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# Pilonidal sinus disease and tuberculosis

P.J. GUPTA

Fine Morning Hospital and Research Center, Laxminagar, Nagpur (India)

**Abstract. – Pilonidal disease is a disease of young people, usually men, which can result in an abscess, draining sinus tracts, and moderate debility for some. It probably results from hair penetration beneath the skin, for reasons that are not totally clear. A pilonidal sinus in the sacrococcygeal region is associated with recurrent infection, abscess formation, cellulitis, and fistulae. The infection is usually chronic and non-specific. However, few reports of granulomatous infection like tuberculosis and actinomycosis in pilonidal sinus have been observed. The presentation of tuberculosis in these sinuses may have wide forms, with atypical and uncharacteristic clinical picture, making it difficult to make a diagnosis preoperatively.**

**This paper discusses about pilonidal sinus disease and tubercular infiltration in these sinuses. The diagnosis of tuberculosis in pilonidal sinus disease is elusive, necessitating a high index of suspicion. The literature on the tubercular affliction of pilonidal sinus is scant. Novel diagnostic modalities such as adenosine deaminase levels and polymerase chain reaction (PCR) can be useful in doubtful situations. Physicians should obtain a thorough history focusing on risk behaviors for human immunodeficiency virus infection (HIV) and tuberculosis.**

**Treatment comprises of standard regimen of antitubercular medication and a modest surgical approach towards the sinuses.**

*Key Words:*

Pilonidal sinus, Tuberculosis, Mycobacterium, Human immunodeficiency virus, Discharge, Sacro-coccygeal.

## Introduction

The name "pilonidal" comes 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[1].

### Pathophysiology

Initially, these cysts were believed to be congenital in nature. One of the more prevalent congenital theories believed that patients with pilonidal disease have persistent remnants of the caudal segment of the neural canal. As the cyst increased in size, they eventually ruptured, resulting in the formation of sinuses tracts. The congenital theory has been called into question for multiple reasons. The male predominance of pilonidal cyst, the presentation in the adolescent period of life, and the recurrence after adequate surgical drainage further encouraged the rejection of the congenital theory[2].

These points lead the way for the modern day acceptance of the acquired theory of pilonidal disease. The acquired theory postulates that pilonidal disease is a result of hair and cellular debris finding a portal of entry into the skin and hair follicles. A vacuum force caused by the touting of skin when the patient bends over is believed to aid in the hair migration. Over time, more and more hair shafts are drawn into the pits by motion from the buttocks. Expulsion in the reverse direction is prevented by barbs on the hair shafts. Keratin accumulation distends the follicle, which eventually forms an epithelialized tube. The entering hair causes an inflammatory reaction and edema. The edema causes occlusion of the skin opening increasing the hair follicle size. This results in a build up in pressure in the hair follicle that eventually spreads its purulent material into the subcutaneous tissue causing a foreign body reaction. This reaction forms multiple micro-abscesses that eventually migrate further into the subcutaneous tissue. These micro-abscesses eventually result in the creation of more sinus tracts and abscesses. When an abscess forms, it drains back to the skin through true sinus tracts. Midline pits are the sine qua non of pilonidal disease and represent hair follicles that have become infected or inflamed. At surgery, however, only 50-75% of all pilonidal cysts were found to contain hair in them[3].

*Corresponding Author:* Pravin J. Gupta, MS; e-mail: drpjg@yahoo.co.in

### Etiology

Pilonidal disease involves loose hair and skin and perineal flora. Risk factors for pilonidal disease include male gender (It is more common in young adult men, a population with a prevalence of 1.1%), hirsute individuals, Caucasians, sitting occupations, existence of a deep natal cleft, and presence of hair within the natal cleft. Family history is seen in 38% of patients with pilonidal disease. Obesity is a risk factor for recurrent disease.

Local trauma is another predisposing factor. Jeep drivers in World War II were subjected to this type of local irritation so frequently that Louis Buie, a Mayo Clinic proctologist, recognized the association and described it in 1944 as "jeep disease".

Pilonidal disease in the general population has a male preponderance. It occurs in the ratio of 3 or 4:1. In children, however, the ratio is the opposite occurring in 4 females for each male it afflicts. Pilonidal disease commonly affects adults in the second to third decade of life. The disease is extremely uncommon after the age of 40 years, and the incidence usually decreases by age 25 years. The average age of presentation is 21 years for men and 19 years for women[4].

