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Case report

Successful treatment of chromoblastomycosis using ALA-PDT in a patient with leukopenia

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ABSTRACT

Chromoblastomycosis is a long-term fungal infection of the skin and subcutaneous tissue, usually presenting as partial hypertrophic and warty plaques. Effective treatment is necessary to control the development of lesions, especially in patients with associated diseases. But till now, the treatment is still a challenge. Photodynamic therapy (PDT) is an efficient and non-invasive treatment option. Here, we reported the case of a 52-year-old male with refractory chromoblastomycosis and leukopenia, who was successfully treated with 5-aminolevulinic acid-based PDT (ALA-PDT). A complete cure, confirmed by clinical improvement and mycological detection, was achieved after six sessions of every-other-week treatment. Post six months follow up no recurrence was observed. The case here suggests that ALA-PDT is a valuable anti-infective therapy for refractory chromoblastomycosis.

1. Introduction

Chromoblastomycosis (CBM) is a chronic implantation fungal infection of the skin and subcutaneous tissue, caused by melanized or brown-pigmented fungi [1]. The characteristic lesions of CBM usually present as partial hypertrophic and warty plaques. We reported the case of a 52-year-old male with refractory CBM and leukopenia, who was treated by 5-aminolevulinic acid-based photodynamic therapy (ALA-PDT). After six sessions of ALA-PDT, the lesions had disappeared entirely.

The case here is evident to improve ALA-PDT is a valuable anti-infective therapy for refractory CBM.

2. Case report

A 52-year-old man was admitted to our hospital with a complaint of long-term verrucous hyperplasia on his left leg (Fig. 1a), which was evolving over the last two years. Physical examination showed multiple, scaly erythematous plaques with erosion and ulcer on the left shin, approximately 10 × 7 cm. The affected individual was also associated with leukopenia, for the white cell count was less than 1×10^9/L for three times. The family history was unremarkable. He was received intermittent antibiotic treatment previously, but without significant improvement. The histological analysis showed hyperplasia and chronic inflammation of the cutaneous, and dark-brown spores were in the dermis (Fig. 2a). Muriform cells were observed through direct mycological examination from the lesion, showing as typical round, brown color, separated thick-walled cells, which was the recognizable feature of CBM (Fig. 2b). Tissue biopsy conducted by culturing the tissue in the Sabouraud's dextrose agar plates showed slow growing, velvet-like, dark brown colonies. Under microscopy, it was visible of dark conidiophores and ovoid conidia located either at the end or at the side of conidiophore (Fig. 2c). DNA was extracted from the cultured colonies, and the base sequence of ITS was identified, which was matched with *Fonsecaea pedrosoi*.

Considering the clinical manifestation and laboratory examinations, the diagnosis of CBM and leukopenia was made. He underwent antifungal therapy (Itraconazole 400 mg/day) for two months, but without visible improvement. Then we treated this patient with ALA-PDT for six sessions, with a one-week interval. The lesions were incubated with freshly prepared 10% ALA cream (Shanghai Fudan-Zhangjiang Bio-Pharmaceutical Co. Ltd., Shanghai, China) for 4 h, and the 633 ± 10 nm red light with the intensity of 80–100 mW/cm^2 was used as the irradiation source for 25 min. Six weeks later, the lesions appeared as thinning plaques, with partial healing ulcers (Fig. 1b). A complete cure, confirmed by clinical improvement and mycological detection, was achieved after six sessions of every-other-week treatment (Fig. 1c). Post six months follow up no recurrence was observed. The case here suggests that ALA-PDT is a valuable anti-infective therapy for refractory CBM.

