Psoriasis and vitiligo are two diseases frequently encountered in dermatological practice. The first case of psoriasis associated with vitiligo was described in 1955 by Selenyi.

We studied four patients (two males, two females), aged between 34 and 72 years old, who were diagnosed with vitiligo and psoriasis. The diagnosis of vitiligo was made based on the clinical aspects. Psoriasis was diagnosed using clinical and histopathological exams. We calculated PASI, DLQI, VASI scores for each patient, in order to assess the seriousness for both conditions. For each patient we performed biological investigations to analyse the comorbidities and, in one of the cases, we used them so that we could monitor the biological therapy. We encountered the following clinical cases of psoriasis: plaque psoriasis (three cases) and guttate psoriasis (one case). All of the four cases that we studied presented non-segmental vitiligo. Considering the four cases we underline the etiopathogenic interferences, aspects which suggest that the association of the two diseases does not seem coincidental.

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Introduction

Psoriasis and vitiligo are two frequently encountered conditions in the dermatological practice. The worldwide psoriasis incidence in the general population is 1-3% and the one for vitiligo is situated between 0.5-1%. The first case of vitiligo associated with psoriasis has been described in 1955 by Selenyi(1). In 1989, Menter et al. (2) described the first case of guttate psoriasis associated with vitiligo, and Dahar and Malak described in 1998 the association of these two conditions for a boy aged 9 years old (3). Within this article, we present the observations made on four patients, where the two skin conditions coexist. On this occasion we bring into discussion common etiopathogenic factors for the two diseases.

Clinical cases

The epidemiological data for each patient are described within table I, followed by the clinical forms of psoriasis and vitiligo (fig. 1, 2, 3), comorbidities and therapeutic means that we applied. The diagnosis of vitiligo was made based on the clinical aspect. Psoriasis was diagnosed using clinical and histopathological exams. We calculated PASI (Psoriasis Area and Severity Index), DLQI (Dermatology Life Quality Index) and VASI scores (Vitiligo Area Severity Index) for each patient, in order to assess the seriousness for both conditions. For each patient we performed biological investigations to specify comorbidities and, only in one case, we used them so that we could monitor the biological therapy.

Discussions

Psoriasis is a chronic inflammatory dermatosis characterised by the hyperproliferation of the evolving whimsical keratinocytes, sometimes disabling. Psoriasis vulgaris represents more than two thirds of the cases, and comprises several aspects: dotted, follicular, guttate, nummular, circinate, plaque psoriasis, universal. In the case of our patients we met the following clinical forms: plaque psoriasis (three cases) and guttate psoriasis (one case).

The etiopathogenesis of psoriasis is multifactorial. Thus, when speaking about the etiopathogenesis of psoriasis, the literature mentions(4): psychological disorders, certain medications, infections, traumas, the endocrine factor, the alcohol excess consumption, smoking, the immunological and metabolic disorders, but also the involvement of genetic factors is nowadays well substantiated.

When referring to the infectious factor, things are clearer regarding the relationship between the guttate psoriasis at young people and the infectious episode, particularly the nasopharyngeal one. The role of the HPV infection in the psoriasis etiopathogenesis is not clear. The idea that some types of HPV can act as superantigens has been postulated, as well as the fact that it may be encountered in latent form and activated by some proinflammatory cytokines(5). For the cases presented, we consider the chronic alcohol consumption (two cases) and the beta-antagonistss medication (for one case, managed for portal hypertension) factors that can be incriminated. Psoriasis is often preceded by the excessive alcohol consumption. The chronic alcohol consumption can interfere in the pathogenesis of psoriasis through the following links: the occurrence of stressful events in the life of the patient, induction of hepatic pathology, immunosuppression induction, increased frequency and severity of infections with the release of proinflammatory cytokines. Vitiligo is a kind of hypomelanosis characterized by the progressive appearance of the hypopigmented macules, related to the rarefaction until the disappearance of the melanocytes within the epidermis(6). The following etiopathogenic theories were invoked: autoimmune, autocyctotoxic, neural, enzymatic, the one of the oxidative stress, transepidermal melanocitragy, the apoptosis. The genetic susceptibility was encountered in 24-38% of the patients diagnosed with vitiligo. The disease occurs more frequently in people with HLA - B12, HLA - B 13, HLA - BQ4. The familial cases are increasing by 4,5 times the risk of disease for descendents. Vitiligo has been associated with several autoimmune diseases, more commonly type I diabetes mellitus, Addison’s disease, pernicious anemia, thyroid diseases and alopecia areata. Thyroid disorders are encountered at approximately 12% of the adults diagnosed with vitiligo, more commonly than for the general population, where the prevalence ranks between 1-2%. Also, the Addison’s disease occurs in 2% of the cases, and the Biermer anemia is 30 times more frequent in the case of the patients with vitiligo. Diabetes mellitus is met in 1-7 % of the cases, and, more particularly, in the case of the insulino-
dependent diabetes. In what comes next, we describe two clinical types of vitiligo: Non-segmental or symmetrically vitiligo, defined by Vitiligo European Task Force as a “chronic acquired disease characterized by hypopigmented macules, often symmetrical, which are growing in size, across the time, and correspond to a significant loss of the melanocytes functionality, located upon the epidermis and also upon the hair follicle” and segmental, with unilateral distribution, that affects one or more dermatomes. All of the four cases that we studied had manifested non-segmental vitiligo.

