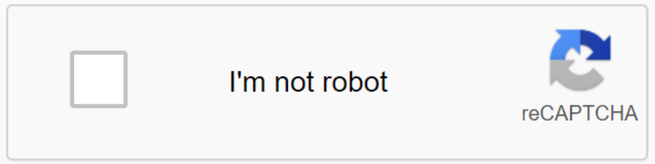

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Opportunistic fungi pdf

These are fungal infections of the body that occur almost exclusively in debilitating patients whose common defense mechanisms are damaged. The organism involved is an international fungus that has a very low inherent toxicity. The increased incidence of these infections and the variety of fungi that cause them, paralleled the emergence of AIDS, the use of more aggressive cancers and post-transplant chemotherapy and antibiotics, cell singles, immunosuppressants, corticosteroids and other macro-destructive procedures that resulted in low resistance of the host. Candidiasis is a primary or secondary fungal infection caused by members of candida genera and other related genera. Clinical symptoms may be acute, subtly or chronic. Participation can be confined to the mouth, throat, skin, scalp, vagina, fingers, nails, bronchial, lungs or gastrointestinal tract, or as in sepsis, endothetitis and meningitis. In healthy individuals, Candida infection is usually due to impaired epithelial barrier function and occurs in all age groups, but is most common in newborns and elderly people. They usually remain superficial and respond easily to treatment. Systemic candidiasis is commonly seen in patients with cell-mediated immunodeficiency and can be seen in patients receiving aggressive cancer treatment, immunosuppression or transplant therapy. Clinical Symptoms: 1. Orobanin candidacy: contains thrush, thyme, stomatitis and hypertaritis. Acute oral candidiasis is rarely seen in healthy adults, but it can occur up to 5% of newborns and 10% of the elderly. However, it is often associated with severe immune disorders due to diabetes, leukemia, lymphoma, malignancy, neutropenia and HIV infection presented as a predictor of clinical progression in AIDS. The use of extensive antibiotics, corticosteroids, cytotoxic drugs, and radiation therapy is also predisposition. Clinically, white plaque resembling a milk curd form on the boucal mucous membranes and tongue, gums, palate or pharynx is less common. Symptoms may include absenteeism or burning or drying of the mouth, loss of taste and pain of swallowing. 2. Curtaneus Candidacy: Includes Intogo, Diaper Candidiasis, Paronicia and Onichomykos. Cunning candidiasis is most commonly seen in saxilas, groin, inter-breast wrinkles, middle folds, digital liver space, and umbilicus. Moisture, heat, friction and macenation of the skin are the principles that cause predisposition in normal patients, but obesity, diabetes, warm water immersion or occlusion of the skin and the use of a wide range of antibiotics are additional factors. The lesion consists of a moist, macular erythema rash with a typical satellite lesion present in the healthy skin around it. Diaper candidiasis is common in infants under unsanitary conditions of chronic moisture and local skin. It is associated with ammotic stimulation due to irregularly altered negative diapers. Once again, the erosion and characteristic erythema lesions with satellite pustules are created, along with noticeable participation of skin wrinkles and wrinkles. The phonichia of finger nails can occur in people whose hands are constantly wet with a sugar solution or contact with flour to retreat nail wrinkles and cuticles. The lesion is characterized by the development of painful and erythemaal swelling on the affected nail. In chronic cases the infection may progress from the nail plate to cause the total separation of the epidermis and onychomycosis. Chronic candida onichomycosis can often be seen in patients with chronic mucosal candidiasis, or other underlying factors that affect the hormonal or immunological condition of the host, which causes complete destruction of nail tissue. These include diabetes mellitus, hypothyroidism, Addison disease, thyroid dysfunction, malnutrition, poor absorption and various malignant tumors. The use of steroids, antibiotics and anti-midorons may also be contributed to factors. 3. Vulvovaginal candidiasis and vulinitis: Vulvovaginal candidiasis is a common condition in women often associated with the use of broad-spectrum antibiotics, third trimester of pregnancy, low vaginal pH and diabetes mellitus. Sexual activity and oral contraception can also be extended to include factors and infections in the perineum, vulva and the entire inguinal area. Chronic internal candid candidiasis associated with oral candidiasis can also be a presentation of HIV infection or AIDS. Symptoms include intense vulvar predation, burning, erythema and dyspareunia associated with discharge like creamy white, curd. In the case of balanitis, diabetes should be ruled out, and sexual partners should investigate vulvabacinitis. Symptoms include erythema, prigo and glyculatulum in glan penis or prepus. Infections are more commonly seen in uncircumcised men and poor hygiene can also be a contributing factor. 