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Subcorneal pustular dermatosis: clinical characteristics and long-term follow-up of seventeen patients

Subkornealna pustulozna dermatoza: kliničke karakteristike i dugotrajno praćenje sedamnaest bolesnika

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Abstract

Background/Aim. Subcorneal pustular dermatosis (SPD) is a rare, relapsing vesiculopustular disease predominantly presenting on the flexor surfaces. Since data about the course and duration of the disease is limited, the aim of the study was to analyze the clinical features and long-term follow-up data on patients with SPD. **Methods.** A hospital database was searched for medical records of patients with SPD hospitalized at the institution between 1985 and 2014. The focus was on clinical characteristics, associated diseases, disease courses, and outcomes. **Results.** Seventeen patients with clinical features of SPD were analyzed – nine females and eight males with a median age at presentation of 45 years (range 18–90 years). Follow-up data were available for 12 patients. The median follow-up time was 9.5 years (1–28 years). In order to establish a histological diagnosis, repeated biopsies were required (on average 1.7 biopsies *per* patient). In one male patient, IgA pemphigus was diagnosed by direct immunofluorescence. In most patients, skin eruption was widespread, while in a smaller number of patients, the changes were present only on the flexor surfaces. Dysproteinemia was evident in three patients. The disease was self-limiting in three patients; five patients had mild flare-ups occurring 1–3 times a year without the need for treatment; four patients had continuous flare-ups requiring treatment. Most patients with SPD responded positively to dapsone. **Conclusion.** SPD is a rare disease, and it usually occurs in a person's fifth decade. While it is self-limiting in some patients, approximately one-third of patients require continuous therapy for continuous flare-ups.

Key words:
biopsy; dapsone; diagnosis; immunohistochemistry; skin diseases, vesiculobullous; treatment outcome.

Apstrakt

Uvod/Cilj. Subkornealna pustulozna dermatoza (SPD) je retko, relapsirajuće vezikulopustulozno oboljenje, koje predominantno zahvata fleksorne površine. S obzirom da su u dostupnoj literaturi podaci o toku i trajanju SPD veoma oskudni, cilj rada bio je da se analiziraju kliničke karakteristike i rezultati dugotrajnog praćenja bolesnika sa SPD. **Metode.** Pretraženi su medicinski kartoni iz bolničke baze podataka bolesnika sa SPD hospitalizovanih u periodu od 1985. do 2014. godine. Fokus je bio na kliničkim karakteristikama, pridruženim bolestima, toku bolesti i ishodima. **Rezultati.** Analizirano je 17 bolesnika sa kliničkim karakteristikama SPD: devet žena i osam muškaraca čija prosečna starost u trenutku manifestacije bolesti je bila 45 godina (raspon 18–90 godina). Podaci o praćenju bolesti bili su dostupni za 12 bolesnika. Srednje vreme praćenja iznosilo je 9,5 godina (1–28 godina). U cilju uspostavljanja histološke dijagnoze bile su potrebne ponovljene biopsije (u proseku 1,7 biopsija po bolesniku). Kod jednog bolesnika je direktnom imunofluorescencijom dijagnostikovan IgA pemfigus. Kod većine bolesnika kožna erupcija bila je rasprostranjena, dok su kod manjeg broja bolesnika promene bile prisutne samo na fleksornim površinama. Disproteinemija bila je prisutna kod tri bolesnika. Bolest je bila samoograničavajuća kod tri bolesnika; pet bolesnika je imalo blage relapse 1–3 puta godišnje, bez potrebe za lečenjem; četiri bolesnika imala su kontinuirane relapse, koji su zahtevali lečenje. Većina bolesnika sa SPD je pozitivno reagovala na dapson. **Zaključak.** Bolest SPD je retka, a obično se javlja u petoj deceniji života. Mada je kod nekih bolesnika samoograničavajuća, kontinuirana terapija je potrebna približno jednoj trećini bolesnika zbog čestih relapsa.

Ključne reči:
biopsija; dapson; dijagnoza; imunohistohemija; koža, vezikulobulozne bolesti; lečenje, ishod.

