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Etiology of Pilonidal Sinus - The Bottom Line

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1. Abstract

Pilonidal sinus disease (PSD) is common among young adults and occurs more often in men than in women (3:1). Pilonidal derives from pilus ("hair") and nidus ("nest") and literally means nest of hair. In practice, PSD is mostly defined as the presence of one or more pits or sinus cavities in the sacrococcygeal area that often contain hair and are prone to infection and abscess formation. The etiology of PSD has been discussed for more than two centuries. Our interest in the etiology of PSD was sparked after two patients visited our outpatient clinic with surprising findings in their pilonidal cavities. These findings motivated us to review the literature to better understand the etiology of this disease. Ten articles contained original research on the etiology of PSD in the IGF and were finally included for this review.

2. Introduction

Pilonidal sinus disease (PSD) is common among young adults and occurs more often in men than in women (3:1). Pilonidal derives from pilus ("hair") and nidus ("nest") and literally means nest of hair. In practice, PSD is mostly defined as the presence of one or more pits or sinus cavities in the sacrococcygeal area that often contain hair and are prone to infection and abscess formation.

The etiology of PSD has been discussed for more than two centuries. For many years, PSD was thought to be of congenital origin. In 1946, Patey and Scarffs were the first to think that PSD is an acquired entity. They firstly recognized that sacrococcygeal PSD is most common in young adults during puberty when they experience hair growth. Secondly, they identified the presence of umbilical PSD and interdigital PSD in barbers and dog groomers [1]. In the following years two main acquired theories were considered: the follicle theory of Bascom that postulates that midline pits are a result of follicle occlusion, inflammation and rupture, where after hairs are able to intrude into enlarged pits [2]; and the penetrating theory of Karydakis and Stelzner suggesting that PSD is caused by intruding hair and debris respectively due to hair-, force- and and/or vulnerability related factors [3-4].

Our interest in the etiology of PSD was sparked after two patients visited our outpatient clinic with surprising findings in their pilonidal cavities. The first patient was a 40-year-old man with recurrent PSD. Physical examination showed three midline pits and one sinus opening cranially. White hair fragments were found in the pits as well as on his clothes, but clearly did not originate from his body or head showing dark hair. Further questioning revealed the white hair belonged to his dog, who also happened to be his hairy bedpartner. The second patient was a 23-year-old woman without hair in her dorsal crest and intergluteal fold (IGF). One long hair fragment of approximately 30cm was found in her sinus cavity and was identical to her occipital hair, suggesting that the sinus hair originated from her scalp and got trapped in her IGF. These findings motivated us to review the literature to better understand the etiology of this disease. We performed a systematic PubMed search on November 23th, 2020. Articles written in English and describing original research on the etiology of PSD in the IGF were included. Studies written in other languages, describing extracoccygeale or congenital PSD or without available fulltext were excluded. The following search resulted in 153 stud-

ies: ((morpholog*[tiab]) OR (etiology[tiab]) OR (cause[tiab]) OR (pathogenesis[tiab]) OR (pathophysiology[tiab])) AND ((pilonidal sinus[tiab]) OR (pilonidal disease[tiab]) OR (pilonidal cyst*[tiab])). Twenty-five were read in full text based on title and abstract. Cross-checking references provided fourteen extra studies. Ten articles contained original research on the etiology of PSD in the IGF and were finally included for this review.