The etiology of pilonidal disease as a foreign body reaction is supported by histological examination. It demonstrates foreign body giant cells associated with hair shafts that are embedded in chronic granulation tissue lining the abscess cavity and sinus tracts.

### Presentation

Pilonidal disease has 2 major types of presentations. (1) Completely asymptomatic sinus tracts that are noticed by the patient or primary care physician, (2) Chronic disease. The average patient has 2 years of disease before seeking medical treatment. More than 80% of presentations of pilonidal disease are exacerbations of a chronic sinus tract. The physical findings in pilonidal disease are dependent on the stage of disease at presentation. In the early stages, the patient can notice a sinus tract or pit in the sacrococcygeal region[5].

The majority of patients present with an acute abscess cephalad in the natal cleft. This position distinguishes the disease from other common anorectal problems, such as perirectal abscesses and anal fistulae, which are typically found near the anus. Midline pits are the distinguishing feature, occurring in 100% of cases, and they can typically be identified 4 to 8 cm from the anus. Hair within the abscess cavity is present in approximately two thirds of cases in men and one third of those in woman[6].

As the acute abscess resolves, whether spontaneously or with treatment, chronic sinus tracts develop toward the skin. Chronic or recurrent abscesses with extensive, branching sinus tracts develop in a small minority of patients. This complex variant of the disease may stem from prolonged neglect of symptoms but also occurs despite appropriate treatment.

Pilonidal disease is a clinical diagnosis. Location is the easiest way to distinguish pilonidal disease from other disease entitles. Differential diagnosis of these sinuses includes anal fistula, hidradenitis suppurativa, perirectal abscess, syphilis, actinomycosis, pyoderma gangrenosum, congenital abnormalities of presacral sinus or dimple and implantation dermoid.

Bacterial colonization of pilonidal sinuses has historically ranged from 50 to 70%, typical isolates including *Staphylococcus aureus* and anaerobes such as *Bacteroides*. Considerable bacterial colonization is also recorded in skin swab samples of the patients.

The bacteria found in infected pilonidal sinuses are polymicrobial in nature and there is predominance of anaerobic bacteria, which outnumbers aerobes in a ratio of 2-4:1. Gram-negative aerobic and facultative bacilli, especially *Escherichia coli*, *Proteus* sp, and *Pseudomonas* sp, are isolated in many instances, while *Staphylococcus* sp were only occasionally recovered. The recovery of Gram-negative bacilli is not surprising as these organisms are part of the normal gastrointestinal flora.[7]

### Tubercular Affection of the Pilonidal Sinus

Tuberculosis is a broad-spectrum disease that may involve pulmonary and extrapulmonary locations. Tuberculosis (TB) is a major public health problem, affecting 8 million persons per year worldwide[8]. The global incidence rate of TB per capita is growing by ≈1.1% per year. Contrary to the increasing number of TB cases in developing countries, the number of cases in industrialized countries is stable or decreasing. Nevertheless, a decreasing trend of the total number of TB patients is seen with an increasing proportion of TB cases with extrapulmonary TB. Both the HIV epidemic and changes in population demographics, with rising numbers of immigrants, are being held responsible for this proportional in-

crease of extrapulmonary TB. Extrapulmonary tuberculosis is responsible for 15% of all cases of tuberculosis[9]. In many countries; patients from Asian origin are known to have a higher incidence of extrapulmonary TB.

*Mycobacterium* (M) may spread through lymphatic or hematogenous dissemination to any tract or through coughing and swallowing to the gastrointestinal tract. Bacteria may remain dormant for years at a particular site before causing disease. Since extrapulmonary TB can affect virtually all organs, it has a wide variety of clinical manifestations, which causes difficulty and delay in diagnosis. Tuberculous infections have been increasing in incidence during the last decades for a variety of reasons, including increasing numbers of patients with immunity-depressive diseases, drug resistance, aging population, and health care worker exposure[10].

