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Chromomycosis

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ABSTRACT

Chromomycosis (CM, ICD-10 code B43) is a chronic deep tissue mycosis occurring predominantly in tropical and subtropical countries. The causative agents of CM are fungi of the genera *Fonsecaea* and *Cladophialophora*, which are plants and soil saprophytes. They enter the human skin during trauma, which is why CM is called implantation (subcutaneous) mycosis. In Europe, the disease tends to be sporadically imported; meanwhile, autochthonous (local) sporadic cases have been described in temperate countries. In endemic regions, diagnosis of CM is often challenging, and most drugs used in treatment remain unavailable due to the underdevelopment of health care, but even in countries with higher levels of health care, there is no gold standard of treatment for CM. The combination of antimycotic pharmacological therapy with physical and surgical methods of treatment is prioritized, as this method provides a good outcome in a short time. Non-surgical treatment is carried out before, during and after the surgery. We present a literature review and our own clinical observations. Also, a case of effectively treated CM caused by *Fonsecaea pedrosoi* in a patient with no previous travel history permanently residing in the Moscow region is presented. Treatment with surgical excision of the lesion and antimycotic therapy in the pre- and postoperative periods resulted in complete clinical recovery.

Keywords: *chromomycosis, Fonsecaea pedrosoi, Cladophialophora carrionii, diagnosis, pharmacological and surgical treatment, Moscow region.*

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Chromomycosis (CM) (synonyms: chromoblastomycosis, blastomycosis, black blastomycosis, Pedroso disease, Lane-Pedroso mycosis, Fonseca disease, verrucosis, verrucous dermatitis), ICD-10 code B43 is a chronic purulent granulomatous inflammation of the skin and subcutaneous tissue, caused by dimorphic saprophytic fungi found in soil and organic material of environment.

CM was first described in 1914 by M. Rudolph [1]. It occurs mainly in tropical and subtropical countries of Asia, Africa, and Latin America, as well as in Australia, more often in people with low income, living in remote rural areas, urban slums, and conflict zones with unfavorable political situations, and poor health care. CM is one of the "neglected" tropical diseases and, as of 2016, is not included in the WHO's list of tropical diseases. According to WHO, it is not part of the list of public health priorities [2, 3]. Due to limited access to health care in endemic regions, diagnosis of CM is challenging, and most drugs used for treatment remain unavailable, but even in countries with a higher level of health care, there is no gold standard of treatment for CM [2, 4].

CM is differentiated with verrucous tuberculosis of the skin and lymph nodes, keloid blastomycosis, squamous cell carcinoma, psoriasis, phaeohyphomycosis, sporotrichosis, leishmaniasis, leprosy, yaws, syphilis, swimming pool granuloma, verrucous dermatitis, gangrenous pyoderma and other diseases [2, 5].

More than 50 cases of CM have been described in Russia, mainly in St. Petersburg and Moscow [6—9]. There are no official statistics.

The causative agents of CM are dimorphic, dark-colored, spherical fungi: *Cladosporium carrionii*, *Fonsecaea compacta*, *F. pedrosoi*, *F. monophora*, *F. dermatitidis*, *Exophiala jeanselmei*, *E. spinifera*, *Phialophora verrucosa* and *Phinocladiella aguaspera*. *Fonsecaea pedrosoi* is found mainly in countries with humid climates and *Cladophialophora carrionii* in semi-arid climates [10, 11]. Nevertheless, the latter occurs worldwide, with the highest prevalence in the Amazon region of Brazil, northern Venezuela, Costa Rica, the Dominican Republic, and Madagascar [1].

In Europe, the disease is usually imported, but Pindycka-Piaszczynska et al. suggested the presence of autochthonous cases based on an analysis of the incidence of CM in Poland. In these patients, the onset of the disease was associated with numerous abrasions from contact with old wood in a coal mine, and the causative agent *Fonsecaea monophora* was isolated from foci on the skin [12]. In Ukraine, the disease has also been reported in coal mine

workers after wood injury [13, 14]. As for European countries, a total of 31 cases of "probable" CM have been identified in Czechoslovakia and Belgium [15, 16]. S. Greven et al. in Germany described a case of CM caused by *Arthrinium arundinis* in a patient receiving immunosuppressive therapy for allogeneic bone marrow transplantation. In Spain, N. A. Richarz et al. diagnosed the first European case of CM caused by *Rhinocladiella similis* in a male who had never left the country [17, 18].

