MYCOLOGY - CHAPTER SIX

DIMORPHIC FUNGI

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WEB RESOURCES
CDC-Blastomycosis

BLASTOMYCOSIS (*Blastomyces dermatitidis*)

Most of the systemic fungi have a specific niche in nature where they are commonly found. *Blastomyces dermatitidis* survives in soil that contains organic debris (rotting wood, animal droppings, plant material) and infects people collecting firewood, tearing down old buildings or engaged in other outdoor activities which disrupt the soil. In addition to an ecological niche, most fungi that cause systemic infections have a limited geographic distribution where they occur most frequently. Blastomycosis (figures 1- 4)occurs in eastern North America (figure 5) and Africa. The vast majority of patients with blastomycosis in South Carolina are infected in the northern part of the state, above the Fall Line (Augusta, GA, Aiken, Columbia, Cheraw, Raleigh, NC).


Figure 1
Skin lesions of blastomycosis.

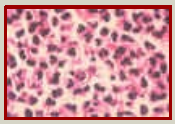


Figure 2.
Histopathology of blastomycosis of skin. Budding cell of *Blastomyces dermatitidis* surrounded by neutrophils. Multiple nuclei are visible. CDC

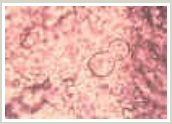


Figure 3.
Smear from foot lesion of blastomycosis showing *Blastomyces dermatitidis* yeast cell undergoing broad-base budding. ASCP/Atlas of Clinical Mycology II / CDC


Figure 4.
Histopathology of blastomycosis. Yeast cell of *Blastomyces dermatitidis* undergoing broad-base budding. Methenamine silver stain. African case. CDC


Figure 5
Map of eastern United States and Canada showing distribution of reported cases of blastomycosis. CDC

Blastomycosis is a chronic granulomatous disease which means that it progresses slowly. Although the pulmonary and skin (figure 1) involvement is the most common, *B. dermatitidis* frequently affects bone, prostate and other organs. More frequently blastomycosis presents as a cutaneous or a respiratory disease. The cutaneous lesions may be primary (usually self-limiting) or secondary (a manifestation of systemic disease). The patient who presents with a complaint of respiratory symptoms will frequently remark about loss of appetite, loss of weight, fever, productive cough, and night sweats. While these symptoms resemble those of TB, it is not this disease. The X-ray (figure 6) shows obvious pulmonary disease. To make the specific diagnosis, the physician must be aware of blastomycosis. Sputum sent to the lab for "culture" will not grow the organism. The lab must be alerted to look for fungal organisms or to look specifically for blastomyces. Some patients have a sub-clinical or "flu-like" response to infection. *B. dermatitidis* can frequently be demonstrated in a KOH preparation of pus from a skin lesion. A typical cutaneous lesion

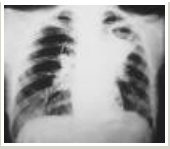


Figure 6
Blastomycosis
This AP chest X-ray demonstrates lung infiltrates due to Blastomycosis, caused by *Blastomyces dermatitidis*. A symptomatic blastomycosis infection (50% of cases) usually presents as a flu-like illness with fever, chills, productive cough, myalgia, arthralgia and pleuritic chest pain.
CDC/Dr. Hardin

shows central healing with microabscesses at the periphery. A pus specimen may be obtained by nicking the top of a microabscess with a scalpel, obtaining the purulent material and making the diagnosis in 5 min. by microscopic examination with KOH. This organism has a characteristic appearance of a double contoured wall with a single bud on a wide base (figures 2 - 4). There are no specific virulence factors for *B. dermatitidis.* Laboratory specimens depend on the manifestation of the disease: If there are skin lesions, send skin scrapings or pus. If there is pulmonary involvement, send sputum. Other specimens include biopsy material and urine. Occasionally, the organism can be isolated from urine as it often infects the prostate.

Mycology

If you request a fungus culture from the microbiology lab, they will incubate the cultures at 37 degrees C and at 25 degrees C because most of the significant pathogenic fungi are dimorphic.

