



CASE REPORT: CHROMOBLASTOMYCOSIS

Melina Handayani^{1*}, Mohamad Mimbar Topik²

¹Department of Medicine, Faculty of Medicine, Malikussaleh University, Lhokseumawe, Indonesia

²Department of Dermatology & Venerology, Faculty of Medicine, RSUD Cut Meutia, Aceh Utara, Indonesia

*Corresponding Author: E-mail: melinlinh19@gmail.com

ABSTRACT

Background: Chromoblastomycosis is a recurring deep mycosis of the skin and subcutaneous towel. Clinical features are characterized by verrucous shrine lesions, especially in the lower extremities. This disease is usually preceded by trauma in the form of a wound which is the initial implantation of the fungus. The fungus will be implanted in the dermis and will infect the skin with the subcutaneous towel. Chromoblastomycosis is caused by a fungus from the Dematiaceae family. Although Chromoblastomycosis occurs worldwide, it is most common (>70%) in tropical and subtropical regions. **Case Presentation:** We present a case in which a 72-year-old man came to the dermatovenereology polyclinic with the main complaint of thickening of the skin in the plantar of the right leg, the surface is rough, the skin looks dry, it doesn't itch and it doesn't hurt, it gets worse over time, widened. Based on the histopathology, the appearance of the epidermis in the form of pseudoepitheliomatous hyperplasia, hyperkeratosis, acanthosis, microabscesses with neutrophil inflammatory cells, lymphocytes, multinucleated giant cells, and spores. In the dermis, microabscesses were found, and no necrosis was seen. **Conclusion:** The diagnosis was chromoblastomycosis. This patient was given oral antimycotic Itraconazole 2 x 200 mg/day. There was a clinical improvement.

Keywords: *chromoblastomycosis; mycosis; hyperkeratosis*

INTRODUCTION

Chromoblastomycosis was first reported by Alexandrio Pedroso in 1911 in Brazil. Chromoblastomycosis is an infection caused by a pigmented (dematiaceous) fungus that originates from the girding terrain. The fungus will be implanted in the dermis and will infect the skin to the subcutaneous towel. This complaint is generally anteceded by trauma in the form of a crack which is the original implantation of the fungus. The course of chromoblastomycosis is habitual.¹

The organisms that beget chromoblastomycosis are saprophytic fungi set up in soil, wood, vegetative shops, and slush. The most common causative organisms include *Fonsecaea pedrosoi*, *F. compacta*, *Rhinoctadiella aquaspersa*, *Cladosporium carrionii*, and *Phialophora verrucosa*. The fungus that causes chromoblastomycosis can be insulated from wood, shops, and soil.²

Although Chromoblastomycosis occurs worldwide, it is most common (>70%) in tropical and subtropical regions. The country with the loftiest frequency rate is Madagascar, Africa. But there are also many cases in Latin America. Chromoblastomycosis has also been reported in Asia, Australia, and several European countries.³

Chromoblastomycosis is usually anteceded by a

trauma, which also results in inoculation of the causative fungus at the point of trauma. The frequency of this complaint is especially in individualities who work barefoot similar as growers, lumberjacks, or diurnal sloggers. The frequency of this complaint is loftiest in men than in women with an age range of 30-60 years.⁴

The clinical donation of Chromoblastomycosis is variable; the original lesion is frequently a sluggishly spreading verrucous pustule or an effortless bump. These early lesions can develop into polymorphic skin lesions. The opinion of Chromoblastomycosis is verified by the presence of muriform cells in the towel and the insulation of the causative agent by culture.⁵

Diagnosis and treatment of Chromoblastomycosis remains a mission for clinicians due to the recalcitrant nature of the grievance, mainly in its extreme form. Beforehand opinion of early lesions is veritably important and affects the prognostic of this complaint.¹

CASE REPORT

A 72-year-old man came to the dermatology and venerology polyclinic at Cut Meutia Hospital with the main complaint of thickening of the skin in the instep area, dry-looking skin, rough surface, no itching, and no pain. This complaint has been felt ±1



Melina Handayani, Mohamad Mimbar Topik

year ago. At first small spots appear, then the patient scratches them so that the surface peels off and causes sores, then the skin dries with a rough surface, progressively widens, and thickens so that complaints like now arise. There was no fever and history of previous fever. Shortness of breath, cough, and chest pain are absent. Appetite within normal limits, weight loss is denied, and night sweats are denied.

