

# Vitamin deficiency and the most common dermatoses in people with ulcerative colitis

## *Niedobór witamin i najczęstsze dermatozy u osób z wrzodziejącym zapaleniem jelita grubego*

### ABSTRACT

In recent years the number of people suffering from colitis ulcerosa (CU) has risen rapidly. The disease is characterized by alternating periods of remission and relapse. Persistent inflammatory activity in the colon impedes the absorption of certain nutrients, which affects in dermatologic manifestations on one's skin, hair and nails. Numerous intestinal symptoms cause discomfort in patients, which negatively affects physical, emotional and psychological well-being.

The aim of this study was the description of CU, indication of deficient components and consequences of impaired absorption manifesting on skin, nail and hair condition.

Patients with CU are among the demanding clients of beauty salons. Properly selected products and treatments can help supplement the deficiencies of selected substances in the body and obtain satisfactory results.

**Keywords:** ulcerative colitis, etiopathogenesis, disorders, deficiency, vitamins

### STRESZCZENIE

W ostatnich latach odnotowuje się coraz większy odsetek osób chorujących na wrzodziejące zapalenie jelita grubego. Choroba przebiega z okresami zaostrzeń i remisji. Toczący się stan zapalny w jelicie grubym utrudnia wchłanianie niektórych składników pokarmowych, co manifestuje się na skórze, włosach i paznokciach. Liczne objawy jelitowe sprawiają duży dyskomfort u osób chorych, przez co zmagają się one także z problemami emocjonalnymi i psychicznymi.

Celem pracy był opis wybranych witamin oraz konsekwencji ich upośledzonego wchłaniania manifestującego się na skórze i jej przydatkach.

Osoby chorujące na wrzodziejące zapalenie jelita grubego są również klientami gabinetów kosmetycznych. Odpowiednio dobrane produkty oraz zabiegi mogą wspomóc uzupełnienie niedoborów wybranych substancji w organizmie oraz uzyskanie zadawalających efektów.

**Słowa kluczowe:** wrzodziejące zapalenie jelita grubego, etiopatogeneza, zaburzenia, niedobór, witaminy

### INTRODUCTION

Ulcerative colitis (*colitis ulcerosa*, CU) has become an ever more common disease in recent years. It affects young people of working age, the group that often uses the services of cosmetology salons. Knowledge of pathophysiology and

specific skincare requirements of this group of customers is extremely important for a cosmetologist.

CU is a chronic disease with periods of exacerbation and remission, with a multifactorial etiology. The lesions cover

the mucosa of the large intestine, extend from the rectum and develop continuously to the proximal parts of the colon [1]. In 30% of cases, only the rectum and sigmoid colon are inflamed. More often, in as much as 50% of patients, the descending and transverse colon is affected [2].

The exact pathogenesis of the disease is not fully understood. It is assumed that it is related to the interaction between genetic, immunological, and environmental factors as well as disturbances of the intestinal microbiota [1].

CU is visualized endoscopically as an inflammatory process that spreads continuously from the rectum towards the mouth. With low disease activity, the intestinal mucosa is rough and granular, with reduced vascular nevi and only mild erythema. During the severe course, confluent ulcers and petechial hemorrhages develop. The transition from normal to inflamed mucosa is usually clearly marked, and the inflammation usually becomes more severe, running distally [3].

To diagnose ulcerative colitis, multiple preliminary examinations should be performed. Laboratory tests include blood parameters like level of hemoglobin, leukocytosis, platelets, Biernacki's test (ESR), ferritin and vitamin B<sub>12</sub>, and biochemical tests: C-reactive protein, liver function tests, albumin and electrolytes, and in the case of absorption disorders - determine the level of calcium and magnesium [4]. The final diagnosis is based on the assessment of the characteristic symptoms confirmed by the endoscopic image and results obtained from histopathological examination of the mucosa of the large intestine taken during colonoscopy [1, 3].

