INTRODUCTION

Sacrococcygeal pilonidal disease is a very common anorectal problem that commonly arises in the hair follicles of the natal cleft of the sacrococcygeal area ***(Khanna, A., & Rombeau, J. L. 2011).***

Sacrococcygeal pilonidal sinus disease dates back to 1833 when Mayo, British physiologist, anatomist and surgeon described it in a female patient as a sinus with hair follicles inside in the sacrococcygeal region ***(Wysocki, A.P. 2015*).**

In 1880, ***Hodges*** named the disease `pilo-nidal` (pilus means hair and nidus, that means nest).” He produced the word “pilonidal” by conjoining the word “pilus” and the word “nidus” ***(De Parades, V., et al. , 2013).***

Regarding the pathophysiology of the disease, it has been commonly thought to be embryonic or acquired. Absence of the exact aetiology explains the diversity of treatment lines and failure of treatment options. ***(Lee, PJ1, et al. 2008).***

Clinical diagnosis is straight forward varying from acute pilonidal abscess, chronic pilonidal sinus, complicated pilonidal sinus and recurrent pilonidal disease accordingly, different treatments have been introduced including non-operative management, excisional and incisional procedures and flaps ***(Topgül, K. 2010).***

Radical treatment has been wide excision of the chronic tracts with or without closure. The main problem is the high rate of recurrence, which can diminish the patient's quality of life ***(Spivak, H., et al. 1996).***

The surgical wound may be left to heal by secondary intention. Advocates of this technique state that eliminated wound tension facilitates trouble-free healing without recurrence if all sinus tracts are fully excised ***(Yoldas, T., et al. 2013).***

Alternatively, the wound may be closed to heal by primary intention. Advocates of primary closure perceive benefits of faster tissue healing . ***(Spivak, H., et al. 1996).***

Methods of primary closure can be broadly categorized as midline closure techniques with the wound lying within the natal cleft or other techniques where the wounds placed out with the midline ***(Singh, H., et al. 2017).***

Skin flaps have been described to cover a sacral defect afterwide excision; this keeps the scar off the midline and flattens the natal cleft. The techniques available include the cleft closure, Karydakis procedure, local advancement flap (V-Y advancement flap), transpositional Limberg flap ***(Spivak, H., et al. 1996).***

Hence, the ideal operation should minimize PSD recurrences and financial cost (patient–community),with short hospital stay, cause minimal pain, associated with a low patient and procedure morbidity rates, and be simple for surgeons. ***(Kanat, B. H., & Sözen, S. 2015).***

Aim of the Work

The aim of this work is to compare between excision with primary closure versus Limberg flap for reconstruction of sacrococcygeal pilonidal disease as regard operative time, postoperative pain, hospital stay, duration of incapacity for work, postoperative complications (infection, flap edema, wound dehiscence), patient satisfaction, and postoperative recurrence.

Chapter One

ANATOMY OF NATAL CLEFT

The prominent and important structures in this area are all musculoskeletal. The bony sacrum forms the posterior part of the bony pelvic ring and is the distal continuation of the vertebral column Figure (1). It is formed by the fusion of the five sacral vertebrae. The posterior surface is roughened and has two paramedian crests "the lateral sacral and intermediate crest" which, with the prominent midline median sacral crest, form points of attachment for fascial and aponeurotic structures. ***(Scott-Conner, C. E. 2009).***

