DOI: https://doi.org/10.17816/dv634008

Case report

Description of a clinical case of bullous scleroderma in a somatically impaired patient

Elena S. Snarskaya, Natalia P. Teplyuk, Lydia M. Shnakhova, Diana A. Myshlyanova, Juliya M. Semiklet

I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russia

ABSTRACT

Bullous scleroderma is a rare variant of localized scleroderma characterized by the formation of subepidermal tense blisters. At the moment, the etiology and pathogenesis of dermatosis is not fully understood, but a number of authors consider the pathology of the endocrine system as a trigger for the development of localized and systemic scleroderma.

Untimely or erroneous diagnosis of bullous scleroderma leads to the risk of developing a systemic process, irreversible cosmetic defects and even disability.

Treatment of bullous scleroderma should be complex and multicourse with the inclusion of systemic glucocorticoids, antibacterial and cytostatic drugs, agents that improve microcirculation, as well as topical glucocorticoids of the 3-4th class of activity, regenerating agents. Physiotherapeutic methods of treatment such as PUVA and UVA therapy are widely used; there are reports on the successful use of a combination of immunosuppressive mycophenolate mofetil and extracorporeal photopheresis, as well as intravenous infusions of N-acetylcysteine. In addition to traditional drug and physiotherapy therapies, the use of biological therapies is currently being considered.

The article presents a rare case of scleroderma bullosa on the background of pronounced chronic endocrine pathology. The interest of this clinical case lies not only in the rare form of the disease and the progressive nature of the course of dermatosis, but also in the probable association of dermatosis with the patient's endocrine gland diseases in the form of panhypopituitarism due to adenohypophysis macroadenoma with the development of secondary hypothyroidism and adrenal insufficiency. In our observation, the steady negative dynamics of the skin process — from localised plaque scleroderma with subsequent transformation into the bullous form, and further into a probable systemic process — drew attention.

Taking into account the severity of the disease course, the presence of concomitant chronic endocrinopathy, as well as positive specific serological reactions for the systemic form of scleroderma, in the presented clinical case the patient needs an interdisciplinary approach and constant dispensary observation.

Keywords: bullous scleroderma; etiology; diagnosis and treatment.

To cite this article:

Snarskaya ES, Teplyuk NP, Shnakhova LM, Myshlyanova DA, Semiklet JM. Description of a clinical case of bullous scleroderma in a somatically impaired patient. *Russian journal of skin and venereal diseases.* 2024;27(4):435–447. DOI: https://doi.org/10.17816/dv634008

Submitted: 02.07.2024 **Accepted:** 31.07.2024 **Published online:** 25.08.2024

DOI: https://doi.org/10.17816/dv634008

Клинический случай

Клинический случай буллёзной склеродермии у соматически отягощённой пациентки

Е.С. Снарская, Н.П. Теплюк, Л.М. Шнахова, Д.А. Мышлянова, Ю.М. Семиклет

Первый Московский государственный медицинский университет имени И.М. Сеченова (Сеченовский Университет), Москва, Россия

АННОТАЦИЯ

Буллёзная склеродермия — редкий вариант локализованной склеродермии, характеризующийся образованием субэпидермальных напряжённых пузырей. В настоящий момент этиология и патогенез дерматоза изучены не до конца, однако ряд авторов рассматривают патологию эндокринной системы как триггер развития локализованной и системной склеродермии.

Несвоевременная или ошибочная диагностика буллёзной склеродермии ведёт к рискам развития системности процесса, необратимых косметических дефектов и даже инвалидности.

Лечение буллёзной склеродермии должно быть комплексным и многокурсовым с включением системных глюкокортикоидов, антибактериальных и цитостатических препаратов, средств, улучшающих микроциркуляцию, а также топических глюкокортикоидов 3–4-го класса активности, регенерирующих средств. Широко применяются физиотерапевтические методы лечения, такие как ПУВА и УФА-терапия; имеются сообщения об успешном применении комбинации иммуносупрессивного микофенолата мофетила и экстракорпорального фотофереза, а также внутривенных инфузий N-ацетилцистеина. Помимо традиционных лекарственных и физиотерапевтических способов терапии в настоящее время рассматривается вопрос применения биологической терапии.

В статье представлен редкий случай буллёзной склеродермии на фоне выраженной хронической эндокринной патологии. Интерес данного клинического случая заключается не только в редкой форме заболевания и прогрессирующем характере течения дерматоза, но и вероятной ассоциации дерматоза с имеющимися у пациентки заболеваниями эндокринных желёз в виде пангипопитуитаризма вследствие макроаденомы аденогипофиза с развитием вторичного гипотиреоза и надпочечниковой недостаточности. В нашем наблюдении обращала на себя внимание неуклонная отрицательная динамика кожного процесса — от локализованной бляшечной склеродермии с последующей трансформацией в буллёзную форму, а далее в вероятный системный процесс.

Учитывая тяжесть течения заболевания, наличие сопутствующей хронической эндокринопатии, а также выявленные положительные специфические серологические реакции для системной формы склеродермии, в представленном клиническом случае пациентке необходим междисциплинарный подход и постоянное диспансерное наблюдение.

Ключевые слова: склеродермия буллёзная; этиология; диагностика и лечение.

Как цитировать:
Снарская Е.С., Теплюк Н.П., Шнахова Л.М., Мышлянова Д.А., Семиклет Ю.М. Клинический случай буллёзной склеродермии у соматически отягощённой пациентки // Российский журнал кожных и венерических болезней. 2024. Т. 27, № 4. С. 435–447. DOI: https://doi.org/10.17816/dv634008

Рукопись получена: 02.07.2024 **Рукопись одобрена:** 31.07.2024 **Опубликована online:** 25.08.2024

BACKGROUND

Bullous scleroderma is a rare variant of localized scleroderma characterized by the appearance of subepidermal tense blisters within plaques of scleroderma. When these blisters rupture, they reveal an extensive eroded surface [1–3]. This form of dermatosis was first described by P.A. Morrow in 1896 [4].

Cases of bullous scleroderma are extremely rare: as of 2012, not more than 100 cases were reported in the literature worldwide. This form accounts for 1.4 to 7.5% of all cases of localized scleroderma [5, 6].

The etiology and pathogenesis of bullous scleroderma remain debatable [7]. A number of authors have identified obstruction of cutaneous lymphatic vessels in the context of the sclerotic process in the dermis as a key factor in blister formation [8], which explains the most frequent localization of eruptions on the skin of the lower limbs [9]. However, this theory does not explain the formation of bullous elements in the absence of lymphatic vessel obstruction and with subsequent lymphangiectasia on histopathological examination of a skin biopsy [7, 10]. However, as early as in 2002, Rencic et al. [6] identified four main pathogenetic mechanisms for the formation of bullous elements in bullous scleroderma: subepidermal edema, secondary lymphangiectasia associated with dermal fibrosis, obstruction of cutaneous lymphatic vessels, and immune-mediated damage to the dermoepidermal junction. Fernandez-Flores et al. [7] described three cases of bullous scleroderma, highlighting mechanical trauma as the leading trigger for the onset of the dermatosis. Also, a number of authors have observed an association between certain autoimmune diseases and endocrine pathology [11, 12]. Thus, in a 2024 study, not only was a link established between thyroid diseases inducing hypothyroidism and scleroderma, but an inverse correlation was also found between thyroid hormone levels and the activity of the autoimmune process [13]. Wu et al. [14] in their systematic review described an increase in prolactin produced by the pituitary in patients with scleroderma compared to the control group. In 2008, a clinical case of scleroderma with erosive-ulcerative skin lesions associated with panhypopituitarism due to probable impaired blood supply to the pituitary vessels was described for the first time in a 53-year-old female patient [15]. Isolated cases of bullous scleroderma in the setting of graft-versus-host disease [16, 17], radiotherapy [18], medications, and other autoimmune conditions have also been described [19–22].

The clinical presentation of bullous scleroderma is a typical pinkish-brown or greyish-whitish plaque ranging from 5 to 20 cm in diameter, surrounded by a rim of erythema, on the surface of which blisters with clear, less often serous or hemorrhagic contents, are located [3, 6, 7, 23]. When the blisters rupture, an extensive eroded surface with poorly defined borders around the ruptured bullous element is revealed [24]. The scarring process is extremely complex, often with secondary infection, subsequent atrophy, and persistent skin hyperpigmentation [3]. Typical subjective sensations are pain, a feeling of tightness, and less often itching in the area of the eruptions [3].

The histological pattern of bullous scleroderma consists in thickening of collagen fiber bundles and hyalinization, dilation of the cutaneous lymphatic vessels, and the formation of subepidermal blisters at the level of the lamina densa. In the epidermis, atrophy is observed, with possible vacuolization of basal keratinocytes and infiltration of the dermoepidermal junction by lymphocytes and plasma cells [1, 25].