CM pathogens are saprophytic and ubiquitous in the environment, especially in decaying wood and plant residues, soil, leaf debris, and grass. In the pathogenesis of the disease, a great role is attributed to the preceding trauma, due to which CM is called implantation ("subcutaneous") mycosis. When infected material enters the skin, the pathogen spreads via lymphogenous and less common hematogenous routes.

Risk groups include agricultural workers, miners, loggers, and sellers of agricultural products. CM is more common for males aged 40—50 years, the ratio of males to females being from 5:1 to 17:1 [1, 11, 19]. The higher incidence of CM in males may be related to hormonal protection in females, as in cases of paracoccidioidomycosis, in which females are protected from clinical manifestations by estradiol, but in CM, hormonal protection is due to progesterone [20].

CM develops slowly, sometimes over years. It involves body parts in contact with infected material: hands, feet, lower legs, knees, buttocks, back of thighs, chest and upper extremities, and, less commonly, the face [6, 21]. After an incubation period of 1 to 6 months, patients with CM develop a red spot at the site of fungus inoculation. Later, the spot turns into a papule with a smooth surface that increases in size and has psoriasis-like flaking, followed by palpable tubercles, papules, induration, and verrucous nodules [1, 22]. Satellite lesions, usually of the papillomatous-verrucous type, gradually occur around the primary site. In the beginning, the disease is asymptomatic or has minimal symptoms; at later stages, it presents with itching. As a result of autoinoculation and lymphatic spread of the pathogen, nodules with a wart-like surface appear on adjacent skin areas. Gradually, they merge into larger nodules and form conglomerates on a large skin surface **(Fig. 1)**.

In later stages, verrucous and papillomatous lesions grow, ulcerate, hyperkeratosis occurs, and the itching becomes severe. The lesions often resemble cauliflower. With a prolonged course, keloid-like scars form **(Fig. 2)**.

In the next stages, hyperkeratosis intensifies, and the papillomatous and verrucous elements continue to ulcerate, crusted, and flat, dark-red, clearly delineated plaques appear among the "cauliflower." The secondary bacterial infection leads to acute inflammation, purulent discharge with an unpleasant odor, and pain. Red to purple infiltrates are observed around the lesions, and cartilaginous scales appear on the surface, sometimes with pronounced fissures **(Fig. 3)**.

In advanced and severe cases, CM can present with elements of several types. Unilateral localization and asymmetric lesions are typical of CM [22, 23]. Hyperpigmented scars may remain on the skin after the resolution.

Involvement of regional lymph nodes has been reported. Hematogenous dissemination involving mucous membranes and the brain is extremely rare. No reports of specific involvement of internal organs in immunocompetent subjects are available [23]. Occasionally, long-term persistent lesions without specific treatment transform into squamous cell skin cancer (1).

According to ICD-10, CM is included in section B43, "Chromomycosis and pheomycotic abscess," and subsections B43.0, "Cutaneous chromomycosis," B43.8, "Other forms of chromomycosis," and B43.9, "Chromomycosis, unspecified."

There is also a clinical classification of CM, reflecting the nature of the skin eruptions, their evolution, and the predominance of certain symptoms: papular



а/a б/b

Fig. 1. **Papulose-papillomatous CM of the left buttock (a), nodular-papillomatous progressive CM with the formation of a conglomerate of nodules and their spread to the adjacent tissues (b).**



Fig. 2. **Papillomatous ulcerative CM of the right buttock (a), verrucous CM with predominant keloid scars of the left buttock (b).**

and tuberculous, nodular, papillomatous-ulcerous, verrucous, with keloid scars. These variants can also be considered as stages of the disease, from spots and papules to verrucous elements and scarring. In practice, mixed forms are more common.

