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Late cutaneous porphyria as a rare case of extrahepatic manifestations of chronic hepatitis C in a patient with a history of syphilis

N.N. POTEKAEV, L.L. KARPOV, O.V. DOLYA, M.N. MARKOVA

Moscow Scientic and Practical Center of Dermatvenerology and Cosmetology. Moscow, Russia

ABSTRACT

The article presents literature data concerning the etiology, pathogenesis, diagnostic and clinical criteria, as well as treatments for cutaneous porphyria. Porphyria (Greek, πορφύριος — scarlet, purple) is a group of diseases based on inherited or acquired impairment of the enzymatic pathway involved in formation of porphyrins, their precursors, and heme in the liver and bone marrow. The pathogenesis of porphyria lies in the fact that the non-protein part of hemoglobin, heme, is not synthesized, and intermediates of its synthesis, porphyrinogens, are instead accumulated in plasma and tissues of the body. Here we report a rare case of late cutaneous porphyria as an extrahepatic manifestation of chronic hepatitis C in a patient who has a history of syphilis.

Keywords: *cutaneous porphyria, classification, clinical manifestations, differential diagnosis.*

N.N. Potekaev — Doctor of Medicine, Professor, Director Moscow Scientic and Practical Center of Dermatvenerology and Cosmetology. Moscow, Russia. https://orcid.org/0000-0002-9331-7714
O.V. Dolya — Doctor of Medicine, Dermatovenereologist. Moscow Scientic and Practical Center of Dermatvenerology and Cosmetology. Moscow, Russia. https://orcid.org/0000-0001-5019-4593
M.N. Markova — Dermatovenereologist. Moscow Scientic and Practical Center of Dermatvenerology and Cosmetology. Moscow, Russia. https://orcid.org/0000-0002-5493-9983
L.L. Karpov — Dermatovenereologist. Moscow Scientic and Practical Center of Dermatvenerology and Cosmetology. Moscow, Russia.

Porphyria (Greek, πορφύριος — scarlet, purple) is a group of diseases based on inherited or acquired impairment of the enzymatic pathway involved in formation of porphyrins, their precursors, and heme in the liver and bone marrow.

Heme is synthesized in all tissues, but the bone marrow and liver are the main organs involved in its synthesis. Heme is required for oxygen binding and transport (in the form of hemoglobin and myoglobin), electron transport (in the form of cytochromes), and for activity of multifunctional oxidases, such as cytochrome $P_{450.}$ Heme biosynthesis is controlled by eight enzymes; this process starts in mitochondria, where δ-aminolevulinic acid is formed from glycine and succinyl CoA with the participation of aminolevulinate synthase enzyme. From mitochondria, δ-aminolevulinic acid moves to the cytoplasm, where intermediate stages of heme synthesis take place with the participation of δ-aminolevulinic acid dehydratase, porphobilinogene desaminase, and uroporphyrinogen decarboxylase. Both final and initial reactions of heme synthesis occur in mitochondria with the participation of coproporphyrinogen oxidase, ferrochelatase (FeC), and protoporphyrinogen oxidase. As the final stage, bivalent iron is added to protoporphyrin IX (with the participation of the FeC enzyme) and heme is formed. Ferritin, an iron-depositing protein, is the source of iron for heme synthesis. Heme is an allosteric inhibitor of the aminolevulinate synthase enzyme, which triggers the reaction of its biosynthesis. Porphyria is associated with heme formation disorders as a result of defects in enzymes; its amount decreases leading to increase in activity of aminolevulinate synthase, which in turn leads to accumulation of heme synthesis intermediates, such as porphyrins or their precursors.

The biological role of porphyrins and their iron complexes in metabolic processes lies in their ability to act as mediators of oxidation reactions, components of hormone metabolism, drugs, and environmental chemical compounds. They are involved in exchange of gases (such as oxygen and carbon dioxide) between the body tissues and external environment.

Porphyrins and heme are daily synthesized in quantities sufficient to meet metabolic needs. Control of heme synthesis is so precise that only micrograms (or even less) of the intermediates of this process are present in the plasma, erythrocytes, urine, and feces under normal conditions.

The pathogenesis of porphyria is based on the fact that the non-protein portion of hemoglobin (heme) is not synthesized and intermediate products of its synthesis, porphyrinogens, are accumulate in the plasma and tissues instead.