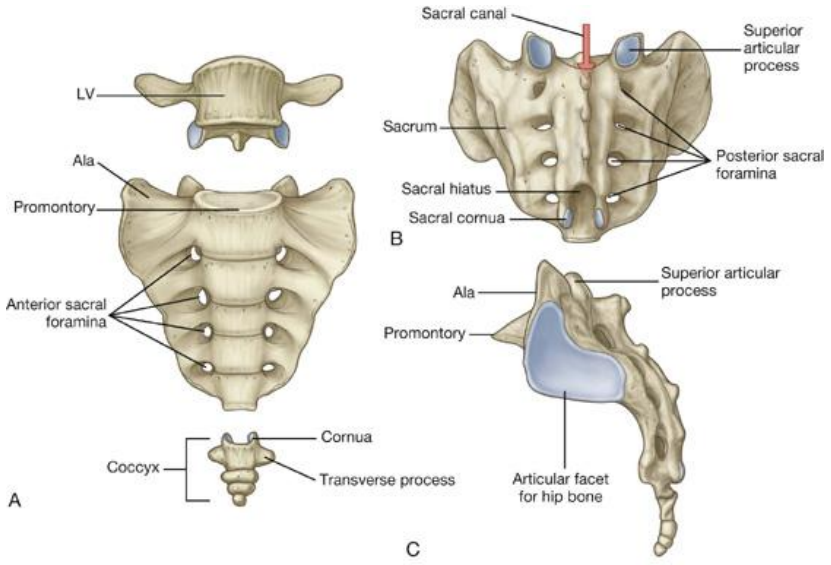

Figure (1): Anatomy of sacrum and coccyx ***(Drake, R.,et al .2014).***

The coccyx is composed of three to five remaining vertebrae (commonly, four). These small, nubbin-like, rudimentary vertebrae articulate with the sacrum. Figure (1).

The gluteus maximus originates primarily from the sacrum, along a roughly diagonal line extending from the tip of the coccyx to the iliac crest.The gluteus medius lies lateral, deep to the gluteal aponeurosis and gluteus maximus. The gluteus minimus, which lies deep to the gluteus medius, has similar functions. Figure (2). ***(Scott-Conner, C. E. 2009).***

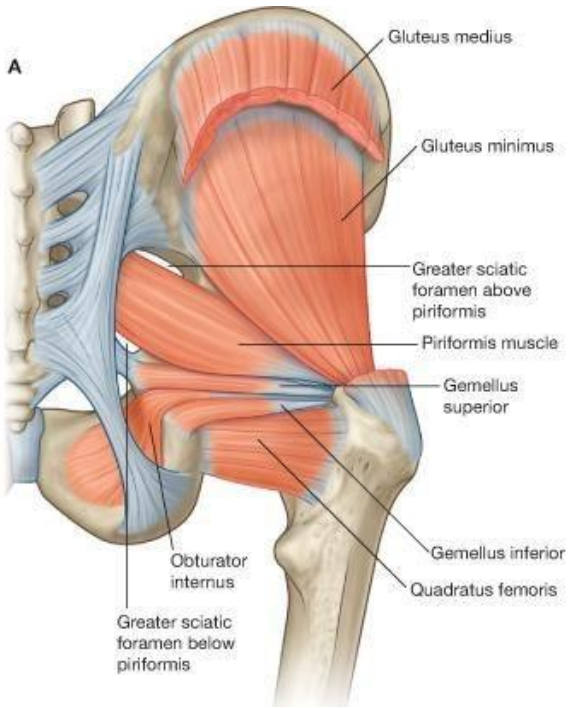

Figure (2): The gluteal muscles***(Drake, R.,et al .2014).***

The midline intergluteal cleft is formed by the infolding of skin and fatty tissue enveloping the gluteal muscles. It extends

from the midsacral level to the anus, blending imperceptibly with the perineum in the region of the anus ***(Scott-Conner, C. E. 2009).***

The skin of this region is thick (although thinner than the skin of the back or buttocks) and is covered with a variable amount of hair. Particularly in hirsute individuals, an increased amount of hair may be present in the intergluteal cleft normally, which may account for the formation of pilonidal cysts in this area ***(Scott-Conner, C. E. 2009).***

Sensory innervation in the region of the intergluteal cleft is derived from branches of the sacral and coccygeal nerves Figure (3). The skin overlying the lower and lateral portions of the gluteal muscles is innervated by gluteal branches of the posterior femoral cutaneous nerve ***(Scott-Conner, C. E. 2009).***