Diagnostic microscopy of native samples taken from the surface of the ulcer or scales treated with KOH shows clusters of spherical, golden brown fungal cells 4-15 μm in diameter, sometimes surrounded by giant fungal cells with transverse and longitudinal septa. The diagnosis is verified by the detection of oval or round cells with a thick yellowish-brown sheath separated by a septum in crusts, secretions, or scrapings. They are found solitary or in groups, and sometimes short, broad septate mycelium is also observed.

When cultured on Sabouraud dextrose agar at 26 °C, uniform dark-colored colonies with aerial mycelium grow on days 7—10; the "velvet" surface of these colonies has a grayish, greenish, or brown tint. Bottom growth on liquid media occurs as black cotton-like balls. On days 5—10, grayish microcolonies are visible; on days 10—15, the colonies mature and become cone-shaped, fluffy, and gray-green colored.

It is possible to distinguish the species of CM pathogens only in culture (hematoxylin-eosin staining) by the conidiophores morphology and the way of co-

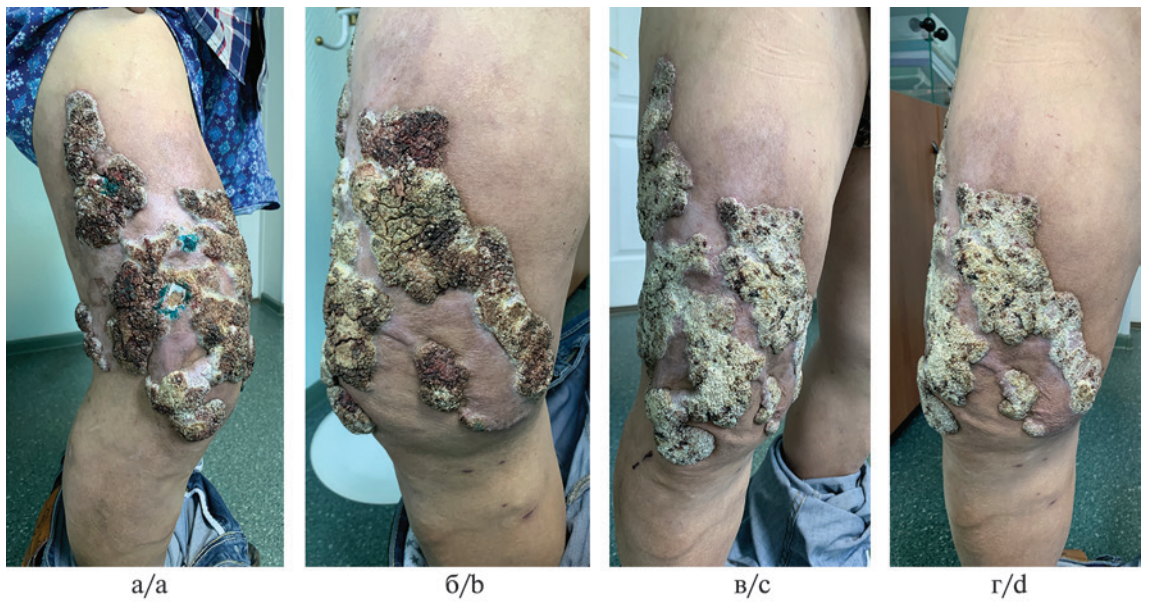


Fig. 3. **Verrucous CM of the skin of the right thigh and knee, anterior and lateral view during pharmacological treatment. a, b — before the treatment, c, d — 1.5 months after the treatment. Inflammation, infiltration, and pain were significantly reduced, and verrucous elements flattened. Restriction of the knee joint mobility has resolved. Step-wise surgical excision is indicated.**



а/a б/b

Fig. 4. **Papillomatous ulcerative CM of the right tibia skin before (a) and after (b) pharmacological treatment. Prepared for surgical treatment.**

nidia (spores) production. When stained by Romanowsky-Giemsa staining, the fungi appear green, while Ziehl–Neelsen stained they are red. There are also molecular biological methods for pathogen detection [1].

