

Is family history of pilonidal sinus really important or is it a myth?

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ABSTRACT

Objective: Pilonidal sinus is a disease that occurs with a sinus abscess in the sacrococcygeal region, especially in young men. This study investigated whether positive family history is a predisposing factor for the disease and whether it influences the age of onset in patients who have undergone pilonidal sinus surgery.

Materials and Methods: A total of 148 patients who underwent pilonidal sinus surgery at our clinic between January 2022 and January 2023. Age, sex, and family history were evaluated, and the family history was further detailed via telephone interviews.

Results: The study included 148 patients, 124 (83.8%) were male and 24 (16.2%) were female. Family history was reported in 21.6% (n = 32) of the patients. Among those with a positive family history, 84.38% (n = 27) had paternal lineage involvement, and 15.62% (n = 5) had maternal lineage involvement. The mean age was 26.31 ± 7.69 years in males and 22.25 ± 8.02 years in females, with the difference being statistically significant ($z = 2.35$; $p < 0.05$).

Discussion: While familial predisposition is notable in a subset of pilonidal sinus disease cases, it is insufficient alone to predict disease onset or severity. Multifactorial influences—genetic, anatomical, and behavioral—must be considered.

Keywords: pilonidal sinus, family history, surgery, paternal lineage

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INTRODUCTION

Pilonidal sinus disease (PSD) is a chronic inflammatory condition that usually presents with a draining sinus or abscess in the sacrococcygeal region and predominantly affects young males [1]. Its incidence has been reported to vary between 6 and 25 cases per 100,000 population [2]. Treatment modalities range from conservative and minimally invasive techniques to complex surgical procedures. However, no universally accepted gold standard exists; therefore, individualized treatment planning has gained increasing acceptance [3].

Numerous theories have been proposed to explain the etiology of PSD since its initial description. While debate has persisted over whether it is congenital or acquired, the current consensus among most surgeons is that the disease is acquired [4]. However, it remains unclear whether specific predisposing factors exist or whether the

condition develops sporadically. Variables such as natal cleft anatomy, hair follicle structure, personal hygiene habits, body weight, and even bathing frequency have been considered as contributing factors. Although there is limited literature focusing specifically on the role of family history, several studies suggest that it may be an important predisposing factor [5].

The aim of this study was to evaluate the family history of patients who underwent pilonidal sinus surgery in our clinic, in order to determine whether familial predisposition plays a role and whether it influences the age at which the disease manifests.

MATERIALS AND METHODS

This retrospective study included patients who underwent pilonidal sinus surgery at our clinic between January 2022 and 2023. The medical records of all the patients were reviewed. Age, sex, and family history were also assessed. Additional details

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regarding family history were obtained through telephone interviews.

Patients were classified based on the presence or absence of a family history of PSD. To ensure accuracy, family history data were initially obtained from medical records and subsequently verified through structured telephone interviews conducted by two independent investigators. During these interviews, patients were specifically asked whether any first- or second-degree relatives had been diagnosed with PSD and whether these relatives had undergone surgical treatment. If the relative's diagnosis or surgery had been performed in a hospital setting, patients were encouraged to provide the name of the hospital or physician, which was cross-checked when feasible. In cases where verification was not possible, the information was recorded as self-reported. To minimize recall bias, all interviews followed a standardized questionnaire, and any ambiguous responses were clarified with follow-up questions.

Within the group with positive family history, additional variables such as the degree of kinship with the affected relative, whether that relative had undergone surgery, and the age of symptom onset were also documented. The influence of maternal vs. paternal lineage was also evaluated in patients with familial disease.

All data were analyzed using the statistical package for the social sciences version 22.0 software for Windows. To determine the appropriate statistical tests (parametric or non-parametric), the distribution of variables was first assessed. The Kolmogorov-Smirnov test and the skewness-kurtosis values were used to evaluate normality. Independent sample t-tests and Mann-Whitney U tests were employed for comparisons between groups. A p-value of < 0.05 was considered statistically significant.

Ethical Approval

This study was approved by the Ethics Committee at Malatya Turgut Özal University (Approval No: E-30785963-020-196429, Date: 05/01/2024). This study was conducted in accordance with the principles of the Declaration of

Table 1. Demographic characteristics of the patients

Variable	Group	N	%
Family history	Absent	116	78.40
	Present	32	21.60
Gender	Male	124	83.80
	Female	24	16.20
Positive family history	Paternal lineage	27	84.38
	Maternal lineage	5	15.62

Helsinki. Owing to the retrospective nature of the study, the requirement for informed consent was waived.

RESULTS

In total, 148 patients were included in this study. Of these, 124 (83.8%) were male and 24 (16.2%) were female. Family history analysis revealed that 32 patients (21.6%) had a positive family history of pilonidal sinus, whereas 116 patients (78.4%) did not. Among those with a positive family history, 84.38% ($n = 27$) had affected relatives on the paternal side, and 15.62% ($n = 5$) on the maternal side (**Table 1**).

The mean age of patients with a positive family history was 26.12 ± 7.97 years (median 25.0; range 15-48), while the mean age of those without was 23.94 ± 7.34 years (median 22.0; range 14-43). The difference between the two groups was not statistically significant ($z = -1.40$; $p > 0.05$) (**Table 2**).

