Pilonidal Disease: Review

Pilonidal Sinüs

Kamil GÜLPINAR, MD,[a]
Süleyman ÖZDEMİR, MD,[a]
S. Erpulat ÖZİŞ, MD,[a]
H. Yahya USLU, MD,[a]
Atilla KORKMAZ, MD[a]

[a]Department of General Surgery
Ufuk University Faculty of Medicine,
Ankara

Geliş Tarihi/*Received:* 29.04.2008
Kabul Tarihi/*Accepted:* 08.09.2008

Yazışma Adresi/*Correspondence:*
Kamil GÜLPINAR, MD
Ufuk University Faculty of Medicine,
Department of General Surgery,
Ankara,
TÜRKİYE/TURKEY
kgulpinar@yahoo.com

ABSTRACT Sacrococcygeal pilonidal disease is a common and well-recognized entity. For many years, the cause of sacrococcygeal pilonidal sinus has been a matter of dispute. There was a common lack of success of the surgical methods of excision regarding morbidity, healing, recurrence and cure. It is most likely that sacrococcygeal pilonidal infection originates within a natal cleft hair follicle, which becomes distended with keratin. The distended follicle becomes inflamed and the resulting folliculitis produces edema, which blocks the follicle opening. The obstructed follicle then expands and finally ruptures into the underlying subcutaneous fat to form a pilonidal abscess. Additionally, hair is a secondary invader, being drawn into the abscess cavity by the suction effect along the gluteal cleft that results from the movement of the buttocks. Pilonidal sinus disease consists of symptoms ranging from asymptomatic pits to a painful draining lesion that are predominantly located in the sacrococcygeal region. Asymptomatic pits do not require treatment. Options for treatment of acute abscess include aspiration, drainage without curettage, and drainage with curettage. The choice of a particular surgical approach depends on the surgeon's familiarity with the procedure and perceived result in terms of low recurrence of sinus and a quick healing of resulting cavity or surgical wound. Conservative nonsurgical management, closed methods, laying open of the track, wide excision and open drainage, wide excision and primary closure, and limited excision are the methods currently used.

Key Words: Pilonidal sinus; etiology; clinical protocols

ÖZET Sakrokoksigeal pilonidal hastalık oldukça yaygın görülen ve iyi bilinen bir rahatsızlıktır. Hastalığın nedeni yıllardır tartılaşan bir konu olmuştur. Lezyonun cerrahi metotlar ile çıkarılması, morbidite, iyileşme süreci, nüks ve iyileşme oranları açısından bakıldığında, başarısızlıkla sonuçlanabilmektedir. Sakrokoksigeal pilonidal sinüs enfeksiyonunun, natal yarık follikülünün keratinle distansiyonu sonucu oluştuğu düşünülmektedir. Gerilmiş olan follikülün iltihaplanmasıyla oluşan follikülit ödeme neden olmakta ve follikül ağzını kapatmaktadır. Sonuç olarak, ağzı tıkanan follikül genişlemekte ve alttaki subkutanöz yağ dokusuna doğru rüptüre olarak pilonidal apseye neden olmaktadır. Ek olarak, kıl ikincil etkili ajandır; gluteal yarık içinde kalça hareketi ile oluşan vakum etkisi, kılı apse kavitesi içine doğru çeker. Pilonidal sinüs hastalığı, asemptomatik küçük bir sivilceden, hayli ağrılı, drene olan lezyona kadar geniş bir semptom profili içerir. Asemptomatik hastalıkta tedavi gerekmez. Akut apse için tedavi seçenekleri ise aspirasyon, drenaj ve drenaj + küretaj şeklinde sıralanabilir. Seçilecek cerrahi tedavi yaklaşımının başarısı, nüks oranının düşük ve iyileşmenin hızlı olması ile değerlendirilmektedir ve cerrahın uygulanacak işlemle ilgili tecrübesiyle doğrudan ilişkilidir. Halen kullanılan yöntemler; konservatif tedavi yaklaşımları, kapalı yöntemler, geniş eksizyonla beraber açık drenaj, geniş eksizyonla beraber primer kapama ve sınırlı eksizyon gibi işlemlerdir.