A culture of *B. dermatitidis* takes 2 to 3 weeks to grow at 25 degrees C. It appears as a white, cottony mold (mycelium) on Sabouraud dextrose agar. Most specimens for fungus culture are plated on Sabouraud's dextrose agar. Microscopically, the mycelia and the fruiting bodies are evident. However, the mold cannot be identified by its fruiting bodies. The fruiting bodies are called microconidia, but they are not distinctive. Other fungal saprophytes and pathogens have similar conidia. At 37 degrees C the yeast form grows in about 7-10 days. It appears as a buttery-like, soft colony with a tan color. Microscopically, we see the typical yeast form of a thick wall and a single bud with a wide base. This wide base is characteristic of *B. dermatitidis*, and it is important to be able to recognize this. The cells are 12-15 microns in diameter. The yeast will convert to the mycelial form when incubated at 25 degrees C, taking from 3 to 4 days up to a few weeks. Similarly, the mycelial growth can be converted to yeast form when incubated at 37 degrees C. In the past, the only way to identify the dimorphic fungi was to convert from one form to the other, but now it is possible to take the mycelial growth (which is the easiest to grow), and confirm the isolate with a DNA probe in a matter of hours.

Histopathology

B. dermatitidis produces both a granulomatous and suppurative tissue reaction

Serology

There are three serological tests used for blastomycosis:

- Immunodiffusion test (precipitin). This requires 2 to 3 weeks to become positive. This test is positive in about 80% of the patients with blastomycosis. When it is positive, there is close to 100% specificity.
- Complement fixation (CF) test. This test requires 2 to 3 months after the onset of disease to develop detectable antibody. Besides the long delay before there is measurable antibody, another disadvantage of the C-F is that it cross reacts with other fungal infections (coccidioidomycosis and histoplasmosis). The advantage is that it is a quantitative test. The physician can follow the patient's response to the disease by monitoring the antibody titer.
- Enzyme Immunoassay (EIA). The latter test has met with mixed acceptance by mycologists. However it is easy to perform and antibody is detected early in the disease process.

Amphotericin B remains is the drug of choice (DOC) although it is very toxic and must be administered intravenously for several weeks. Itraconazole is also being used in mild cases. Voriconazole is showing promise.

HISTOPLASMOSIS (*Histoplasma capsulatum*)

Histoplasmosis is a systemic disease, mostly of the reticuloendothelial system, manifesting itself in the bone marrow, lungs (figure 12-17), liver, and the spleen. In fact, hepatosplenomegaly is the primary sign in children, while in adults, histoplasmosis more commonly appears as pulmonary disease. This is one of the most common fungal infections, occurring frequently in South Carolina, particularly the northwestern portion of the state. The ecological niche of *H. capsulatum* is in blackbird roosts, chicken houses and bat guano. Typically, a patient will have spread chicken manure around his garden and 3 weeks later will develop pulmonary infection. There have been several outbreaks in South Carolina where workers have cleared canebrakes which served as blackbird roosts with bulldozers. All who were exposed, workers and bystanders, contracted histoplasmosis. Histoplasmosis is a significant occupational disease in bat caves in Mexico when workers harvest the guano