The inflamed intestine, as a result of the disease, does not absorb some nutrients, which causes malnutrition or aggravates the disease. Underweight and malnutrition weaken the immune system, make the intestine more susceptible to infections, and disturb the process of its regeneration [5]. Patients most often have a deficiency of fat-soluble vitamins: A, D, E, K, and water-soluble vitamins: B<sub>9</sub>, B<sub>12</sub>, C, H [5-7]. The study aimed to indicate the cosmetic consequences of vitamin deficiencies and the most common dermatoses in patients with UC.

## VITAMIN DEFICIENCIES IN CU PATIENTS

### Vitamin A

One of the most important fat-soluble vitamins for the proper condition of the skin is vitamin A. Deficiency of this vitamin strongly influences the formation of skin lesions, which are manifested by dryness, hyperkeratosis, and peeling of the epidermis. Retinol, a derivative of vitamin A, stimulates the reactions of transforming low-activity fibroblasts into cells with relatively high collagen production. Increasing the number of these cells affects the connective tissue of the dermis, improving its firmness, elasticity, and hydration. It also affects the production of type I and III collagen fibers and the

elimination of damaged elastin fibers and contributes to the growth of new blood vessels. Vitamin A belongs to the group of substances with anti-wrinkle and anti-aging properties. It smoothes and eliminates fine wrinkles, reduces discoloration and signs of aging, and contributes to the proper breakdown of melanin in the skin. The appropriate level of this substance regulates the work of the sebaceous glands, accelerates the healing of wounds and epidermis damage [8]. A characteristic symptom of deficiency is dry mouth, inflammation of the corners of the mouth, and mucosa atrophy [9]. Retinol deficiency can disrupt hair and nail growth. The main symptom of vitamin A shortage level is manifested as excessive brittleness of the nails as well as their longitudinal furrowing. There is increased follicular keratosis with accompanying dryness and flaking of the scalp. Hair may become unreasonably brittle, and occasionally increased hair loss is also observed [9, 10].

In the event of skin symptoms indicating vitamin A deficiency, the use of cosmetic preparations containing this vitamin can significantly improve the condition of the skin. With deeper deficiencies, a specialist consultation is recommended.

### Vitamin D

Vitamin D is a fat-soluble steroid hormone with pleiotropic biological properties, including the regulation of cell proliferation, differentiation, and apoptosis. It has anti-inflammatory activity, affects the condition of the immune and nervous systems [11], and is also crucial for the proper metabolism of adipose tissue [12]. It is assumed that its deficiency may exacerbate the picture of CU [13]. Vitamin D<sub>1</sub> (calciferol) is responsible for the condition of hair and nails and its deficit in the body leads to baldness, brittle hair, and increased seborrhea [14]. It cannot be used externally, and the oral administration should be adjusted to the individual needs of the organism [13]. Moreover, nutricosmetic preparations do not contain vitamin D<sub>1</sub>.

### Vitamin E

Vitamin E, which is one of the most important vitamins of antioxidant properties, affects the proper hydration of the epidermis and reduces transepidermal water loss (TEWL). It strengthens the epidermal barrier and limits the penetration of external substances, which helps to prevent irritation and reduce the risk of inflammation [15]. A deficiency of tocopherol, which is included in vitamin E, may manifest itself in petechiae, hyperkeratotic follicular papules, pale skin, frinoderma, or be associated with the production and deposition of lipofuscin in the tissues. In the oral cavity, the mucosa may become pale [9]. Deficiencies may predispose to severe skin damage after exposure to solar radiation [16].