Anahtar Kelimeler: Pilonidal sinüs; etiyoloji; klinik tedavi protokolleri

Turkiye Klinikleri J Med Sci 2009;29(4):959-67

Sacrococcygeal pilonidal disease is a common and well-recognized entity that stands for a debilitating, chronic disease of the natal cleft. Pilonidal disease is most common in the third decade of life; its incidence peaks between 16 and 20 years of age and remains high until the age of 25, thereafter declining rapidly. Men are affected three to four times more commonly than women.[1] It is generally assumed that most patients with pilonidal sinus are hairy and overweight of dark complexion with poor body hygiene.[2]

As patients who are young adults of working age often miss a considerable number of days from work before and after surgery, it attracts considerable attention in terms of human resources and economics.[3]

The condition was first described by Mayo in 1833 and it was suggested to result from the infection of subcutaneous rests of tissue that were present because of imperfect separation of the ectodermal and mesodermal layers during embryological development.[3] The term 'pilonidal' was first used associated with this condition by Hodges in 1880. It comes from the Latin *pilus* meaning 'hair' and *nidus* meaning 'nest'. This term was used to describe the situation of trapped hair in this unusual form of chronic natal cleft skin infection involving the skin and the subcutaneous tissue.[4] It was initially believed that the disease was congenital in origin and various theories were developed, such as the preen gland theory, the neural canal theory and the sacral skin in-drawn theory. But in the last 50 years it has been suggested that the condition is acquired.[5] Pilonidal disease was a major reason for hospitalization during World War II and was felt to be related to the mechanical trauma of riding in jeep, truck, and tank, which supports the idea that the condition is acquired.[6] The infrequent incidence of pilonidal cysts in locations other than the natal cleft that are subject to local trauma from hair, such as the hands of barbers, sheep shearers, and dog groomers also supports the theory of acquired disease.[7,8] There are also case reports of pilonidal cysts at other hairy locations, such as the umbilicus and the scalp.[9,10]

It is most likely that sacrococcygeal pilonidal infection originates from a natal cleft hair follicle, which becomes distended with keratin. The distended follicle becomes inflamed and the resulting folliculitis produces edema, which blocks the follicle opening. The obstructed follicle then expands and finally ruptures into the underlying subcutaneous fat to form a pilonidal abscess.[11] Additionally, hair is a secondary invader, being drawn into the abscess cavity by the suction effect along the gluteal cleft that results from the movement of the buttocks. Studies of surgical specimens have shown the presence of hairs within the cysts, often surrounded by foreign body giant cells.[12] Davage et al proposed that the hair follicles were never found within the cysts, suggesting that the hairs were introduced from outside the lesion.

Bascom suggested that, shafts of hairs apparently are not the source of most pilonidal disease cases; instead, follicles of hairs seemed to be the source.[13] He reported that pilonidal disease progressed through five stages. Accordingly, an infection initially forms about the base of the follicle where keratin breaks through and hairs punch through. Infection generates edema, which swells the mouth of the follicle that eventually closes and this blocks the escape of follicular contents. It also conceals the mouth of the follicle from sight. Follicular contents either are pushed toward fat by the pressure of secretion accumulating in the closed follicle or are pulled toward the fat by vacuum effect. The bottom of the follicle breaks out, releasing infected contents into fat. As a result, an acute pilonidal abscess is formed.and subsequently is drained. As edema subsides, the mouth of the follicle reopens. The follicular remnant that is open at both ends, forms the outer part of the chronic abscess cavity, a cavity which blows and sucks.[13]

The cause for the development of these distorted hair follicles is unclear. Gravity and motion of the gluteal folds have been suggested to create a vacuum force that pulls on the follicles. Local inflammation by bacteria, debris and edema, which occludes the mouth of the follicle, leads to further expansion of the follicle which then ruptures resulting in a foreign body reaction and microabs-

cesses that develop into acute and chronic pilonidal abscesses along with laterally displaced and epithelialized tracts. Once the microabscess becomes a burrowing infection, the disease is defined as a pilonidal sinus.[14]

In dealing with the pathogenesis of pilonidal sinus disease, Karydakis attributed the hair insertion process to three main factors:[15] The invader, which is the loose hair, the force, which causes the insertion, and the vulnerability of the skin to the insertion of hair at the depth of the natal cleft.