On physical examination found composentis mindfulness, blood pressure 120/80 mmHg, pulse 89 times per minute, respiratory rate 22 times per minute, body weight 80 kg, height 170 cm. Dermatological status in the pedis region found plaques with a verrucous surface, grayish in color, blackish brown spots above, hyperkeratosis, localized distribution, size 9 cm x 6 cm, serpiginous spreading, no enlarged glands seen.

The differential diagnosis in this patient is Chromoblastomycosis, TB cutis verrucosa, and verruca vulgaris. The supporting examination is a skin biopsy for histopathological examination. Based on the results of the histopathological examination obtained a description of the epidermis in the form of pseudoepitheliomatous hyperplasia, hyperkeratosis, acanthosis, microabscesses with inflammatory cell inclusions of neutrophils, lymphocytes, multinucleated giant cells, and spores. In the dermis found microabscesses, but no necrosis was found.

Based on the history, physical examination, dermatology, and supporting examinations, the working diagnosis in this patient is Chromoblastomycosis. Patients and families have explained the disease, its causes, treatment options, prevention, and education for foot skin hygiene. The patient was advised to return to the dermatovenereology polyclinic at Cut Meutia Hospital 1 week later to evaluate the treatment.

DISCUSSION

Chromoblastomycosis is a fungal infection of the skin, frequently caused by colorful pigmented fungi (dematiaceous) similar as *Fonsecaea pedrosoi*, *Fonsecaea compactum*, *Wangiella dermatitidis*, *Phialophora verrucosa*, and *Cladophialophora carrionii*. The majority of infections are caused by *F. pedrosoi*.⁶

In this case, the patient is a 72-year-old man. Based on the literature, Chromoblastomycosis

generally occurs in countries with tropical climates and is predominantly found in men, especially at the age of 30-60 years, and infrequently set up in women or children. The ratio of the incidence of chromoblastomycosis in men and women is 4:1, this is because men work more and the type of work is often outdoors so the risk of contact with the fungus that causes chromoblastomycosis is higher.⁷

Based on the literature, individuals who are susceptible to Chromoblastomycosis are those whose work is often in contact with soil or vegetables, and farmers account for nearly 75% of patients with this disease. This is because the fungus that causes Chromoblastomycosis is saprophytic and can live in soil, rotting vegetables, or on wood chips which are the normal habitat of this fungus. The most common site of predilection for chromoblastomycosis is the feet. However, the disease can also involve other exposed areas such as the hands, trunk, buttocks, neck, and face.⁷

The patient's main complaint in this case was skin thickening in the plantar area, the skin looks dry, the surface is rough, it doesn't itch and it doesn't hurt. At first small spots appear, then the patient scratches them so that the surface peels off, then the skin dries out with a rough, gray surface with black spots on it, getting thicker and wider over time. The clinical symptoms found in chromoblastomycosis vary, but the most common are nodules which then expand slowly to become spots and lumps with a verrucous surface (rough surface). Sometimes found cauliflower-like florets. In chronic lesions, it can even spread to the bone layer under the skin, causing osteolysis, and can spread hematogenously and lymphogenously, accompanied by elephantiasis⁸, or surrounded by a reddish halo area and black spots. This sign corresponds to the patient's, which is in the form of a grayish patch with a rough surface and has been getting bigger since 1 year ago on the right instep.⁴

The emergence of clinical symptoms due to an infectious process that occurs through traumatic inoculation of the causative agent into the host tissue such as through a thorn or wood splinter, which is often not realized by the patient. After the pathogen reaches the towel, the fungus undergoes a metamorphosis from a filamentous form to a parasitic form, known as fumagoid cells/muriform bodies. Mature fumagoid cells suffer fragmentation



Melina Handayani, Mohamad Mimbar Topik

and new fumagoid cells are formed from each scrap. When phagocytic cells (macrophages and polymorphonuclear cells) are unfit to destroy the parasitic form of this fungus, it'll beget a thick stringy response in the dermis and subcutaneous towel which originally serves as a host defense medium to help complaint development. The result is a thickening of the case's skin.²