A retrospective, cross-sectional and comparative study, made on 740 patients with chronic dermatoses, that were undergoing phototherapy, observed that 35,27 % of the cases manifested vitiligo (segmental 15,3%) and 44,72 % had psoriasis. Vitiligo associated with psoriasis was observed only on 9 patients (3,44 %). The latter were analyzed having in mind the following parameters: sex (6M and 3F), mean age (32 years, between the interval 14-75 years), phototype (phototype III prevailed, for four patients).

The family history of vitiligo was encountered only in a single case, the same as allergic rhinitis and asthma. The Koebner phenomenon and the poliosis were encountered in three or four cases. The commonly affected areas were the head (four cases), face (two cases) and in one case, several areas were affected: forearms, shoulders or a generalized vitiligo form was encountered. In the cases of 5 patients, from a chronological aspect, vitiligo debuted firstly, and the strictly developed psoriasis on vitiligo areas was found in two cases.

### Table 1. Psoriasis and vitiligo diagnosed patients - epidemiological, clinical and therapeutical data

<table>
<thead>
<tr>
<th>Crt. No</th>
<th>Sex</th>
<th>Age</th>
<th>Family history of psoriasis</th>
<th>Family history of vitiligo</th>
<th>Vitiligo onset age</th>
<th>Psoriasis onset age</th>
<th>Vitiligo Type</th>
<th>Psoriasis clinical form</th>
<th>Other comorbidities</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>66 years old</td>
<td>neg.</td>
<td>neg.</td>
<td>40 years</td>
<td>60 years</td>
<td>Non-segmental vitiligo; VASI 79,8</td>
<td>Disseminated plaques PASI 16,2 DLOI 21</td>
<td>-Obesity gr. II</td>
<td>-Enbrel - Etanerceptum - Calcipotriol 50 microg/betamethasone dipropionate 0,5 mg/g gel</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>34 years</td>
<td>neg.</td>
<td>neg.</td>
<td>13 years</td>
<td>34 years</td>
<td>Non-segmental vitiligo; VASI 10,8</td>
<td>Disseminated plaques PASI 18,9 DLOI 24</td>
<td>-Epilepsy (grand mal) - Chronic ethanol hepatitis</td>
<td>-Mometasone furoate 0,1% / 5%Salicylic acid - Calcipotriol 50 microg / betamethasone dipropionate 0,5 mg / g gel</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>72 years</td>
<td>neg.</td>
<td>neg.</td>
<td>52 years</td>
<td>62 years</td>
<td>Non-segmental vitiligo; VASI 67,5</td>
<td>Disseminated plaques PASI 8 DLOI 14</td>
<td>-Von RecklinghaussenNeurofibromatosis - Decompensated cirrhosis, portal and parenchymal</td>
<td>-Betamethasone dipropionate 0,05% / Salicylic acid, 3% ointment</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>47 years</td>
<td>neg.</td>
<td>pos.</td>
<td>22 years</td>
<td>47 years</td>
<td>Non-segmental vitiligo; VASI 29</td>
<td>Guttate psoriasis PASI 7,5 DLOI 15</td>
<td>No</td>
<td>-Tinefon 2x2 capsules/ daily - Calcipotriol 50 microg/betamethasone dipropionate 0,5 mg/g gel - Betamethasone dipropionate 0,05%/salicylic ointment 3%</td>
</tr>
</tbody>
</table>

Psoriasis and vitiligo: four clinical cases.

Case Presentation
If we report our conclusions on the four cases that we presented, we can emphasize the equal gender distribution. The onset of vitiligo (mean age 31.75 years) preceded with 20 years the onset of psoriasis mean age of (51.75 years). The family history was psoriasis-negative for all the patients and vitiligo-positive in one of the patients.

Psoriasis/vitiligo: etiopathogenic interferences

The explanation for the psoriasis - vitiligo coexistence is not clear. In 1982, Koransky and Roenig(7) conducted a literature review and presented 25 cases of vitiligo associated with psoriasis, concluding that the association of these two conditions is relatively rare. Chronologically, in all our cases vitiligo preceded psoriasis. It was suggested that the melanin decrease or absence may be a predisposing factor for the psoriasis plaques development(8).

It is known that both dermatoses have an autoimmune component concerning the pathogenesis. The level of TNF-α is increased within the psoriasis lesions, as well as in perilesional skin of the patients with vitiligo, fact that can lead to the conclusion that TNF-α may be a link between psoriasis and vitiligo. In vitro studies(8) have shown the increased production of TNF-α at the level of the melanocytes in vitiligo. The favorable results obtained for both diseases after the treatment with cyclosporine and tacrolimus support this hypothesis(9).