The first original studies favoring the theory of an acquired origin were published in 1954 and 1962 [5;6]. Davage re-examined the microscopic sections of 463 PSD cases. Epithelial lining of variable degrees was found in 51% of patients. Hair shafts were found in 72% of patients, either lying loose in granulation tissue or deep in scar tissue. No hair follicles were found in the walls of these sinuses [5]. Frankowiak and Jackman examined specimens of 354 PSD patients. Inflammation was found in all but three of their cases and was reported as chronic in 72% due to increased amounts of scar tissue, lymphocytes and plasma cells. Epithelial lining was found in 28%; in 9% of patients the entire sinus was lined with epithelium. Hair was found in 27% of cases and described as fragments of broken hair shaft without root structures [6]. Following studies aimed to further examine the etiology of acquired PSD. In 1970, Millar (n=112) demonstrated the sinus tracking in the same direction as the direction of hair follicles, suggesting that the primary pit originates from a follicle rather than penetration of intact skin by hair. Lateral traction will then open the follicle and enable foreign material to enter. Negative pressure then creates suction effect in to the pit [7]. This follicle theory is also believed by von Laffert et al. (n=27) who found follicle hyperkeratosis, hyperplasia of the follicular epithelium, perifolliculitis and follicle ruptures in 92%, 80%, 64% and 40% of cases respectively. Dislocated hair shafts were seen in 74%. The authors conclude that the start of PSD seems to take place at terminal hair follicles due to keratin plugs and debris causing inflammation, after which sinus tract formations follow as secondary events [8]. Similar results were found by Wortsman and colleagues (n=43) who performed sonographic examinations. All pilonidal sinus communicated with widened hair follicles and contained retained hair tracts [9]. The authors, however, doubt the hypothesis of an embedding process of multiple hair fragments penetrating the skin through considerably small follicular ostia. They favored the theory of ectopic sites of production of hair and keratin debris within collections and fistulous tracts [9]. One study believes that the follicle theory and penetrating theory come together. Histologic examination of Sondenaa and Pollard (n=53) showed keratin plugs and debris creating pits that were about to penetrate the skin, with and without connection to hair follicles. They concluded that PSD is a chronic inflammatory process of the skin creating pits and sinuses that may be related to follicles [10].

Recent studies of Doll et al. provided evidence favoring the penetrating theory. A morphological analysis (n=10) showed that sinus hair originated from three different body regions in or cranially to the IGF: occiput (n=5), lower back (n=5) and/or IFG (n=4); three patients had one or more morphological allocations [11]. These results imply that sacrococcygeal PSD is common due to the deep IGF and intergluteal hair that may help catch and retain hair from higher up. This also illustrates the importance of avoiding loose hair fragments to reach the IGF. In an additional study, Doll harvested hair from all three body regions in seventeen PSD patients and seventeen matched controls and compared its vertical strength. Hairs from all three body regions were able to sustain a larger vertical force than hairs of matched controls respectively ($p<0.01$). Within the sinus of the seventeen PSD patients, an average of 21 hairs (range 1-415) were found, mostly fragments with sharp cut ends [12].

The latter result highlights another point of debate; whether hair penetrates the skin with its root end or with sharp fragments. Bosche et al. evaluated pilonidal hair using a scanning electron microscopy. Of the 624 hairs found in the pilonidal cavities of 20 PSD patients, 74% was rootless. All hair showed cut ends, none had broken or split ends. Seventy percent of all hairs were ≤1 cm in length; 93% were ≤ 2cm in length. None of the patients reported razor depilation of their back or IGF, suggesting the significance of (cut) occipital hair [13]. In contrast with the 74% rootless hairs in this study, Gosselink et al. illustrated that in fifteen out of seventeen patients, hair was oriented with its root end into the pit. All hairs showed a hook structure, encouraging the hair to be driven deeper into the pit [14]. This finding was also reported by Sondenaa and Pollard, who demonstrated that hair found in the sinus opening (n=7) was oriented with its root end into the sinus [10]. It can be concluded that PSD has an acquired origin in which hair fragments, keratin and debris create pits, sinuses and eventually sinus tracts. Every kind of hair can enter the pit, yet cut hairs are a greater risk factor than broken or split ends, stiff hair more than soft hair, and those entering with the root end might insert faster and deeper due to a scale structure. Debate persists whether the primary pit is caused by penetration in intact skin or entry of hair and debris in dilated follicles. It is not unthinkable that both theories are plausible and the etiology of sacrococcygeal PSD is multifactorial. Our patients have illustrated the remarkable finding that every hair, even from your dog, has potential to reach your bottom line.

3. Statements and Declarations

The authors declare no conflicts of interest.

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