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Introduction

Subcorneal pustular dermatosis (SPD) is a rare, relapsing neutrophilic dermatosis first described by Sneddon and Wilkinson [1]. It can occur at any age but is more commonly observed in middle-aged and older women [2–4]. The causes and pathogenesis of SPD are still unknown. Since the disease is characterized by sterile, subcorneal accumulation of neutrophils, it has been suggested that immunologic mechanisms are involved in pathogenesis. It has been shown that tumor necrosis factor (TNF)-α, interleukin-8, and complement component C5a levels are raised in affected tissue samples [2]. In a subset of patients, intraepidermal deposits of immunoglobulin (Ig) A have been detected on direct immunofluorescence (DIF) [5, 6].

Clinical findings include discrete, oval, flaccid pustules on normal or slightly erythematous skin, predominantly distributed on the flexor surfaces and proximal limbs. Lesions may be grouped or isolated and have a characteristic appearance with the accumulation of pus in the lower half of the pustules [1, 2, 7, 8]. SPD has been described in association with IgA-paraproteinemia, IgA multiple myeloma [9–13], inflammatory bowel diseases [14, 15], rheumatoid arthritis [16, 17], Sjögren's syndrome [18], pyoderma gangrenosum [19–21], pustular psoriasis [22], and *Mycoplasma* (*M.*) *pneumoniae* infection [23, 24].

Data regarding the disease course and long-term outcomes are scarce. The aim of this study was to evaluate the clinical features, associated diseases, therapeutic options, and disease course and outcomes in patients with SPD after a long-term follow-up.

Methods

The Military Medical Academy, Belgrade, Serbia, database was searched for medical records of patients with SPD treated at the institution between 1985 and 2014. Demographic data, as well as data on clinical manifestations, associated diseases, treatments, and follow-ups, were recorded and analyzed in a retrospective study format. Diagnoses were based on clinical appearance and histopathological and DIF analysis of skin samples. Patients' records were reviewed, and cases fulfilling the diagnostic criteria proposed by Lutz et al. [7] were included in the analysis. These criteria included the onset of a pustular eruption without systemic symptoms, flaccid pustules with pus filling the lower half of lesions, absence of existing psoriasis or other stigmata of psoriasis, subcorneal neutrophilic pustule without spongiosis, and response to dapsone if challenged. Laboratory investigations encompassed the following: full blood cell count with differential, chemistry studies, quantitative serum IgA, IgM, IgG levels, urine, and serum electrophoresis, complete urinalysis, syphilis serology, microbiological examination of swabs, and other tests for excluding other specific dermatoses. Descriptive statistics were used to estimate patient characteristics and data processing.

Results

Clinical characteristics, treatments, and follow-ups of the patients are presented in Table 1. Between 1985 and 2014, 17 patients were diagnosed with SPD – nine females and eight males. Follow-up data were available for 12 patients. The median age at presentation was 45 years (range 18–90 years). The median disease duration from diagnosis was four years (range 1–28 years).

Pustular lesions were the characteristic finding in patients with SPD. Twelve patients exhibited widespread pustular eruptions, while five presented with lesions solely on flexural surfaces (Figures 1 and 2). Skin biopsies, as seen in Figure 3, were performed for all 17 patients to confirm the diagnosis. The initial biopsy confirmed SPD in 10 (58.8%) patients. For the others, multiple biopsies were required for diagnosis. DIF and indirect immunofluorescence (IIF) examinations were performed on 13 patients to rule out other autoimmune bullous dermatoses. Twelve of these patients tested negative. However, one patient displayed intercellular IgA deposits in the upper third of the epidermis, and an IIF examination verified the presence of IgA autoantibody deposits in the serum (titer of 1:20). This led to a diagnosis of IgA pemphigus, SPD type. Immunoblotting of human epidermal extracts revealed that sera clearly reacted with the 160 kD *pemphigus foliaceus* antigen.

Bacteriological examinations of pustule smears showed no bacterial growth in 11 patients. In contrast, *Staphylococcus* (*S.*) *aureus* was identified in six patients. Notably, despite this identification, the disease followed a chronic course and did not respond to short-term antibiotic treatment. This aligns with the idea that the presence of *S. aureus* was due to colonization of the diseased skin and not an active infection. Comorbidities were observed in 7 (41.2%) out of the 17 patients. Specific findings included polyclonal hypergammaglobulinemia in two patients, decreased levels of IgA in one, and benign monoclonal gammopathy (MG) IgA in one patient diagnosed with IgA pemphigus. Single instances of hypothyroidism, diabetes mellitus, and seronegative polyarthritis were also documented.