When exposed to light, porphyrinogens turn into porphyrins, which are able to absorb ultraviolet (as well

Corresponding author: Dolya Olga Valentinovna — Moscow Scientic and Practical Center of Dermatvenerology and Cosmetology. Moscow, Russia. olga-d7@yandex.ru

as visible) radiation, leading to photosensitization. Additionally, porphyrins are oxidized when interacting with air oxygen, forming active radicals that damage skin cells and cause inflammation of the skin. It should be noted that all porphyrins have red fluorescence (this phenomenon is used for diagnostic tests).

Neurovisceral symptoms of porphyria are due to neurotoxic properties of porphyrin precursors (δ-aminolevulinic acid and porphobilinogen). Additionally, heme deficiency in the nervous tissue may be observed as a result of defects in enzymes involved in heme synthesis. Thus, peripheral and autonomic nervous system are particularly susceptible to neuropathy caused by porphyrin and its precursors.

There are two groups of porphyria that differ in location of the primary defect of expression of specific enzymes responsible for heme synthesis: erythropoietic and hepatic one. The main criteria for distinguishing between these porphyrias include characteristics of porphyrin metabolism disorders and the nature of clinical manifestations, including onset of the disease.

The development of erythropoietic porphyrias is based on genetically determined defects of enzymatic systems, leading to impaired porphyrin metabolism in bone marrow erythroblasts. The development of hepatic porphyrias is mainly associated with the influence of various hepatotropic factors, such as drugs, intoxication with salts of heavy metals and other chemical compounds, liver diseases, and alcohol.

According to clinical manifestations, neurovisceral (in the form of acute attacks) and cutaneous forms are distinguished. However, it should be mentioned that two types (mixed or variegate porphyria and hereditary coproporphyria) can have both neurovisceral and cutaneous manifestations. Classification of porphyria is shown in Table 1.

Porphyriais occur in all countries around the world, but its forms are unequally represented in different regions.

Erythropoietic porphyrias are characterized by hereditary nature of the disease, onset in early childhood, and the absence of preceding action of triggering factors. Three forms of erythropoietic porphyria are distinguished: uroporphyria, protoporphyria, and coproporphyria. Each of these forms is characterized by accumulation of certain types of porphyrins, i.e. uroporphyrins, protoporphyrins, and coproporphyrins, respectively. X-linked dominant protoporphyria also belongs to erythropoietic ones.

Erythropoietic uroporphyria (Gunter's congenital erythropoietic porphyria, named after the German physician Hans Gunter who described the disease in 1911). This is a rare congenital form of porphyria, which, unlike most other forms, is characterized by autosomal recessive inheritance (patient's parents, who are carriers of impaired genes, do not suffer from porphyria). It is caused by deficiency of uroporphyrinogen III cosynthase, an enzyme located in patient's pathological erythroblasts, resulting in disturbed heme biosynthesis and increase in the level of uroporphyrinogen I in the patient's body (in erythrocytes, urine, and feces). Uroporphyrin deposition in the skin leads to photosensitization. The disease manifests immediately at birth or within the first year of life. Itchy erythema appears on the open areas of the skin exposed to sunlight followed by formation of vesicular and bullous elements with serous or serous-hemorrhagic contents. Opening of blisters result in formation of difficult-to-heal erosion and ulceration, which are often complicated by secondary bacterial infection. Ulceration healing is accompanied by scarring with hyperpigmentation foci, which is more pronounced on the face and opisthenar. Long and chronic course of the disease results in involvement of deep tissues in the pathological process; ear mutilation is observed. Nails are dystrophic, thickened, deformed, and eventually fall off. X-ray examination of the osteo-articular system shows osteoporosis, complete or partial contracture of ligaments. Eyes can also be involved (conjunctivitis, symblepharon, etc.). Erythrodontia is also observed, i.e. maroon teeth. The teeth produce bright purple-red fluorescence when examined under ultraviolet light. There is hypertrichosis on the face, especially on the forehead. Patients have very thick eyebrows and long eyelashes. Urine is red, which is often the first sign of the disease. Most patients demonstrate splenomegaly, and some individuals have hepatosplenomegaly. Additionally, poikilocytosis, anisocytosis, spherocytosis, and thrombocytopenia are observed. The level of uroporphyrins in the 24-hour urine of patients increases several

