Clinical phenotypes and risk factors of hidradenitis suppurativa: A retrospective study of 57 Moroccan patients

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Abstract

Background: Hidradenitis suppurativa (HS) is a chronic inflammatory dermatosis. It is a challenging disease characterized by a clinical phenotypic heterogeneity. Clinical phenotypes of HS have different epidemiological and evolutive features, with a significant impact on the therapeutic management. To our days, literature data concerning clinical phenotypes of HS still scarce.

Methods: We conducted a retrospective descriptive study in a Moroccan population, to identify the main clinical phenotype of HS and its risk factors, as a primordial step towards a personalized approach in the management of HS.

Results: Fifty-seven patients were included. Gluteal phenotype (LC2) was the most represented (85.96%). The mean age of onset of symptoms in this group was 31 years old. A male predominance (77.41%) was observed. Acne in adolescence was found in 18 patients (36.73%). Thirty-nine patients (79.59%) were active smokers. Fourteen patients (29.16%) had a prolonged sitting position. Their professional occupation was: drivers (n=10), shopkeepers (n=3) and secretaries (n=1). Five patients (10.20%) had a family history of hidradenitis suppurativa. Mean body mass index was 23.87. Follicular phenotype (LC3) was observed in 8 patients (14.03%). The average age of onset of symptomatology was 37 years old. A female predominance was noted (62.5%). Acne was found in 3 patients (37.5%), active smoking in 2 patients (25%). Mean body mass index was 22.83. Axillary-mammary phenotype (LC1) was observed only in a 15-year-old non-smoking girl in overweight presenting intermammary fold fistulas.

Conclusions: The predominance of the gluteal phenotype in our population may be related to genetic factors, intestinal microbiota and prolonged gluteal friction. Large-scale analytical studies are needed to support these hypothesis.

Keywords: Hidradenitis suppurativa; Clinical phenotypes; Gluteal phenotype LC2.

1. Introduction

Hidradenitis suppurativa (HS) is a chronic and progressive dermatosis [1]. It is characterized by recurrent painful nodules, draining sinus tracts, and abscesses, mainly affecting the axillary, intergluteal, perineal, inguinal, intermammary, and inframammary folds [2]. The clinical presentation is very heterogeneous with several clinical phenotypes. Based on Latent Class Analysis (LCA) classification, three clinical phenotypes can be distinguished: Axillary-mammary phenotype LC1, gluteal phenotype LC2, and follicular phenotype LC3 [3], characterized by a different
epidemiological and clinical outcomes and a distinguished evolutive profil. The aim of our study is to identify the predominant clinical phenotype in our population, and the main factors incriminated in its predominance.

2. Methodology

A retrospective descriptive study was conducted in the dermatology department of Casablanca university hospital, including patients followed up for HS over a 10-years period (between January 2012 and December 2022). Clinical information's were collected from medical records specifying: age, sex, professional occupation, history of acne and family history of HS, toxic habits, age of onset of symptomatology, body mass index, inaugural elementary lesions and their initial topographies as well as elementary lesion and topography at the time of diagnosis, and the disease severity stage according to the Hurley's classification. Clinical phenotypes were determined based on Latent Class Analysis (LCA) classification.

3. Results

Fifty-seven patients were included. Gluteal phenotype LC2 was observed in 48 patients (84.21%), follicular phenotype LC3 in 8 patients (14.03%) and axillary-mammary phenotype LC1 in only one patient (1.75%) (figure 1).

![Figure 1](image.png)

Figure 1 Distribution of the different clinical phenotypes of hidradenitis suppurativa in our population

3.1. Gluteal phenotype LC2

The mean age of onset of symptoms in this group was 31 years, with a male predominance (77.41%). Acne in adolescence was reported in 18 patients (36.73%). Thirty nine patients (79.59%) were active smokers. Prolonged sitting was noted in 14 patients (29.16%), their professional occupations were drivers (n=10), shopkeepers (n=3) and secretaries (n=1). Five patients (10.20%) had a family history of HS. The mean body mass index (BMI) was 23.87, corresponding to a normal build. The initial symptomatology was a fistula (74.19%), an abscess (38.70%), observed in the intergluteal fold (80.64%) (figure 2), the axillary fold (70.96%). At the time of diagnosis, hypertrophic scars (46.93%), retractile scars (30.61%) were noted. A pilonidal sinus was associated in 12 patients (24.48%). The Hurley3 stage was the most represented (71.42%).
Figure 2(a,b) Gluteal phenotype (LC2) with fistulas and retractile scars in the intergluteal fold

3.2. Follicular phenotype LC3

The mean age of onset of symptoms was 37 years, with a predominance of females (62.5%). Three patients (37.5%) had acne during adolescence, only 2 (25%) were active smokers, and the mean BMI was 22.83. In this group, inaugural elementary lesions were fistula (75%), and pustule (50%) starting in the axillary fold (100%) (figure 3). At the time of diagnosis, hypertrophic scars (37.5%) and retractile scars (25%) were noted. Trunk folliculitis was observed in 3 patients (37.5%). Hurley stage 2 was predominant (75%).