An extensive fibrotic process assisted by a chronic inflammatory infiltrate and increasing secondary infection can lead to impaired lymphatic flow and lymphedema beneath the affected area. Circulatory disorders due to chronic lymphedema can then lead to skin and soft tissue degenerate, deformity, and joint ankylosis which ends in permanent disability.¹

The host defense mechanism in chromoblastomycosis is not fully understood. Research shows that CD4+ lymphocytes are the main cells for controlling Chromoblastomycosis. Cellular immunity in patients with chronic Chromoblastomycosis is impaired so that the individual is unable to form an adequate immune response against fungal antigens resulting in the persistence of the fungus in the tissues.¹

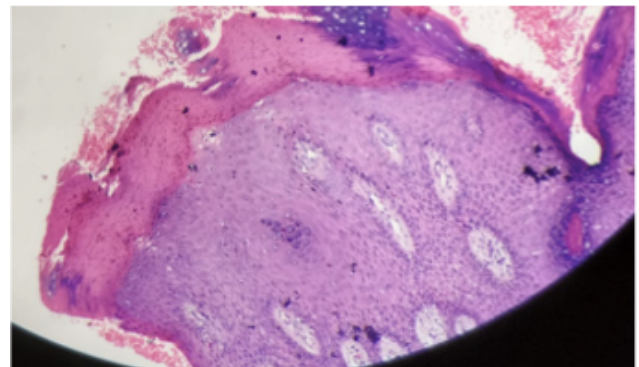
In this case, the patient belongs to the elderly group, in which the group has experienced a decrease in physiological function, including a decrease in the resistance of the immune system. The theory of immunodeficiency states that with increasing age, the body becomes increasingly difficult to defend itself from pathogens. When there is a pathogen attack, the immune response that is formed is inadequate. This can make the patient susceptible to various infections, including yeast infections.⁹

In the literature, the histopathological picture obtained can be in the form of pseudoepitheliomatous hyperplasia in the epidermis, lymphomononuclear infiltrates in the dermis, neutrophil infiltration characterized by microabscess formation, granulomas with giant cells found in the middle found fungal cells. In the abscess, brown sclerotic cells with a round shape are also found, which is a sign that is very specific for chromoblastomycosis.^{1,10}

The histopathological picture, in this case, showed that the epidermis was found with pseudoepitheliomatous hyperplasia, hyperkeratosis, acanthosis, and microabscesses with inflammatory

cells, neutrophils, lymphocytes, multinucleated giant cells, and spores. There are microabscesses in the dermis, but no necrosis was seen (picture 1). So that the diagnosis of Chromoblastomycosis can be enforced.

In the literature, the histology is that of a foreignbody granuloma with insulated areas of microabscess conformation. In the organized granuloma, substantially within giant cells, groups of fungal cells may be seen. They're groaner or golden brown and thus can be fluently distinguished in the insinuate. The cells characteristically divide into several aeroplanes of division by thick septa and are nominated muriform or sclerotic cells. There's pronounced pseudoepitheliomatous hyperplasia of the epidermis. In some areas, apparent transepidermal elimination of fungal cells, which are in the stratum corneum. The towel between the granulomatous nodes shows habitual fibrosis. When ulceration has passed, there's generally a secondary bacterial infection.²



Picture 1. Pseudoepitheliomatous hyperplasia, hyperkeratosis, acanthosis, microabscesses with inflammatory cells, neutrophils, lymphocytes, multinucleated giant cells, and spores.

This patient was given oral antimycotic Itraconazole 2 x 200 mg/day. Itraconazole is a broad-spectrum antifungal agent; it has an active metabolite; hydroxyitraconazole. Itraconazole inhibits ergosterol conflation, which helps maintain the cell membrane in fungi. Lanosterol must suffer a 14 alpha-demethylation response to become ergosterol, which is catalyzed by fungal 14 alpha-demethylase. Itraconazole blocks this response by interacting with the fungal 14 alpha-demethylase substrate-binding point. This disabled ergosterol conflation leads to fungal membrane abnormalities that increase permeability and disrupt fungal cell

Melina Handayani, Mohamad Mimbar Topik

membrane integrity, changing membrane-bound enzyme exertion.²

Based at the literature the primary remedies for chromoblastomycosis are Itraconazole 200 mg daily, Terbinafine 250 mg each day, and in extensive cases amphotericin B IV (up to 1 mg/kg day by day). The lesions can spread surgically, so this option is only used as adjunctive therapy after drug therapy. The response of these fungi to other antifungal sellers did not look like drastically extraordinary despite the fact that there is a few evidence that *C. Carrionii* replied extra swiftly to Terbinafine and Itraconazole. Remedies have to be persisted till there's medical decision of the lesions, normally numerous months. Giant lesions frequently do now not respond to conventional treatment, so a combination of antifungal drugs can be an option.¹¹



a.



b.