Table 1. **Classification of porphyria**

Porphyria	Porphyria type depending on the site of primary defect of enzyme expression	Porphyria type depending on clinical manifestations
Late cutaneous (LCP)	Hepatic	Cutaneous
Hepatoerythropoetic	Hepatic	Cutaneous
Acute intermittent	Hepatic	Neurovisceral
Mixed (variegated)	Hepatic	Cutaneous, neurovisceral
Hereditary coproporphyria	Hepatic	Cutaneous, neurovisceral
ALA-dehydratase-deficient (plumboporphyria)	Hepatic	Neurovisceral
Erythropoietic protoporphyria	Erythropoietic	Cutaneous
X-linked dominant protoporphyria	Erythropoietic	Cutaneous
Congenital erythropoietic	Erythropoietic	Cutaneous

hundred times compared to the normal value, reaching 140—160 mg, while coproporphyrin level reaches 30—52 mg (high values in urine as opposed to hepatic form of porphyrins are characteristic only of congenital erythropoietic porphyria).

The disease is characterized by chronic course with exacerbations in the spring-summer period. Previously, the prognosis of congenital erythropoietic porphyria was unfavorable, patients died at the age under 30 years due to various intercurrent diseases and hemolytic anemia. Currently, the prognosis of the disease is favorable, but patients cannot be fully cured.

Erythropoietic protoporphyria was described in 1953 by W. Kosenow and A. Treids. The authors noted increased level of protoporphyrins in feces, signs of photosensitization, and fluorescence of blood erythrocytes in two infant patients and called this disease protoporphyrinemic photodermatosis. Metabolism of this disease was thoroughly studied in 1961 and L. Magnus classified it as a form of porphyria. Erythropoietic porphyria is characterized by an autosomal dominant inheritance.

The disease is caused by decrease in activity of ferrochelatase, resulting in hyperproduction of protoporphyrin, which is accumulated in the bone marrow, erythrocytes, and skin. The presence of protoporphyrin in the skin causes photosensitization, similarly to deposition of uroporphyrin. Additionally, this type of porphyria is associated with secondary skin changes, which are observed in patients during clinical remission. Obviously, this fact is associated with formation of free radicals as a result of protoporphyrin decay process, which damage the skin. The relative protoporphyrin-to-coproporphyrin ratio in feces play an important role in the diagnosis of the latent form of erythropoietic protoporphyria.

The disease usually manifests at the age of 4—5 years, sometimes later. Most patients develop edema (resembling Quincke' ones) and erythema at the exposed skin areas accompanied by burning, which usually persists for 1—2 days. There are several forms of the disease: suppressed form, which presents only with subjective sensations; urticarial form characterized by localized skin swelling and itching; edematous erythematous form is the most common one; eczema-like form presents with eczema-like skin changes; bullous-hemorrhagic form is characterized by development of purpura and blisters on the edematous-erythematous background.

Secondary dermatological signs of erythropoietic protoporphyria are characterized by moderate pigmentation, thickening and induration of the skin, enhancement of its pattern at the perioral and periorbital areas, on the alae and dorsum of the nose, on the back of the hands, mainly above the small joints, and on the back of the neck. In some patients, there are small atrophic scars, which give the skin on the dorsum and alae of the nose a specific grainy look. The general condition is usually normal. However, patients often suffer from gallstone disease and may develop liver cirrhosis.

Erythropoietic coproporphyria is caused by a defect of coproporphyrinogen oxidase, which leads to increase in the level of coproporphyrin. Clinical presentation is similar to that of erythropoietic protoporphyria.

Hepatic porphyrias are more common and are represented by four nosological forms: acute intermittent, late cutaneous, variegated, and hereditary coproporphyria. Hepato-erythropoietic porphyria and ALA-dehydratase-deficient porphyria (plumboporphyria) are also referred to as hepatic forms.

Acute intermittent porphyria is characterized by an autosomal dominant inheritance and it is caused by decrease in activity of porphobilinogen deaminase. The disease is more common in females and manifests only when the patient is exposed to some triggering factors, such as certain pharmaceuticals, hormones (estrogens), and alcohol. Some females suffering from acute porphyria during pregnancy demonstrate worsening of the disease up to death. Clinical presentation includes numerous symptoms in the digestive tract (abdominal pain, nausea, vomiting, constipation, diarrhea); nervous system (paralysis, paresis, epileptiform seizures; mental disorders — emotional lability, depression, etc.); cardiovascular system (tachycardia, arterial hypertension). The skin is not involved. The diagnosis is established on the basis of clinical presentation and laboratory data: high level of porphyrin precursors (porphobilinogen and d-aminolevulinic acid) in urine.