In the majority of patients, systemic treatment was necessary to control the disease, while in only one patient with mild symptoms, topical corticosteroids were sufficient to control flare-ups. Dapsone 1–3 mg/kg was used as monotherapy in 6 (35.4%) patients and in combination with systemic steroids (initial dose of 0.5 mg/kg) in six patients. Treatment with dapsone was combined with ultraviolet B (UVB) therapy in one patient. For two patients, systemic steroids were combined with etretinate and cyclosporine. In two particularly challenging cases (including the case of IgA pemphigus), multiple treatments were explored, including etretinate, a combination of psoralen and long-wave ultraviolet radiation (PUVA), cyclosporine A, colchicine, methotrexate, acitretin, and narrow-band UVB (Table 1). The median duration of active treatment for these two patients was 5.95 years.

Table 1

Clinical characteristics, treatment, and follow-up of the patients with subcorneal pustular dermatosis

Patient number	Gender / age (years)	Localization	Disease duration from the onset (years)	Disease duration from diagnosis (years)	MG	DIF	IIF	Treatment	Associated diseases	Follow-up
1	F/55	generalized	36	23	no	ND	ND	etretinate	hypergammaglobulinemia	23 years after diagnosis, continues to have a rare intermittent pustule
2	M/41	flexor surfaces	4	4	no	neg	ND	dapsone with systemic Cs	IgA hypoglobulinemia	last 8 years in remission after dapsone treatment
3	F/52	generalized	2	/	no	neg	neg	dapsone	diabetes mellitus	lost to follow-up
4	M/45	flexor surfaces	2	/	no	ND	ND	dapsone with systemic Cs	no	lost to follow-up
5	M/19	generalized	1	/	no	neg	neg	dapsone	no	lost to follow-up
6	F/40	generalized	11	3	ND	neg	1:10	dapsone	hypergammaglobulinemia	14 years of disease in remission
7	M/21	generalized	1	/	no	neg	neg	dapsone	no	lost to follow-up
8	F/35	generalized	28	28	no	ND	ND	dapsone	no	28 years after having a rare pustular lesion in the summer period
9	F/62	flexor surfaces	3	1	no	neg	neg	dapsone	angina pectoris, bronchial asthma	one year disease controlled with dapsone without new eruption during 18 years of follow-up period
10	M/19	generalized	12	/	no	neg	neg	dapsone + nbUVB	no	lost to follow-up
11	F/18	flexor surfaces	7	1	no	neg	neg	topical Cs	no	last 12 years without the occurrence of disease
12	F/54	generalized	6	3	no	neg	neg	dapsone with systemic Cs	seronegative polyarthritis	3 years after dapsone treatment very rare mild eruption once a year
13	F/68	generalized	6	6	no	neg	ND	dapsone with systemic Cs	no	6 years after diagnosis, frequent eruption of pustules
14	F/34	generalized	10	8	no	neg	neg	acitretin dapsone nbUVB cyclosporine A methotrexate	hypothyreosis	4 years after multiple treatment modalities has an occasional eruption
15	M/90	generalized	4	1,5	no	ND	neg	cyclosporine A with systemic Cs	angina pectoris	died
16	M/48	generalized	24	4	no	neg	neg	PUVA dapsone with systemic Cs	arterial hypertension	4 years on dapsone therapy with low dose Cs intermittent flares 3 times a year
17	M/50	flexor surfaces	26	8	IgA	IgA[#]	1:20	dapsone + Cs methotrexate PUVA etretinate cyclosporine A colchicine	IgA MG	last 18 years continues to have intermittent flares twice a year

MG – monoclonal gammopathy; DIF – direct immunofluorescence; IIF – indirect immunofluorescence; F – female; M – male; ND – not done; neg – negative; Cs – corticosteroids; nbUVB – narrow-band ultraviolet B; PUVA – psoralen + long-wave ultraviolet radiation. [#] – IgA in the upper epidermis.