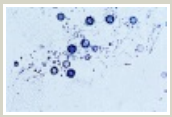


Figure 7
Spores of Histoplasma
Dr Arthur DiSalvo

for fertilizer. In the endemic area the majority of patients who develop histoplasmosis (95%) are asymptomatic. The diagnosis is made from their history, serologic testing or skin test. In the patients who are clinically ill, histoplasmosis generally occurs in one of three forms: acute pulmonary, chronic pulmonary or disseminated. There is generally complete recovery from the acute pulmonary form (another "flu-like" illness). However, if untreated, the disseminated form of disease is usually fatal. Patients will first notice shortness of breath and a cough which becomes productive. The sputum may be purulent or bloody. Patients will become anorexic and lose weight. They have night sweats. This again sounds like tuberculosis, and the lung x- ray also looks like tuberculosis, but today radiologists can distinguish between these diseases on the chest film (histoplasmosis usually appears as bilateral interstitial infiltrates). Histoplasmosis is prevalent primarily in the eastern U.S. In S.C., a histoplasmin skin test survey of lifetime, one county residents, white males, 17 to 21 years old, was performed on Navy recruits. The greatest number of positive skin tests appeared in the northwestern part of the state. A similar study of medical students conducted at Medical University of South Carolina, about 25 years ago, bore the same distribution.The skin test is NOT used for diagnostic purposes, because it interferes with serological tests. Skin tests are used for epidemiological surveys.

Clinical specimens sent to the lab depend on the presentation of the disease: Sputum or Bronchial alveolar lavage, if it is pulmonary disease, or biopsy material from the diseased organ. Bone marrow is an excellent source of the fungus, which tends to grow in the reticulo-endothelial system. Peripheral blood is also a source of visualizing the organism histologically. The yeast (figures 7-11) is usually found in monocytes or in PMN's. Many times an astute medical technologist performing a white blood cell count will be the first one to make the diagnosis of histoplasmosis. In peripheral blood, *H. capsulatum* appears as a small yeast about 5-6 microns in diameter. (Blastomyces is 12 to 15 microns). Gastric washings are also a source of *H. capsulatum* as people with pulmonary disease produce sputum and frequently swallow their sputum.

Mycology

When it is grown on Sabouraud dextrose agar at 25 degrees C, it appears as a white, cottony mycelium after 2 to 3 weeks. As the colony ages, it becomes tan. In the mold form, *Histoplasma* has a very distinct spore called a tuberculate macroconidium (figure 7). The tubercles are diagnostic, however there are some non-pathogens which appear similar. A medical mycologist will be able to distinguish them. Grown at 37 degrees, C the yeast form appears. It is a white to tan colony. The yeast cell is 5-6 microns in diameter and slightly oval in shape. This is not diagnostic. To confirm the diagnosis, one must convert the organism from yeast to mycelium or vice-versa or use the DNA probe.

Serology

Serology for histoplasmosis is a little more complicated than for other mycoses, but it provides more information than blastomycosis serology.

There are 4 tests:

- Complement Fixation
- Immunodiffusion
- EIA (antibody)
- EIA (antigen)

Each of these serological tests has different characteristics that make them useful.

The complement fixation test is like the one for blastomycosis, except there are 2 antigens, one to the yeast form of the organism and the other to the mycelial form. Some patients react to one form and not the other, while some individuals react to both. The reason for the different responses is not clear. One disadvantage is that complement fixing antibody develops late in the disease, about 2 to 3 months after onset. A second disadvantage is that it cross reacts with other mycotic infections. An advantage of the C-F test is that it is quantitative, so the physician can follow the course of the disease by observing the titer of several samples. The interpretation of the immunodiffusion test is a little more complicated than with blastomycosis because there are two bands which may appear. An H band indicates active disease and will appear in 2 to 3 weeks. An M band can indicate past or present disease, or result from a skin test. This is one reason why skin tests are not used for diagnosis because they can interfere with other tests. Skin tests will also affect the complement fixation test.

Recently, a radioimmunoassay for histoplasma polysaccharide antigen has been developed. This is a proprietary test so the evaluation of the results have been questioned. The drug of choice (DOC) is amphotericin B, with all its side effects. Itraconazole is now also being used for mild cases.