Laboratory diagnosis of bullous scleroderma includes complete blood count, clinical chemistry test, and clinical urinalysis [26, 27]. Additional patient examination methods involve determining the levels of antinuclear factor, anti-topoisomerase I antibodies, anticentromere antibodies, and anti-RNA polymerase III antibodies to exclude systemic involvement [28–30], as well as testing for anti-Borrelia antibodies [31, 32]. Instrumental diagnostic methods include computed tomography and magnetic resonance imaging to exclude deeper tissue involvement [33, 34], dermatoscopy as a method for assessing the degree of process activity [35], and pathological examination of biopsy material [36].

The diagnosis of bullous scleroderma is made based on the patient's history, clinical presentation of the disease, and histological examination findings. Patients are recommended to undergo a thorough examination and consult a rheumatologist to rule out systemic scleroderma and other connective tissue diseases. When cosmetic defects form, a consultation with a plastic surgeon is conducted to determine the possibility of surgical correction [30].

During diagnostics, bullous scleroderma should be differentiated from the bullous form of lupus erythematosus, pemphigus vulgaris, and bullous pemphigoid of Lever (Table 1) [1, 3, 7, 25, 37–39].

Treatment of the bullous form of scleroderma should be comprehensive and involve multiple courses, including systemic glucocorticoids, antibacterial and cytostatic agents, medications that improve microcirculation, as well as topical glucocorticoids of potency classes III–IV and regenerative agents [6, 40, 41]. Non-pharmacological treatment methods are widely used, such as PUVA (psoralen + ultraviolet A) and UVA therapy [6, 42]. In 2008, Schlaak et al. [43] reported successful use of a combination of the immunosuppressive drug mycophenolate mofetil at a dose of 2 g/day and extracorporeal photopheresis in a patient with extensive bullous and erosive-ulcerative skin lesions. Successful treatment of a skin ulcerative defect in a patient with bullous scleroderma using intravenous infusions of N-acetylcysteine at a dose of 15 mg/kg per hour over 5 hours daily for 14 days has been described [44]. In addition to conventional pharmacological and non-pharmacological methods, the use of biologics is currently being considered [45].

Table 1. Differential diagnosis of scleroderma bullosa

Diagnosis	Scleroderma bullosa	Lupus erythematosus bullosa	Vulgar vesicles	Lever's bullous pemphigoid
Age, years	40–50	Young people	40–60	⩾70–80
Gender	More common in women	More common in women	Similar incidence	Similar incidence
Localisation of rashes	More often affecting the lower limbs, but can be generalized	Generalized	Mucous membranes, generalized distribution on the skin	More commonly generalized
Clinical picture	Ivory-colored plaques, on the surface of which are blisters filled with clear or hemorrhagic fluid. When these blisters rupture, an eroded surface is revealed with indistinct borders at the periphery of the ruptured bulla	Tense vesiculobullous lesions on normal or erythematous skin, tending to group. Erosions that form upon blister rupture leave prolonged depigmentation after epithelialization	On mucous membranes: mild solitary blisters on apparently unchanged mucous membranes, rapidly transforming into erosions. On the skin: blisters on unchanged skin with serous contents	Tense blisters in the setting of edematous erythema with serous or serous-hemorrhagic content. After the blisters rupture, erosions rapidly epithelialize, forming yellow-brown crusts
Subjective symptoms	Tenderness and a feeling of tightness	Often pruritus	Burning, tenderness	Pruritus, burning
Pathohistology	In the dermis: thickening of collagen fibers, hyalinization, and dilation of lymphatic vessels at the level of the lamina densa. In the epidermis: atrophy and vacuolization of basal cells	In the dermis: neutrophilic microabscesses in dermal papillae, basophilic degeneration of collagen, mucinosis, perivascular infiltrates. In the epidermis: vacuolization of basal cells	Intraepidermal blisters, basal cells tightly attached to the basement membrane ("tombstone appearance"). Acantholytic cells may be present in the blister cavity	In the dermis: inflammatory infiltrate consisting of eosinophilic and neutrophilic leukocytes, subepidermal blister
Specific studies	Antinuclear factor (HEp-2), anti-topoisomerase I antibodies (anti-Scl 70), anticentromere antibodies, anti-RNA polymerase III antibodies, and antibodies to Borrelia burgdorferi	Antinuclear antibodies, anti-extractable nuclear antigens antibodies, anti-native DNA antibodies, anti-cytoplasmic antigens antibodies (SS-A, SS-B), antiphospholipid antibodies, anti-cardiolipin antibodies, anti-nucleosome antibodies, lupus anticoagulant, complement component testing (C3, C4), anti- collagen type VII antibodies	Circulating anti-desmoglein 3 and anti-desmoglein 1 antibodies (ELISA), anti-130 kDa and anti-160 kDa antibodies (immunoblot), "fish-scale" antibody deposition around keratinocytes in the spinous layer of the epidermis (direct IF)	Circulating antibodies (indirect IF), anti-BP180 and BP230 antibodies (ELISA), anti-180 kDa and anti-230 kDa antibodies (immunoblot), antibody deposition along the lamina lucida (direct IF)

Note. ИФА (ELISA) — enzyme-linked immunosorbent assay; РИФ — immunofluorescence reaction.

DOI: https://doi.org/10.17816/dv634008

CASE DESCRIPTION

Patient information

Patient T., a 48-year-old woman, presented with complaints of extensive eruptions located on the skin of the face, trunk, upper and lower extremities, accompanied by tenderness, a feeling of tightness, and pruritus.

History. The patient considered herself unwell from March 2019, when, following an injection of an ibandronic acid product, initial eruptions appeared on the abdominal skin accompanied by pruritus. Upon consulting a dermatologist in her locality, a diagnosis of allergic dermatitis was made, and she was prescribed dexamethasone at a dose of 4 mg twice intramuscularly, antihistamines, and topical glucocorticoids. On therapy, a slight positive response was noted, as evidence by a reduced erythema intensity of the eruptions. However, by October–November 2019, the patient observed a spread of eruptions in the form of bluish-burgundy patches on the trunk and both upper and lower limbs, and reported a sensation of tightness in the affected areas.

In December 2019, upon a follow-up visit to the dermatologist, a clinical diagnosis of scleroderma was first established, and a consultation with a rheumatologist was recommended. Following laboratory investigations for antinuclear antibodies (HEp-2, titer: 1:640 sp), a systemic pathological process was excluded, and active topical therapy with glucocorticoids and calcineurin inhibitors was advised.

Throughout 2020, the dermatitis continued to progress steadily amid the unfavorable epidemiological situation (COVID-19 pandemic). The patient did not receive vaccination and contracted COVID-19, without complications.

In November–December 2021, the patient noted the appearance of bullae on the existing lesions, which ruptured when damaged by clothing, resulting in moist erosions. She self-administered antihistamines, gently applied Fucorcin topically to the eroded areas, and used Solcoseryl gel.

In March 2022, due to the extent, progression, and severity of the skin condition, she was hospitalized at the V.A. Rakhmanov Clinic of Dermatology and Venereology, where a diagnosis of "localized scleroderma, bullous form" was established. Systemic glucocorticoid therapy with prednisolone at 40 mg/day was initiated, followed by dose tapering with appropriate corrective therapy, as well as antibacterial therapy. During therapy, a marked positive response was observed, including regression of bullous eruptions and epithelialization of erosions. No evidence of systemic involvement was detected upon further evaluation.

Since December 2022, the patient has experienced another exacerbation of the dermatitis, which occurred following mechanical trauma to the temporal region of the head. Subsequently, she noted an increase in the lesion in the temporal area to 3 cm in diameter, as well as the appearance of new lesions and enlargement of existing lesions on the trunk and limbs.

Comorbidities: pituitary macroadenoma for which non-radical adenectomy was conducted in 2011; panhypopituitarism with secondary hypothyroidism and adrenal insufficiency; secondary hypogonadism; amenorrhea; osteoporosis.

Allergic history: Non-contributory.

Physical, laboratory, and instrumental findings

Status localis: The skin involvement is extensive and of a chronic inflammatory nature, presenting multiple lesions localized on the temporal regions, neck, abdominal skin, lower back, and thighs.