For a long time, it was believed that George III, King of England, suffered from acute intermittent porphyria, but later on, when the evidence of increased sensitivity of the King's skin to sunlight was found, the diagnosis was changed to variegated porphyria.

Late cutaneous porphyria (urocoproporphyria) is caused by a defect of uroporphyrinogen decarboxylase, which results in almost twofold increase in the level of this enzyme in the tissues. It is regarded as a quite rare disease, although it is the most common form of porphyria in the world. According to various sources, the incidence rate is 1.5—20 cases per 100 thousand population. In the case of the hereditary form of the disease, enzyme deficiency is observed in the liver and erythrocytes; in the case of sporadic form — only in the liver. Sharp increase in urine and plasma level of uroporphyrin was detected, resulting in orange-red urine fluorescence under the Wood's lamp. Alcohol, estrogens, salts of heavy metals and iron preparations, as well as aromatic hepatotoxic hydrocarbons, hepatitis C virus, and HIV infection are believed to be triggering agents of the disease. Numerous studies confirm that porphyria is rarely caused by only one specific factor, but it is rather a multifactorial disease and the set of risk factors is individual for each patient. Late cutaneous porphyria, as a rule, develops in the individuals (more often in males before oral contraceptives were widely used) older than 30 years and is characterized by tense blisters with serous or serous-hemorrhagic contents on the intact or moderately hyperemic skin. Rash occurs after insolation or injury and

is localized on the exposed skin areas. Thin caps of the blisters are easily ruptured soon afterwards, resulting in erosions that quickly become crusted. Positive Nikolsky's sign may be detected when stretching fragments of epidermis around ruptured blisters. Fragility of the skin leads to formation of blisters, erosions, and abrasions, which is especially often observed on the dorsum of the hands. At the initial stage, blisters form only in response to mechanical injury, but progressive course of the disease may result in spontaneous formation of blisters. Superficial scars remain at the site of opened bubbles, where milium-like elements are often formed, which tend to group.

In addition to blisters, late cutaneous porphyria is often associated with pigmentation of exposed skin areas, which is initially reddish and then brown color becomes more intense and persistent. Hypertrichosis is observed in 70% of patients. As a rule, it occurs on the face and it is especially pronounced at the temporo-zygomatic area, on the auricles and nose; there is also increased growth of eyelashes and eyebrows.

White indurated scleroderma-like plaques can form both at exposed and protected from light skin areas. It was proved that uroporphyrin I stimulates collagen synthesis in human skin fibroblasts.

The following forms of the disease are considered as atypical ones: scleroderma-like, sclero-vitiliginous, and sclero-lichenoid, melanodermic, infiltrative-plague, and porphyrin-induced cheilitis.

Although cutaneous manifestations mostly occur at the areas exposed to sunlight, patients often do not realize that sunlight plays an important role in the development of these lesions, since acute pain and burning sensation caused by sunlight, which is characteristic of erythropoietic porphyria, is rarely observed in the case of LCP. However, most patients note worsening of the skin process in the spring and summer season.

Patients with late cutaneous porphyria often demonstrate hepatomegaly. Changes in the liver evolve in the manner characteristic of chronic hepatitis and cirrhosis; the latter is 28 times more common than in the whole population, and liver cancer is 130 times more common. Involvement of the cardiovascular system often manifests in the form of intermittent claudication and myocardial infarction. Some patients develop diabetes-like syndrome.

Validation of the diagnosis is based on detection of significantly elevated plasma porphyrin levels (usually 5—100 times higher than normal), elevated coproporphyrin level in the stool and uroporphyrin level in urine. Sometimes, LCP patients have normal or slightly elevated daily porphyrin levels in urine, and fluorescence screening test for urine porphyrins is also often negative. In these patients, plasma porphyrin levels should be determined and daily uroporphyrin and coproporphyrin levels in urine and feces should be assessed using high-resolution liquid chromatography.

Almost all LCP patients demonstrate excessive amount of total iron, which manifests in the form of increase in serum iron level andor increase in hepatocellular iron level. Mild erythrocytosis is sometimes observed. Biochemical blood analysis shows elevated serum levels of transaminases and gamma-glutamyl transferase.