Figure 3 Follicular phenotype (LC3) with fistulas, pustules and retractile scars in the axillary fold.
3.3. Axillary-mammary phenotype LC1

The axillary-mammary LC1 phenotype was observed in only a 15-year-old female patient, with no acne or family history of HS, non-smoker, obese, presenting pustules and fistulas in the intermammary fold (figure 4).

![Fistulas in the intermammary fold](image)

Figure 4 Fistulas in the intermammary fold
Table 1 Epidemiological features, risk factors and clinical characteristics among the 3 phenotypes of HS

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Number of patients</th>
<th>Mean age of onset of symptoms (years)</th>
<th>Sex</th>
<th>Acne in adolescence (patients)</th>
<th>Active tabagisme (patients)</th>
<th>Prolonged sitting (patients)</th>
<th>Family history of HS (patients)</th>
<th>Mean BMI</th>
<th>Initial elementary lesion</th>
<th>Initial topography</th>
<th>Hurley stage</th>
<th>Associated follicular disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axillary-mammary phenotype LC1</td>
<td>1 (1.75%)</td>
<td></td>
<td>Female</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>No</td>
<td>27.57</td>
<td>Fistula</td>
<td>Intermammary fold</td>
<td>2</td>
<td>--</td>
</tr>
<tr>
<td>Gluteal phenotype LC2</td>
<td>48 (84.21%)</td>
<td>31</td>
<td>Male (77.41%)</td>
<td>18 (36.73%)</td>
<td>39 (79.59%)</td>
<td>14 (29.16%)</td>
<td>Yes (50.20%)</td>
<td>23.87</td>
<td>Fistula (74.19%)</td>
<td>Intergluteal fold (80.64%)</td>
<td>3 (71.42%)</td>
<td>Pilonidal sinus (24.48%)</td>
</tr>
<tr>
<td>Follicular phenotype LC3</td>
<td>8 (14.03%)</td>
<td>37</td>
<td>Female (62.5%)</td>
<td>3 (37.5%)</td>
<td>2 (25%)</td>
<td>1 (12.5%)</td>
<td>No</td>
<td>22.83</td>
<td>Fistula (75%)</td>
<td>Axillary fold (100%)</td>
<td>2 (75%)</td>
<td>Trunk folliculitis (37.5%)</td>
</tr>
</tbody>
</table>
4. Discussion
The gluteal phenotype LC2 is the most frequent in our population. As in our study, this phenotype is characterized by a predominance of smoker normocorpulent males, with associated pilonidal cysts, and moderate to high disease severity [3]. However, the presence of familial cases suggests the role of genetic factors. Sun Q et al reported that common variants related to HS located near the SOX9 and KLF5 genes were associated with HS risk. These or other nearby genes may be involved in the genetic risk of the disease and the development of clinical features including the clinical phenotype [4]. The initial topography in intergluteal fold, in the majority of our patients, could suggest the role of the intestinal microbiota in the expression of this phenotype. The human gut microbiome is defined as a complex ecosystem consisting of bacteria, fungi, and viruses, all taking part in its host’s health [5]. It is recently incriminated in the pathogenesis of HS [6]. McCarthy et al reported a difference between the gut microbiome of individuals with HS and healthy controls [7], consolidating this hypothesis. Prolonged sitting, as a mechanical factor favoring friction, could also explain the frequency of the gluteal phenotype in our population. In fact, mechanical stress and the possible effect of pressure and shear forces onto the skin in HS have been consistently reported. Consequently, patients are advised to avoid friction onto their skin, and specific underwear are recommended [8]. The follicular phenotype (LC3), which is the second represented phenotype in our study, is characterized by a higher proportion of females, normal/over-weight patients, and had the lowest Hurley stage of severity scores, with a later disease onset and a shorter disease duration. While the axillary-mammary phenotype (LC1), seen in only one patient, is distinguished by a higher proportion of smoker females, a larger proportion of obese patients, a more severe disease stage, earlier disease onset, and longer disease evolution [3].

5. Conclusion
Our study suggests the role of genetic factors, intestinal microbiota and prolonged friction in the predominance of the gluteal phenotype in our population. However, large-scale analytical studies with genetic and microbiological monitoring are needed to confirm these hypotheses.

Compliance with ethical standards

Acknowledgments
The researchers acknowledge and appreciate all patients who participated in this study.

Disclosure of conflict of interest
No conflict of interest.

Statement of informed consent
Informed consent was obtained from all individual participants included in the study.

References