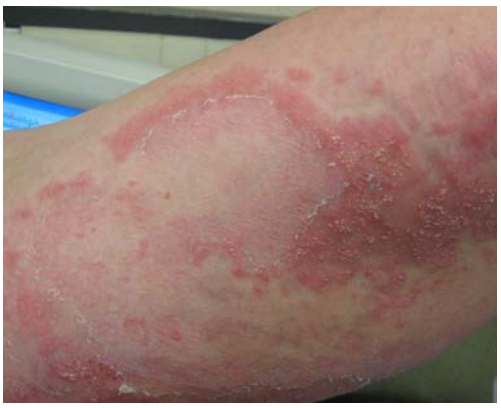

Fig. 1 – Coalescence of pustules on erythematous skin, which form a circinate pattern on the flexor side of the upper limbs.

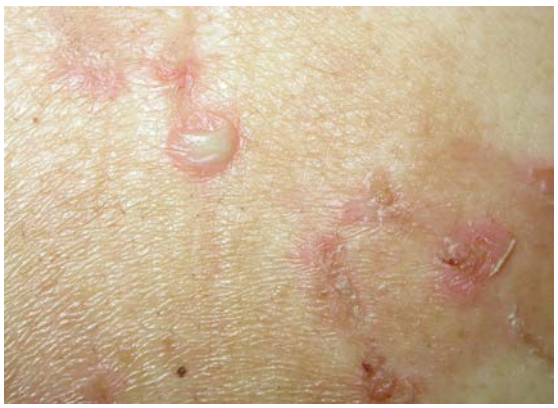

Fig. 2 – Newly formed flaccid pustule with hypopyon formation.

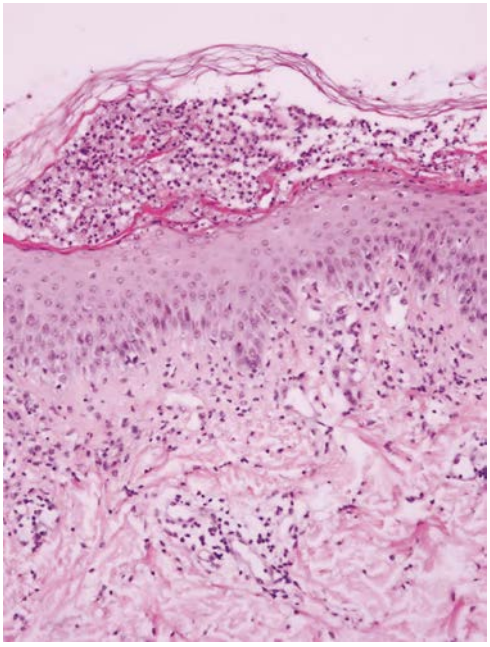

Fig. 3 – Histopathological examination shows a subcorneal accumulation of neutrophils. The dermis contains a sparse, mostly mononuclear, perivascular, and interstitial inflammatory infiltrate (hematoxylin-eosin, ×400).

A long-term follow-up spanning 1–28 years (median 9.5 years) revealed that most patients experienced lesion recurrence and needed ongoing or intermittent therapy to manage flare-ups (Table 1). Five patients had periodic flare-ups occurring one to three times a year, which were managed with intermittent topical corticosteroid treatment. Summer flare-ups were reported by one female patient. For three female patients (aged 18, 40, and 62 years), the disease spontaneously resolved, with a median duration of seven years and no relapse in the past ten years. Two patients experienced continuous flare-ups and were managed with dapsone and low-dose systemic steroids. Among the four patients diagnosed with hypo- or hyper-gammaglobulinemia, no progression to hematologic disease was observed.

Discussion

SPD is a rare but challenging clinicopathologic entity. It is a chronic, benign, pustular eruption, usually affecting adults, mostly middle-aged women. New lesions have the tendency to coalesce and often form annular or *serpiginous* patterns. Flaccid pustules or vesicles with an accumulation of pus in the lower half of lesions on clinically normal or slightly erythematous skin are characteristic. These lesions typically appear symmetrically, affecting submammary areas, axillae, groin, flexor side of the limbs, and the abdomen. No differences have been described in the clinical characteristics or prognosis of the disease in children and adults [1, 2, 7, 8].