WEB RESOURCES
CDC-Histoplasmosis

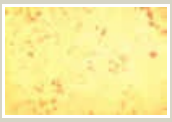


Figure 8.
Histopathology of histoplasmosis showing yeast forms of *Histoplasma capsulatum*. This fungus shows thermal dimorphism: mold form at 25°C and yeast form at 37°C. CDC

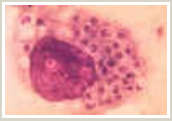


Figure 9.
Histiocyte containing numerous yeast cells of *Histoplasma capsulatum*. Dr. D. T. McClenan / CDC

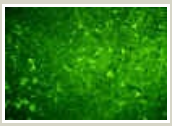


Figure 10.
Histopathology of histoplasmosis in open lung biopsy. FA stain reveals numerous yeast cells of *Histoplasma capsulatum*. CDC/Dr. Leo Kaufman, Maxine Jalbert lek1@cdc.gov

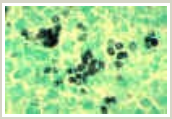


Figure 11.
Methenamine silver stain reveals *Histoplasma capsulatum* fungi. CDC/Dr. Edwin P. Ewing, Jr. epe1@cdc.gov

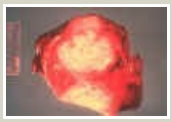


Figure 12.
Gross pathology specimen of lung showing cut surface of fibrocaseous nodule due to Histoplasma capsulatum.
ASCP Atlas of Clinical Mycology II / CDC

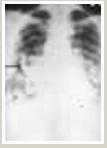


Figure 13.
Chest radiograph showing miliary densities in both lung fields plus thin-walled cavity with fluid level. Histoplasmosis. ASCP Atlas of Clinical Mycology II /CDC

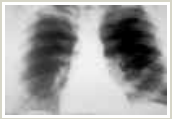


Figure 15.
Chest radiograph showing single pulmonary nodule of histoplasmosis.
Case 49-03. Mass Gen Hosp Case Records. CDC


Figure 16.
Computed tomography scan of lungs showing classic snowstorm appearance of acute histoplasmosis CDC

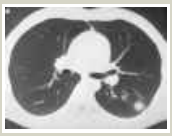


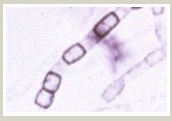
Figure 17.
Computed tomography scan showing single pulmonary nodule of histoplasmosis. Case 49-01. Mass Gen Hosp Case Records. CDC

C. COCCIDIOIDOMYCOSIS (*Coccidioides immitis*)

Coccidioidomycosis is primarily a pulmonary disease. About 60 % of the infections in the endemic area are asymptomatic. About 25 % suffer a "flu-like" illness and recover without therapy. This disease exhibits the typical symptoms of a pulmonary fungal disease: anorexia, weight loss, cough, hemoptysis, and resembles TB. CNS infection with *C. immitis* is more common while it is less frequent with the other fungal diseases. The ecological niche of *C. immitis* is the Sonoran desert, which includes the deserts of the Southwest (California, Arizona, New Mexico, Nevada, Utah and Texas) and northern Mexico (figure 18). It is also found in small foci in Central and South America.


Figure 18
Map of United States showing geographic variation in the prevalence of coccidioidin sensitivity in young adults CDC

Desert soil, pottery, archaeological middens, cotton, and rodent burrows all harbor *C. immitis*. *C. immitis* is a dimorphic fungus with 2 life cycles. The organism follows the SAPROPHYTIC cycle in the soil and the PARASITIC cycle in man or animals. The saprophytic cycle starts in the soil with spores (arthroconidia) that develop into mycelium. The mycelium then matures and forms alternating spores within itself. The arthroconidia are then released, and germinate back into mycelia (figure 19). The parasitic cycle involves the inhalation of the arthroconidia by animals which then form spherules filled with



Figure

19
Coccidioides immitis - arthroconidia
Dr Arthur DiSalvo

endospores (figure 20). The ambient temperature and availability of oxygen appear to govern the pathway The organism can be carried by the wind and therefore spread hundreds of miles in storms so the distribution is quite wide. In 1978, cases were seen in Sacramento 500 miles north of the endemic area, from a dust storm in Southern California. The spores of the organism are readily airborne. The cases that occur in South Carolina are usually in patients who have visited an endemic area and brought back pottery, or blankets purchase from a dusty roadside stand, or in Navy and Air Force personnel who were exposed when they were stationed in the endemic area. The disease manifests itself after they are transferred to a base in South Carolina. A few interesting cases occurred in cotton mills in Burlington and Charlotte, N.C. The cotton, grown in the desert of the Southwest, was contaminated with the fungus spores and the mill workers inhaled the spores while handling the raw cotton and developed coccidioidomycosis.