Another factors described as being responsible for this coexistence are the Koebner phenomenon, commonly met to both diseases, but also NALP1 gene association, and an activation of the Th1 and Th17 cellular immune system pathways(10). AIS 1 locus, susceptible for vitiligo, is located on the level of the chromosome 1p31.3 - p32.2 which is closely situated to the locus PSORS7 of psoriasis. However, the possibility that these two loci are identical, is minimized by the low encountered prevalence of psoriasis in vitiligo patients(11). AIS 1 was found only in the case of patients with generalized vitiligo and it is possible that it may not be present in other vitiligo forms.

Recently, Prignano et al(12) suggested that both diseases are immune-mediated, with a genetic connection, and Zhu et al.(13) found that both psoriasis and vitiligo have an ordinary common locus, within the major histocompatibility complex (MHC). We did not find data in the literature regarding the triple combination, including psoriasis, vitiligo and neurofibromatosis type I, pathology seen in one of our cases. The association between psoriasis and neurofibromatosis type I is rare and the authors could not determine whether the existence is purely coincidental or is determined by common genetic defects(14). Within table II we summarized psoriasis, vitiligo and type I neurofibromatosis data (the type I neurofibromatosis was found in one of our cases), facilitating the separation of certain commonly points related to the pathogenesis of the three diseases. Based upon data from the speciality literature related on the two diseases, we can say that T lymphocytes, keratinocytes, melanocytes and cytokines have an essential role for the psoriasis and vitiligo coexistence. Satisfactory results may be obtained in these patients by using the following therapies: PUVA therapy, treatment with UVB – narrow band (twice weekly) associated with tacrolimus ointment 0,1% (twice weekly), xenon laser 308 nm chloride excimer, potent topical corticosteroids, vitamin D3 derivatives, calcineurin inhibitors (tacrolimus, pimecrolimus), calcipotriol 50 microg/betamethasone dipropionate 0,5mg, overall corticosteroids therapy. The therapeutic results in the four cases were satisfactory, if we are judging them from the perspective of controlling psoriasis flares, but without beneficial effects on the depigmented areas. For the female patient who received biologic therapy with Etanercept, after four months from the treatment initiation,
en confetti pigmentation occurred, on the upper limbs and trunk, without any improvement of the aesthetics.

Conclusions

The psoriasis/vitiligo coexistence is rarely encountered in medical practice. Extensive epidemiological studies are needed in order to know the real rate of these two diseases association. Future pathogenesis research for each of the two conditions may bring new data in order to confirm that there is not a coincidental coexistence or even to contradict this hypothesis.

Table 2. Psoriasis, Vitiligo and von Recklinghausen Neurofibromatosis: comparative data

<table>
<thead>
<tr>
<th>Genetic predisposition</th>
<th>Vitiligo</th>
<th>Psoriasis</th>
<th>von Recklinghausen Neurofibromatosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polygenic AD transmission; multiple susceptible loci and candidate genes have been involved</td>
<td>Polygenic transmission can involve multiple genes situated on different locus.</td>
<td>AD monogenic transmission. Variable expressivity. 100% penetrance.</td>
<td></td>
</tr>
<tr>
<td>Involved genes from the following chromosomes 1, 2, 3, 6, 7, 8, 10, 11, 12, 14, 17, 21, 22, 28</td>
<td>PSORS2 Genes on 17q, PSORS1 6p21.3, PSORS3 pe 4q, PSORS4 pe 1q21.3, PSORS5 pe 3q21, PSORS6 pe 19p, PSORS7 pe 1p, PSORS8 pe 16q, PSORS9 pe 4q28 Q32 si PSORS10 pe 18p11.26, 27</td>
<td>NF1 gene located on the long arm of chromosome no. 17 and codates the neurofibromin-protein, which is linked to RAS gene</td>
<td></td>
</tr>
<tr>
<td>Genetic Markers - antigens</td>
<td>HLA-B12, HLA-B13 and HLA-B04</td>
<td>HLA-B13, B17 and HLA-CW6, BW57, psoriasis vulgaris, HLA-B27 highly emphasized to the patients diagnosed with artropathy psoriasis; HLA-DQB1 alpha for generalized pustular psoriasis</td>
<td></td>
</tr>
<tr>
<td>Antibodies</td>
<td>AC antimelanocytes</td>
<td>AC corneous antistat</td>
<td></td>
</tr>
<tr>
<td>Environment factors</td>
<td>The stress contribution is significant</td>
<td>The environment factors contribution is a significative one: infections (streptococcal, HIV, etc.), medicines, traumatism, smoking, stress, etc.</td>
<td></td>
</tr>
</tbody>
</table>

Conflicts of interest: none declared

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Bibliography


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