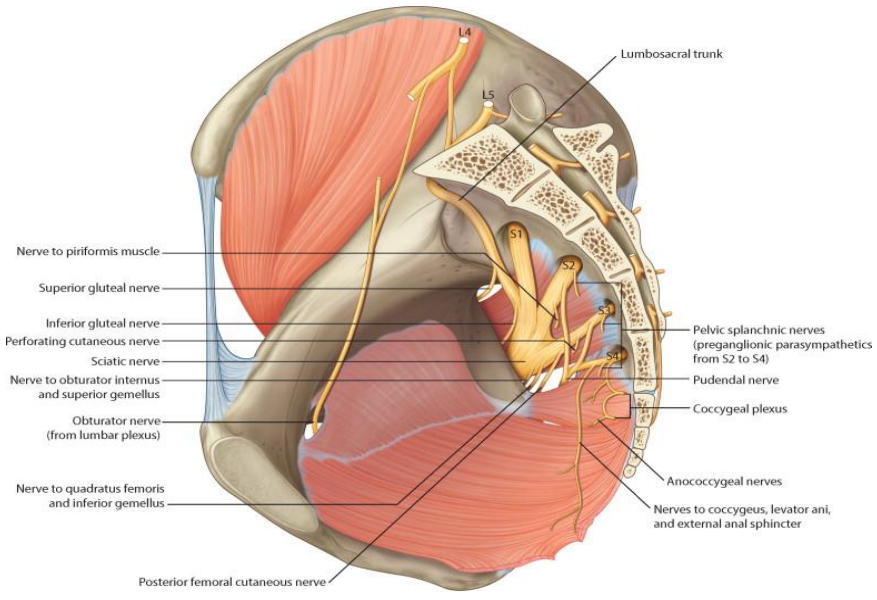

Figure (3): Sensory innervation***(Drake, R.,et al .2014).***

The natal cleft posses a poor blood supply derived mainly from small medial twigs of posterior penetrating arteries from sacral foramina. Secondary tracts of the sinus may enter the buttock along these twigs. They run upwards on either side from coccygeal branches of the inferior gluteal artery, superior and inferior branches of lateral sacral artery ***(Onishi and Maruyama, 2010).***

Although the network of superficial veins exists here, as elsewhere in the body, it is rare to encounter even small veins in the midline. Hence, dissection can proceed swiftly and is attended with a little risk ***(Scott-Conner, C. E. 2009).***

Chapter Two

EPIDEMIOLOGY & PATHOPHYSIOLOGY OF SACROCOCCYGEAL PILONIDAL DISEASE

Peak incidence is between the ages of 15 and 24 years. Risk begins to decreases after age 25 years; rarely occurs over age of 45 years ***(Hull,T. L., & Wu,J. 2002).*** Males are affected more than females (3:1 ratio). Believed to be due to more hirsute nature ***(Bendewald, F. P., & Cima, R. R. 2007).***

Family history of pilonidal disease is present in 38% of cases**.** The most common predisposing factors are: hairy body, excessive weight gain (BMI > 25 kg/m^2), thick skin, deep gluteal cleft, neglecance of hygiene,previous family history and spending several hours per day taking the seated position ***(Doll, D. 2014).*** Hyperhidrosis can be associated with pilonidal disease ***(Onder, A., et al. 2012).***

The most common site for PNS is the upper natal cleft. However; it may appear in other sites with irregular skin surface and pressure applied to that region. It might be seen in the finger webs especially in barbers and hairdressers due to dealing with hair. Also, PNS was noticed in many other areas like the foot interdigital spaces, pulps of the fingers, the periungual area, umbilicus, intermammary area,clitoris, penis,

and the perineal regions ***(Abdulwahab, B. A., & Harste, K. (2010).***

PNS has high incidence among certain communities, which can be explained by the different hair distribution and its growth pattern. The condition is more common in Caucasians than in Asians and Africans ***(Baker, T. ,et al. 2008).***

Aetiology of pilonidal disease

There are two main theories that have been established to explain the occurrence of sacrococcygeal pilonidal sinus disease and yet none is accepted or supported. These two main theories, congenital and acquired theories, are surrounded by much controversies ***(Duman, K., et al. 2017).***

a) Congenital theory:

According to the congenital theory, pilonidal sinus disease occurs when there is still residual epithelium in the spinal canal and skin or when hair follicles enter to interspace formed by imperfect fusion occurring as a result of a defective union of the skin layer during the early embryonic period ***(Ackerman, L. L., & Menezes, A. H. 2003).***

This theory has never been proven and failed to explain pilonidal sinus diseases located in the regions away from the sacrococcygeal area . In addition, successive clustering of hairs with the same length, thickness, color, and position could not

be explained by this congenital theory ***(Bascom, J., & Bascom, T. (2007).***

b) Acquired theory:

