Chromoblastomycosis

A primary (1°), 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; 1st 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

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 orginally from Australia & S. Africa**

1972 **Borelli *Rhinocladiella aquasperma***
etc?
***Now considered one of main agents of phaeohyphomycosis; originally named *Hormiscium dermatitidis* by Kano (Japan)**
****Most have changed name from form-genus *Cladosporium***

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)**

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**

5. **molecular IDs possible by some groups**

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 trearment 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 tendancy to be considered primary chronic infections of normal hosts and systemic in compromised hosts.**
5. **often, but not exclusively, caused by dematiaceous fungi.**
6. **etc. see notes page 46.**

Focus: Subcutaneous Dematomycoses*
Chromoblastomycosis
Phaeohyphomycosis
Dematiaceous Mycetoma

***diseases caused by melanized (black/dematiaceous) fungi.**