Pathomorphological examination, shows that LCP is characterized by formation of subepidermal blisters with uneven scalloped bottom. There is little or no inflammatory infiltrate in the dermis. The walls of the papillary vessels are thickened as evidenced by the periodic acid Schiff reaction.

Direct immunofluorescence shows deposition of C3 component of the complement and immunoglobulin G at the boundary between the epidermis and dermis, as well as in the vascular walls around them.

Involvement of superficial vessels of the dermis and dermal-epidermal junction indicates that structural changes triggered by porphyrin photosensitization may cause LCP-specific fragility of the skin.

LCP should be differentiated from other forms of porphyria, pseudoporphyria, hydroa vacciniforme, scleroderma, and acquired epidermolysis bullosis.

Variegated porphyria is caused by decrease in the level of protoporphyrinogen oxidase characterized by autosomal dominant inheritance. The disease is widespread in Europeans in South Africa. Clinical presentation is characterized by symptoms of both acute intermittent and late cutaneous porphyria and consists of abdominal symptoms, cardiovascular and nervous disorders, as well as skin lesions. The disease mostly manifests before the age of 30 years and is usually triggered by the use of barbiturates, analgesics, associated intercurrent diseases, and pregnancy. As a rule, skin changes occur 3—4 years before somatic symptoms. Similarly to late cutaneous porphyria, rash is represented by pigmentation, bullous elements, erosions, atrophic changes, and scars. Patients suffer from increased skin fragility. High level of protoporphyrin (to a lesser degree coproporphyrin) in feces and porphobilinogen in urine is detected.

Hereditary coproporphyria is caused by a defect of coproporphyrinogen oxidase. It is characterized by the mildest course and clinical symptoms. During the acute phase (onset) patients may experience nausea and vomiting, and, more rarely, neuropsychiatric disorders. Mild skin fragility and, as a consequence, formation of blisters, is possible. There is increased coproporphyrin level in feces and porphobilinogen and coproporphyrin level in urine.

Treatment

Patients suffering from porphyria should avoid insolation and apply photoprotective agents and beta-carotene preparations (at a dose of 60—180 mg). Chingamins (rezokhin, plaquenil, chloroquine), riboxin, and iron-removing drugs (deferoxamine) should be prescribed. It is advisable to use therapeutic doses B vitamins (B_1, B_6, B_{12}), nicotinic acid. Along with this, folic acid (0.01 g TID), riboflavin

Table 2. **The results of laboratory diagnostic methods**

Value	10.06.2016	03.07.2016	22.07.2016
Complete blood count			
Hemoglobin	14.4	13.3	15.7
Red blood cells	4.75	4.15	5.13
MCH	30.3	32	30.3
MCV	90.1	91.9	90.1
Hematocrit	42.3	38.1	44.3
Platelets	320	284	197
Leucocytes	12.2	10.7	10.9
Stab cells	2	3	4
Segmented leucocytes	54.2	61	66.9
Lymphocytes	31.3	20	20.0
Monocytes	9.3	10	9.7
Basophils	0.9	0	0.9
ESR	63	33	54
Biochemical blood analysis			
ALT	230	189.7	117
AST	101.5	78.8	76.9
Total bilirubin	317.6	170.1	23.3
Conjugated bilirubin	260.2	86.9	9.1
GGTP	57	72.7	181.0
ALP	235.4	153.2	206.1
Albumin	28.7	31.6	37.5
Creatinine	101.6	107.6	64.0
Urea	5.9	4.6	3.47
Na	136.2	140.6	135.8
K	5.06	5.6	3.86
Prothrombin	66.4	76.3	71.9
INR	1.33	1.154	1.23

Note. MCH — mean content of hemoglobin in a single erythrocyte in absolute units; MCV — mean red blood cell volume; ALT — alanine aminotransferase; AST — aspartate aminotransferase; GGTP — gamma-glutamyl transpeptidase; ALP — alkaline phosphatase; INR — international normalized ratio.

(0.005 g TID), ascorbic acid (0.1 g TID), aevit (1 capsule BID or TID), methionine (0.5-0.75 g per day), syrepar (intramuscular at a dose of 2—3 ml, 50—60 injections per course) are recommended. Topical corticosteroid ointment and solcoseryl are also administered. Bone marrow transplantation can also be used to treat patients with Gunther's erythropoietic porphyria.

In the case of HCV or HIV, appropriate therapy should be prescribed, which also results in regression of LCP simptoms.

