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Evaluation of Histopathology Findings of Clinically Confirmed Psoriasis Vulgaris

Sawitri[1], Muhammad Yulianto Listiawan[1], Arisia Fadila[1], Priangga Adi Wiratama[2], Dwi Murtiastutik[1], Evy Ervianti[1], Linda Astari[1], Damayanti[1], Diah Mira Indramaya[1], Afif Nurul Hidayati[1], Medhi Denisa Alinda[1]

[1]Department of Dermatology and Venereology, Faculty of Medicine/Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

[2]Department of Pathology and Anatomy, Faculty of Medicine/Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

ABSTRACT

Background: Psoriasis vulgaris can be diagnosed clinically. A biopsy is usually used to confirm non-classic cases. The findings of the histopathology feature are not usually present, thus clinical relevance is needed to confirm the diagnosis. **Purpose**: To describe the histopathology findings from tissue samples of histopathologically confirmed psoriasis vulgaris reported in the Department of Pathology and Anatomy of Dr. Soetomo General Hospital. **Methods**: A descriptive-retrospective study of pathology-ascertained tissue samples of histopathologically confirmed psoriasis vulgaris was reported in the Department of Pathology and Anatomy from patients who were also previously diagnosed clinically with psoriasis vulgaris in the Dermatology and Venereology Outpatient Unit of Dr. Soetomo General Academic Hospital over a period of 2 years. Morphological parameters were observed after histopathology sections were stained with hematoxylin and eosin. **Result**: Thirty-three tissue samples were examined. Parakeratosis (86%) and hypogranulosis (70.3%) were the most observed findings. Club-shaped rete ridges, suprapapillary plate thinning, and spongiform pustules of Kogoj were the least observed findings (each by 2.7%). Three tissue samples (8.1%) showed only parakeratosis. Only 1 tissue sample (2.7%) showed 4 features (absence of the granular layer, parakeratosis, microabscess of Munro, and spongiform pustules of Kogoj). No tissue sample showed every histopathology finding of psoriasis vulgaris. **Conclusion**: All of the histopathology features of psoriasis vulgaris are rarely found in one tissue section. Albeit histopathology is believed to be the gold standard diagnosis of psoriasis vulgaris, relevance with clinical findings is still mandatory to support the diagnosis.

Keywords: psoriasis vulgaris, histopathology, human & health.

Correspondence: Sawitri, Department of Dermatology and Venerology Faculty of Medicine, Universitas Airlangga / Dr. Soetomo General Academic Hospital, Surabaya, Jl. Mayjen Prof. Dr. Moestopo No. 6-8 Surabaya 60131, Indonesia. E-mail: sawitri.rh@gmail.com, Phone: 628123033993.

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BACKGROUND

Psoriasis vulgaris is a chronic inflammatory papulosquamousskin disease characterized histopathologically by epidermal hyperplasia. The prevalence is estimated to be between 0.1 and 3 percent in all over the world, and the variation is influenced by age, sex, geography, ethnicity, genetics, and enviromental factors.[1,2] Psoriasis vulgaris has a worldwide distribution and affects equally men and women of all ages, races, and social economic strata. The symptoms could be deleterious, stigmatizing, and affect the patient's quality of life. Psoriasis vulgaris is currently not only considered as skin disorder but also a systemic condition that is associated with metabolic, joints

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cardiovascular, and psychological morbidities.[1] The pathogenesis has not yet been clearly established although it is known to be a combination of immune, genetics, and enviromental factors.[2]

The diagnosis is usually clinical, a histopathology examination is indicated for non-classic cases.[2-4] The classic clinical manifestations of psoriasis vulgaris are usually typical, which are well-defined, thick, silvery white scales; the presence of a smooth, red membrane with bleeding points beneath the scales by manual removal (Auspitz sign); the typical predilection of the lesions (Köebner area) such as the scalp, elbows, knees, and gluteal region, despite the fact that any skin surface can be involved; and the appearance of a new lesion after trauma, known as isomorphic response or Köebner sign.[1,2] Establishing the diagnosis of psoriasis vulgaris in cases with a lack of those classic clinical and/or histologic criteria will be confusing because there is still no other method to establish the diagnosis without those prototypical findings.