Picture 2. a. Before treatment, b. after 1 week of treatment

The prognosis for Chromoblastomycosis is generally good, especially for small-to-moderate and localized lesions. If the affected area is large enough, healing is difficult to achieve and recurrences may occur even if the disease is controlled. Death from chromoblastomycosis is a rare event. Morbidity is immediately associated with the severity of the disease. In the papular or nodular phase, the disease is asymptomatic. However, when the nodules coalesce, they can form large plaques and sometimes can involve the whole body, which allows complications to arise. Complications generally

include ulceration, lymphedema, and secondary infection.¹

CONCLUSION

One case of Chromoblastomycosis has been reported in a 72-year-old man. The diagnosis is based on anamnesis which obtained the main complaint in the form of thickening of the skin in the instep of the right leg, the surface is rough, the skin looks dry, it doesn't itch and it doesn't hurt, it gets wider and wider.

Based on clinical examination, it was found in the form of a grayish plaque with verrucous surface, blackish brown spots above, hyperkeratosis, localized distribution, size 9 cm x 6 cm, serpiginous spreading, no enlarged glands seen.

Based on the histopathology obtained, epidermal features include pseudoepitheliomatous hyperplasia, hyperkeratosis, acanthosis, microabscesses with inflammatory cell inclusions of neutrophils, lymphocytes, multinucleated giant cells, and spores. In the dermis, microabscesses were found, and no necrosis was seen.

This patient was given oral antimycotic Itraconazole 2 x 200 mg/day. There was a clinical improvement.

REFERENCES

1. Horo R, Made L, Rusyati M, Luh N, Ratih P, Karna V. Kasus kromoblastomikosis yang awalnya diduga furunkel pada seorang perempuan. *Intisari Sains Medis*. 2022;13(1):362–6.
2. de Brito AC, Bittencourt M de JS. Chromoblastomycosis: An etiological, epidemiological, clinical, diagnostic, and treatment update. *An Bras Dermatol*. 2018;93(4):495–506.
3. Santos DWCL, de Azevedo C de MP e. S, Vicente VA, Queiroz-Telles F, Rodrigues AM, de Hoog GS, et al. The global burden of chromoblastomycosis. *PLoS Negl Trop Dis*. 2021;15(8).
4. Intan SA, Agus S, Putriyuni A. Kromomikosis yang menyerupai karsinoma sel skuamosa: suatu kasus jarang. *Maj Kedokt Andalas*. 2018;41(3):112.
5. Santos DWCL, Vicente VA, Weiss VA, Sybren de Hoog G, Gomes RR, Batista EMM, et al. Chromoblastomycosis in an



Melina Handayani, Mohamad Mimbar Topik

- endemic area of brazil: A clinical-epidemiological analysis and a worldwide haplotype network. *J Fungi*. 2020;6(4):1–14.
6. Qomariah LN, Purwanto H, Damayanti N, Siswati AS. Case Report Diagnosis and therapy of chromoblastomycosis. 2022;32(2):443–8.
 7. Rositawati A, Suyoso S. Chromomycosis Treatment With Combination Of Itraconazole And Terbinafine. *Period Dermatology Venereol*. 2017;29(2):168–73.
 8. Lasut M V., Tanamal RS, Kapantow GM. Kasus Kromoblastomikosis Pada Seorang Perempuan. *J Biomedik*. 2015;7(1):62–9.
 9. Marisa YT, Mulyana R. Infeksi Jamur Pada Geriatri. *Hum Care J*. 2020;5(1):328.
 10. Sharma K, Negi SR, Joshee R, Rathore K. Cutaneous Chromoblastomycosis: A Case Report. *J Med Sci Heal*. 2016;02(03):36–9.
 11. Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K. *Fitzpatrick's Dermatology in General Medicine*. 8th ed. Mc Graw Hill; 2012.