He also identified secondary factors and expressed these in an equation.[15,16] Some of these factors had been postulated earlier, but Karydakis showed their relevance in formulating a successful surgical approach.[17] These two theories by Karydakis and Bascom propose the best explanations of pathogenesis of pilonidal sinus disease.[13,15]

CLINICAL FEATURES AND DIAGNOSIS

Pilonidal disease may manifest in various forms such as small asymptomatic pilonidal cysts or sinuses, acute abscess, or complex chronic disease with multiple sinus tracts.[18] They usually occur at midline toward the top of the natal cleft. Asymptomatic disease is recognized by the patient with a painless cystic lesion at the top of the natal cleft. Acute abscess usually presents with acute severe aching and swelling mass over the coccyx following usually the trauma or inflammation of the previous asymptomatic cyst. Pain is the major complaint usually without fever if abscess is localized.[19] Chronic disease may be followed by one or more acute attacks and is characterized by painless drainage from sinus or sinuses connected to the pilonidal cyst. The rate is reported to be 50% for acute abscess and 50% for chronic disease.[20]

The condition may easily be diagnosed with the clinical appearance and complaint of the patient with an acute painful and swelling mass localized roughly at the top of the natal cleft. This condition should be differentiated from some other inflammatory lesions, which may have some similar histological features such as tracts (which could be epithelialized or even contain hair), granulation tissue, foreign-body giant cells and granuloma.[18] These lesions are:

1. Anal fistula, which presents as a perianal extension from an internal opening within the anal canal and is associated with the absence of a midline opening in the postanal region.

2. Hidradenitis suppurativa. These lesions may contain hair shafts.

3. Pyoderma gangrenosum sometimes contains hair shafts.

4. Congenital abnormalities like (i) sacrococcygeal sinus (vestigial remnant of the medullary canal), (ii) presacral sinus or dimple (traction dermoid), (iii) inclusion dermoid, and (iv) treaties.[18]

Neural tube defects like spina bifida should also be considered in the differential diagnosis. Spina bifida refers to a cleft in the spinal column. Spina bifida may be closed (the skin covering the defect is intact) or open (not covered by skin). Spina bifida occulta is the simplest form in which there is failure of the dorsal portions of the vertebrae to fuse with one another. This abnormality, usually localized to the sacrolumbar region, is covered by skin and is not noticeable on the surface except for the presence of a small tuft of hair or other dermal lesion over the affected area; it may resemble pilonidal sinus. If suspected the diagnosis can easily be made by radiographs of the spinal vertebrae.

Although anal abscesses and fistulas can be ruled out by physical examination without difficulty, in extensive disease with deep sinuses magnetic resonance imaging may be required. Mangetic resonance imaging (MRI) features of perianal and deep-seated sepsis, characteristic of fistula in ano, are also found in patients with pilonidal sinus, but the absence of intersphincteric sepsis or enteric opening allows reliable MRI distinction between the two.[21]

Computed tomography (CT) permits structures outside the bowel lumen to be visualized, and is valuable for evaluating suspected perianal abscesses and inflammation.[22,23] In one study, for example, CT (with intravenous and when possible, rectal contrast) was useful in differentiating perirectal ab-

scesses from severe perirectal cellulitis and correctly identified 13 surgically proven abscesses in 10 patients.[24] This imaging technique may be helpful in the differential diagnosis of pilonidal disease and perianal abcess and fistulas.

Nuclear medicine also has an important role in the evaluation of infection and inflammation. FDG (Fluorine 18 fluorodeoxyglucose) PET imaging is a novel promising modality in the diagnosis of infection and inflammation.[25] However, PET scan is rarely used for the identification of pilonidal disease. Recently Ilknur Ak reported a case which illustrated a pitfall associated with F-18 FDG imaging. She presented the images of a 57-year-old woman with non-Hodgkin's lymphoma that showed intense accumulation of F-18 FDG in a sacrococcygeal pilonidal sinus that could indicate a lymphomatous involvement from a primary disease.[26]

Ultrasonography (USG) in the assessment of pilonidal disease is uncommon, but in suspected patients, a sinus tract can be visualized by USG. Moreover, it has also been reported that sacrococcygeal fat thickness measured by USG, as a local factor, is closely associated with pilonidal disease.[27] Endosonography, both blind transrectal (TRUS) and endoscopic ultrasound (EUS) have also been used to evaluate perianal fistulas in the differential diagnosis.[28]

Malignant degeneration of pilonidal disease is a rare condition and occurs in approximately 0.1% of patients who have chronic pilonidal disease.[29] In the literature to date, 68 cases of carcinoma originating from chronic pilonidal disease was reported, three of which occurred in immunosuppressed patients.[30]