Case study

We report our own clinical observation of a patient with late cutaneous porphyria.

Patient Sh.M.S. born in 1970 applied to the Lyublinskiy branch of the Moscow Scientific and Practical Center of Dermatovenerology and Cosmetology of the Moscow Healthcare Department for consultation on the possibility of staying at the somatic hospital. Medical history shows that the patient received specific outpatient treatment for syphilis at the dermatovenerologic dispensary in Dagestan at the place of residence in 2005.

The survey at the Lyublinskiy branch showed negative microprecipitation test, passive hemagglutination reaction 4+; positive enzyme immunoassay (*Ig* total), CP 8,2; HIV — negative, HCV(+) — positive. A day before, the patient was consulted by an infectious disease specialist and therapist in Moscow clinic. Hospitalization to hepatology department of somatic hospital was recommended.

The patient complained of darkening of the skin, itching, skin rash on exposed skin areas, pain in the right hypochondrium and at the area of rash, weight loss of 4 kg for 2 months, sleep disturbance, suicidal thoughts.

Medical history. aHCV was first detected in 1999, the patient did not receive treatment. Moderate alcohol consumption. In 2013, chronic hepatitis C was diagnosed in the Infectious Diseases Hospital No 1, replication stage, genotype 1c. Antiviral therapy was not carried out. The patient received courses of hepatoprotectors (Essentiale, Phosphogliv). Mild rash on the limbs for the first time appeared in 2014. The patient was consulted by an infection disease doctor, but the diagnosis was not established. In the summer of 2015, the patient developed rash after