Eruptions on the skin of the left temporal region are represented by pink erosions with rounded contours, surrounded by yellow-brown crusts (Figure 1). The eruptions on the abdominal skin extend to the skin of the mammary glands and present as pink-red and greyish-white extensive areas with rounded shapes and blurred borders, measuring up to 15–20 cm in diameter. The lesion surface displays isolated bullous elements with flaccid upper layer, containing clear and serous-hemorrhagic fluid, alternating with reddish erosive areas ranging from 1 to 10 cm in diameter, the majority of which are covered with large dark brown crusts (Figure 2). On the skin of the lateral surfaces of the thighs and partially on the buttocks and sacral region, eruptions are represented by white-pink plaques, on the surface of which there are red erosions measuring up to 5 cm in diameter with dark brown crusts at the periphery of the lesions (Figures 3, 4). On the

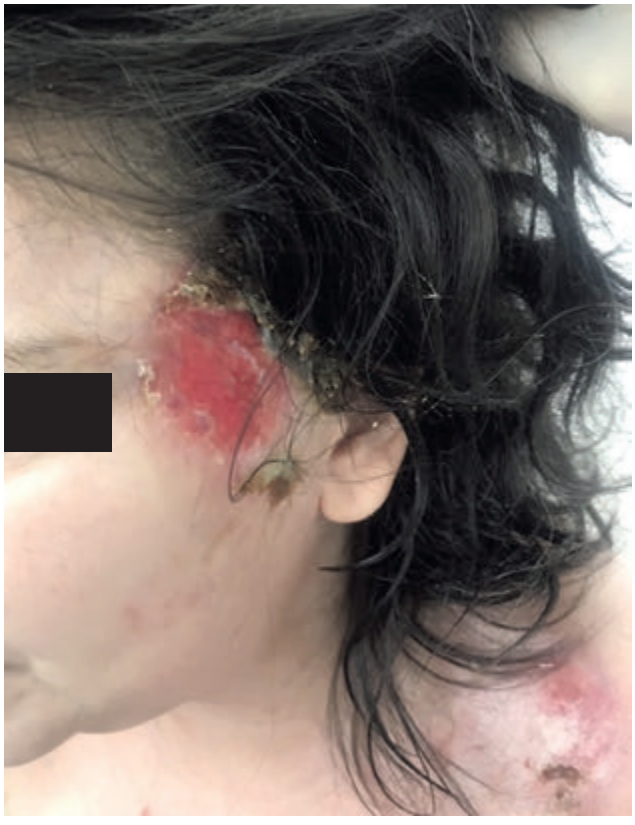

Fig. 1. Bullous scleroderma: foci on the skin of the left temporal region.

DOI: https://doi.org/10.17816/dv634008

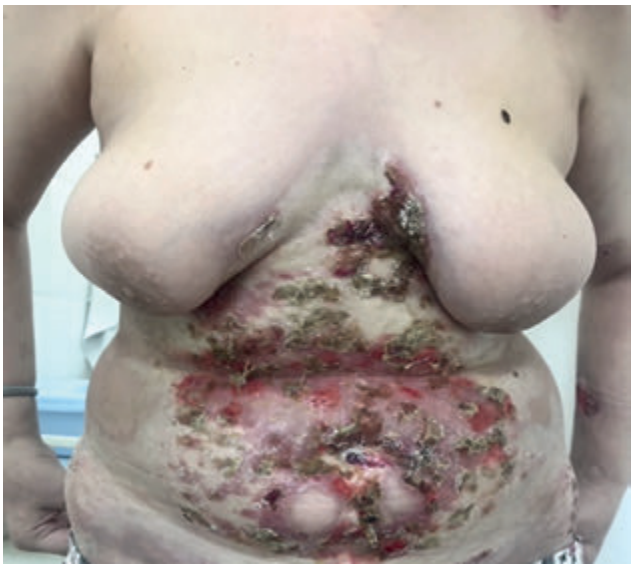

Fig. 2. Multiple foci on the skin of the abdomen and mammary glands (general view).

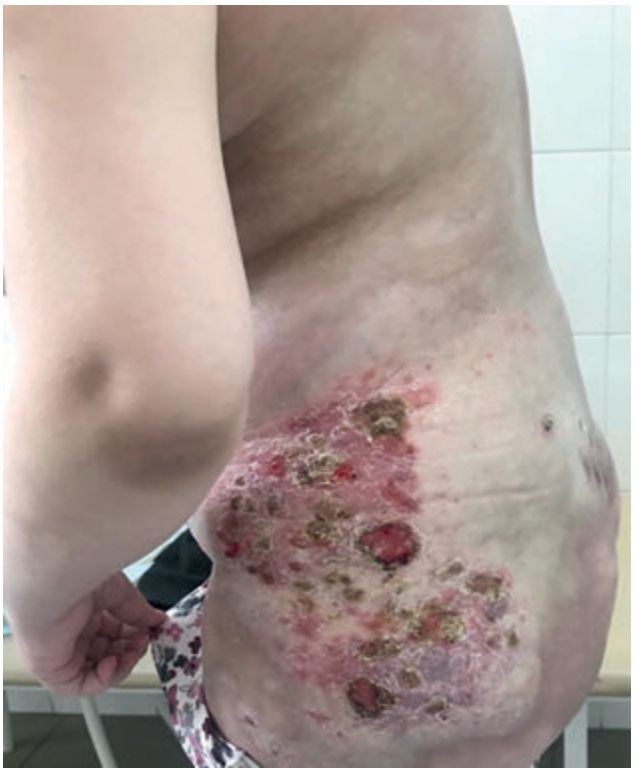

Fig. 3. Multiple foci on the skin of the left lateral surface of the trunk.

skin of the anterior surfaces of both thighs, there are linear elongated plaques with blurred borders, on which areas of greyish atrophy, burgundy erosions, and large greyish-brown crusts alternate (Figure 5). On the neck, symmetrical plaques of rounded and irregular shapes with clear borders are observed. The plaque on the right side of the neck is white with a smooth, shiny surface measuring up to 12 cm in diameter (Figure 6, *a*). The plaque on the left side of the neck is whitish-pink with isolated brown crusts on the surface (Figure 6, *b*).

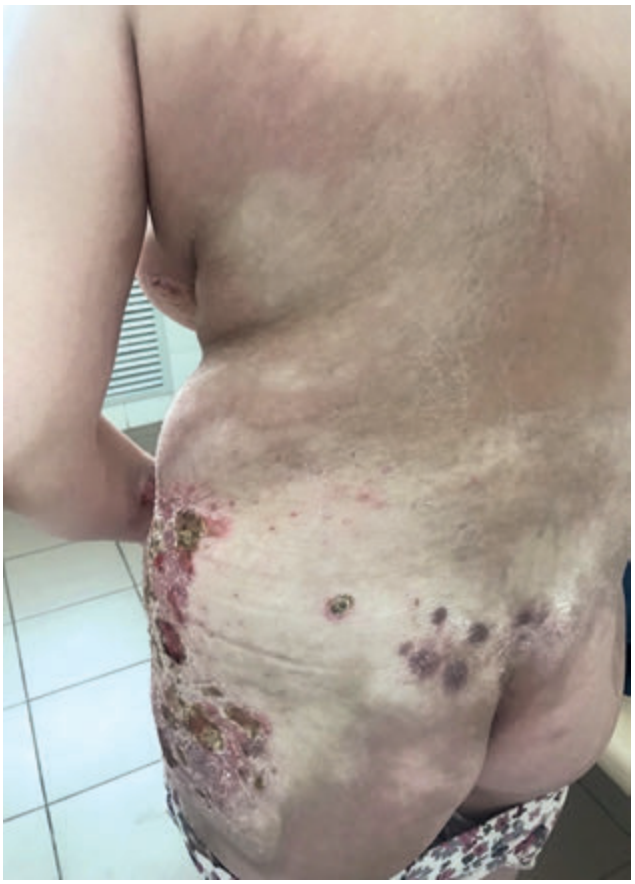

Fig. 4. Foci in the sacral region.

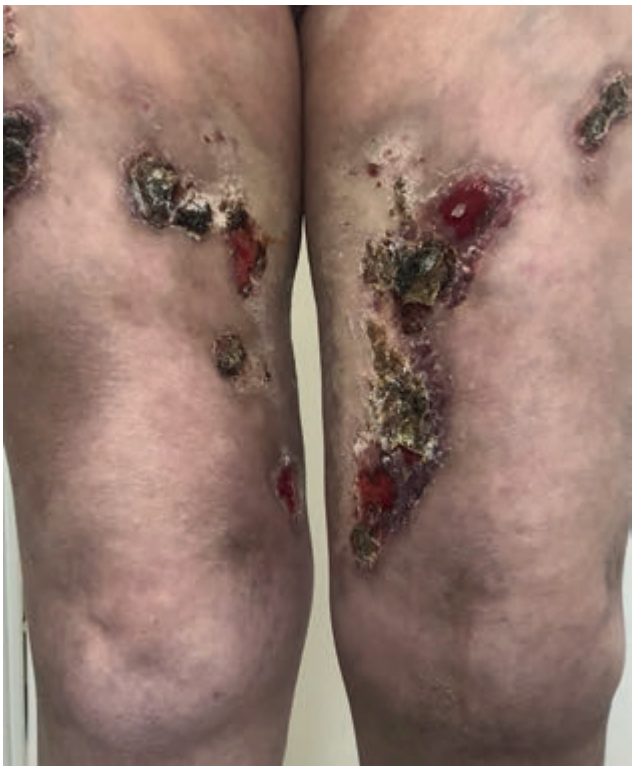

Fig. 5. Multiple foci on the anterior surface of the thighs.

Subjectively, the patient reports a feeling of tightness, tenderness, and itching.

