Recent Trends in Management of Pilonidal Sinus Disease

Essay

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List of abbreviations

Abbreviations	Terms
PS	Pilonidal Sinus
PSD	Pilonidal Sinus Disease
MRI	Magnetic Resonance Imaging
HS	Hidradenitis Suppurativa
HIV	Human Immunodeficiency Virus
HPV	Human Papilloma Virus
SGA	Superior Gluteal Artery
S-GAP	Superior Gluteal Artery Perforator
PSIS	Posterior Superior Iliac Spine
GT	Greater Trochanter
PM	Piriformis Muscle
CX	Coccyx
LAFTF	Lumber Adipo-Fascial Turnover Flap

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Introduction And Aim of The Work

Introduction

“Pilonidal disease” refers to a subcutaneous infection occurring in the upper half of the gluteal cleft. It typically presents in the second decade of life, but also occurs in teenagers and in patients in their thirties.

It affects men more often than women and is more common in individuals with more body hair **(Silva, 2000).** Incidence varies between different countries and races. In an American population study, 1.1 percent of young male students and 0.11 percent of female students suffered from the condition, whereas in Mediterranean countries a higher incidence (9–30 percent) has been reported in young men **(Abdelrazeq et al., 2008).**

The term pilonidal is usually referred to as jeep disease, a well-documented phenomenon of hirsute jeep drivers during World War II **(Kafali et al., 2008).**

The first reported case of pilonidal disease was published in 1833 by Mayo, but it was Anderson who first described his management of the disease in an 1847 article entitled "Hair Extracted from an Ulcer." In 1880, Hodges coined the term pilonidal disease, which is in use today. The term literally means "nest of hair” **(Solla et al., 1990).** It is most common in the gluteal cleft but can occur in bizarre places like web spaces of fingers (barbers) and toes, umbilicus, perineum, axilla, amputation stumps and the clitoris **(Rao et al., 2006).**

Risk factors for Pilonidal disease are white race, male sex, family predisposition, increased sweating, activity associated with sitting and

buttock friction, sedentary lifestyle, poor personal hygiene, obesity, and local trauma **(Jukic et al., 2006).**

It was thought to be of congenital origin, congenital theories suggest that pilonidal sinuses are vestigial structures, cystic remnants of the medullary canal or the result of dermal inclusions caused by faulty development of the median coccygeal raphe or as a result of congenital abnormal inward growth of hair, but now most experts believe it to be mainly the result of acquired conditions **(Fazeli et al., 2006).** Pilonidal sinus is thought to be originated by healthy hair penetrating the skin using preformed sinus or creating new ones. Consecutive foreign body reaction, partial epithelization of tracts, and chronic infection are the tissue responses underlying chronic fistulating or abscess-forming disease **(Doll et al., 2008).**

The lesion is asymptomatic until it becomes infected. In 20 percent of cases, the disease is observed as an acute abscess, whereas in the remaining cases it presents as a chronic sinus, in which there are discharging orifices **(Fazeli et al., 2006).** Common complications of pilonidal disease are infection, abscess formation, and recurrent sinus disease after surgery. Less common complications include sacral osteomyelits and meningitis. Malignant transformation is a rare but well-known complication of pilonidal disease and occurs in approximately 0.1% of patients with recurrent pilonidal disease and most commonly in men **(Mentes et al., 2008).**

Management of this disease remains controversial, and "gold standard" surgical treatments have not yet been described. A number of

operative and nonoperative techniques are currently used for the management of pilonidal disease **(Rao, 2006).**The standard surgical procedures are associated with a significant risk, both of delayed healing and of recurrent disease. When the disease is confined to the midline one option is to excise the affected area and close it in an attempt to achieve primary healing. When this fails a large wound then has to granulate. Even if primary healing is successful, the patient remains at great risk of recurrent disease as there's now a scar deep within the natal cleft throw which hairs may penetrate with greater ease than throw undamaged skin. The alternative of leaving the wound open for secondary healing, the disadvantage though are of low healing and it will be at least six weeks before the wound has filled and epithelised. All heal with a wide midline scar which vulnerable to recurrent disease, and despite meticulous postoperative attention the occasional patient develops a persistently unhealed midline wound. Recent advances in treatment have focused on the avoidance of a midline scar **(Farquharson and Moran, 2005).**

Aim of the work

Since the concepts about the pilonidal sinus disease and surgical management have changed markedly during the last decades, the aim of this work is to put special concern on these novel procedures highlighting to which degree they may be promising in decreasing complications and lowering recurrence rates of the disease.