Some previous reports regarded histopathology evaluation as the gold standard to confirm the psoriasis vulgaris diagnosis. Chau et al. (2015) named 8 classic histopathology findings of psoriasis vulgaris, which are regular acanthosis, hypogranulosis, club-shaped rete ridges, thinning of the suprapapillary plate, edema and elongation of the dermal papillae, the microabscess of Munro, and the spongiform pustule of Kogoj. Chau also mentioned some non-classic features that can be encountered in a psoriasis vulgaris histopathology examination: irregular acanthosis, hypergranulosis, orthokeratosis, necrotic keratinocytes in the stratum spinosum and stratum basale, lichenoid interface and junctional vacuolar alterations, spongiosis, neutrophils in the dermal layer, eosinophils, plasma cells, and fibrosis of the papillary dermal. However, the classic histopathology findings usually outnumber the non-classic findings in psoriasis vulgaris tissue samples and are mostly characteristic of psoriasis vulgaris.[5,6] The non-classic findings are not to be discussed in the current study.

A single cut of psoriasis vulgaris tissue rarely shows all features of classic histopathology findings. In order to collect more features, multiple sections or a larger number of samples need to be examined. Those classic histopathology findings are said to represent the disease's activity. Along with the dynamic process, there are microscopic differences between each lesion. The most typical findings of psoriasis vulgaris, which are microabscesses of Munro and spongioform pustules of Kogoj, are rarely found. In such cases, other classic epidermal features can substitute for the confirmation of psoriasis vulgaris along with the previously mentioned representative clinical findings.[5] Kim et al. (2015) reported that parakeratosis, hypogranulosis, regular acanthosis, and edema and elongation of the dermal papillae are found more frequently in psoriasis vulgaris histopathology examinations. However, because the evaluation of the tissue samples is usually only done in one section, the finding of those microscopic features is also limited. Hence, the diagnosis of psoriasis vulgaris needs to be supportive clinically and histopathologically.[7]

Trozak (1994) mentioned 10 microscopic findings that are characteristic of psoriasis vulgaris: regular acanthosis, club-shaped rete ridges, edema and elongation of the dermal papillae, perivascular mononuclear infiltrate in the upper dermis of papillae, hypogranulosis, parakeratosis, thinning of the suprapapillary plate, mitosis above the stratum basale, microabscesses of Munro, and spongioform pustules of Kogoj.[6] This retrospective study is aimed at describing those findings from psoriasis vulgaris tissue samples of clinically confirmed psoriasis vulgaris cases in the Dermatology and Venereology Outpatient Unit of Dr. Soetomo General Academic Hospital Surabaya.

METHODS

A total of 37 samples which were clinically diagnosed as psoriasis vulgaris, another morphology type of psoriasis vulgaris or those suspected with other dermatoses and recorded in the Outpatient Unit of Dermatology and Venerology and the Department of Pathology and Anatomy Faculty of Medicine, Universitas Airlangga / Dr. Soetomo General Academic Teaching Hospital, Surabaya, throughout a period of 2 years (January 2019–December 2020), were analysed. Each slide was taken from an active lesion (erythematous, raised, and scaly patches) from a different patient; 37 patients in total. Each slide consists of three serial sections. The best section that still represented a lesion was chosen. Slides were reviewed by two examiners. Paraffin blocks were retrieved, sectioned, stained with Hematoxylin and Eosin (H&E), and recorded for their morphological parameters 1391/112/4/IV/2022

RESULT

In a total of 37 samples, the most common age

group was found to be between 45 and 60, followed by 25 to 44 years old. The study showed female predominance (21 females, 16 males). At the time of the biopsy, all patients had complained of erythema, itch, scales, and recurrence (100%), 15 patients had dandruff (40.54%), and 2 patients had joint pain (5.4%). We did not have the data about the patients previous diseases due to a lack of data. Psoriasis Area Severity Index (PASI) records were obtained for 33 patients; 4 patients (10.8%) did not have PASI data. Most of the recorded PASI was not obtained at the time of biopsy. Most outpatients (62.2%) had severe psoriasis vulgaris (PASI>10), followed by moderate psoriasis vulgaris (PASI 3-10) (21.6%), and mild psoriasis vulgaris (PASI <3) (5.4%). Documented therapy at the time of biopsy included 89 patients receiving topical corticosteroids and 10% (4/36) receiving topical corticosteroids and Methotrexate (MTX). We did not have the data about patients duration of therapy due to a lack of data (Table 1).