4. Chronic mucosal candidiasis: Chronic mucosal candidiasis is a form of persistent candidiasis commonly caused by C. albicans of the skin, nails and mucous membranes, which occur in patients with various metabolic disorders with cell-mediated immunity. These include thyroid function or injury function, Addison disease, thyroid function, diabetes, thyroid and functional disorders of multi-life autoimmune diseases. The patient is usually children. Candida mesothenoal is a serious localization form that can occur regardless of the presence or absence of endometriosis characterized by a significant hyperawakening of the middle of the species lesion. 5. Newborn and congenital candidiasis: Underweight and age, prolonged intravascatheterization and the use of antibiotic drugs are the principle of treating conditions for systemic candidiasis in newborns. Blood It is often positive and there is also a high incidence of meningitis. Kidney complications can also occur due to the formation of fungal balls in the urethra or kidney pelvis. Congenital candidiasis acquired from the uterus is usually limited to the skin in the form of a generalized erythematosus sanctuary rash, but intrauterine candidiasis can lead to an abortion. 6. Esophageal candidiasis: esophageal candidiasis is often associated with AIDS and severe immunosuppression after treatment for leukemia or solid tumors. Accompanied oral candidiasis often exists. Esophagitis can also lead to sepsis and propagated candidiasis. Symptoms include burning pain in the sub-region, outdoors, nausea and vomiting. Clinical diagnosis usually relies on radiation and endoscopic findings showing white mucosal plaques with erythema, similar to those seen in oral candidiasis. Herpes simplex or cell megalovirus (CMV) infection may also be present, clinical diagnosis needs to be confirmed by histopathology and culture. 7. Gastrointestinal candidiasis: Patients with acute leukemia or other hematologic malignant tumors may have numerous ulcers of the gastrointestinal tract and less commonly duodenum and intestines. Perforation can lead to regression and hematopoietic spread to the liver, spleen and other organs. Colonization and intrusion of the gastrointestinal or intestinal mucosa is often accompanied by a large number of yeast folds, which can be detected in the feces. 8. Pulmonary candidiasis: Pulmonary candidiasis can be obtained by the prevalence of hematopoietic disease, which causes diffuse pneumonia, or bronchial extension in patients with oropharyncity candidas. Yeast aspirations in the oral cavity have also been reported in infants. Pulmonary candidiasis is difficult to diagnose due to non-specific radiation and culture results and granular cellular phenonia present in most patients, especially during autopsy. The presence of yeast in alveolar labeige or sputum specimens is not specific and blood culture can be negative. Unfortunately, only hisoria can provide a definitive diagnosis, and not always in patients with clotting problems. 9. Peritonitis: Candida peritonitis can be caused by colonization of compounding catheters used for ulcers, ceylstor colitis, peritoneal dialysis (CAPD) or gastrointestinal gland due to surgery or abdominal neoplasms. Symptoms include fever, abdominal pain, tenderness and cloudy peritoneal dialysis that includes more than 1000 hundred leukocytes/mm3. Candida peritonitis is usually localized in the abdominal cavity unless the patient is severely immunosuppressed. 10. Urinary tract candidiasis: Transient asymptomatic candidamay occur during antibiotic or corticosteroid therapy to promote the growth of Candida, throughout the gastrointestinal tract and genital area, the lowest urinary tract infection occurs in the local spread of yeast at this site. The condition is common in women. Candida cystitis or bladder colonies can be caused by accompanying antibiotic treatment, diabetes and glycomya, anatomical urinary donation, previous bladder endoscopy or surgery, chronic interference from diabetic neurological bladder, prostate hypertrophy, or pelvic and long-term catheterization of cervical cancer. Kidney candidiasis (pyelonephritis) is usually the result of elevated infection or more often, hematopoietic prevalence in other organ focal points. Symptoms include fever, severity, lumbar pain and abdominal pain. The development of fungal balls in the kidney pelvis, although it can rarely complicate the infection. Predisposition for this includes contraction of the urinary tract, state-owned necrosis, urethra or bladder catheterand diabetes. Although, up to 80% of patients with spread candidiasis are not a reliable method for the diagnosis of kidney infection and related candidacy, urine culture alone. The real problem with Candida patients is the distinction between colonization and/or contamination and infection. Therefore, it is important to determine whether renal function exists or whether the infection is confined to the bladder. Cliché findings are generally crucial to making clinical parameters important. The following criteria are hints of kidney infection; Isolation of yeast from urine samples is obtained by suprapubic aspirations, benign blood culture and positive immune fusion precipitation test results or serological conversion in patients with iatrogenic profactorand /or underlying diseases. It should be noted that many clinicians do not recommend suprapubic aspiration because they require additional expertise in invasive and especially immune-impaired patients. The lab is also advised on the need to report the isolation of any yeast from urine samples obtained from high-risk immunosuppressive patients. 