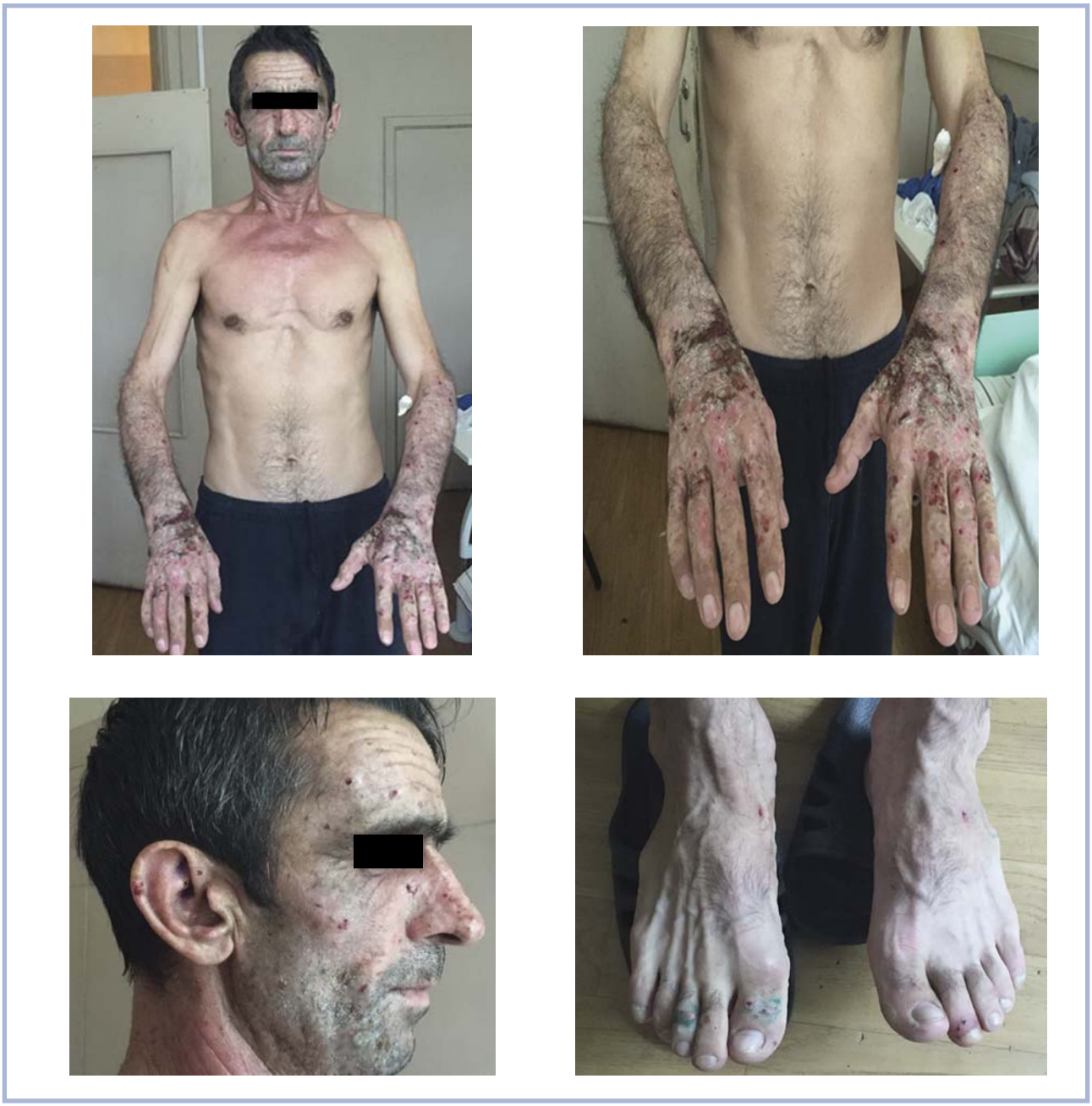

Fig.1. **Patient Sh.M.S., clinical manifestations of late cutaneous porphyria.**

insolation. He was treated with 10% Linimenti Zincioxydi (30 ml), which was almost ineffective.

After prolonged insolation in 2016, skin rash spread to the face, neck, limbs; it was accompanied by pain and fever up to 38°C. The patient was consulted by a dermatologist and diagnosed with cutaneous porphyria.

The results of examination are shown in Table 2.

Daily urine spectroscopy: total porphyrin level — 9194 nmol/l (limit — less than 300 nmol/l).

Instrumental methods. Esophagogastroduodenoscopy: axial hiatal hernia, superficial gastritis, duodenogastric reflux.

Ultrasound examination of abdominal organs: diffuse changes in the liver, splenomegaly.

Examination showed that the patient has asthenic body type. The general condition of moderate severity due to severe pain at the area of skin rash and the presence of hyperthermia. Local status: extensive inflammatory skin lesions localized on the face and lower third of the upper and lower extremities. Rash elements are represented by tense blisters, erosions at the site of vesicles and blisters, and serous hemorrhagic crusts against the background of unchanged skin. The face is bluish-pink, the color is especially pronounced at the periorbital area.

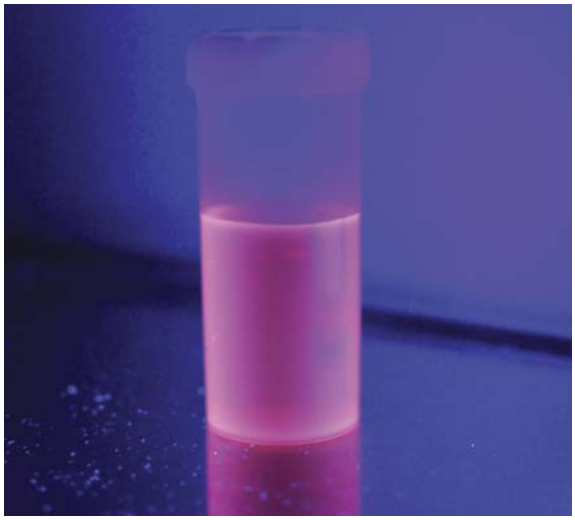

Fig. 2. **Express urine test (coral-red fluorescence under Wood's lamp).**

Diffuse hyperpigmentation of exposed areas of the body. Hypertrichosis of the face **(Fig. 1)**.

Express test was performed to validate the diagnosis of cutaneous porphyria: coral-red urine fluorescence under the Wood's lamp was detected **(Fig. 2)**.

The patient was admitted to the hepatology department of the somatic hospital, where he was diagnosed with chronic HCV, replication stage, moderate biochemical activity with extrahepatic manifestations in the form of late dermal porphyria. Inpatient treatment resulted in elimination of pain and asthenic syndrome; the pathological skin lesions regressed to post-inflammatory hyperpigmentation; signs of cytolytic and cholestatic syndromes were normalized; total porphyrin level in 24-hour urine decreased. The patient was discharged in satisfactory condition. Recommendations included avoiding insolation, photoprotection, and non-interferon antiviral therapy to prevent recurrence of skin manifestations.

The reported case of LCP is undoubtedly interesting for clinicians.

The authors declare no conflict of interest.

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