DOI: https://doi.org/10.17816/dv634008

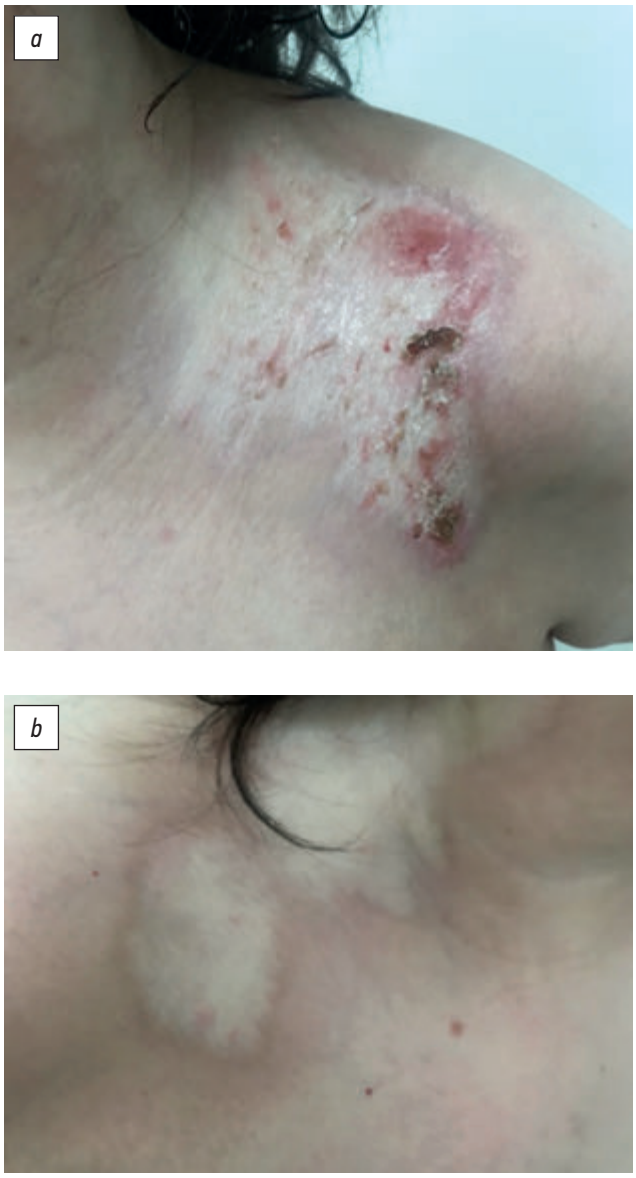

Fig. 6. Skin foci on the left shoulder (*a*) and right shoulder and neck (*b*).

Laboratory investigations: Complete blood count: erythrocytes, 4.65×10^12/l*;* hemoglobin, 139 g/l*;* hematocrit, 42%*;* platelets, 381×10^9/l*;* leucocytes, 7.74×10^9/l*;* lymphocytes, 33.4%*;* neutrophils, 59%*;* monocytes, 2.3%; eosinophils, 4.3%*;* basophils, 1.1%*;* erythrocyte sedimentation rate, 14 mm/h*;* color index, 0.9*;* clinical chemistry test: glucose, 5.86 mmol/l; total protein, 74 g/l; albumin, 46.1 g/l; urea, 4.5 mmol/l; uric acid, 399.50 μmol/l; creatinine, 82 μmol/l; total bilirubin, 17.9 μmol/l; direct bilirubin, 3 μmol/l; cholesterol, 5.7 mmol/l; triglycerides, 0.91 mmol/l; high-density lipoproteins, 1.6 mmol/l; alanine aminotransferase, 62 u/l; aspartate aminotransferase, 71 u/l; lactate dehydrogenase, 132 u/l; alkaline phosphatase, 138 u/l; gamma-glutamyl transferase, 156 u/l; c-reactive protein, 11.8 mg/l; rheumatoid factor, 165 u/ml; urinalysis, color, yellow; specific gravity, 1.021; transparency, slightly cloudy; ph, 5; protein, negative; glucose, negative; ketones, negative; nitrites, negative; urobilinogen, normal; leucocytes, 3–6 per field of view; erythrocytes, negative; mucus, slightly present; bacteria, slightly present; coagulation profile, activated partial thromboplastin time, 1.19; prothrombin (quick), 85%; international normalized ratio, 1.09; prothrombin time, 12 seconds; fibrinogen, 4.72 g/l; notably, there is a tenfold increase in rheumatoid factor levels, along with elevated liver transaminases, uric acid, cholesterol, and C-reactive protein.

Borrelia infection testing: IgG antibodies to *Borrelia burgdorferi*: negative;

Antinuclear factor (HEp-2), 1/640; anti-topoisomerase I antibodies (Scl-70), 54 IU/L; anticentromere antibodies, 12 IU/L;

Conclusion: A fourfold increase in antinuclear factor has been detected, along with elevated reference values for anti-DNA topoisomerase I antibodies and anticentromere antibodies, which may indicate a probable development of a systemic process.

Hormone blood testing: Adrenocorticotropic hormone, 5 pg/ml; cortisol, 38 nmol/l; thyroid-stimulating hormone, 0.1 miu/l; triiodothyronine, 0.23 nmol/l; follicle-stimulating hormone, 0.12 miu/ml, luteinizing hormone, 0.09 miu/ml; prolactin, 560 miu/l; estradiol, 27 pmol/l.

Conclusion: a total decrease in hormone levels produced by both the anterior pituitary and peripheral endocrine glands is observed, with a slight increase in prolactin reference values.

Endocrinologist consultation: Due to decompensated hypothyroidism and secondary adrenal insufficiency, replacement therapy with thyroid hormones (Euthyrox 150 mg in the morning) and adrenal cortex hormones (Cortef 5 mg in the morning and 2.5 mg in the evening) is recommended under the supervision of an endocrinologist at the place of residence and with monitoring of thyroid and adrenal hormone levels.

Histopathological examination: Histological examination of the skin flap biopsy revealed epidermal atrophy, focal vacuolization of basal keratinocytes, and edema of the papillary dermis, leading to the formation of subepidermal blisters. In the dermis, hyalinization and thickening of collagen bundles with subsequent sclerosis and dilation of lymphatic vessels were observed. Lymphoplasmacytic infiltrate was noted in the perivascular space and at the dermoepidermal junction (Fig. 7).

Conclusion: The morphological picture is consistent with the diagnosis of “localized scleroderma, bullous form.”

Diagnosis

Based on the clinical presentation of the dermatosis, results of histopathological examination, medical history and disease course, a rare form of localized scleroderma — bullous scleroderma — was diagnosed.

Treatment

Treatment included systemic glucocorticoids (prednisolone 40 mg daily with dose tapering) with adjuvant

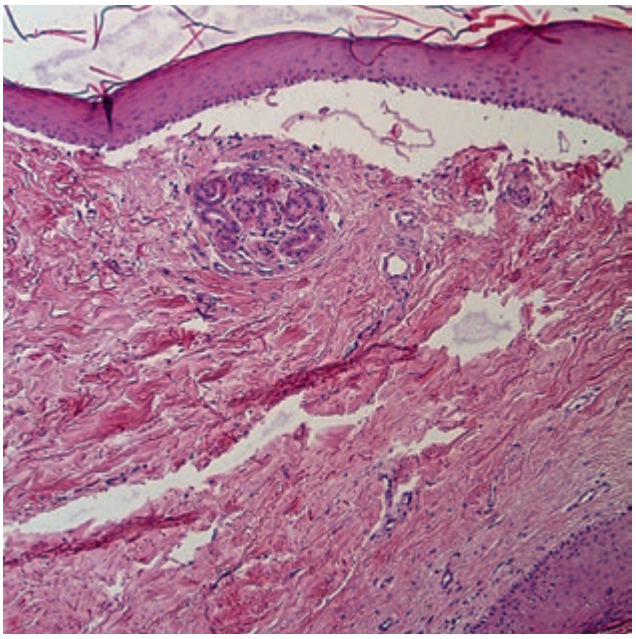

Fig. 7. Pathomorphological picture presents epidermal atrophy, focal vacuolisation of basal keratinocytes, swelling of the papillary layer of the dermis and subepidermal bullae.

therapy consisting of proton pump inhibitors, potassium, magnesium and calcium supplements combined with vitamin D; antibiotic therapy (ceftriaxone 1,000 mg once daily intramuscularly for 10 days); topical combined corticosteroids with antimicrobial component (Celestoderm with Garamycin) and tissue regeneration-promoting agents (methyluracil ointment applied to erosions and crusts twice daily).

Follow-up status

Following comprehensive therapy, the patient showed marked improvement in skin lesions, with epithelialization and scarring of erosions resulting in ivory-colored skin atrophy zones with irregular borders consistent with the size of bullous scleroderma lesions, and shedding of crusts from eroded areas (Figures 8–12). No new elements appeared. Subjective symptoms resolved.

Post-treatment blood tests for serological reactions showed a two-fold decrease in antinuclear factor (HEp-2 1/320), as well as normalization of anti-DNA topoisomerase I antibodies (17 IU/mL) and anticentromere antibodies (8 IU/mL).