It is suggested that this progression is secondary to impaired DNA repair mechanisms seen in chronic inflammation as well as the release of free oxygen radicals from activated inflammatory cells.[29,31]

The malignant lesions tend to be deeply invasive and bone involvement is detected in 8% of patients on diagnosis.[29]

If malignancy is diagnosed, preoperative workout should carefully be initiated to recognize the extent of invasion and presence of metastases. Physical examination of the lesion and inguinal area should be done. Extension of the rectum should be assessed by endoscopy. In addition, CT or MRI can be used to determine the local extent of the disease, occurrence of intraabdominal metastases, and involvement of iliac or para-aortic nodes. PET scan is also a practical modality to identify metastases.[30,31]

Histopathological tissue diagnosis is also helpful in classification and differential diagnosis of pilonidal disease. By histopathological reports, acute and chronic inflammation can be distinguished by the amount and the type of inflammatory cells and tissue reaction. Pure chronic inflammation is characterized by the presence of lymphocytes, plasma cells and histiocytes including foreign body macrophages, with lack of granulocytes. Purely acute inflammation is characterized by the presence of a predominantly neutrophilic exudate, without the obvious presence of chronic inflammatory cells.

MANAGEMENT

The management of chronic pilonidal disease is variable, debatable, and problematic. Eradication of the sinus tract, complete healing of the overlying skin and prevention of recurrence are the principles of treatment. During the last 25 years, many treatments have been suggested for pilonidal sinus but no consensus has emerged and few studies have compared relative treatment costs, discomfort and time off work. The treatment goal of pilonidal sinus is to provide a high chance of cure with a low recurrence rate while avoiding hospital admission and general anesthesia, causing minimal inconvenience, complication rate and time off work for the patient.[32,33]

The treatment of sacrococcygeal pilonidal cyst, despite being considered a well-defined clinical entity has some controversial aspects. There was a frequent lack of success of the surgical methods of excision regarding morbidity, healing, recurrence and cure. All these factors rendered the acquired thesis of pilonidal sinus disease to be more accepted.

Surgery is the principal method of treatment, and several techniques have been proposed, all with the aim of reducing morbidity, offering conditions of fast cicatrisation, reducing recurrence rate and offering cure.

Asymptomatic pits do not require treatment and it is also possible that minimally symptomatic cases may sometimes resolve with meticulous removal of hair from the area by shaving or the use of depilatories.[1]

In most cases, however, either a simple incision and drainage or a more definitive surgical procedure will be required.

ANTIBIOTICS

Routine use of antibiotics are not indicated for the treatment of pilonidal disease. A systemic review of antibiotic use for chronic wounds could not exclude a small benefit in terms of more rapid postoperative healing when perioperative antibiotics were given, but the magnitude of any benefit was modest.[34]

If a significant component of cellulitis is present, antibiotics may be appropriate. If possible, culture from an abscess should be obtained to direct antimicrobial therapy.

The infection of pilonidal disease is generally polymicrobial due to enteric facultative aerobic and anaerobic bacteria (aerobic/anaerobic= 1:5).[35] The predominant anaerobic organisms were gram-negative bacilli (including *Bacteriodes fragilis* group and pigmented *Prevotella* and *Porphyromonas* spp.), gram-positive anaerobic cocci, *Fusobacterium* spp., and *Clostridium* spp. The main aerobic organisms are *Escherichia coli*, *Enterococcus* spp., *Proteus* spp., and *Pseudomonas* spp.[36] The antimicrobials effective against anaerobes include: clindamycin, cefoxitin, chloramphenicol, or metronidazole. An aminoglycoside, a quinolone or third-generation cephalosporins 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 sulbactam or clavulanic acid) may be adequate.[36]

INCISION AND DRAINAGE

For patients who present with an acute pilonidal abscess, simple incision and drainage, which can be done by a primary care physician in the office with local anesthesia, is rationale for initial management. The drainage of abscess with curettage of the cavity has the advantage of quick relief of symptoms and early return to work in all cases.[37] Complete healing occurs in 60% of patients. Recurrence occurs in 25% of those with initial healing and the overall cure rate at 18 months is 75%.[38]

In a study of 73 consecutive patients with a first episode of acute pilonidal abscess who were treated by simple incision and drainage, 42 (58 percent) healed primarily, with a median time to healing of five weeks; 9 of the 42 developed recurrence of pilonidal disease during follow-up (median follow-up 60 months) with an overall cure rate of 45%.[39]

Surgical referral, rather than initial incision and drainage, may be appropriate if the patient is unlikely to change dressings as frequently as required after incision and drainage. In this case, initial surgical excision with primary closure may be preferable. Patients with complex disease with multiple sinus tracts should also be initially referred for surgery.

