IJMAMR 7 (2020) 1-3 ISSN 2053-1834

International Journal of Modern and Alternative Medicine Research
www.bluepenjournals.org/ijmamr

Overlapping bullous pemphigoid with chronic arthropatic psoriasis

doi.org/10.33500/ijmamr.2020.07.001

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Article History
Received 02 September, 2019
Received in revisedform 30 January, 2020
Accepted 03 February, 2020

Keywords:
Psoriasis vulgaris, Bullous pemphigoid, Multiple autoimmune syndrome.

Article Type:
Case Report

ABSTRACT

The pathophysiological mechanism of autoimmune diseases has always been a point of interest for physicians worldwide. Approximately one out of every four patients with an autoimmune disease will develop another type of autoimmune disease. The coexistence of psoriasis and bullous pemphigoid (BP) is not uncommon, but the cause is still unclear. Multiple autoimmune syndromes describe the association between more than three autoimmune diseases, but the coexistence and the way these diseases influence one another is not fully understood, with more and more theories arising every day. The case of a patient with a particular cutaneous bullous eruption, shortly after the interruption of long-term biological treatment for psoriasis is hereby presented. Patient adherence to a long ongoing treatment of an autoimmune disease is an important factor in the causality of another autoimmune disease, and should be taken into consideration when thinking of multiple autoimmune syndrome. Cessation of treatment may trigger the onset of multiple autoimmune syndrome and should be avoided.

INTRODUCTION

Psoriasis and bullous pemphigoid (BP) are two distinct chronic inflammatory diseases. The association between psoriasis vulgaris (PV) and BP is due to a poorly understood autoimmune process. Both entities emerge from the dermal-epidermal junction, with specific alteration for each form of disease (Caca-Biljanovska et al., 2015). The mechanism of action in psoriasis involves the dermal-epidermal layers of the skin, because the premature maturation of the keratinocytes starting in the basal layer of the epidermis is induced by an inflammatory cascade in the dermis, involving dendritic cells, macrophages and T-cells.

Psoriasis is an inflammatory, T-cell mediated disease which affects the skin and joints, and is associated with many other cutaneous and systemic diseases. The inflammatory activity of psoriasis may affect the antigenic properties of the basal membrane of the epithelial skin cells (Colmenero et al., 2015). Approximately one out of every four patients with an autoimmune disease will develop another type of autoimmune disease (Ohata et al., 2015). The coexistence of these two particular disorders is not rare, and more often patients with psoriasis will develop BP than the general population (Cojocaru et al., 2010). According to Iskandarli et al. (2015), psoriasis induced BP appears to be dependent on the inflammatory status of the main pathology. Moreso, the association of more than three autoimmune diseases is known as the multiple autoimmune syndrome (MAS), BP being classified as a Type 1 MAS, along with pemphigus vulgaris and myasthenia gravis.

Type 1 MAS is comprised of myasthenia gravis, bullous autoimmune diseases of the skin, thymoma, polymyositis, giant cell myocarditis. Type 2 MAS is comprised of Sjögren's syndrome, rheumatoid arthritis, primary biliary cirrhosis, scleroderma, and autoimmune thyroid disease. Type 3 MAS is comprised of autoimmune thyroid disease, myasthenia gravis and/or thymoma, Sjögren's

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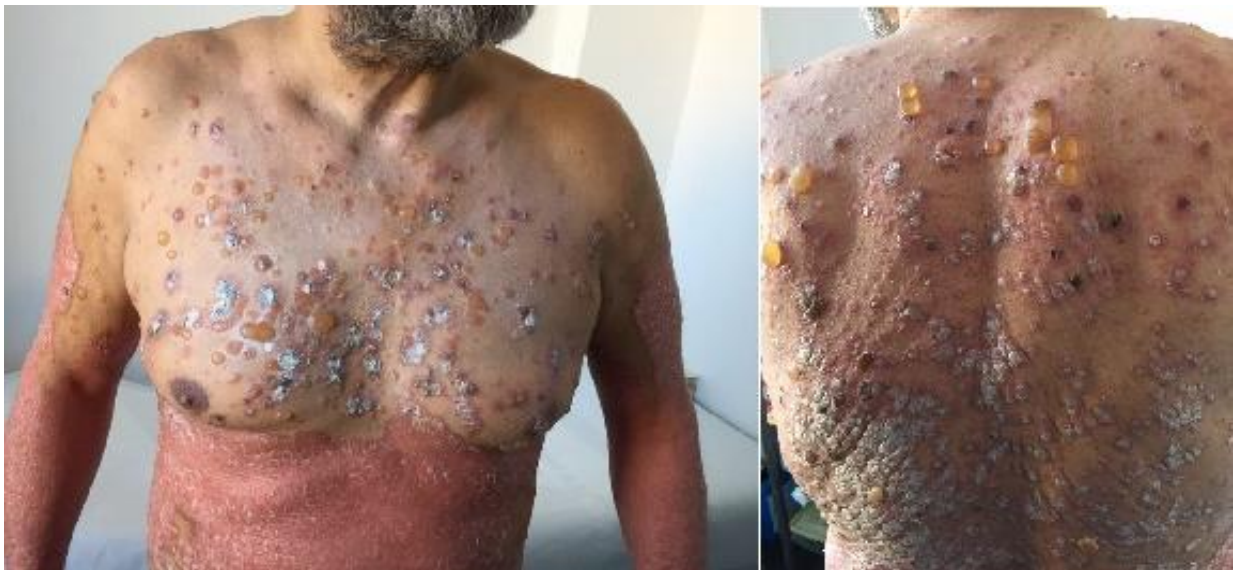

Figure 1. Clinical presentation of both BP and arthropathic psoriasis. Multiple bullous lesions overlapping with erythematous and squamous plaques affecting the trunk and upper limbs.

syndrome, pernicious anemia, idiopathic thrombopenic purpura, Addison's disease, Type 1 diabetes mellitus, vitiligo, autoimmune hemolytic anemia, systemic lupus erythematosus, and dermatitis herpetiformis (Cojocaru et al., 2010).