The patient was recommended follow-up with a rheumatologist for regular monitoring of specific antibody levels, as well as endocrinologist follow-up to monitor pituitary and peripheral endocrine gland hormone levels for appropriate replacement therapy.

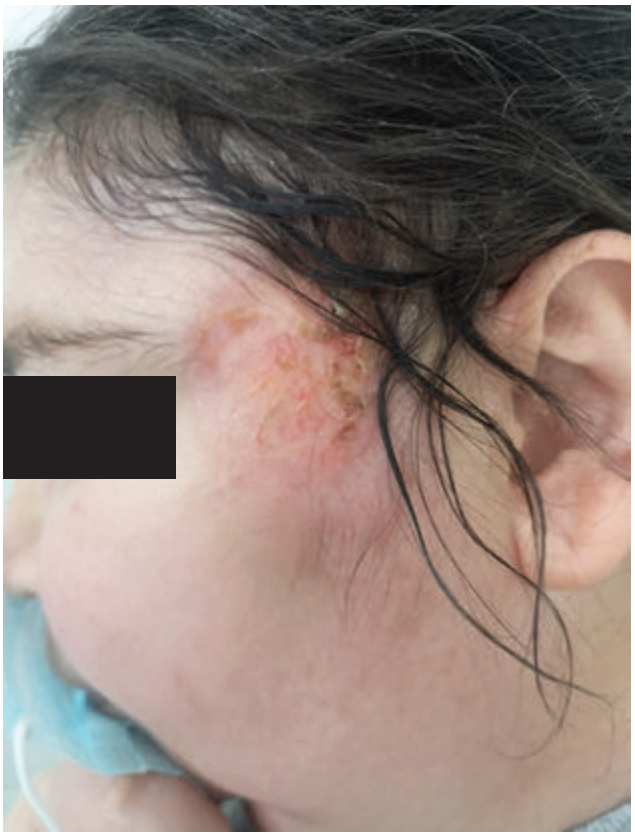

Fig. 8. Exodus of the nidus into skin atrophy with telangiectasia on the surface and single light brown crusts.

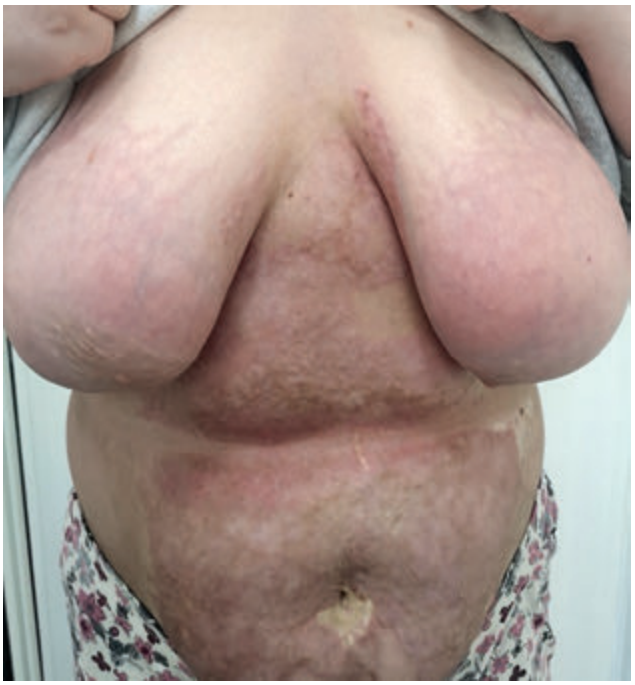

Fig. 9. Exodus of multiple foci on the abdomen and mammary glands into widespread skin atrophy with irregular edges; presence of areas of hyperpigmentation.

DISCUSSION

This article presents a rare clinical case of bullous scleroderma in the context of significant chronic endocrine pathology. The interest of this case lies not only in the rare form of the disease and progressive nature of the dermatosis, but also in the probable association with the patient's endocrine disorders, namely panhypopituitarism due to

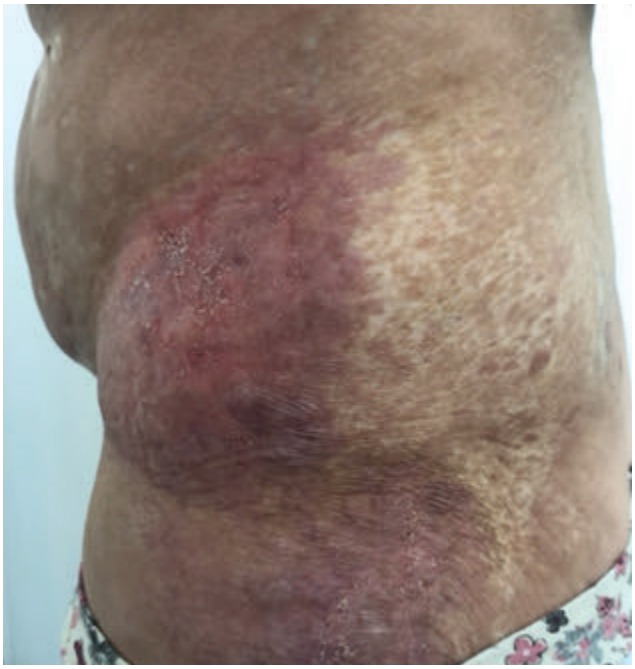

Fig. 10. Exodus of foci on the skin of the left lateral surface of the torso.

pituitary macroadenoma with secondary hypothyroidism and adrenal insufficiency.

Our observation highlights the steady progression of the skin condition from localized plaque scleroderma to bullous form, and potentially to a systemic process. According to several studies, decompensated endocrine pathology exacerbates the course and prognosis of scleroderma.

Thus, considering the severity of the disease, presence of concomitant chronic endocrinopathy, and positive specific serological reactions for systemic scleroderma, this case requires an interdisciplinary approach and continuous follow-up.

CONCLUSION

Bullous scleroderma is a relatively rare phenomenon. Increased awareness among clinicians about the bullous form of localized scleroderma will contribute to timely diagnosis, detection of systemic features, and appropriate interdisciplinary management of patients.

ADDITIONAL INFORMATION

Funding source. This study was not supported by any external sources of funding.

Competing interests. The authors declare that they have no competing interests.

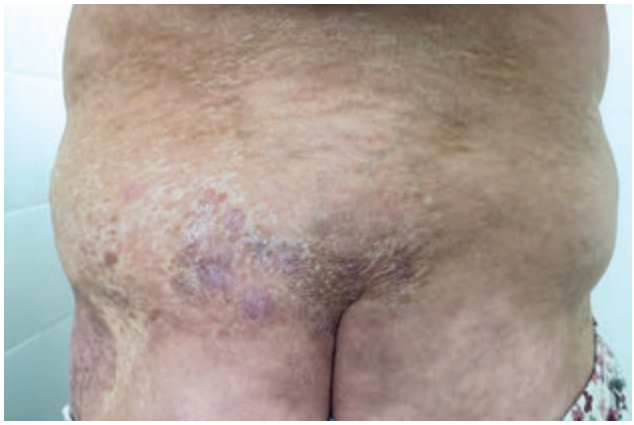

Fig. 11. Exodus of foci on the skin in the sacral region.

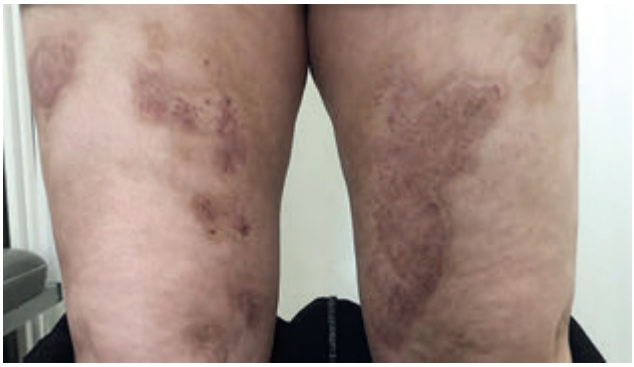

Fig. 12. Exodus of foci on the skin of the lateral inner surface of the thighs into atrophy and hyperpigmentation along the contour of the area of skin atrophy.

Authors' contribution. All authors made a substantial contribution to the conception of the work, acquisition, analysis, interpretation of data for the work, drafting and revising the work, final approval of the version to be published and agree to be accountable for all aspects of the work. E.S. Snarskaya, N.P. Teplyuk — concept and design of the study, making significant (important) corrections to the manuscript in order to increase the scientific value of the work, writing the article, approval of the final manuscript; L.M. Shnakhova — making significant (important) corrections to the manuscript in order to increase the scientific value of the work, writing the article, approving the final manuscript; D.A. Myshlyanova — acquisition, analysis of data and interpretation of results, writing of the article, approval of the final version; Y.M. Semiklet — obtaining, analyzing data and describing a clinical case, writing an article, approving the final version.