11. Meningitis: Candida meningitis is a rare entity found mainly in low-weight newborns with sepsis and in traceable prosthetic devices such as hematologic malignant tumors, complex neurosurgery or ventricular blood clots. Symptoms include fever and meningococcal stimulation. The diagnosis of a newborn requires a high index of suspicion by clinicians on the possibility of meningitis as a sequel to sepsis. Detection of candida cells in stains and isolation in CSF is often difficult. 12. Liver and soy candidiasis: Liver candidiasis occurs in patients with severe neutropenia, usually acute leukemia. Symptoms include fever, increased blood levels of epileptic elixir and alkaline phosphate. Histopathology shows the proliferation of spleen necrotic lesions or abscesses, which include a veterinary inthe number of doctors. But blood and biopsy culture is usually culture. A sure diagnosis is often difficult due to the inability to properly biopsy these patients. 13. Endothelial itis, myocarditis and pericarditis: endothelial is the most common form of cardiac candidiasis. Pre-existing Balbi disease with intravenous catheterization and antibiotic treatment, intravenous substance abuse, heart surgery and valve prosthesis are the most common predisposing factors. Clinical symptoms include fever, murmuring, congestion heart failure, anemia and spleen. Blood culture is often positive for the detection of candida antibodies (immunofusion acupuncture tests), and echocardiography is another useful diagnostic procedure. Myocardial abscess, arterial embolization and purulent arteritis are further rare complications of Candida sepsis or surgery. 14. Candidamia (Candida sepsis) and radio candidas: Candida was defined as the presence of yeast in the blood with or without visceral involvement. Hematopoietic prevalence can occur in one or more different organ systems with the formation of numerous micropesticides. Candida species have been reported to cause up to 15% of cases of sepsis seen in hospital patients. Predisposition includes intravenous catheters, antibacterial drug use, urinary catheters, surgical procedures, corticosteroid therapy, neutropenia, severe burns, parental nutrition and co-operative or chemotherapy-induced disorders of the gastrointestinal mucosa. The characteristic presentation is stellar heat in neutrophil patients with tachycardia and shortness of breath. Hypotension is also common and skin lesions may also occur. Diagnosis is simple when yeast is isolated in the blood or in tissue biopsies, but this is often not the case. Blood culture often remains negative, especially in granucellular patients, even in patients dying from propagated candidiasis proven. As much as possible, suspected posh should be asach, including joints, peritoneum, CSF or vital specimens. And liver and lung biopsies should also be performed. However, histopathology is not a viable option because more often biopsies are contraindicated because of patients with underlying diseases. Finally, finding yeast in more accessible aseptic sites such as urine is too common in diagnostic values. In this situation, clinically unproven radio candidiasis, only sulfuric acid and/or eye lesions can be quickly confirmed. Specific reliable serological tests are not yet available. Empirical antifungal treatment usually begins in these cases. 15. Ocular candidiasis: Candida insufficiency is often associated with candidamia, residential catheteror substance abuse, but patients with severe neutropenia are rare. Lesions are often confined near the macular, and the patient complains of blurred vision. Exogenous Candida insositis is rare, but cases have been reported. Eye trauma or surgery. Similarly, conjunctival and corneal infections have also been recorded following trauma. 16. Osteoarthritis: Arthritis may be the late sequel to Candidamia in newborns or neutrophils. Prosthetics or rheumatoid joints are also susceptible to hematopoietic spread during surgery or are directly infected by inoculation or by corticosteroid injections within the joint. The knee is the main site associated with weight bearing or pain in the entire expansion. Diagnosis depends on the isolation of yeast from the joint fluid obtained from needle aspiration synovial or synovial biopsy. 