Clinical Specimens

Clinical specimens include sputum, pus from skin lesions, gastric washings, CSF, and biopsy material from skin lesions.


Figure 20A
Smear of exudate showing spherules of *Coccidioides immitis.* Experimental infection of mouse with soil sample. CDC

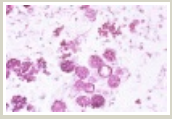


Figure 20B
Coccidioides immitis - spherules
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Mycology

C. immitis is a dimorphic fungus (figure 19-24). Cultured on Sabouraud's agar at 25 degrees C it grows as a mold in 2 to 3 weeks. Characteristically, the mycelia develop arthroconidia. ("By their fruits ye shall know them"). It is a barrel-shaped (smaller at the edges, wider at the middle) asexual spore. Typically, the arthroconidia alternate with non spore-forming cells in the mycelium. When grown *in vitro* at 37 degrees C, there is no yeast form!! *C. immitis* is a dimorphic fungus; *in vivo*, (pus or tissue) one sees the pathogenic or invasive form B which is a spherule. The organism develops into spherules (30-60 microns) that are filled with endospores which are 3 to 5 microns in diameter (see figure 20A and B). A spherule will develop endospores within, then break apart, releasing the endospores. This is the tissue form seen in pus or histological sections: spherules and loose endospores. They can also be seen in a KOH preparation of sputum. It is pathognomonic for coccidioidomycosis.

Histopathology

The inflammatory reaction is both purulent and granulomatous. Recently released endospores incite a polymorphonuclear response. As the endospores mature into spherules, the acute reaction is replaced by lymphocytes, plasma cells, epithelioid cells and giant cells.

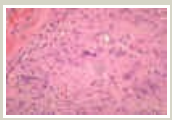
Figure 21
Histopathology of coccidioidomycosis, retroperitoneal area. Coccidioides immitis fungi are visible within granuloma. CDC/Dr. Edwin P. Ewing, Jr. epe1@cdc.gov

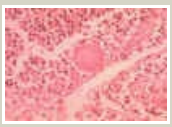
Figure 22
Histopathology of coccidioidomycosis of lung. Mature spherule with endospores of Coccidioides immitis, intense infiltrate of neutrophils. CDC/Dr. Lucille K. Georg

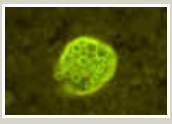
Figure 23
Histopathology of coccidioidomycosis. Spherule of Coccidioides immitis with endospores. Mercy Hosp Toledo OH/Brian J. Harrington

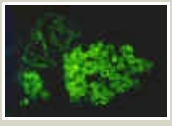
Figure 24
Histopathology of coccidioidomycosis of lung showing spherule with endospores of Coccidioides immitis. FA stain. Endospores, not spherule wall, are stained. CDC

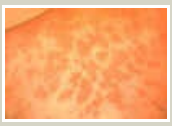
Figure 25
Erythema nodosum lesions on skin of back due to hypersensitivity to antigens of Coccidioides immitis CDC/Dr. Lucille K. Georg

Serology

There are four tests for diagnosis:

- Complement-Fixation
- Slide agglutination

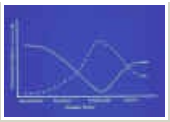
Figure 24
Immune responses during coccidioidomycosis. Line graph showing immunologists' concept of the interplay between humoral and cell-mediated immune responses during coccidioidomycosis. TX State Chest Hosp/Dr. Rebecca A. Cox

- Immunodiffusion
- EIA C-F antibody is slow to rise and develop in about 1 month. This test is excellent for coccidioidomycosis because it is quantitative. However, these antibodies cross-react with some other fungi (Blastomyces and Histoplasma). The C-F test is also a PROGNOSTIC test. If the titer keeps rising, then the patient is responding poorly and the course may be fatal. If the C-F titer is dropping then the prognosis for that patient is favorable. A titer of greater than 1:128 usually indicates extensive dissemination. Life-long immunity usually follows infection with *C. immitis.* There is a much greater mortality rate in dark-skinned people (Mexicans, Filipinos, and Blacks). They are 25 times more likely to develop progressive disease and death. The reason for this is obscure.