Consent for publication. The patient voluntarily signed informed consent for the publication of personal medical information in anonymized form in the journal "Russian Journal of Skin and Venereal Diseases".

REFERENCES

1. Lahouel M, Soua Y, Njim L, et al. An unusual presentation of localized bullous morphea. *Dermatol Online J.* 2020;26(6):13030/qt6hm3k4g9
2. Bakulev AL, Galkina EM, Karakaeva AV, Litvinenko MV. A case of localized bullous scleroderma. *Vestnik dermatologii i venerologii.* 2016;92(3):97–101. EDN: WGBBOP doi: 10.25208/0042-4609-2016-92-3-97-101
3. *Illustrated guide to dermatology. For preparation of doctors for accreditation*. Ed. by O.Y. Olisova, N.P. Teplyuk. Moscow: GEOTAR-Media; 2023. 376 p. (In Russ). doi: 10.33029/9704-7375-7-DER-2023-1-376
4. Jia H, Chen XH, Shi JH, et al. A case of bullous morphea reported in mainland China. *Int J Dermatol.* 2002;41(12):949–950. EDN: BEXCVN doi: 10.1046/j.1365-4362.2002.16672.x
5. Venturi M, Pinna AL, Pilloni L, et al. Bullous morphoea: A retrospective study. *Clin Exp Dermatol.* 2017;42(5):532–535. doi: 10.1111/ced.13127
6. Rencic A, Goyal S, Mofid M, et al. Bullous lesions in scleroderma. *Int J Dermatol.* 2002;41(6):335–339. EDN: EUHWSB doi: 10.1046/j.1365-4362.2002.01360.x
7. Fernandez-Flores A, Gatica-Torres M, Tinoco-Fragoso F, et al. Three cases of bullous morphea: Histopathologic findings with implications regarding pathogenesis. *J Cutan Pathol.* 2015;42(2):144–149. doi: 10.1111/cup.12418
8. Kavala M, Zindanci I, Demirkesen C, et al. Intertriginous bullous morphea: A clue for the pathogenesis? *Indian J Dermatol Venereol Leprol.* 2007;73(4):262–264. doi: 10.4103/0378-6323.33640
9. Daoud MS, Su WP, Leiferman KM, Perniciaro C. Bullous morphea: Clinical, pathologic, and immunopathologic evaluation of thirteen cases. *J Am Acad Dermatol.* 1994;30(6):937–943. doi: 10.1016/s0190-9622(94)70113-x
10. Ahn SK, Won JH, Lee SH, et al. 'Swiss cheese' lymphangiectasia in a case of solitary morphea profunda. *J Dermatol.* 1997;24(10):666–669. doi: 10.1111/j.1346-8138.1997.tb02313.x
11. Chakravarty SD, Markenson JA. Rheumatic manifestations of endocrine disease. *Curr Opin Rheumatol.* 2013;25(1):37–43. doi: 10.1097/BOR.0b013e32835b4f3f
12. Markenson JA. Rheumatic manifestations of endocrine diseases. *Curr Opin Rheumatol.* 2010;22(1):64–71. doi: 10.1097/BOR.0b013e328333dd38
13. Cherim A, Petca RC, Dumitrascu MC, et al. Thyroid disorders in systemic sclerosis: A comprehensive review. *J Clin Med.* 2024;13(2):415. EDN: RJURFZ doi: 10.3390/jcm13020415
14. Wu Y, Li ML, Han HJ, et al. Elevated plasma/serum levels of prolactin in patients with systemic sclerosis: A systematic review and meta-analysis. *Medicine (Baltimore).* 2020;99(38):e22239. doi: 10.1097/MD.0000000000022239
15. Katsantonis JC, Protopsaltis J, Kokkoris S, et al. Systemic sclerosis associated with generalized vasculitis and hypopituitarism. *Rheumatol Int.* 2008;28(11):1165–1168. doi: 10.1007/s00296-008-0586-8
16. Moreno JC, Valverde F, Martinez F, et al. Bullous scleroderma-like changes in chronic graft-versus-host disease. *J Eur Acad Dermatol Venereol.* 2003;17(2):200–203. EDN: BGSQMB doi: 10.1046/j.1468-3083.2003.00606.x
17. Jachiet M, de Masson A, Peffault de Latour R, et al. Skin ulcers related to chronic graft-versus-host disease: Clinical findings and associated morbidity. *Br J Dermatol.* 2014;171(1):63–68. doi: 10.1111/bjd.12828
18. Petersen E, Yazdani L, Hymes SR. A case of radiation-induced bullous morphea/lichen sclerosus overlap in a breast cancer patient. *Rep Pract Oncol Radiother.* 2018;23(1):47–49. doi: 10.1016/j.rpor.2017.11.004
19. Wooten MD, Scott JW, Miller AM, Boh E. Chronic myelogenous leukemia and porphyria cutanea tarda in a patient with limited systemic sclerosis. *South Med J.* 1998;91(5):493–495. EDN: ENLPOP doi: 10.1097/00007611-199805000-00019
20. Wu KH, Dai YS, Tsai MJ, et al. Lichen sclerosus et atrophicus, bullous morphea, and systemic lupus erythematosus: A case report. *J Microbiol Immunol Infect.* 2000;33(1):53–56.
21. Iskandarli M, Gerceker Turk B, Yaman B, Unal I. Rare association of morphea with vesicular pemphigoid. *G Ital Dermatol Venereol.* 2016;151(5):562–563.
22. Maglie R, Baffa ME, Montefusco F, et al. Case report: Bullous pemphigoid associated with morphea and lichen sclerosus: Coincidental diseases or pathogenetic association? *Front Immunol.* 2022;13:887279. doi: 10.3389/fimmu.2022.887279.
23. Sánchez-Pérez S, Escandell-González I, Pinazo-Canales MI, Jordá-Cuevas E. [Bullous morphea: Description of a new case and discussion of etiologic and pathogenic factors in bulla formation. (In Spanish)]. *Acta Dermosifiliogr.* 2017;108(1):75–76. doi: 10.1016/j.ad.2016.07.010.
24. Su WP, Greene SL. Bullous morphea profunda. *Am J Dermatopathol.* 1986;8(2):144–147. doi: 10.1097/00000372-198604000-00009
25. Samtsov AV, Belousova IE. *Bullous dermatoses: monograph*. Saint Petersburg: Kosta; 2012. 144 p. (In Russ).
26. Gorkiewicz-Petkow A, Kalinska-Bienias A. Systemic involvement in localized scleroderma/morphea. *Clin Dermatol.* 2015;33(5):556–562. doi: 10.1016/j.clindermatol.2015.05.009
27. Arkachaisri T, Fertig N, Pino S, Medsger TA. Serum autoantibodies and their clinical associations in patients with childhood- and adultonset linear scleroderma. A single-center study. *J Rheumatol.* 2008;35(12):2439–2444. doi: 10.3899/jrheum.080098
28. Krieg T, Takehara K. Skin disease: A cardinal feature of systemic sclerosis. *Rheumatology (Oxford).* 2009;48(3):14–18.
29. Desbois AC, Cacoub P. Systemic sclerosis: An update in 2016. *Autoimmun Rev.* 2016;15(5):417–426. EDN: WPTHQP doi: 10.1016/j.autrev.2016.01.007
30. Van den Hoogen F, Khanna D, Fransen J, et al. 2013 Classification criteria for systemic sclerosis: An American College of Rheumatology / European League against Rheumatism collaborative initiative. *Arthritis Rheum.* 2013;72(11):1747–1755. doi: 10.1136/annrheumdis-2013-204424
31. Xie CB, Cowper S, Odell ID. Morphea after Borrelia-induced facial nerve palsy. *J Scleroderma Relat Disord.* 2021;6(1):111–113. doi: 10.1177/2397198320956892
32. Șandru F, Popa A, Petca A, et al. Etiologic role of Borrelia burgdorferi in morphea: A case report. *Exp Ther Med.* 2020;20(3):2373–2376. doi: 10.3892/etm.2020.8815