Other patients who should be referred for surgery are those who have recurrent disease or failure of healing after treatment with simple incision and drainage.

For chronic and recurrent sinuses, various techniques have been reported. Allen-Mersh did an extensive review in 1990 of over 90 papers dealing with the treatment of pilonidal sinus.[11] He concluded, “virtually without exception these studies are flawed because of one or more defects in the study design like lack of randomization, comparison of healing time, recurrence rate, follow up time, etc.” However, he suggested that the choice of a particular surgical approach was dependent on the surgeon’s experience and perceived results in terms of low recurrence of sinus and of quick healing of resulting cavity or surgical wound.

SURGICAL EXCISION

The definitive treatment of pilonidal disease often requires surgery and the condition is often treated by removing the diseased skin and subcutaneous tissue completely. The surgical wound either is closed with sutures (for healing by primary intention) or is packed open (to heal by second intention). Both treatments can lead to recurrences and unhealed wounds.[18] So many clinical trials have compared whether it is better to pack the wound open or to close it.[40]

The cyst and any associated sinus tracts are excised en bloc. The wound may be left open to heal by secondary intention or may be closed primarily. A retrospective study of 272 patients referred for surgical management of nonrecurrent pilonidal disease over ten years in Saudi Arabia compared the open and closed approaches and found that time to healing was significantly longer with the open approach (48 versus 10 days), but hospital stays were shorter (5 versus 10 days).[41] In the primary closure group, there was a 10 percent failure rate because of infection, whereas none of the open wounds failed to heal. There was no significant difference in recurrence (7.3 versus 9.1 percent). The authors concluded that the slightly lower infection rate and shorter hospital stays did not outweigh the benefit of the earlier return to full function generally provided by the primary closure approach. However, the failure rate for primary closure in this study was lower than in some other studies. As an example, in a large case series of 1129 patients treated with primary closure, the failure rate was 16%.[42] Techniques to improve primary closure such as the use of a gentamycin collagen fleece implant have not yet demonstrated clear benefits.[43]

Marsupialization, where the edges of the wound are sutured to the underlying fascia, has been suggested as a compromise between open and closed techniques, as it generally results in a smaller wound than when the incision is left completely open. Reported time to healing with the marsupialization technique is 20 to 35 days.[44] A small randomized trial found that sinus excision led to shorter hospital stays and time off work and better patient satisfaction than marsupialization.[45] In contrast, a larger randomized trial found shorter time to return to work with marsupialization, as well as decreased recurrence rates, but longer time to healing.[46]

In a meta-analysis by McCallum I. et al., relative effects of open healing compared with primary closure were determined. The meta-analysis included 18 randomized controlled trials that assessed the effects of open healing versus closed surgical treatment and the optimal method of closure (midline versus off-midline) for pilonidal sinus. The outcomes were time to healing, surgical site infection, and recurrence rate.[19] The review found no significant difference between open and closed healing in the rates of surgical site infection, and recurrence was significantly less likely after open healing (relative risk 0.42, 95% confidence interval 0.26 to 0.66). Data were inconclusive for time to wound healing. Compared with off-midline surgical closure, midline closure significantly increased healing time, recurrence, and infection of the surgical site. The authors concluded that based on current evidence, off-midline closure should become standard management of pilonidal sinus.

A variety of more complex operations has been used for patients with unusually extensive disease or those who have failed simpler operations. Generally, these procedures involve the use of flaps, which allow for the removal of greater amounts of tissue as well as a decrease in tension in the healing wound. The most commonly used flap techniques are as follows.

The Limberg flap is a series of communicating equilateral triangles. All angles are 60°, which means that every side of both the defect and the flap is equal in length. This orientation creates a flap that is the same size as the defect to be excised. Any flap angle other than 60° theoretically involves either widening or compression of the flap. An elliptical defect at the donor site remains after the flap is rotated to fill the rhomboid defect. This defect can be closed primarily with appropriate undermining of the surrounding tissue.