With severe deficiencies, only topical application may not be sufficient to strengthen the antioxidant defense of the skin,

especially the dermis. To prevent or reduce the changes caused by photoaging, nutricosmetics should be added. Studies on vitamin E indicate the mechanisms and routes of distribution of this component after oral administration and its effect on individual layers of human skin. Human sebum is high in  $\alpha$ -tocopherol, and the sebaceous glands provide an important physiological route for the delivery of  $\alpha$ -tocopherol to the skin. This is especially accurate of areas rich in these glands, such as facial skin. However, if this route must be efficient, the saturation of the body with vitamin E must be correct. Studies on oral supplementation show that proper saturation is achieved after 2-3 weeks of regular intake of the preparation [16]. The second distribution mechanism involves the penetration of vitamin E into the cells and the slow migration of vitamin E along with the cells to the stratum corneum layer.

### Vitamin K

Vitamin K is involved in the synthesis and maintenance of the correct concentration of coagulation factors. It has also been proven to be active in other extrahepatic tissues. Deficiency increases the risk of developing osteoporosis, atherosclerosis, neoplastic diseases, and Alzheimer's disease [17]. The effect of hypovitaminosis is prolonged prothrombin time, which is associated with decreased blood clotting, which will manifest itself in the skin with visible subcutaneous extravasations and impaired wound healing [18]. This is especially important in the context of laser treatments. In 2002, placebo-controlled studies on the effects of vitamin K on the prevention and removal of laser-induced purpura were conducted. The preparation was applied to the skin of one half of the face before the laser treatment, and the skin of the other half of the face - after the treatment. Cream with vitamin K applied after the procedure reduced the severity of bruises compared to the side of the face premedicated with a preparation containing vitamin K [19]. Such annotations were observed by another research team [20]. Cream with vitamin K combined with retinol removed laser-induced spots faster. These reports call for greater vigilance when performing laser treatments in people with possible vitamin K deficiency, including patients with UC.

### Vitamins B<sub>9</sub>, B<sub>12</sub>

Vitamin B<sub>9</sub> (folic acid) is involved in many metabolic processes in the body. Its deficiency leads to impaired production of erythrocytes, increase in their volume, shortened survival time, and bone marrow destruction. The consequence may also be leukopenia, thrombocytopenia, and megaloblastic anemia triggered by disturbances in the synthesis of nucleic acids resulting from disturbances in the synthesis of purine precursors [21]. Hypovitaminosis is manifested by a slowdown in the synthesis of deoxyribonucleic acid (DNA) and cell replication, often enlargement of the liver and spleen, and in advan-

ced cases, increased muscle tone [22]. The skin turns pale, the tongue is red with ulcerations, inflammation appears on the tongue and the mucosa of the lips. Deficiency also affects the hair which becomes early and sudden graying [23].

Due to the possibility of storing vitamin B<sub>12</sub> in the liver, clinical symptoms related to its deficiency may be noticeable only after several years [24]. The most characteristic symptom is a disturbance in the process of hematopoiesis [25], as a result of which the skin becomes lemon-colored, pale, and hyperpigmentation foci appear in about 10% of cases. Within the oral cavity, glossitis, the so-called "Buffalo tongue" can be noticed, characterized by a smooth and shiny surface. In addition cheilitis, oral ulcerations, painful mucosal atrophy, recurrent mouth ulcers may appear, as well as a tendency to yeast infections. Vitamin B<sub>12</sub> deficiency for hair, similarly to vitamin B<sub>9</sub>, is manifested by premature graying [9, 24].

### Vitamin C

Vitamin C (ascorbic acid) can affect several skin features, including the hydration level. Studies based on cell lines indicate that vitamin C administration increases the lipid barrier and induces keratinocyte differentiation. Ascorbic acid increases the stratum corneum, and thus retains water to a greater extent [26]. It promotes collagen synthesis and acts as a cofactor for lysine and proline hydrolases, which stabilize this protein. In vitro studies on fibroblasts have shown that the lack of vitamin C reduces both the total collagen synthesis and its cross-linking, which increases the risk of fiber breakage, slower healing of wounds and weakening of capillaries with microcarcinoma [26]. The skin symptoms are follicular purpura, follicular hyperkeratosis, ecchymosis and limb edema. The characteristic condition is corkscrew hair, and nails show linear subungual petechiae, resembling splinters [9].