Table 1. Characteristics of psoriasis vulgaris patients.

Variable	Number of Cases	Percentage (%)
Sex		
Male	16	42.2
Female	21	56.76
Age (years old)		
<15	3	8.1
15-24	3	8.1
25-44	10	27
45-60	16	43.2
>60	5	13.5
Symptoms		
Erythema	37	100
Itch	37	100
Dandruff	15	40.5
Joint pain	2	5.4
PASI		
Mild (<3)	2	5.4
Moderate (3-10)	8	21.6
Severe (>10)	23	62.2
No data	4	10.8
Therapy		
Topical steroid	33	89.2
Topical steroid + MTX	4	10.81
NBUVB	0	0

PASI: Psoriasis area severity index; MTX: Methotrexate; NBUVB: Narrowband ultraviolet B

Table 2. Parameters of histopathology findings.

Criteria	Number of Cases	Percentage
Regular acanthosis	3	8.1
Club-shaped rete ridges	1	2.7
Elongation and edema of dermal papillae	5	13.5
Perivascular mononuclear infiltrate in the upper papillae dermis	11	29.7
Hypogranulosis	20	70.5
Parakeratosis	32	86.5
Suprapapillary plate thinning	1	2.7
Mitosis above the stratum basalis	4	10.8
Microabscesses of Munro	7	18.9
Pustules spongiform of Kogoj	1	2.7

The most prevalent features were parakeratosis and hypogranulosis. The least prevalent features were club-shaped rete ridge, suprapapillary thinning, and spongioform pustules of Kogoj. In our study, papillomatosis was seen in perivascular infiltrates in 11 cases (29.7%), 5 cases (13.5%), mitosis above the stratum basale in 4 cases (10.8%), and elongation of rete ridges in the remaining 3 cases (8.1%) (Table 2).

DISCUSSION

Thirty-seven psoriasis vulgaris tissue samples were analyzed in our study. In the present study, psoriasis vulgaris was slightly more prevalent in female patients (56.76%). The incidence of psoriasis was reported to be equal in males and females.[2,8] A few studies had reported that the prevalence of psoriasis vulgaris was slightly higher in men than women due to life style.[9]

Psoriasis vulgaris was found in people of all ages, with the most common age group being 45-60 years old. The youngest age was 4 and the highest age was 69 years old, with the mean age being 42.65±16.8 years old. Kassi K. et al. in 2013 reported that the average age of psoriasis was 39.6±3.3 years old, with extremes of 4 and 77 years.[10] In 2013, Alhumidi et al. reported in their study that the age of onset of psoriasis vulgaris was found to be between 6 and 83 years old, with a mean age of 31.5 years old.[11] According to Coimbra et al. (2012), psoriasis vulgaris can occur at any age and has a bimodal onset, with the first onset typically occurring between the ages of 16 and 22 and the second onset typically occurring between the ages of 57 and 60. The age of onset is thought to be slightly earlier in women than in men.[12]

Limited studies have been performed regarding The histopathological changes in psoriasis vulgaris. In our study, the classic histopathological features of parakeratosis and hypogranulosis were found to be prominent in all cases (86% and 70.3%, respectively). Alternatively, club-shaped rete ridges, suprapapillary thinning, and pustules of Kogoj were less evident (each by 2.7%). In 2013, Alhumidi et al. reported in their study that the most common features were acanthosis (75%), microabscess of Munro (70%), elongation and edema of dermal papillae (62%), and hypogranulosis (40%).[11] In 2015, Chau et al.reported that the most common features were club-shaped rete ridges (96%) and hypogranulosis (40%).[5] In 2013, Kassi K. et al.reported that loss of the granular layer and parakeratosis were the exclusive epidermal features of psoriasis vulgaris (100%), followed by thinning of the suprapapillary plate (90.9%) and microabscess of Munro (72.2%).[10]