Although previous studies have suggested that the disease affects females more frequently than males [2, 8], an equal gender distribution was found between males and females in the current analysis. Most patients in this study were middle-aged, though four were young adults aged 18–21. Follow-up data were unavailable for three of these patients. In two out of the total of three cases in whom the disease was self-limiting (an 18-year-old and a 40-year-old female), the disease lasted for seven years from the first onset without recurrence during a 12-year follow-up period.

In a recent case report of younger patients with SPD associated with *M. pneumoniae* [23, 24], it was postulated that SPD could be a self-limiting dermatosis occurring as a reaction to an infective agent in younger adult patients. Further case studies with a larger number of patients are needed to answer this question, as the available literature on follow-up data is limited. In our case series, no difference was found between patients younger and older than 40 years in relation to the clinical features, localization, or disease duration. As expected, associated diseases were more common in older patients.

The histopathologic hallmark of SPD is subcorneal pustules filled with neutrophils, occasionally eosinophils, and acantholytic cells [25]. In 1983, Sanchez et al. [22] reviewed more than 20 cases of SPD and postulated that it is clinically and histopathologically associated with psoriasis. They suggested the need for longer follow-ups to differentiate these conditions better. However, further studies did not confirm these assumptions, and SPD was classified as a neutrophilic dermatosis associated with gammopathies and inflammatory bowel disease.

There have been many case reports of SPD accompanied by IgA gammopathy [10–13], IgA myeloma [9, 19], Crohn's disease [14, 15], pyoderma gangrenosum [19–21], aplastic anemia [26], and more. In a study of 10 patients with SPD, Lutz et al. [7] found that four patients had monoclonal paraproteinemia, and three were of the IgA type. A comparison was made with a control population of 20 patients with pustular psoriasis in which no MG was detected. In the present case series, hematologic disorders were found in four patients, and of these, benign IgA MG was found in one without evidence of progression in long-term follow-up.

IgA pemphigus must be sought out in patients with the clinical appearance of SPD. A small number of cases of IgA pemphigus have been reported in the literature, with a slight prevalence of the SPD type [27, 28]. IgA pemphigus associated with MG is rare and, until now, it has beendescribed in 20% of patients. In 1992, Wallach [5] reviewed 29 cases. Six cases were associated with IgA paraproteinemia; of these, two patients had myeloma, one had B-cell lymphoma, and one could not be examined, while two patients were diagnosed with benign gammopathy. MG can appear concurrently with the skin lesions or appear between 4 to 27 years before or after them. This association was found exclusively in the SPD subtype and has never been found in the intraepidermal neutrophilic IgA dermatosis subtype.

SPD–type IgA pemphigus was found in one patient, and it was associated with IgA MG at diagnosis.

The drug of choice for SPD treatment is dapsone at a dose of 50–150 mg/d. The response is slower than in dermatitis herpetiformis but most often obtained [1, 8]. In the study of 10 patients, Lutz et al. [7] proposed that one of the diagnostic criteria for SPD should be a response to dapsone. The majority of patients in the present study had a satisfactory therapeutic effect after the administration of dapsone as monotherapy or in combination with systemic steroids, as opposed to those with IgA pemphigus, which was more resistant to dapsone therapy. Two patients, one diagnosed with the IgA pemphigus SPD variant, underwent several therapeutic regimens based on literature data, including methotrexate, etretinate [29, 30], cyclosporine A [31], colchicine [32], PUVA [33], and acitretin [13]. There is evidence that TNF-α may be involved in the pathogenesis of SPD, and, also, there is an association between SPD and inflammatory bowel disease, pyoderma gangrenosum, and rheumatoid arthritis. For the patients with recalcitrant SPD in which multiple treatments were attempted without benefit, there were few reports on patients responding to etanercept [34] and chimeric anti-TNF-α antibody (infliximab) [35, 36] .

Conclusion

After achieving long-term remission, the majority of SPD patients experienced rare recurrences that were very mild in comparison to the disease at first presentation. However, in resistant cases of the disease, the need for long-term treatment should be included in the treatment plan.

Conflict of interest

The authors declare no conflict of interest.

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