Surgical Anatomy

Surgical Anatomy

The natal cleft is maintained as the thin midline skin is attached to the underlying ligamentous and aponeurotic fibers on the dorsum of sacrum and coccyx by a dense well defined collagenous fascia. Caudally, this fascia is continuous with a dense mass at the tip of the coccyx where the natal cleft enters the anal zone. Laterally, each layer of the natal cleft fascia is continuous with the superficial fascia of the buttocks and its fibrous septa **(Carol and Conner, 2003).** This buttock fascia is the most dense and least resilient in the body, and the fibrous septa invades the fat attached deeply to the gluteal aponeurosis covering the muscles. Proximally, the fascia widens at the level of the posterior superior iliac spines to join the superficial fascia of the flanks, the right layer hugged the midline while the left deviated at a lower level toward the posterior spine. Some surgeons believe that spread of pilonidal sinus is governed by the local arrangement of this fascia of the natal cleft **(Davis and Starr, 1945).**

Blood supply of the lumbosacral area

The arterial supply of the lumbosacral area comes from the dorsal branches of the lumbar arteries, the dorsal branch of the median sacral artery, the dorsal branches of the lateral sacral arteries, and the superior and inferior gluteal arteries. These arteries make a rich anastomotic network in the lumbosacral area and supply blood to the thoracolumbar (lumbar) fascia, which lies under the subcutaneous fat tissue and covers the deep muscles of the back and trunk **(Turan et al., 2007).**

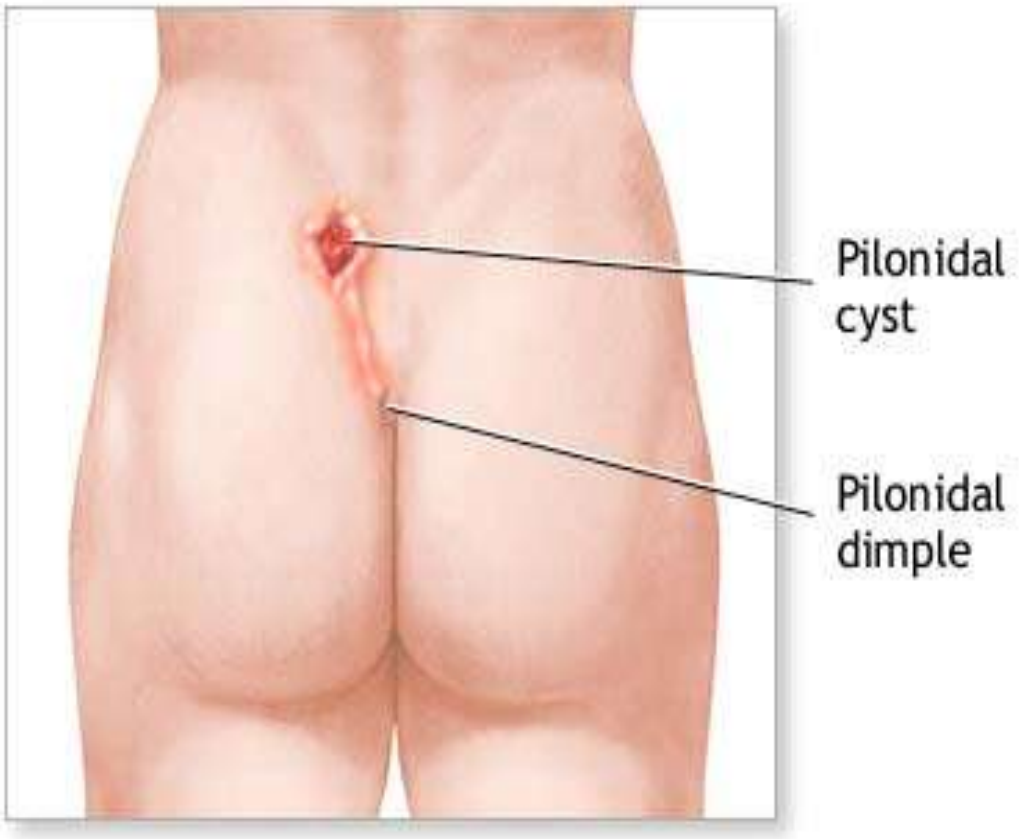

Fig. (1): Pilonidal cyst and dimple.