Hypogranulosis is considered a highly sensitive finding in psoriasis.[5]Hypogranulosis findings are accordant with defective keratinocyte proliferation due to abnormal regulation of T cells, which is the pathogenesis of psoriasis vulgaris. The defective keratinocytes promote the activation of cytokine release that becomes a loop, resulting in the hyperplasia of the epidermis. Keratinocyte proliferation becomes rapid as the cells' nuclei retent in the terminally differentiated keratinocytes, causing parakeratosis and variable changes. In the stratum from hypogranulosis to agranulosis.[13] Parakeratosis started to form in the early stages and became more confluent in the advanced stages. Most of our biopsies (70.3%) showed parakeratosis and hypogranulosis was one of the prominent findings in our study. These two findings are reported to occur in a cyclical fashion.[6]

The presence of club-shaped rete ridges is characteristic of psoriatic epidermis, which helps rule out seborrheic and nummular dermatitis, which both usually show squared-off instead of club-shaped ridge tips. Suprapapillary plate thinning strongly supports the diagnosis of psoriasis vulgaris, which is commonly seen in fully developed lesions, and rarely present other psoriasiform conditions.[6] This was not in line with our study and can be attributed to the small section that was evaluated in this study and the variability of the findings themselves. In 2022, Yelamos et al.reported that only about 0.1% of a 4-mm specimen actually gets presented in a glass side.[14] Especially for suprapapillary plate thinning, careful sectioning must be established at the epidermal surface.[6]

The microabscess of Munro and the spongiform pustule of Kogoj are the most diagnostic features of psoriasis vulgaris because those are notably uncommon in other skin diseases, yet both are reported to be present only in a diminutive way (except when associated with the psoriasis group of diseases).[6,13,15] Munro microabscess was found to be 18.9% in our study,while spongiform pustules of Kogoj were found to be 2.7% of the cases in our samples. Kim et al. in 2015 reported that microabscess of Munro and spongiform pustules of Kogoj were found regardless of the lesion size due to both are not perpetually located in the psoriatic epidermis. The examined section may not contain these 2 findings, even if the whole biopsy

specimen contained both findings. Contrarily, the other classic findings are commonly found anywhere in the psoriatic lesion; therefore, their frequencies are surely increased when larger lesions are examined.[7]

In 2016, Bai et al. reported that the fundamental histopathological findings of psoriasis vulgaris are a combination of acanthosis, parakeratosis, suprapapillary thinning, papillomatosis, intercellular edema, mitosis above the stratum basale, hypogranulosis, tortuous capillaries in dermal papillae, perivascular infiltration of lymphocytes, microabscess of Munro, and spongiform pustules of Kogoj. But all these characteristic features may not be found in one section alone. However, some of these findings can also be seen in non-psoriatic conditions.[13] In the present study, there were 3 tissue samples that only showed 1 classic feature, feature, which is parakeratosis, and 1 single biopsy in our series demonstrated none of the features of psoriasis vulgaris. On the other hand, there was 1 tissue sample that showed most of the 4 classic features of psoriasis vulgaris: hypogranulosis, parakeratosis, microabscess of Munro, and spongiform pustules of Kogoj.

The variability in histopathology features of psoriasis vulgaris means that all classic findings from one specimen are seldom seen on a single cut; hence, a large number of sections need to be studied to obtain an indicative picture of the evaluation.[6] This can also explain why, since the possibility of histopathology findings in a psoriasis tissue section is not always showing all of the classic parameters of psoriasis vulgaris, clinical judgement is crucial in diagnosing the diseases. However, there are several limitations in our study. The first limitation is that our study was using old paraffin blocks, and the second limitation is the nature of the retrospective design, so we could not obtain exact information about patients' clinical data and previous histories.

All the classic histology features of psoriasis vulgaris are rarely found in one tissue section. Histopathological parameters along with clinical assessment are mandatory to support the diagnosis of psoriasis vulgaris. Microabscess of Munro and spongiform pustules of Kogoj were the pathognomonic features in psoriasis vulgaris, yet they are rarely present. In the absence of the latter two, other classic epidermal features are feasible as confirmatory evidence of psoriasis vulgaris.

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