports of hair causing pilonidal sinus in the interdigital clefts, mammary ducts, penis, axilla and umbilicus.

Currently, the debate on the etiology of pilonidal disease centers on two main theories. One theory is that pilonidal disease is the result of a foreign-body reaction to hairs embedded in the skin, commonly in the midline sacrococcygeal area. Patey and Scarff (1946); noted that although pilonidal tracks contain hair, they do not always contain hair follicles. This would suggest that the hair and not the follicle of the source of the disease. Keratin plugs and other debris may contribute to the inflammation. The inflammation around the hair follows the path of least resistance and often tracks in a cephalic and lateral direction, thus forming secondary tracks and openings **(Karulf & Perry, 1998).**

Bascom, (1980; 1983; 1993) proposed an alternative theory, that the origin of pilonidal disease is in the hair follicles of the pilonidal disease, however hair is, surprisingly, found on only half of the patients. Midline holes, however, appear in nearly every case of pilonidal disease. It has been established that these holes or pits are distorted hair follicles. The normal hair follicles enlarge in the following manner. Keratin accumulates concentrically around the intrafollicular portion of the hair shaft. This generates outward pressure on the follicular wall, thereby increasing its diameter. The follicle enlarges to become a cavity, after which a vacuum eject is created from normal natal cleft motion; when a person is in a sitting position, hair and loose debris (e.g., keratin) are expelled out of the dilated follicle as the gluteal tissues are pushed against the sacrum. Creating a vacuum within the enlarged follicle **(Helmy & Khalil, 1989).**

As the dilated follicle diameter increases, so does its wall tension, which makes it more prone to rupture. The most likely point of rupture is the bottom of the follicle. Keratin begins to bulge out through a break in the wall of the enlarged follicle. Inflammatory cells move up to deal with keratin and a microabscess will soon result **(Bascom, 1983).**

A Similar pathology, called " perforating folliculitis " has been described **(Mehregan & Coskey, 1968).**

The associated edema occludes the follicle's skin orifice, causing pus to accumulate within the ruptured follicle and underlying fat creating an acute pilonidal abscess. When the edema subsides, the follicle's skin orifice becomes patent, allowing pus and follicular debris to drain spontaneously. Epithelium grows slowly down into the follicular remnant and underlying abscess cavity to become a chronic pilonidal sinus **(Bascom, 1983).**

The epithelial lining developed in long standing ceases has no skin structures or hair follicles. These epithelial tubes are usually only 2-5 mm in length (Bascom, 1983).[fig; 1,2].

Hair seems to play three separate roles in pilonidal disease. Hair that remain in the follicle that gave them birth ***play the first role.*** They are shed down through the floor of the follicle and penetrate the follicle wall with keratin sharing the inflammatory response. Gathered hairs ***play the second role*** from the head and back that accumulate in the natal cleft (catch basin effect). Buttock friction aligns the loose hairs into a drill – shaped configuration (Brearley, 1955). These hairs do not penetrate intact skin but enter through an existing hole in the depth of the cleft. Hairs that remain attached to surrounding skin ***play the third role***; they enter by their tips into open pilonidal wounds. These hairs interferer with skin healing **(Bascom, 1983).**