As one of the neglected tropical diseases, CBM is hard to cure and easy to be recurrent. Previously, we successfully treated two cases of

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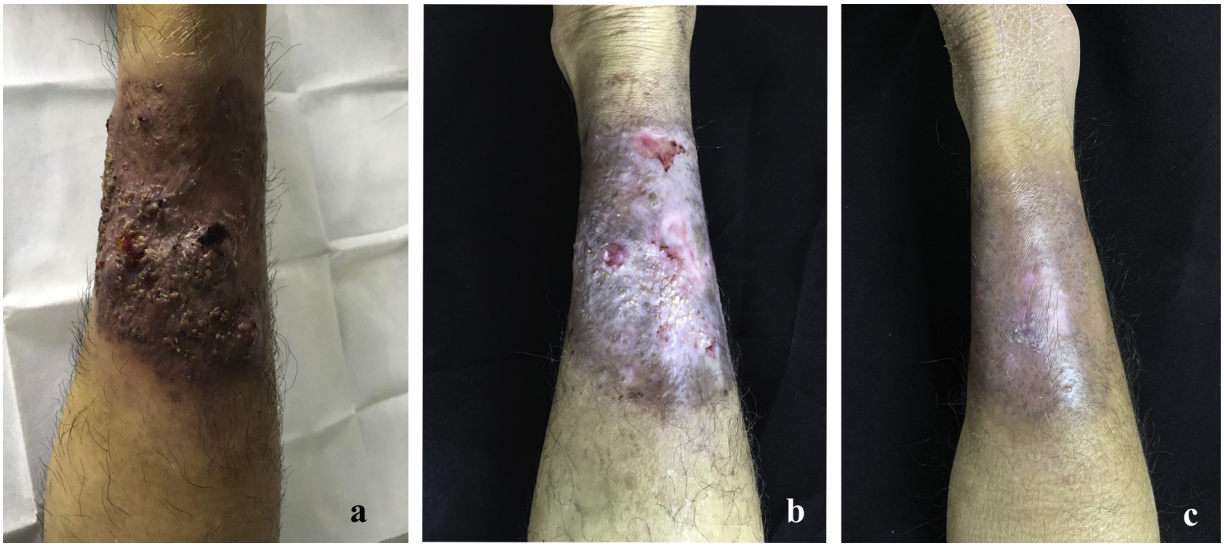

Fig. 1. Lesions on the left thin before treatments with ALA-PDT (a), after three sessions of ALA-PDT (b), two months after six sessions of ALA-PDT (c).

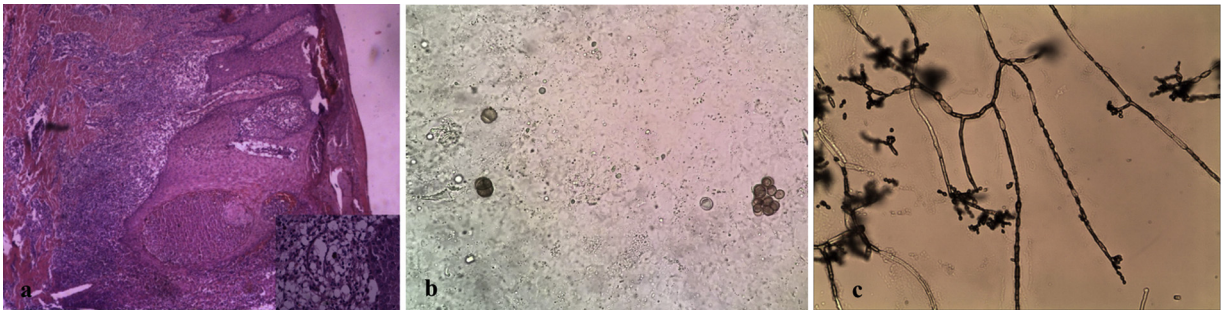

Fig. 2. The biopsy specimen showed pseudoepitheliomatous hyperplasia and chronic granulomatous inflammation, and dark-brown spores in the upper dermis (H&E, ×40) (a). Direct mycological examination showed large and brown separated muriform cells from the lesion (KOH, ×400) (b). Brownish conidia from conidophore in the slide culture of *F. pedrosoi* (×400) (c).

CBM by ALA-PDT combined with terbinafine 250 mg/day [2,3]. And in the following studies, the fungicidal effect of ALA-PDT against the pathogen was shown great promise [2]. And it also demonstrated that ALA-PDT inactivated *F. monophora* (one of the primary causative agent of CBM) through directly killing and ROS-dependent oxidative damage in macrophages [4]. The present case applied ALA-PDT subsequent with the first-line antifungal drug, itraconazole, and showed a positive effect. It's the preferred therapy in refractory cases of CBM.

Declaration of interest

None declared.

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