Perianal tuberculosis is rare, but the disease is now endemic throughout the world. As the rate of patients with extra pulmonary tuberculosis has increased globally in the last few years, the anoperianal localization is also increasing in similar proportion. Tuberculosis should be suspected in patients with complex or recurrent perianal septic lesions. The most frequently encountered anorectal tuberculous lesions are suppurations and fistulae.[11] Tuberculosis is a neglected cause of anal sepsis, not often recognized and, therefore, does not get the desired treatment. Tuberculous cutaneous infection may result from direct inoculation or hematogenous dissemination from a primary focus such as the lung.

It is difficult to explain the possible cause of tubercular affection in the pilonidal sinuses. Cutaneous tubercular abscess can occur from extension of an embolism to subcutaneous tissue (such as pulmonary foci or direct skin inoculation) or from extension of an underlying lymphadenitis, synovitis, or osteomyelitis[12]. TB has also been described following subcutaneous or intramuscular injection. Either the syringe, needle or fluid to be injected has been contaminated or the medical attendant has exhaled tubercle bacilli into the patient's skin, which are then introduced by the injection. It may be due to a direct inoculation from the stool of the patient, which may be containing tubercular bacilli[13]. Another possibility is that the pre-existing sinuses get infected with tubercle bacilli either by way of finger or by the use of toilet paper[14]. As tuberculosis in the pilonidal sinus is rarely diagnosed before operation on the basis of the clinical picture, the histological examination of the tract of the sinus is mandatory for the correct diagnosis[15].

There should be a strong clinical suspicion of tuberculosis in endemic areas with such presentations as *Mycobacterium* is one of the causes of granulomatous diseases of the skin and subcutaneous tissues. Patients with such presentations are treated several times in the past by the family physicians considering it as boils or abscess. On occasions it is squeezed and drained and at other times it may be treated with antibiotics. The treatment often results in arresting the symptoms for the time being, but would recur after few weeks with the similar symptoms and presentations[16].

## Diagnosis

Diagnosing tuberculosis in a pilonidal sinus can be difficult, so a high index of suspicion remains important. In immigrants from countries with highly endemic TB, a medical history, physical examination, basic laboratory tests, and chest radiograph can lead to a diagnosis. Tuberculosis should be kept in mind for all patients with prolonged or repeatedly recurrent discharging sinuses. The discharge is usually thin and watery contrary to thick and yellowish from a sinus with pyogenic infection[17].

Perianal cutaneous suppuration in tropical countries has multiple causes: bacterial, viral, and parasitic. Infections, such as amoebiasis and actinomycosis have to be explored in priority. Emphasis should be put on the diversity of clinical presentations including acute parasacral abscess, chronic ulcer, and fistula in ano. Clinical presentation of tubercular pilonidal sinus is atypical. This entity may not even be considered in the initial list of differential diagnosis. Because of nonspecific symptoms and non-characteristic radiological and endoscopic features, the diagnosis of tuberculosis rests mainly on histological evidence of the classical tubercle in a surgical biopsy specimen[18]. Crohn's disease and tuberculosis pose major diagnostic problems for clinicians where these conditions coexist.

Definitive diagnosis of tuberculosis involves demonstration of *M. tuberculosis* by microbiological, cytopathological or histopathological methods. Histological examination of the biopsy usually shows epithelioid granulomas, Langhans' type multinucleated giant cells, caseous necrosis, and acid-fast bacilli[19]. Even when adequate tissue is procured, the pathological findings may be suggestiveof "granulomatous infection" which

encompasses a wide range of differential diagnoses rather than "definitive tuberculosis". Therefore, one has to rely upon the clinical impression, radiological and non-conventional diagnostic methods[20].

Sinus tract biopsy and culture and sensitivity of the discharge should both be performed in such patients. Recurrent abscess drainage procedures are likely to be required in these patients and sepsis may persist after anti-TB therapy.

A number of non-conventional diagnostic methods are often resorted to for diagnosing tuberculosis[21]. These test results are relied upon as 'positive evidence' to initiate or withhold antituberculosis treatment. Enzyme linked immunosorbent assay (ELISA) for detecting mycobacterial antigens, antibodies and immunecomplexes in the blood and pus have been used in the diagnosis of tuberculosis. Similarly, detection of antimycobacterial antibodies to A60 antigen in serum or the polymerase chain reaction (PCR) to detect various sequences representing the DNA of *M. tuberculosis* have been described[22]. It should however, be remembered that a positive non-conventional test may perhaps "rule in" a diagnosis, but certainly a negative test cannot "rule out" a diagnosis of tuberculosis. The sensitivity and specificity of these tests ranges between 50 and 85%. Thus, the PCR alone must not be the sole evidence on which antituberculosis treatment is initiated or withheld[23].