Recent studies have reported that lateralization of the natal cleft is important for the prevention of midline recurrences. The Karydakis flap

technique was performed to lateralize the natal cleft. An asymmetric ellipsoid excision was made lateral to the natal cleft. After excising the pilonidal sinus down to the level of the sacral fascia, the flap was mobilized on the level of the gluteal fascia. After mobilizing the flap, a Redon-drainage was inserted and a double layer Vicryl 3-0 suture of the subcutaneous tissue closed the defect. Skin closure was made by single knots of 4–0 polypropylene. The drainage was removed routinely on the second postoperative day.

Two randomized trials (n = 100 and n = 50) compared a flap procedure (Limberg flap) with primary closure.[47,48] Both trials found that the Limberg flap procedure was superior in terms of pain, healing, complications, and return to work.

In a study that compared marsupialization, primary midline closure and skin flap interventions in the treatment of pilonidal disease reported that hospital stay for the skin flaps method was longer than marsupialization and primary closure methods. The average time to return to work after marsupialization was significantly longer than the primary closure or the skin flaps methods. There was no difference in wound infection rate or recurrence rates. And the study concluded that there were no differences in terms of wound infection or recurrence rates between the three groups and the period to return to work was relatively shorter; thus the excision and repair techniques were useful in the surgical treatment of pilonidal disease.[49]

Regardless of the surgical technique concerned, standard principles of wound care are essential with repeated depilation of the natal cleft, removal of hair and any debris from the wound bed and keeping the wound edges separated using an appropriate dressing.

Novel treatment modalities are still being studied for the prevention of recurrent disease. In a recent study laser epilation as an adjunctive therapy to surgical excision of the pilonidal sinus was evaluated and was reported to be effective for the treatment of pilonidal disease. Although not curative in and of itself, the removal of hair allows better healing and decreases the chance of recurrence by removal of a significant etiology of pilonidal disease.[50]

The review of studies reveals that various methods are still investigated and no method is universally acceptable. Recurrence rates vary with the technique, operator and length of follow-up. The ideal operation should be performed as a day case or on outpatients, should be simple to perform and have a low recurrence rate with rapid and complete healing with minimal requirement for after care such as shaving or dressings.

FUTURE PROSPECTS

The ideal therapy for pilonidal disease should be quick, allowing patients to return rapidly to normal activity, with minimal morbidity and a low risk of complications. For this purpose, new minimal invasive techniques have been studied for the treatment of pilonidal disease. Some examples are mentioned below.

Using negative pressure wound therapy following surgery for complex pilonidal disease is reported to be a successful technique for recurrent pilonidal disease.[51]

One of the conservative treatments for pilonidal disease is the injection of sclerosing agents. The treatment includes injection of 80% phenol into the sinus tract after depilation and excavation under local anesthesia.[52] Crystallized phenol treatment is also a simple and inexpensive method that can be readily applied on an outpatient basis, decreasing both the recurrence rate and lost work time. This method was suggested as the first-line treatment of any type of pilonidal sinus.[53]

Radiofrequency sinus excision by a radiofrequency device is another new method reported to be performed as a day care surgery in a pilot study. With reduced postoperative pain and early resumption to work, patient satisfaction is better in comparison to excision and marsupialization techniques.[54]