The histological pattern of CM is a mixed purulent and granulomatous inflammation of the skin and subcutaneous tissue. In the tissues, the pathogens appear as thick-walled dark brown cells with a double-shaped envelope; they are called sclerotic bodies, but a more accurate term is "muriform cells"; they may be located outside or inside the giant Langhans cells [24]. Many small keratolytic abscesses are filled with these dark-colored spherical fungi and their branching yellow hyphae (mycelium).

Pseudoepitheliomatous and pseudocarcinomatous hyperplasia, hyperkeratosis, and irregular acanthosis alternating with areas of atrophy and infiltrates of inflammatory cells forming epidermal abscesses prevail in the epidermis [25]. In the dermis and subcutaneous tissue, mixed reaction, granulomatous inflammation with varying degrees of fibrosis, clusters of granulocytes, giant cells, Langhans cells, "foreign bodies," and "muriform cells" around microabscesses are observed. At the same time, the acute inflammatory process is minimal despite a pronounced granulomatous inflammation [26, 27].

Treatment of CM lasts from 6 months to 2 years. A combination of medical and surgical treatment is a priority because it gives a favorable outcome in a short time. Conservative antifungal therapy is used before, during and after surgery.

Systemic antifungals (itraconazole and terbinafine, 200—400 and 250—500 mg, respectively), and in recent years, posaconazole 0.1—0.3 g/day has also been used [25, 28—32]. In case of secondary bacterial infection, exacerbation of the inflammatory process, and abscesses, antibacterials are administered for 1-2 weeks. Topical antifungal, antibacterial and antiseptic agents are used in the form of ointments, creams, solutions, sprays and powders **(Fig. 4)**.

The cure rate of CM ranges from 15 to 80%, depending on the severity and duration of the disease. If monotherapy with antifungal drugs is not effective, it is combined with radical surgical excision of the affected tissues,



а/a б/b

Fig. 5. **CM in the knee joint after adequate pharmacological treatment (a) and the result of extensive excision of the lesion within the normal tissue (b).**



а/a б/b

Fig. 6. **CM of the inner surface of the lower third of the right thigh after pharmacological treatment (a), extensive excision of the lesion, and preparation for autodermoplasty (b).**

physical methods (removal of lesions with carbon dioxide laser, cryodestruction, photodynamic therapy, etc.), low-dose acitretin (20 mg/day) and topical imiquimod [33, 34].

The extent of surgical intervention depends on the age of the disease, localization, and spread of the lesions. After preoperative medication preparation, stepwise radical excision of abnormal lesions with minimal inflammatory component and clear delimitation from healthy tissues is performed. An effective combined approach for CM treatment has been developed and is widely used in the Moscow Deep Mycosis Center **(Fig. 5, 6)**.

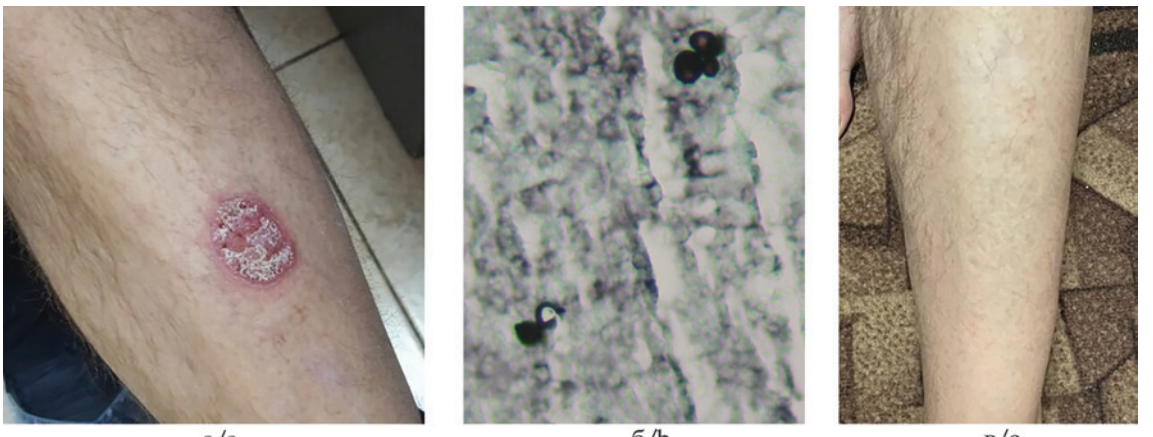
We describe a clinical case of high efficacy of timely treatment in a 73-year-old male with an initial stage of CM.