Amphotericin B, fluconazole and itraconazole are the drugs of choice.

PARACOCCIDIOIDOMYCOSIS (*Paracoccidioides brasiliensis*)

This is a chronic granulomatous disease of mucous membranes, skin, and pulmonary system. This disease occurs from the middle of Mexico (North America) to Central and South America. Most cases are reported from Brazil. The ecological niche of this organism is probably the soil. A common triad of symptoms that are seen in Latin America is pulmonary lesions, edentulous mouth (figure 25 and 26), and cervical lymphadenopathy (figure 25). Prior to the recognition of this disease, patients in Latin America with paracoccidioidomycosis were often sent to TB sanitariums, just as patients with histoplasmosis were in the U.S. The organisms invade the mucous membranes of the mouth causing the teeth to fall out. White plaques are also found in the buccal mucosa, and this along with the triad are now used to clinically differentiate between TB and. This disease has a long latency period. 10-20 years may pass between infection and manifestation of the infection in the non-endemic areas of the world. Typically, a case of paracoccidioidomycosis seen in the U.S. occurs in someone who worked in South America for some period of time and then they return to the U.S. and years later, develop this disease. The patient does not realize the importance of this past history. Almost all diagnoses of fungal diseases depend upon careful questioning and a probing history. The clinical material which should be sent to the lab for examination is sputum, biopsy material, pus, and crust from the lesions. Examination of sputum or crust from one of the lesions with KOH reveals a yeast because this is a dimorphic fungus. In contrast to the other yeasts, particularly Blastomyces, Paracoccidioides has multiple buds, a thin cell wall, and a narrow base. At 25 degrees C, the colony is a dense, white mycelium (figure 28), not loose and cottony like the others. On Sabouraud's agar (figure 29), it takes 2-3 weeks to grow. When cultured at 37 degrees C, it is slow growing with a white-tan, thick colony. Microscopically, these yeasts appear as described above ranging in size from 5 to 15 microns.

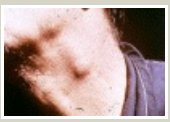
Figure 25
Cervical lymphadenopathy
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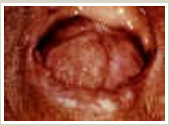
Figure 26
Paracoccidioidomycosis: Mouth Mucosa in man © Bristol Biomedical Image Archive. Used with permission


Figure 27
Tongue lesion of paracoccidioidomycosis. CDC/Dr. Lucille K. Georg

Histopathology

Histologically, one sees multiple buds forming a "Captain's wheel." This is diagnostic of paracoccidioidomycosis. In this case, the mother cell is 40-50 microns in diameter and the buds are 2-5 microns in size (figure 30-32).

Serology

The best serological test for paracoccidioidomycosis is the immunodiffusion test. It is better than 99% specific and almost 85% sensitive.

Therapy

The drug of choice is Amphotericin B. Sulphonamide-trimethoprim has also been used. Presently Itraconazole appear to provide the best recovery.

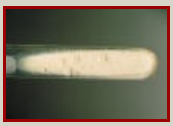
Figure 28
Lowenstein-Jensen slant culture of the fungus *Paracoccidioides brasiliensis* grown at 37°C. CDC/Dr. William Kaplan


Figure 29
Sabouraud dextrose agar slant culture of the fungus *Paracoccidioides brasiliensis* grown at 37°C CDC/Dr. William Kaplan

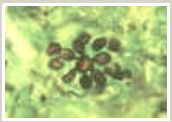
Figure 30
Histopathology of paracoccidioidomycosis. Budding cell of *Paracoccidioides brasiliensis*. Methenamine silver stain. CDC