REFERENCES

1. Hull TL, Wu J. Pilonidal disease. Surg Clin North Am 2002;82(6):1169-85.
2. Søndenaa K, Andersen E, Nesvik I, Søreide JA. Patient characteristics and symptoms in chronic pilonidal sinus disease. Int J Colorectal Dis 1995;10(1):39-42.
3. Mayo H. Observations on Injuries and Diseases of the Rectum. 1st ed. London: Burgess and Hill; 1833: p.45-6.
4. Hodges R, Pilonidal sinus. Boston Med Surg J 1880;103:485-6.
5. Talbott T. Pilonidal disease. In: Veidenheimer MC, ed. Seminars in Colon and Rectal Surgery. 1st ed. Philadelphia: WB Saunders; 1990. p.224–6.
6. Buie LA. Jeep disease (pilonidal disease of mechanized warfare). South Med J 1944;37: 103-9.
7. Mohanna PN, Al-Sam SZ, Flemming AF. Subungual pilonidal sinus of the hand in a dog groomer. Br J Plast Surg 2001;54(2): 176-8.
8. Patel MR, Bassini L, Nashad R, Anselmo MT. Barber's interdigital pilonidal sinus of the hand: a foreign body hair granuloma. J Hand Surg Am 1990;15(4):652-5.
9. Allegaert WJ. Pilonidal sinus of the umbilicus. Br J Clin Pract 1967;21(4):201.
10. Moyer DG. Pilonidal cyst of the scalp. Arch Dermatol 1972;105(4):578-9.
11. Allen-Mersh TG. Pilonidal sinus: finding the right track for treatment. Br J Surg 1990;77(2): 123-32.
12. Davage ON. The origin of sacrococcygeal pilonidal sinuses based on an analysis of four hundred sixty-three cases. Am J Pathol 1954;30(6):1191-205.
13. Bascom J. Pilonidal disease: origin from follicles of hairs and results of follicle removal as treatment. Surgery 1980;87(5):567-72.
14. Bascom J. Pilonidal disease: long-term results of follicle removal. Dis Colon Rectum 1983;26(12):800-7.
15. Karydakis GE. New approach to the problem of pilonidal sinus. Lancet 1973;2(7843):1414-5.
16. Karydakis GE. Easy and successful treatment of pilonidal sinus after explanation of its causative process. Aust N Z J Surg 1992;62(5): 385-9.
17. Patey DH, Scarff RW. Pilonidal sinus in a barber's hand with observations on postanal pilonidal sinus. Lancet 1948;2(6514):13.
18. Golladay ES. Outpatient adolescent surgical problems. Adolesc Med Clin 2004;15(3):503-20.
19. McCallum IJ, King PM, Bruce J. Healing by primary closure versus open healing after surgery for pilonidal sinus: systematic review and meta-analysis. BMJ 2008;336(7649): 868-71.
20. Jones DJ. ABC of colorectal diseases. Pilonidal sinus. BMJ 1992;305(6850):410-2.
21. Taylor SA, Halligan S, Bartram CI. Pilonidal sinus disease: MR imaging distinction from fistula in ano. Radiology 2003;226(3):662-7.
22. Fishman EK, Wolf EJ, Jones B, Bayless TM, Siegelman SS. CT evaluation of Crohn's disease: effect on patient management. AJR Am J Roentgenol 1987;148(3):537-40.
23. Yousem DM, Fishman EK, Jones B. Crohn disease: perirectal and perianal findings at CT. Radiology 1988;167(2):331-4.
24. Guillaumin E, Jeffrey RB Jr, Shea WJ, Asling CW, Goldberg HI. Perirectal inflammatory disease: CT findings. Radiology 1986;161(1): 153-7.
25. Love C, Tomas MB, Tronco GG, Palestro CJ. FDG PET of infection and inflammation. Radiographics 2005;25(5):1357-68.
26. Ak I. F-18 FDG imaging of an asymptomatic sacrococcygeal pilonidal sinus in a patient with malignant disease. Clin Nucl Med 2007;32 (10):822-4.
27. Balik O, Balik AA, Polat KY, Aydinli B, Kantarci M, Aliagaoglu C, et al. The importance of local subcutaneous fat thickness in pilonidal disease. Dis Colon Rectum 2006;49(11):1755-7.
28. Deen KI, Williams JG, Hutchinson R, Keighley MR, Kumar D. Fistulas in ano: endoanal ultrasonographic assessment assists decision making for surgery. Gut 1994;35(3):391-4.
29. de Bree E, Zoetmulder FA, Christodoulakis M, Aleman BM, Tsiftsis DD. Treatment of malignancy arising in pilonidal disease. Ann Surg Oncol 2001;8(1):60-4.
30. Malek MM, Emanuel PO, Divino CM. Malignant degeneration of pilonidal disease in an immunosuppressed patient: report of a case and review of the literature. Dis Colon Rectum 2007;50(9):1475-7.
31. Cilingir M, Eroğlu S, Karacaoğlan N, Uysal A. Squamous carcinoma arising from chronic pilonidal disease. Plast Reconstr Surg 2002;110(4):1196-8.
32. Bilgin OF, Bengisun U, Eryavuz Y, Bayar S, Atalay A. Aras N. [The various surgical techniques in pilonidal sinus]. Turkiye Klinikleri J Med Sci 1997;17(3):200-2.
33. Guyuron B, Dinner MI, Dowden RV. Excision and grafting in treatment of recurrent pilonidal sinus disease. Surg Gynecol Obstet 1983;156 (2):201-4.
34. O'Meara SM, Cullum NA, Majid M, Sheldon TA. Systematic review of antimicrobial agents used for chronic wounds. Br J Surg 2001;88(1):4-21.
35. Brook I. Microbiology of infected pilonidal sinuses. J Clin Pathol 1989;42(11):1140-2.
36. Brook I. The role of anaerobic bacteria in cutaneous and soft tissue abscesses and infected cysts. Anaerobe 2007;13(5-6):171-7.
37. Füzün M, Bakir H, Soylu M, Tansuğ T, Kaymak E, Haŕmancioğlu O. Which technique for treatment of pilonidal sinus--open or closed? Dis Colon Rectum 1994;37(11):1148-50.
38. Deans, GT, Spence RAJ, Love AHG. Colorectal Disease for Physicians and Surgeons. 1st ed. Oxford: Oxford University; 1998. p.294-5.
39. Jensen SL, Harling H. Prognosis after simple incision and drainage for a first-episode acute pilonidal abscess. Br J Surg 1988;75(1):60-1.
40. Søndenaa K, Nesvik I, Andersen E, Søreide JA. Recurrent pilonidal sinus after excision with closed or open treatment: final result of a randomised trial. Eur J Surg 1996;162(3):237-40.
41. Chiedozi LC, Al-Rayyes FA, Salem MM, Al-Haddi FH, Al-Bidewi AA. Management of pilonidal sinus. Saudi Med J 2002;23(7): 786-8.
42. Foss MV. Pilonidal sinus: excision and closure. Proc R Soc Med 1970;63(8):752.
43. Holzer B, Grüssner U, Brückner B, Houf M, Kiffner E, Schildberg FW, et al.; EMD study group. Efficacy and tolerance of a new gentamicin collagen fleece (Septocoll) after surgical treatment of a pilonidal sinus. Colorectal Dis 2003;5(3):222-7.
44. da Silva JH. Pilonidal cyst: cause and treatment. Dis Colon Rectum 2000;43(8):1146-56.
45. Oncel M, Kurt N, Kement M, Colak E, Eser M, Uzun H. Excision and marsupialization versus sinus excision for the treatment of limited chronic pilonidal disease: a prospective, randomized trial. Tech Coloproctol 2002;6(3): 165-9.
46. Gencosmanoglu R, Inceoglu R. Modified lay-open (incision, curettage, partial lateral wall excision and marsupialization) versus total excision with primary closure in the treatment of chronic sacrococcygeal pilonidal sinus: a prospective, randomized clinical trial with a complete two-year follow-up. Int J Colorectal Dis 2005;20(5):415-22.