Patient L., 73 years old, had been ill for 10 months when he first noted an asymptomatic rounded plaque about 2 cm in diameter on the anterior surface of the lower leg. It rapidly increased in size and flaked. He attributed the onset of the disease to his work on a garden plot in Crimea. He consulted a dermatologist at the MHCVD and was referred for admission to the dermatovenerology department of M. F. Vladimirsky Moscow Regional Research Clinical Institute.

On admission: on the skin of the anterior surface of the middle third of the tibia, there was a 2×3 cm pink plaque with gray-white scales on the surface and a red peripheral ridge. Around this lesion, there was irregularly protruding hyperemia up to 2-3 mm from its borders **(Fig. 7)**.

Laboratory test data included the following: HIV, AT to HCV, HbAS, seroreactivity to syphilis (all negative), general urine analysis, complete blood count, biochemical blood analysis, hemostasiogram were within reference ranges.

EKG: sinus rhythm, heart rate 66 beats per minute, first degree atrioventricular block, left anterior fascicular



а/a б/b в/c

Fig 7. **CM of the left tibia. Plaque with gray-white scales, elevated inflammatory infiltrate, and perifocal hyperemia (a). CM pathogen *Fonsecaea pedrosoi* in histological slide (b). No relapse 2years after the disease onset (c).**

block, partial right bundle branch block, changes in the left atrial component, left ventricular myocardial changes.

Histological examination of postoperative material of the excised plaque: pseudocarcinomatous hyperplasia of the epidermis with the presence of intraepidermal microabscesses and acantholytic lesions were observed in the removed skin fragments. In the upper and middle dermis layers (up to the level of sweat glands), productive granulomatous inflammation with an abundance of giant cells and microabscesses was observed. Multiple rounded budding corpuscles with a thick brown sheath were observed in the epidermis and giant multinucleated cells in the dermis. Conclusion: the histological pattern is consistent with skin chromomycosis.

Before the surgery, the patient received itraconazole 100 mg b.i.d. for 1 month, actinolizate 3 mL b.i.w. total 10 injections, supportive therapy, and topical antifungal ointments. After pharmacological treatment, radical surgical excision of the abnormal lesion focus (plaque) on the anterior surface of the left tibia within apparently healthy tissues was performed.

After the surgery, the patient received itraconazole for 2 months, ceftriaxone 2 ml IM b.i.w., omeprazole 20 mg orally QD in the evening, essential phospholipids, and dressings with levomecol.

Discussion

This clinical case is interesting due to the identification of CM in a resident of the Moscow region who did not travel to endemic zones but engaged in agricultural work in Crimea in a moderately continental climate. The probability of such infection is confirmed by the reported CM cases in Central Russia [5—9, 13, 14].

We believe that histological and mycological examinations should be performed in all patients with persistent, poorly treatable, pruritic papulosquamous, and verrucous eruptions, regardless of the immune states.

The used surgical treatment after drug therapy with the aim of complete cure, in this case, was preferable and adequate since the lesion was well separated from healthy tissues, the disease was at the initial stage (disease duration 10 months), and the patient received antimycotic and general supportive therapy for a long time before surgery. In cases of large lesions and widespread forms of CM, in advanced stages and with a long duration of the disease (up to several years), when excising the lesion in combination with further skin grafting, there is a risk of spread of the infection. In addition, it is not possible to excise the affected tissues in a single step and a single block because of the large anatomical spread, so in these cases, long-term antimycotic therapy with terbinafine and itraconazole remains the main treatment, and the surgery is divided into stages or remains adjuvant. Moreover, the above clinical case demonstrates the high effectiveness of timely treatment in the initial stage of the disease after early diagnosis and rational choice of the treatment regimen.

The authors declare no conflict of interest.

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