17. Other forms of candidiasis: Candidiasis can usually occur in patients with endogenous, nosocomial infections, especially in patients with endogenous, nosocomial infections. For example, reported macular, eye and arthritis symptoms reported in heroin addicts; Fever, rash and bone associated with leukemia patients; Candida cholecystitis; Candida prostatitis; Pancreatic abscess; Name laryngitis and osteomyelitis, a few. Summary of clinical groups and/or predispositions to invasive candidiasis. Neutropenia (especially >7 days). Hematologic malignant tumors. Solid tumor malignant tumors. Post-operative intensive care. Prolonged intravenous catheterization. Broad spectrum or multiple antibiotic therapy. Diabetes. parental nutrition. Severe burns. Neonatal.Cortico steroid therapy. Intravenous drug abuse. Laboratory diagnosis: 1. Clinical material: skin and nail scratching; urine, phlegm and bronchial washing; Cerebrospinal fluid, pleural fluid and blood; Tissue biopsy from various visceral organs and residential catheter tips. 2. Direct microscopy: (a) skin and nails should be examined using 10% KOH and parka ink or calcofluor white mount. (b) Obscene and bodily fluids must be centrifuged and inspected using 10% KOH and parker ink or calcofluer white mount and/or gram stained stains. (c) The tissue section should be dyed using PAS Digest, Grocott's Metenamin (GMS) or gram stain. See Candida H&H E You may be missed in the stained section. Examine samples for the presence of small, round oval, thin walls, clusters (blastoconidia) of budding yeast cells and branch doctors. Dr. Candida Doctor Hypei can be difficult to distinguish aspergillus high damage when the cell cells are not observed to occur frequently in liver biopsies. Interpretation: In general, benign direct microscopy in sterile sites, especially tissue biopsies, should be considered important even if the laboratory cannot culture yeast. In addition, the demo of physician hydration in the scraping or smear from the color, oral, esophagus and vaginal lesions should be considered important if the clinical symptoms support the diagnosis. However, finding that budding yeast cells from such substances has little diagnostic importance. Note, doctor's moisture C. glabrata is involved and from the stain when the diagnosis requires additional supporting evidence. Direct microscopy of sterile fluids such as CSF, vitreous humor, joint fluid and peritoneal fluid is not relatively sensitive and positive culture will generally be required for diagnosis. 3. Culture: The colony is usually white in cream, soft on the wax surface and colored in glabrous. Interpretation: A positive culture in the blood, or other sterile body fluids, or tissue biopsies should be considered important. Lysis centrifugation is currently the most sensitive method for isolating Candida from the blood. However, benign cultures of non-aching specimens such as phlegm, bronchial lava, esophageal brushing, urine, feces and surgical drains have little diagnostic value. Similarly, the culture of skin or mucosal lesions is not a diagnosis without direct microscopic examination supporting evidence. Candida species usually separate from the mouth, vaginal, anus, less frequently, the moist skin surface of normal individuals without candidiasis. 4. Vertical: A variety of introductional procedures have been devised to detect the presence of Candida antibodies, from immunofusion to more sensitive tests such as counter-immune electrostatic electricity (CIE), enzyme-linked immunosuction assay (ELISA), and radiation immunoassay (RIA). However, these are often negative in immune compromised patients, especially at the start of infection. Production of four or more precipitate lines in CIE tests was reported as a diagnosis of candidiasis in predisposing patients. Tests for antigen circulation by immunological or non-immunological means have also been developed. Of non-immunological techniques, it was most useful to use a gas liquid chromography (GLC) to detect the manno derivative of the cell wall or metabolic by-products. Although antigen detection was used by immunological methods such as ELISA or RIA, small laboratory latex aggregation tests for glycorotien antigens have been reported to be variable, but have proven to be the most useful. It should be emphasized that the interpretation of serum tests for Candida, especially in patients with neutrophils, is often difficult and should be correlated with other diagnostic methods. False negatives and false positives occur. Hopwood and Evans (1991) provide a great review of the current cethenological methods. 