33. Fett N, Werth VP. Update on morphea: Part II. Outcome measures and treatment. *J Am Acad Dermatol.* 2011;64(2):231–242. doi: 10.1016/j.jaad.2010.05.046
34. Schanz S, Fierlbeck G, Ulmer A, et al. Localized scleroderma: MR findings and clinical features. *Radiology.* 2011;260(3):817–824. doi: 10.1148/radiol.11102136
35. Florez-Pollack S, Kunzler E, Jacobe HT. Morphea: Current concepts. *Clin Dermatol.* 2018;36(4):475–486. doi: 10.1016/j.clindermatol.2018.04.005
36. Onajin O, Wieland CN, Peters MS, et al. Clinicopathologic and immunophenotypic features of eosinophilic fasciitis and morphea profunda: A comparative study of 27 cases. *J Am Acad Dermatol.* 2018;78(1):121–128. doi: 10.1016/j.jaad.2017.06.148
37. Sprow G, Afarideh M, Dan J, et al. Bullous systemic lupus erythematosus in females. *Int J Womens Dermatol.* 2022;8(3):e034. EDN: AIPDXW doi: 10.1097/JW9.0000000000000034
38. Malik AM, Tupchong S, Huang S, et al. An updated review of pemphigus diseases. *Medicina (Kaunas).* 2021;57(10):1080. doi: 10.3390/medicina57101080
39. Pratasava V, Sahni VN, Suresh A, et al. Bullous pemphigoid and other pemphigoid dermatoses. *Medicina (Kaunas).* 2021;57(10):1061. doi: 10.3390/medicina57101061
40. Yasar S, Mumcuoglu CT, Serdar ZA, Gunes P. A case of lichen sclerosus et atrophicus accompanying bullous morphea. *Ann Dermatol.* 2011;23(Suppl 3):S354–359. doi: 10.5021/ad.2011.23.S3.S354
41. Micalizzi C, Parodi A, Rebora A. Generalized bullous morphoea. Efficacy of salazopyrin. *Clin Exp Dermatol.* 1996;21(3):246–247. doi: 10.1111/j.1365-2230.1996.tb00080.x
42. El-Mofty M, Zaher H, Bosseila M, et al. Low-dose broad-band UVA in morphea using a new method for evaluation. *Photodermatol Photoimmunol Photomed.* 2000;16(2):43. doi: 10.1034/j.1600-0781.2000.d01-1.x
43. Schlaak M, Friedlein H, Kauer F, et al. Successful therapy of a patient with therapy recalcitrant generalized bullous scleroderma by extracorporeal photopheresis and mycophenolate mofetil. *J Eur Acad Dermatol Venereol.* 2008;22(5):631–633. doi: 10.1111/j.1468-3083.2007.02403.x
44. Rosato E, Veneziano ML, di Mario A, et al. Ulcers caused by bullous morphea: Successful therapy with N-acetylcysteine and topical wound care. *Int J Immunopathol Pharmacol.* 2013;26(1):259–262. doi: 10.1177/039463201302600128
45. Martin LK, Kirsner RS. Ulcers caused by bullous morphea treated with tissue-engineered skin. *Int J Dermatol.* 2003;42(5):402–404. EDN: BGSCKH doi: 10.1046/j.1365-4362.2003.01763.x

СПИСОК ЛИТЕРАТУРЫ

1. Lahouel M., Soua Y., Njim L., et al. An unusual presentation of localized bullous morphea // Dermatol Online J. 2020. Vol. 26, N 6. P. 13030/qt6hm3k4g9
2. Бакулев А.Л., Галкина Е.М., Каракаева А.В., Литвиненко М.В. Случай локализованной буллезной склеродермии // Вестник дерматологии и венерологии. 2016. Т. 92, № 3. С. 97–101. EDN: WGBBOP doi: 10.25208/0042-4609-2016-92-3-97-101
3. Иллюстрированное руководство по дерматологии. Для подготовки врачей к аккредитации / под ред. О.Ю. Олисовой, Н.П. Теплюк. Москва: ГЭОТАР-Медиа, 2023. 376 с. doi: 10.33029/9704-7375-7-DER-2023-1-376
4. Jia H., Chen X.H., Shi J.H., et al. A case of bullous morphea reported in mainland China // Int J Dermatol. 2002. Vol. 41, N 12. P. 949–950. EDN: BEXCVN doi: 10.1046/j.1365-4362.2002.16672.x
5. Venturi M., Pinna A.L., Pilloni L., et al. Bullous morphoea: A retrospective study // Clin Exp Dermatol. 2017. Vol. 42, N 5. P. 532–535. doi: 10.1111/ced.13127
6. Rencic A., Goyal S., Mofid M., et al. Bullous lesions in scleroderma // Int J Dermatol. 2002. Vol. 41, N 6. P. 335–339. EDN: EUHWSB doi: 10.1046/j.1365-4362.2002.01360.x
7. Fernandez-Flores A., Gatica-Torres M., Tinoco-Fragoso F., et al. Three cases of bullous morphea: Histopathologic findings with implications regarding pathogenesis // J Cutan Pathol. 2015. Vol. 42, N 2. P. 144–149. doi: 10.1111/cup.12418
8. Kavala M., Zindanci I., Demirkesen C., et al. Intertriginous bullous morphea: A clue for the pathogenesis? // Indian J Dermatol Venereol Leprol. 2007. Vol. 73, N 4. P. 262–264. doi: 10.4103/0378-6323.33640
9. Daoud M.S., Su W.P., Leiferman K.M., Perniciaro C. Bullous morphea: Clinical, pathologic, and immunopathologic evaluation of thirteen cases // J Am Acad Dermatol. 1994. Vol. 30, N 6. P. 937–943. doi: 10.1016/s0190-9622(94)70113-x
10. Ahn S.K., Won J.H., Lee S.H., et al. 'Swiss cheese' lymphangiectasia in a case of solitary morphea profunda // J Dermatol. 1997. Vol. 24, N 10. P. 666–669. doi: 10.1111/j.1346-8138.1997.tb02313.x
11. Chakravarty S.D., Markenson J.A. Rheumatic manifestations of endocrine disease // Curr Opin Rheumatol. 2013. Vol. 25, N 1. P. 37–43. doi: 10.1097/BOR.0b013e32835b4f3f
12. Markenson J.A. Rheumatic manifestations of endocrine diseases // Curr Opin Rheumatol. 2010. Vol. 22, N 1. P. 64–71. doi: 10.1097/BOR.0b013e328333dd38
13. Cherim A., Petca R.C., Dumitrascu M.C., et al. Thyroid disorders in systemic sclerosis: A comprehensive review // J Clin Med. 2024. Vol. 13, N 2. P. 415. EDN: RJURFZ doi: 10.3390/jcm13020415
14. Wu Y., Li M.L., Han H.J., et al. Elevated plasma/serum levels of prolactin in patients with systemic sclerosis: A systematic review and meta-analysis // Medicine (Baltimore). 2020. Vol. 99, N 38. P. e22239. doi: 10.1097/MD.0000000000022239
15. Katsantonis J.C., Protopsaltis J., Kokkoris S., et al. Systemic sclerosis associated with generalized vasculitis and hypopituitarism // Rheumatol Int. 2008. Vol. 28, N 11. P. 1165–1168. doi: 10.1007/s00296-008-0586-8
16. Moreno J.C., Valverde F., Martinez F., et al. Bullous scleroderma-like changes in chronic graft-versus-host disease // J Eur Acad Dermatol Venereol. 2003. Vol. 17, N 2. P. 200–203. EDN: BGSQMB doi: 10.1046/j.1468-3083.2003.00606.x
17. Jachiet M., de Masson A., Peffault de Latour R., et al. Skin ulcers related to chronic graft-versus-host disease: Clinical findings and

associated morbidity // Br J Dermatol. 2014. Vol. 171, N 1. P. 63–68. doi: 10.1111/bjd.12828

18. Petersen E., Yazdani L., Hymes S.R. A case of radiation-induced bullous morphea/lichen sclerosus overlap in a breast cancer patient // Rep Pract Oncol Radiother. 2018. Vol. 23, N 1. P. 47–49. doi: 10.1016/j.rpor.2017.11.004

19. Wooten M.D., Scott J.W., Miller A.M., Boh E. Chronic myelogenous leukemia and porphyria cutanea tarda in a patient with limited systemic sclerosis // South Med J. 1998. Vol. 91, N 5. P. 493–495. EDN: ENLPOP doi: 10.1097/00007611-199805000-00019

20. Wu K.H., Dai Y.S., Tsai M.J., et al. Lichen sclerosus et atrophicus, bullous morphea, and systemic lupus erythematosus: A case report // J Microbiol Immunol Infect. 2000. Vol. 33, N 1. P. 53–56.

21. Iskandarli M., Gerceker Turk B., Yaman B., Unal I. Rare association of morphea with vesicular pemphigoid // G Ital Dermatol Venereol. 2016. Vol. 151, N 5. P. 562–563.

22. Maglie R., Baffa M.E., Montefusco F., et al. Case report: Bullous pemphigoid associated with morphea and lichen sclerosus: coincidental diseases or pathogenetic association? // Front Immunol. 2022. Vol. 13. P. 887279. doi: 10.3389/fimmu.2022.887279.

23. Sánchez-Pérez S., Escandell-González I., Pinazo-Canales M.I., Jordá-Cuevas E. [Bullous morphea: Description of a new case and discussion of etiologic and pathogenic factors in bulla formation. (In Spanish).] // Acta Dermosifiliogr. 2017. Vol. 108, N 1. P. 75–76. doi: 10.1016/j.ad.2016.07.010.

24. Su W.P., Greene S.L. Bullous morphea profunda // Am J Dermatopathol. 1986. Vol. 8, N 2. P. 144–147. doi: 10.1097/00000372-198604000-00009

25. Самцов А.В., Белоусова И.Э. Буллезные дерматозы: монография. Санкт-Петербург: Коста, 2012. 144 с.