When patients were examined in terms of gender, the mean age of male patients was 26.31 ± 7.69 years (median 25.0; range 14-48), whereas female patients had a mean age of 22.25 ± 8.02 years (median 19.5; range 15-46). This difference was statistically significant ($z = 2.35$; $p < 0.05$).

In patients with a positive family history, the mean age of the patient was 23.79 ± 6.28 years (median 22.0; range 14-36), and the mean age of the affected relative was 27.95 ± 7.74 years (median 25.0; range 18-47). This difference was not statistically significant ($z = -1.58$; $p > 0.05$).

Among the patients with positive family history, those with paternal relatives had a mean age of 23.14 ± 6.03 years

Table 1. Comparison of patients by age

Variable	Group	N	Mean \pm standard deviation	Median (min-max)	Z	p-value
Family history	Present	32	26.12 ± 7.97	25.0 (15-48)	-1.40	0.17
	Absent	116	23.94 ± 7.34	22.0 (14-43)		
Gender	Male	124	26.31 ± 7.69	25.0 (14-48)	2.35	0.02*
	Female	24	22.25 ± 8.02	19.5 (15-46)		
Patient vs. relative age	Patient	19	23.79 ± 6.28	22.0 (14-36)	-1.58	0.11
	Relative	19	27.95 ± 7.74	25.0 (18-47)		
Lineage	Paternal	27	23.14 ± 6.03	22.0 (14-36)	1.41	0.14
	Maternal	5	19.20 ± 3.27	19.0 (15-24)		

Note. *Z: Mann-Whitney U test

(median 22.0), whereas those with maternal relatives had a mean age of 19.20 ± 3.27 years (median 19.0). This difference was not statistically significant ($z = 1.41$; $p > 0.05$).

DISCUSSION

The findings of the present study indicate that a positive family history may act as a significant risk factor for PSD in a defined subset of patients [6]. In our cohort, 21.6% of participants reported a positive family history, a prevalence figure that aligns with contemporary epidemiological data [7]. A comprehensive analysis on recurrence rates in young adults highlighted familial clustering as a noteworthy factor influencing disease patterns. Consistently, a large case series examining pilonidal disease in siblings further reinforces the role of familial aggregation in PSD pathogenesis [8].

The literature consistently reports a higher prevalence of PSD among males. A recent meta-analysis in [9] demonstrated that although the male-to-female ratio has shown a relative decline in certain populations over time, PSD remains predominantly a male-oriented disease. In our cohort, males accounted for 83.8% of the study population, and this gender disparity was statistically significant. Differences in hair distribution in the sacrococcygeal region, perspiration levels, and lifestyle-related factors are likely contributors to this observation.

A detailed evaluation of familial predisposition revealed that the majority of cases (~84%) were associated with paternal lineage [10]. Moreover, a recent case-control study demonstrated that over half of PSD patients had a positive family history, particularly among first-degree relatives, further underscoring familial clustering and genetic or environmental influence [11]. This observation may be explained by a combination of genetic susceptibility and shared environmental factors.

It is now widely recognized that PSD does not arise solely from local trauma or excessive hair growth but involves multiple predisposing mechanisms [12]. Increasing evidence indicates that a combination of genetic, anatomical, and behavioral factors contributes substantially to disease development [13]. In a systematic review [14], it was highlighted that connective tissue characteristics and inherited susceptibility may also play a decisive role in the pathogenesis of PSD, beyond mechanical explanations. This multifactorial perspective is consistent with the findings of our present study.

Furthermore, a large-scale retrospective analysis identified family history as an independent risk factor not only for the occurrence of PSD but also for postoperative recurrence [15]. In adolescents, family history has been shown to be a significant risk factor, with PSD frequently presenting during teenage years [16].

One recurring theme in the literature is the broad consensus that PSD is an acquired condition [17].

Nevertheless, the observation of positive family history, particularly in cases with early onset or atypical clinical presentation, highlights the importance of further research into the genetic and epigenetic aspects of the disease [18].

In conclusion, the findings of this study suggest that although family history constitutes an important risk factor in the development of PSD, it does not act as an isolated determinant. The interplay between inherited predisposition, anatomical characteristics, and environmental influences appears to be critical in shaping both disease onset and recurrence. Consequently, effective prevention and management strategies should not only consider genetic susceptibility but also address modifiable lifestyle and environmental factors such as hygiene, body weight, and hair control. Future studies with larger cohorts and molecular analyses are warranted to clarify the underlying genetic and epigenetic mechanisms and to guide the development of more targeted interventions.

Limitations

This study has certain limitations. First, family history information was partially based on patient self-report and telephone interviews, which introduces the possibility of recall bias. Although structured interviews and partial cross-checking with available hospital or surgical records were performed, complete verification of relatives' medical histories was not always feasible. Second, the number of patients with a positive family history was relatively small, and subgroup analyses—such as comparisons between maternal and paternal lineage—were therefore limited in statistical power. These factors should be considered when interpreting the findings. Future prospective studies with larger cohorts and objective validation methods, including genetic analyses and registry-based data, are warranted to provide stronger evidence regarding the role of familial predisposition in PSD.

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AI statement: The authors stated that no artificial intelligence or artificial intelligence-supported programs were used.

Declaration of interest: No conflict of interest is declared by authors.

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