### Vitamin H

Vitamin H, also known as biotin, plays a crucial role in the body functioning. It is a cofactor of carboxylases, involved in the catabolism of amino acids as well as the biosynthesis and metabolism of fatty acids, thus contributing to the improvement of the appearance of the skin. It stimulates skin cells to grow, improves its metabolism and regenerative abilities, thus reducing fine lines. Due to vitamin H, the skin's protective barrier is also strengthened as a result of accelerating the synthesis of fatty acids [28].

Biotin deficiency may cause alopecia, brittleness of nails, skin inflammation and secondary fungal infection in the body caused by *Candida albicans* [29]. Hypovitaminosis can lead to scaly skin inflammation on the hands, legs and arms. They are characterized by drying and discoloration of the skin and mucous membranes. In addition, a red rash may appear around the eyes, nose and mouth, referred to as "biotin deficient face" [30].

## SKIN MANIFESTATIONS IN CU PATIENTS

The skin symptoms in patients with UC are usually specific, reactivation changes, symptoms related to nutrient malabsorption or treatment regimen. They can also be pathological changes.

Studies have shown that in children and adolescents with UC, oral involvement may occur in up to one-third of cases and usually consists of non-specific lesions [31]. During exacerbation of the disease, aphthous stomatitis often occurs [32]. Clinically, it is characterized by numerous circular or oval painful ulcers with a yellow pseudomembranous base and erythematous margins [33]. Vitamin deficiencies (B1, B12, B9, D) may be the etiological factors [34].

Erythema nodosum is the most common skin lesion in patients with UC. It is an acute inflammatory skin disease characterized by the sudden appearance of symmetrical, erythematous, warm, painful and non-ulcerative nodules. They have a raised, slightly red or purple color and a diameter of 1 to 5 cm. They occur mainly on the front part of the lowest limbs, although they can occur anywhere on the body [33, 35]. Literature data do not link this disease entity with vitamin deficiencies.

Pyoderma gangrenosum is the most severe and debilitating skin manifestation in patients with UC. The changes can occur anywhere in the body, but the most common sites are legs and peristomal sites [34]. Lesions begin as pimples or papules that ulcerate quickly and form painful areas with the typical violet-purple raised margins. The ulcer bed is necrotic and may be affected by a secondary infection. Changes can be isolated or multiple. It is accompanied by systemic symptoms such as fever, joint pain and malaise [33]. Studies link pyoderma gangrenosum with deficiency of B vitamins (the role of vitamin B12 and folate is most often reported) [36].

*Pyodermatitis pyostomatitis vegetans* is a mucocutaneous symptom that affects men more often than women with UC. Vesicular and maculopapular lesions are mainly located in the scalp, face, armpits and groin [33]. High doses of corticosteroids are used in therapy, and vitamin and mineral supplements are often recommended, which indicates that deficiencies of these ingredients may contribute to the pathomechanism of the disease or extend its duration.

Sweet's syndrome is a neutrophilic dermatosis characterized by painful erythematous plaques or nodules on the face, neck and extremities, and frequent fever and leukocytosis [37]. The main histopathological feature is a dense aseptic neutrophilic infiltrate in the reticular layer of the dermis, which may be accompanied by spongy and vesicles in the epidermis and swelling of the papillary layer of the dermis [35]. In etiopathogenesis, the role of vitamin D deficiency is indicated.

## SUMMARY

Ulcerative colitis is a chronic inflammatory intestine disease with periods of exacerbation and remission. The inflamed intestine is less able to absorb some nutrients, and their deficiencies may be manifested on the skin, hair and nails. Disturbances in the intestinal microflora are also observed in the course of the disease.

For a cosmetologist, a CU client is the one that needs special attention. In order to select the appropriate treatment and to obtain the desired effects, it is important to know about possible deficiencies in the pathomechanism of this disease. In addition to the use of appropriately selected preparations applied to the skin, oral supplementation may often be needed.

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