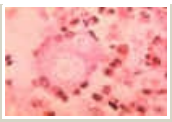
Figure 31
Histopathology of paracoccidioidomycosis, skin. Budding cell of *Paracoccidioides brasiliensis* within multinucleated giant cell. CDC/Dr. Lucille K. Georg


Figure 32
Histopathology of paracoccidioidomycosis. Budding cells of *Paracoccidioides brasiliensis*. Methenamine silver stain. CDC/Dr. Lucille K. Georg


Figure 33
This patient presented with sporotrichosis affecting the skin of the thumb. CDC

SPOROTRICHOSIS (*Sporothrix schenckii*)

Sporotrichosis is usually a chronic infection of the cutaneous or subcutaneous tissue which tends to suppurate, ulcerate and drain. In recent years, a pulmonary disease has been seen more frequently. Occasionally, infection with *S. schenckii* may result in a mycetoma. Sporotrichosis is caused by another dimorphic fungus. The infection is also known as "rose growers disease." The ecologic niche for this organism is rose thorns, sphagnum moss, timbers and soil. A study on the occupational distribution of sporotrichosis showed that forest employees accounted for 17% of the cases, gardeners and florists, 10%; and other soil-related occupations another 16%. Sporotrichosis occurs worldwide. Every aspect of this disease (clinical, pathology, mycology, ecology) was investigated during an epidemic of 3,000 patients in a gold mine in South Africa during the 1940's. Patient history is very important in this disease also. It is often seen in gardeners and begins with a thorn prick on the thumb (figure 33). A pustule develops and ulcerates. It infects the lymphatic system and then the disease progresses up the arm with ulceration (figure 39), abscess formation, break down of the abscess with large amounts of pus followed by healing. Progression usually stops at the axilla. Clinical material to be sent to the lab may be pus, biopsy material, or sputum from pulmonary patients. The yeast form of this fungus in tissue or in culture, can be round (6 - 8 *u*m) or fusiform (figure 34). The fusiform shape is not the usual form but if a cigar-shaped yeast (figure 34) is observed in tissue, it is usually diagnostic of sporotrichosis. *S. schenckii* does not stain with the usual histopathological stains. If sporotrichosis is suspected, the pathologist must be informed so he can use special stains. Histologically asteroid bodies, a tissue reaction (also known as Splendori reaction) may be seen around the yeast cell. At 25 degrees C, this colony is white-cream and very membranous (figure 36-38), but as it ages for 2-3 weeks it becomes black and leathery (figure 36). Microscopically, the mycelium is branching, septate and very delicate, 2-3 um in diameter (figure 35). The pyriform conidia, 2-4 um form a typical arrangement in groups at the end of a conidiophore called "daisies" (figure 35). Serologic tests are not commercially available. For the systemic form the drug of choice is itraconazole or amphotericin B.

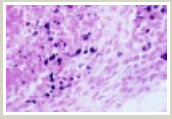
Figure 34
Yeast form of *Sporothrix schenckii*
Dr Arthur DiSalvo


Figure 35
conidiophores and conidia of the fungus Sporothrix schenckii
CDC/Dr. Libero Ajello


Figure 36
SABHI agar plate culture of *Sporothrix schenckii* grown at 20°C CDC/Dr. William Kaplan

WEB RESOURCES
CDC-Sporotrichosis


Figure 37
SABHI agar slant culture of the fungus *Sporothrix schenckii* grown at 37 degrees
CDC/Dr. William Kaplan


Figure 38
Sabouraud's dextrose agar plate culture is growing the fungus *Sporothrix schenckii*
CDC/Dr. Lucille K. Georg

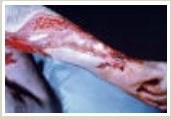
Figure 39
This patient's arm shows the effects of the fungal disease sporotrichosis, caused by the fungus *Sporothrix schenckii*
CDC/Dr. Lucille K. Georg

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Page maintained by Richard Hunt