

## Topic 10: Chromoblastomycosis

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### Chromoblastomycosis

A primary (1<sup>o</sup>), chronic, localized granulomatous mycosis of the skin and subcutaneous tissue characterized by verrucoid, ulcerated and crusted lesions that may be flat or raised and is caused by 4 (possibly 6) form-species of Dematiaceae

- \*1. *Phialophora verrucosa*\*
- \*2.3. *Fonsecaea pedrosoi*\* & *F. compacta*\*
- \*4. *Cladophialophora carrionii*\*
- ?5. *Rhinocladiella aquasperma*\*

# of cases in literature to 1974 = 1,970; 24 in U.S.

#survey between 1980 & 1982 suggest ~ 25 cases/year U.S.

Costa Rica ~1 case/12,000 people

U.S. ~ 1 case/12 million people

Many S. American cases reported in Brazil, Venezuela, State of Falcon, Venezuela 16/1000; 1<sup>st</sup> case in China reported 1951, ~ 400 since, 300 in Sandong. Also Australia, Thailand, etc.

\*Tissue dimorphism characterized by the production of "sclerotic" bodies in subcutaneous granuloma: agents considered to be hyphomycetous molds.

Tissue morphologies usually sclerotic bodies

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### History - Chromoblastomycosis

- 1915 Medlar & Lane -U.S. case *Phialophora verrucosa* (Taxter)
- 1914 Rudolph - Brazilian case (probably 1st case, but fungus etiology not established)
- 1920 Pedroso & Gomes -4 more Brazilian cases *Fonsecaea pedrosoi*
- 1936 Carrion *Fonsecaea compacta* Puerto Rico
- 1937 Kano *Wangiella dermatitidis*\* (*Exophiala dermatitidis*\*)
- 1954 Trejos *Cladophialophora carrionii*\*\* Isolates originally from Australia & S. Africa
- 1972 Borelli *Rhinocladiella aquasperma*  
etc?

\*Now considered one of main agents of phaeohyphomycosis; originally named *Hormscium dermatitidis* by Kano (Japan)

\*\*Most have changed name from form-genus *Cladosporium*

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### Pathology

1. **trauma or puncture wound → site of initial lesion**
2. **initially small, raised erythematoid papule, which is rarely pruritic (itchy)**
3. **papules or pustules become more violaceous and have modest cell infiltration**
4. **lesions next often become scaly (fungi in lesions may be present as distorted hyphal elements, from which sclerotic bodies develop)**
5. **lesions may become raised (1-3 >mm) and begin to coalesce (possibly into large eruptive masses 3 or > cm) into cauliflower-type skin structures resulting from extensive granuloma formations in skin and subcutaneous tissues.**
6. **invasion of new areas occurs at slow chronic rate (mechanisms of spread unclear)**

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### Diagnosis of Subcutaneous Chromoblastomycosis

1. **observations of characteristic lesions**
2. **observations of sclerotic bodies, golden brown, thick-walled, muriform structures (usually 10 µm or more in diameter with, from two-to- many, vertical and horizontal septa) in granulomatous structures**
3. **culture of fungus and identification by observing conidial structures**
4. **serodiagnosis poor, but can be done by personnel at CDC**

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### Therapy

1. **surgery - early lesions**
2. **medical management or supportive therapy\***
3. **antimycotics for advanced cases; EBIs, e.g. Itraconazole, or whatever works\*\***

\*including antibacterial therapy for secondary infections if required.

\*\* new treatment for children, topical 5-FU (an antineoplastic cream) & topical Ajoene (alcoholic extract of onion, as reported in Med. Myco. 44; 467, 2006)

The subcutaneous mycoses are characterized by:

1. development of lesions at the site of inoculation
2. being initiated by traumatic implantation of the fungus into the skin or subcutaneous tissue
3. limited spread which, if it occurs, is usually associated with the lymphatics or autoinoculation and not hematogenous spread
4. by the tendency to be considered primary chronic infections of normal hosts and systemic in compromised hosts.

Focus today: Subcutaneous Dematomycoses

Chromoblastomycosis

Phaeohyphomycosis

Dematiaceous Mycetoma