5. Identification: Candida genus is characterized by globos, which lengthen cells such as yeast or blastoconidia, reproduced by multilateral budding. Most Candida species are also characterized by the presence of well-developed physicians, but these characteristics may be absent, especially in species officially contained in torulopsis. Joint coniac, ballytoconidia and colonial pigmentation do not always exist. In Candida Genus, Fermentation, Nitrate Assimilation and Inositol Although it may be present or absent, all inositol-positive bacteria produce scoliosis. Cause: Candida Albicans Candida Catenura Candida Candida Candida Candida Candida Candida Glabratha Complex Candida Haemulonii Candida Inconspikua Candida Candida Parasite Complex Candida Lugosa Candida Candida Da tropical isClavispora lusitaniae (formerly Candida Lucitania) Cyberlindnera fabianii (formerly Candida Fabiani) Devayomis Hanseni (formerly Candida Pamata) Cluibero Misis Marxian Us (formerly Candida Kefir) Meyerozyma Guillermondi (formerly Candida Guillermondi) Piccia Kudriavze (formerly Candida Cruzey) Phelia Norvegensis (formerly Candida Norvegen Sissy) Lulaspora Delbrueki (formerly Candida Colliculosa) Wickerhamomis Anomalus (formerly Candida Feliculosa) Yarouria Polytica (formerly Candida Polytica) Management: See Treatment Guidelines section. AmFurther Read: Azzelo L and R.J. Hay. 1997. Medical Mycological Vol 4 Topless && Wilson's microbiology and infectious infections. 9th edition, Arnold London.Barnett et al. 1990. 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Fungal infection in damaged patients. 2nd Edition. John Wylie & Son Cryptococis Cryptococcosis is a chronic, acute lung subacute, systemic or meningococcal disease, started by the inhalation of infectious propagation (basidiospores and/or dry yeast cells) in the environment. Primary lung infection has no diagnostic symptoms and is usually clinical. On the prevalence, the fungus usually shows a tendency for the central nervous system, but the skin, bones and other visceral organs may also be involved. Although C. neoformans and C. gattii are considered principle pathogenic species, Cryptococcus albidus and C. laurentii have sometimes been involved in human infections. Clinical Symptoms: Cryptococcus is a fungal like encapsulated body diomycete yeast with a tendency for the respiratory and nervous system of humans and animals. Two species, C. neoformans and C. gattii, are distinguishable biochemically by molecules In humans, C. neoformans mainly affect immune-impaired hosts and are the most common cause of fungal meningitis; Worldwide, 7-10% of AIDS patients are affected. HIV-related cryptococosis account for 50% of all cryptococcal infections reported annually and usually occur in HIV patients when their CD4 lymphocyte count is below 200/mm3. Meningitis is the most common symptom and is a leading clinical presentation with fever and headaches. Secondary salt infection occurs in up to 15% of patients with spread cryptococcosis and often indicates a bad prognosis. Lesions usually begin with a small field of ulcers afterwards, but may also appear as an abscess, erythema nodules, or cellulite. This diversity is found around the world. In contrast, the distribution of cryptococosis due to cryptococcus gatti is geographically limited, the non-immune damage host is generally affected, large mass lesions of the lungs and / or brain (cryptococcomas) is characterized by a high and high foreign exchange rate from neurological diseases. Human diseases occur in Australia, Papua New Guinea, Africa, the Mediterranean region, India, Southeast Asia, Mexico, Brazil, Paraguay and Southern California. 1. Pulmonary cryptococcosis: Asymptomatic transportation of Cryptococcus has been reported in the respiratory tract, especially in the skin of healthy people as a result of sputum and normal environmental exposure. In addition, patients with chronic lung disease, such as bronchitis and bronchitis, may have asymptomatic colonies with Cryptococcus, which is separated from the phlegm for many years. Clinical typical pulmonary disease can occur as a result of environmental exposure, and normal individuals can experience self-limiting pneumonia with a sense. Most primary infections of this type have no diagnostic symptoms and are usually only discovered by routine chest X-rays. When present, symptoms include cough, low fever and leukocyte pain. Invasive pulmonary cryptococcosis can occur in some patients when primary infections may not be easily addressed in some patients, leading to more chronic pneumonia, which progresses slowly for several years. Patients can become pyrexic and may have an accompanying cough, but many lung lesions are often asymptomatic, when chronic granulomas are formed. Chronic lung cryptography also increases the risk of prevalence in the central nervous system. 