Pilonidal sinus disease is now considered as a widely accepted disease to be an acquired phenomenon. Several lines of evidence that suggest an acquired nature of pilonidal disease include the following: This disease does not present at birth but occurs in young adults; and to explain this acquired theory, pilonidal disease occurs as a result of inflammatory process resulting from foreign-body reaction occurring secondary to the entry of hairs in the subdermal area after trauma ***(Harlak, A., et al .2010).***

Pathogenesis of pilonidal disease:

Pilonidal sinus disease is more frequent in men of hirsute complexion; and some researchers documented that hair follicles become infected under the effect of sex steroids when they are filled with keratin during puberty; as a result of this infection an inflamed tract is formed. Hairs by getting their way through the tract pierce the skin while they are still attached to the skin and are pulled into subcutaneous tissues under the vacuuming effect of gluteal muscles as a result of friction to form a sinus***(Harlak, A., et al .2010).***

Karydakis GE suggests that hairs move towards the follicle and enter into deeper regions like a drill. This theory is validated by the fact that the disease occurs in interdigital areas, the umbilicus, clitoris, and anal canal, and it recurs even after radical excisions ***(Karydakis, G. E. 1992).***

Certain occupations that involve prolonged sitting, or professional driving, predispose people to develop pilonidal diseas***)*** as the vacuuming effect is mostly observed in the sitting position ***(Majeski, J., & Stroud, J. 2011).***

A foreign-body reaction develops, which is accompanied by infection and the subsequent development of the primary pilonidal sinus. Interesting research studies demonstrated that a deep natal cleft is the location of the main problem for establishing the sinus formation where the epidermis is exposed to moisture, anaerobic conditions, hair, debris, and bacteria. Hair follicles in the natal cleft become distended with keratin, which is prone to infection ***(Khanna, A., & Rombeau, J. L. 2011 ; Sarhan, A. E., et al. 2016).***

Infected material in the follicle is then pulled and pushed into deeper subcutaneous fat to create the acute abscess of pilonidal disease. A chronic abscess often persists and matures after drainage of the acute pilonidal abscess ***(Bascom, J.1980).***

The external openings described as midline pits are usually located 4–5 cm above the anus in the midline and this

midline pit extends into the subcutaneous tissue as a fibrous tract to form the pilonidal sinus and this pilonidal sinus frequently develops a secondary cutaneous opening, located off the midline. This cutaneous fistula, leading from the pilonidal abscess sinus, usually opens 1–4 cm from the midline and in approximately 90% of cases opens cephalad to the midline pits ***(Majeski, J., & Stroud, J. 2011).***

Chapter Three

CLINICAL PRESENTATION

Pilonidal disease may arise in one of four forms:

Quiescent phase (Asymptomatic form), acute abscess formation, sinus tracts formation (Chronic form) or complex disease characterized by chronic or recurrent abscesses with extensive and branching sinus tracts ***(Bendewald, F. P., & Cima, R. R. 2007).***

During quiescent phase -

Pilonidal sinus is characterized by the existence of a midline pit figure (4) in the natal cleft identified4 to 8 cm from the anus in typical cases. The skin enters the sinus giving a smooth edge to the opening.

Figure (4): Single pit congenital pilonidal sinus
(De Parades, V., et al. 2013).

There is connection between this primary tract and a subcutaneous cavity, which contains granulation tissue and a nest of hairs figure (5) that is present in two thirds of cases in men and in one third of those in women and may be seen projecting from the skin opening. In many patients, there is secondary lateral openings 2 to 5 cm above the midline pit. Squamous cell epithelium line the skin opening and the superficial portion of the tract, but not the deep cavity and its extensions ***(*Varnalidis, I., *et al. 2014).***

Figure (5): Hair debris ***(De Parades, V., et al. 2013).***

In asymptomatic type, figure (6) patients have no complaints and are diagnosed incidentally. There ratio was found to be 11%among all patients with pilonidal sinus ***(el-Khadrawy, O., et al. 2009).***

Figure (6): Pilonidal pit ***(De Parades, V., et al. 2013).***

Acute abscess formation in pilonidal sinus tract(s) figure (7)

The common form of pilonidal disease is an acute abscess along with sinus tract formation. This position distinguishes the disease from other common anorectal problems, Such as perirectal abscesses and anal fistulae, which are typically found near the anus ***(Bendewald, F. P., & Cima, R. R. 2007).***

Symptoms related to tense pointing abscess are mainly pain, but fever is rare. It is very common to find bleeding or