47. Akca T, Colak T, Ustunsoy B, Kanik A, Aydin S. Randomized clinical trial comparing primary closure with the Limberg flap in the treatment of primary sacrococcygeal pilonidal disease. Br J Surg 2005;92(9):1081-4.

48. Ertan T, Koc M, Gocmen E, Aslar AK, Keskek M, Kilic M. Does technique alter quality of life after pilonidal sinus surgery? Am J Surg 2005;190(3):388-92.

49. Aydede H, Erhan Y, Sakarya A, Kumkumoglu Y. Comparison of three methods in surgical treatment of pilonidal disease. ANZ J Surg 2001;71(6):362-4.

50. Schulze SM, Patel N, Hertzog D, Fares LG 2nd. Treatment of pilonidal disease with laser epilation. Am Surg 2006;72(6):534-7.

51. Bendewald FP, Cima RR, Metcalf DR, Hassan I. Using negative pressure wound therapy following surgery for complex pilonidal disease: a case series. Ostomy Wound Manage 2007;53(5):40-6.

52. Hegge HG, Vos GA, Patka P, Hoitsma HF. Treatment of complicated or infected pilonidal sinus disease by local application of phenol. Surgery 1987;102(1):52-4.

53. Dogru O, Camci C, Aygen E, Girgin M, Topuz O. Pilonidal sinus treated with crystallized phenol: an eight-year experience. Dis Colon Rectum 2004;47(11):1934-8.

54. Gupta PJ. Radiofrequency sinus excision: better alternative to marsupialization technique in sacrococcygeal pilonidal sinus disease. J Natl Med Assoc 2005;97(7):998-1002.