26. Gorkiewicz-Petkow A., Kalinska-Bienias A. Systemic involvement in localized scleroderma/morphea // Clin Dermatol. 2015. Vol. 33, N 5. P. 556–562. doi: 10.1016/j.clindermatol.2015.05.009

27. Arkachaisri T., Fertig N., Pino S., Medsger T.A. Serum autoantibodies and their clinical associations in patients with childhood- and adultonset linear scleroderma. A single-center study // J Rheumatol. 2008. Vol. 35, N 12. P. 2439–2444. doi: 10.3899/jrheum.080098

28. Krieg T., Takehara K. Skin disease: A cardinal feature of systemic sclerosis // Rheumatology (Oxford). 2009. Vol. 48, N 3. P. 14–18.

29. Desbois A.C., Cacoub P. Systemic sclerosis: An update in 2016 // Autoimmun Rev. 2016. Vol. 15, N 5. P. 417–426. EDN: WPTHQP doi: 10.1016/j.autrev.2016.01.007

30. Van den Hoogen F., Khanna D., Fransen J., et al. 2013 Classification criteria for systemic sclerosis: An American College of Rheumatology / European League against Rheumatism collaborative initiative // Arthritis Rheum. 2013. Vol. 72, N 11. P. 1747–1755. doi: 10.1136/annrheumdis-2013-204424

31. Xie C.B., Cowper S., Odell I.D. Morphea after Borrelia-induced facial nerve palsy // J Scleroderma Relat Disord. 2021. Vol. 6, N 1. P. 111–113. doi: 10.1177/2397198320956892

32. Șandru F., Popa A., Petca A., et al. Etiologic role of Borrelia burgdorferi in morphea: A case report // Exp Ther Med. 2020. Vol. 20, N 3. P. 2373–2376. doi: 10.3892/etm.2020.8815

33. Fett N., Werth V.P. Update on morphea: Part II. Outcome measures and treatment // J Am Acad Dermatol. 2011. Vol. 64, N 2. P. 231–242. doi: 10.1016/j.jaad.2010.05.046

34. Schanz S., Fierlbeck G., Ulmer A., et al. Localized scleroderma: MR findings and clinical features // Radiology. 2011. Vol. 260, N 3. P. 817–824. doi: 10.1148/radiol.11102136

35. Florez-Pollack S., Kunzler E., Jacobe H.T. Morphea: Current concepts // Clin Dermatol. 2018. Vol. 36, N 4. P. 475–486. doi: 10.1016/j.clindermatol.2018.04.005

36. Onajin O., Wieland C.N., Peters M.S., et al. Clinicopathologic and immunophenotypic features of eosinophilic fasciitis and morphea profunda: A comparative study of 27 cases // J Am Acad Dermatol. 2018. Vol. 78, N 1. P. 121–128. doi: 10.1016/j.jaad.2017.06.148

37. Sprow G., Afarideh M., Dan J., et al. Bullous systemic lupus erythematosus in females // Int J Womens Dermatol. 2022. Vol. 8, N 3. P. e034. EDN: AIPDXW doi: 10.1097/JW9.0000000000000034

38. Malik A.M., Tupchong S., Huang S., et al. An updated review of pemphigus diseases // Medicina (Kaunas). 2021. Vol. 57, N 10. P. 1080. doi: 10.3390/medicina57101080

39. Pratasava V., Sahni V.N., Suresh A., et al. Bullous pemphigoid and other pemphigoid dermatoses // Medicina (Kaunas). 2021. Vol. 57, N 10. P. 1061. doi: 10.3390/medicina57101061

40. Yasar S., Mumcuoglu C.T., Serdar Z.A., Gunes P. A case of lichen sclerosus et atrophicus accompanying bullous morphea // Ann Dermatol. 2011. Vol. 23, Suppl 3. P. S354–359. doi: 10.5021/ad.2011.23.S3.S354

41. Micalizzi C., Parodi A. Rebora A. Generalized bullous morphoea. Efficacy of salazopyrin // Clin Exp Dermatol. 1996. Vol. 21, N 3. P. 246–247. doi: 10.1111/j.1365-2230.1996.tb00080.x

42. El-Mofty M., Zaher H., Bosseila M., et al. Low-dose broadband UVA in morphea using a new method for evaluation // Photodermatol Photoimmunol Photomed. 2000. Vol. 16, N 2. P. 43. doi: 10.1034/j.1600-0781.2000.d01-1.x

43. Schlaak M., Friedlein H., Kauer F., et al. Successful therapy of a patient with therapy recalcitrant generalized bullous scleroderma by extracorporeal photopheresis and mycophenolate mofetil // J Eur Acad Dermatol Venereol. 2008. Vol. 22, N 5. P. 631–633. doi: 10.1111/j.1468-3083.2007.02403.x

44. Rosato E., Veneziano M.L., di Mario A., et al. Ulcers caused by bullous morphea: Successful therapy with N-acetylcysteine and topical wound care // Int J Immunopathol Pharmacol. 2013. Vol. 26, N 1. P. 259–262. doi: 10.1177/039463201302600128

45. Martin L.K., Kirsner R.S. Ulcers caused by bullous morphea treated with tissue-engineered skin // Int J Dermatol. 2003. Vol. 42, N 5. P. 402–404. EDN: BGSCKH doi: 10.1046/j.1365-4362.2003.01763.x

AUTHORS' INFO

*** Diana A. Myshlyanova;**
address: 4/1 Bolshaya Pirogovskaya street, 119991 Moscow, Russia;
ORCID: 0009-0006-0801-2227;
eLibrary SPIN: 9002-2365;
e-mail: dina.myshly@gmail.com

Elena S. Snarskaya, MD, Dr. Sci. (Medicine), Professor;
ORCID: 0000-0002-7968-7663;
eLibrary SPIN: 3785-7859;
e-mail: snarskaya-dok@mail.ru

Natalya P. Teplyuk, MD, Dr. Sci. (Medicine), Professor;
ORCID: 0000-0002-5800-4800;
eLibrary SPIN: 8013-3256;
e-mail: teplyukn@gmail.com

Lydia M. Shnakhova, MD, Cand. Sci. (Medicine);
ORCID: 0000-0003-3000-0987;
eLibrary SPIN: 5549-5823;
e-mail: lika-slm@mail.ru

Juliya M. Semiklet;
ORCID: 0000-0001-7615-3917;
eLibrary SPIN: 3245-4770;
e-mail: semiklet.jul@mail.ru

* Corresponding author / Автор, ответственный за переписку

ОБ АВТОРАХ

*** Мышлянова Диана Алексеевна;**

адрес: Россия, 119991, Москва, ул. Большая Пироговская, д. 4/1;
ORCID: 0009-0006-0801-2227;
eLibrary SPIN: 9002-2365;
e-mail: dina.myshly@gmail.com

Снарская Елена Сергеевна, д-р мед. наук, профессор;
ORCID: 0000-0002-7968-7663;
eLibrary SPIN: 3785-7859;
e-mail: snarskaya-dok@mail.ru

Теплюк Наталия Павловна, д-р мед. наук, профессор;
ORCID: 0000-0002-5800-4800;
eLibrary SPIN: 8013-3256;
e-mail: teplyukn@gmail.com

Шнахова Лидия Мухамедовна, канд. мед. наук;
ORCID: 0000-0003-3000-0987;
eLibrary SPIN: 5549-5823;
e-mail: lika-slm@mail.ru

Семиклет Юлия Михайловна;
ORCID: 0000-0001-7615-3917;
eLibrary SPIN: 3245-4770;
e-mail: semiklet.jul@mail.ru

ECO • VECTOR

SCIENTIFIC JOURNALS PORTAL

Eco-Vector's Scientific Journals Portal is a hosting platform being an access point to journals and articles for readers. It is also an editorial and publishing system allowing automated digital workflow across all aspects of the publishing lifecycle: starting from easy online manuscript acquisition to publishing

https://journals.eco-vector.com

- The editorial and publishing system provides the user with ample possibilities to customize the design and functionality of a scientific journal's website
- Flexible workflow management system, that can be tailored to needs of any journal or publisher
- Online manuscript acquisition and collaboration between Authors and Editorial Boards
- Various publishing models: Gold Open Access, Delayed Open Access, Hybrid, and Subscription
- Interface in 6 languages: Russian, English, Chinese, Brazilian Portuguese, Kazakh, Spanish

PLATFORM FEATURES

- All Crossfer services: DOI, Cited-By, CrossMark, FundRef, CrossCheck (SimilarityCheck)
- Fast Track and Online First
- Publication of full texts in PDF, HTML, XML JATS
- Cooperation with eLibrary.ru, EBSCO, DOAJ, SCOPUS, CrossRef, Google Scholar, PubMed, PubMed Central, CNKI, DataCite, mEDRA, ResearchBib
- Article level statistics: Dimensions, PlumX, Crossref Cited-By, Altmetrics
- Online payment options for subscription and editorial services
- Multilingual publications (1 article – 1 URL for all language versions)