A high index of suspicion of tuberculosis should be borne in mind in cases of pilonidal sinus disease with vague etiology or with diagnostic problems, which should be confirmed by histological and bacteriological analysis and treated specifically. An association with pulmonary tuberculosis should be searched for, as the coexistence could be as high as 75%[24]. With the advent of computed tomography (CT) scan and the magnetic resonance imaging (MRI), tremendous progress has been achieved in precise anatomical localization of tubercular lesions.

### Treatment

Treatment of tubercular pilonidal sinus disease included two parts: conventional surgical treatment of sinuses and specific medical antituberculosis treatment[25].

Antituberculosis treatment is the mainstay in the management of tubercular sinuses. However, the ideal regimen and duration of treatment have not yet been resolved. Since 1982, the American Thoracic Society and the Centers for Disease Control have recommended a nine-month course of isoniazid and rifampicin for the routine treatment of TB in the United States[26]. However, a shorter course of four or six months of chemotherapy has been recommended for the treatment of tuberculosis.

The treatment of choice is chemotherapy using three to four anti-TB drugs for up to six to nine months. Isoniazid, rifampicin, and pyrazinamide, with or without ethambutol, are normally used initially for six to 12 weeks. After the six to 12-week course, isoniazid and rifampin are used for an additional three to six months[27]. It should be remembered that many reports of resistance to rifampicin, isoniazid, and streptomycin are coming up[28].

While the six months treatment may be sufficient for many patients, each patient has to be individually assessed and, where relevant, treatment duration may have to be extended for a given patient. An extension of the anti-mycobacterium treatment course to 9-18 months may be required for patients with complicated disease presentations or with co-existing disease elsewhere[29]. Patients receiving antituberculosis treatment should be carefully monitored for adverse drug reactions, especially drug-induced hepatotoxicity.

### Antiretroviral Drugs

In co-existent HIV infection, the CD4+ and CD8+ T lymphocyte counts must be estimated and highly active antiretroviral treatment (HAART) must be administered when indicated. The CD4 counts are usually, but not always, less than 200 in such patients, and antiretroviral therapy is indicated, usually after tuberculosis therapy is completed[30].

Patients with tuberculosis especially those who are co-infected with HIV may develop paradoxical reactions while on antitubercular treatment. The paradoxical worsening and the immune reconstitution syndrome when HAART treatment is started must be distinguished from poor response due to treatment failure, drug resistance or due to an alternate diagnosis. When rifampicin is co-administered along with antiretroviral drugs, by inducing the hepatic P450 pathway, rifampicin may result in dangerously low levels of the antiretroviral agents[31]. In this situation, the available therapeutic options include deferring HAART until standard antituberculosis treatment is completed; or, discontinuing HAART and treating with a standard short-course regimen; deferring or discontinuing

HAART during the initial two month intensive phase when rifampicin is used; using a nonrifampicin containing regimen for the maintenance phase and using HAART among others.

### Surgical Treatment of Pilonidal Sinuses

The therapy to pilonidal sinus disease should be simple to require minimal wound care, and allow rapid return to normal activity. No treatment meets all these ideal goals. Therefore, starting with a simple surgical approach and progressing to other options if failure occurs despite meticulous wound care and hair shaving is the logical approach[32].

The infected cavity should be vigorously curetted, debriding the walls of embedded hairs, and the surrounding skin should be meticulously depilated at the time of operation and over subsequent weeks. Trimming, shaving, and plucking are typically recommended for hair removal[33].

## Conclusions

High index of clinical suspicion, timely judicious use of invasive diagnostic methods and confirmation of the diagnosis, suitable surgical intervention, early institution of specific antituberculosis treatment and close clinical monitoring for adverse drug reactions are the key to the successful management of tubercular pilonidal sinus disease. Increased awareness among physicians about the changing clinical picture and upto date knowledge about diagnosis of TB is warranted. Similarly, patients having perianal sepsis and a concurrent tubercular lesion elsewhere should be suspected to have a tubercular origin to avoid undesirable delays in the diagnosis and treatment of this disease.

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