Some researchers consider psoralen and ultraviolet A(PUV) therapy used for psoriasis patients to be the trigger for a subclinical BP, by producing antibodies against the cell membrane (Caca-Biljanovska et al., 2015). Other reports suggest that the use of biological treatment for psoriasis increases the incidence of autoimmune diseases associated, in the duration of treatment (Nakayama et al., 2015). Topical or systemic use of steroids, anthralin or coal tar, may also be triggers of the BP in psoriasis patients (Ohata et al., 2015).

METHODOLOGY

The case of a 48 years old Romanian man diagnosed with arthropatic psoriasis (AP) at the age of 14, who came to Dermatology Department of County Emergency Clinical Hospital of Oradea, Romania with generalised vesiculobullous eruption, associated with erythemato-squamous plaques is presented (Figure 1). The last chronic AP treatment was Etanercept 50 mg/week associated with Methotrexate 10 mg/week (during this time, the patient was in the care of the Rheumatology Department of County Emergency Clinical Hospital of Oradea, Romania), which had been discontinued for approximately two months before attending the Dermatology Department of County Emergency Clinical Hospital of Oradea. Prior to Methotrexate and Etanercept treatment, the patient underwent topical therapy with steroid and emulsion ointments, UV-B therapy, but with no improvement of the simptomathology. The Romanian National Health Service, which re-evaluates the condition of each patient with AP every six months, to ensure suitability of treatment on a case by case basis, caused a delay of two months to approve treatment, not only for this patient, but for other patients suffering from AP. Therefore he could not obtain the medication needed to continue his chronic treatment. One month after the treatment was discontinued, the patient described confluent hyperkeratosis plaques, more pronounced in the antero-posterior trunk and lower limbs regions. Shortly after, the sudden onset of BP was described as a generalized cutaneous and buccal mucosa eruption. At the moment of the first dermatological consultation since the patient discontinued his chronic treatment, psoriasis area and severity index (PASI) score was 36 points. The patient presents multiple articular involvements in the form of symmetrical distal joint arthritis, interphalangeal hand and foot ankylosis, maintaining minimal digital grasp, with dactylitis and nails involvement.

If some studies mention antigen altering factors as drugs, UV treatment, vaccinations, surgeries and organ transplant to be the BP triggers in psoriasis patients (Lesniewska et al., 2016), the cessation of treatment is considered to be the cause.

In this particular case, the clinical onset of BP was without any obvious trigger, the patient was not taking any medication, and the bullous eruption appeared on psoriasis affected skin as well as on normal cutaneous

surface. The onset of BP was after a short period of AP treatment discontinuation, during which time the cutaneous presentation of psoriasis aggravated without locomotor function altering.

RESULTS

Paraclinic findings at hospital admission noted: leukocytosis (12.140×10³ /uL), neutrophilia (8.033 × 10³/uL), increased hepatic transaminases (GPT 113 U/L, GOT 50 U/L), anemic syndrome (Hb 12 g/dl, HCT 40%, MCHC 30 g/dl), low serum iron and calcium levels (Ca 8.2 mg/dl), inflammatory syndrome (VSH 40mm/h), Tzanck smear: 50% neutrophils, 40% monocytes, 10% eosinophils. IgG anti BP 180 antibodies > 500U/mL^3.

Skin biopsies from both bullous lesion and hyperkeratosic plaque were performed.

1. Subepidermic vesicle with fibrin and leukocytes (eosinophiles) with characteristic appearance of BP.
2. Acanthotic epidermis, orthokeratosis and parakeratosis, with superficial polymorphonuclear infiltration and dermal vascular ectasis, suggestive psoriasis aspect.

DISCUSSION

In the present case, the evolution was rapidly favorable, with the complete cessation of bullous eruption after 2 weeks of treatment with Etanercept 50mg/week, Methotrexate 10 mg/week, Prednisone 70mg/day and total lesion remission at 1 month after onset. His body weight was 90 kg, 0.77 mg prednisone/kg body weight. Psoriasis had a good evolution, the lesions improved after 3 to 4 weeks of specified treatment.

It is of interest to note that despite the fact that BP can arise from a multitude of environmental, medication or pathogenous triggers, the sudden withdrawal of anti-AP treatment can act as a possible cause, thus making BP a sign of active aggravatedAP. The antigenicity of the basement membrane in psoriasis plaques may stimulate autoantibody formation and BP development(Vaccaro et Kabashima, 2018). If the theory put forward proves to be true based on further research, attention is therefore drawn to the fact that cessation of AP therapy may cause the onset of a new autoimmune disease, which may be avoided with continual therapy. The theory of this report is based on an observational study of one patient only, and as such more similar cases are required to cement this theory.

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