2. Central nervous system: Prevalence of the brain and meningococcal is the most common clinical manifestation of cryptococoxysis and includes meningitis, encephalitis or enlargement of cryptococcoma. Meningitis is a common clinical form that accounts for up to 85% of the total number of cases, but clinical indications are almost dramatic. Symptoms usually develop slowly for several months, and initially include drowsiness, dizziness, irritability, confusion, nausea, vomiting, neck stiffness and focus following neurological headaches Like aloss. Decreased vision and coma can also occur in the later stages of infection. Cases of acute onset can occur especially in patients with a wide range of diseases, which rapidly worsen and can lead to death in a matter of weeks. Meningitis caused by infestation of the cerebral cortex, brain stems and cerebellum is a rare, fast fullness infection, often leading to a coma within a short time. Symptoms include treatment and a slow response to signs of brain edema or narrow ness, especially papilledema. Cryptococcoma is a rare entity, topical, solid, characterized by tumor-like mass, commonly found in the cerebral hemisphere or cerebellum, or more rarely in the spinal cord. Symptoms are consistent with enlarged intracranial mass and include headaches, drowsiness, nausea, vomiting, mental changes, slurry speech, double vision, instability of gait, incoma, numbness and oneway. This phenomenon can mimic cerebral neoplasms that can delay a true diagnosis. 3. Behind-the-scenes cryptococcosis: Basic added crypto coccosis in the form of ulcerous lesions or cellulite sometimes occurs, especially in immunosuppressive patients. This lesion can be resolved spontaneously or through systemic antifungal treatment. However, all patients with skin lesions should be carefully monitored for possible dissemination of the central nervous system. Secondary salt infection occurs in up to 15% of patients with spread cryptococcosis and often indicates a bad prognosis. Lesions usually begin with a small field of ulcers afterwards, but may also appear as an abscess, erythema nodules, or cellulite. In AIDS patients, skin expression represents the second most common site of spreading cryptococintosis. Lesions often occur in the head and neck and can appear as lesions that simulate syntax, nodules, plaques, ulcers, abscesses, yellow ulcer plaque, herpesfoam lesions, moluscum transmission and caposi sarcoma. ulcers can also occur. 4. Bone cryptococosis:Osseous cryptococosis occurs in up to 10% of cases and may involve bone protrusions, skull bones and vertebrae. The lesion is lytic without a periosteal reaction and symptoms of dull pain in motion are reported. Occasional cases of arthritis have also been reported, mainly involving knee joints. 5. Eye cryptococoxysis: eye symptoms of cryptococoxysis most commonly include papilled edema and vision due to intracranicy pressure. Other eye signs of cryptocosysis are not uncommon and usually occur as a result of prevalence. 6. Other forms of Cryptococcosis: Cryptococcus neoformans are often isolated from the urine of patients with infectious infections. Sometimes, signs of pielonepneumonia or prostatitis may be observed. Other rare forms of cryptococococsis include corticosteroids, endothetitis, hepatitis, sinusitis and localized esophageal lesions. Laboratory diagnosis: 1. Substances: cerebrospinal fluid (CSF), biopsy tissue, phlegm, bronchial washing, pus, blood and urine. 2. Direct microscopy: (a) endometriosis and fluids use indian ink to demonstrate encapsulated yeast cells to create a thin wet film under the cover slip. Phlegm and pus may need to be digested with 10% KOH before dyeing indian ink. (b) FOR the organization section, pas digest, GMS and H&amp Using E, muscamine stains are also useful for demonstrating polysaccharides. Globos is tested on budding yeast cells surrounded by a wide range of gelatin capsules. Note, unencapsulated deformations are rare, but they can also occur. Interpretation: Demonstrationof CSF, biopsy tissue, encapsulated yeast cells in the blood or urine should be considered important even in the absence of clinical symptoms. Positive phlegm specimens should be considered potentially important, although cryptococcus can also occur in respiratory secretions with sapropituatero. Basically, all patients with a positive microscopy for cryptococci, should be investigated for diseases spread at any site, especially by cultural and anti-origin detection. 3. Culture: Samples are inoculated in primary isolation media, such as Sabulau's Dextros cloth. Find translucent and smooth gelatin colonies, later becoming very mucous membranes and creams in color. Interpretation: At any site, the isolation of C. neoformans or C. gattii should be considered important, and patients without clinical symptoms should thoroughly investigate the disease. The positive culture of CSF is decisive. However, the positive culture of respiratory secretions, especially in patients without clinical symptoms, needs to be interpreted carefully until further support ingevidence becomes available. Isolation of cryptococcus albidus or C. laurentii, is also Jong-eun Lee a rare pathogen and should be interpreted carefully because once again, additional support clinical and microscopic evidence is needed. 4. Vertical paper: it should be noted that the detection of cryptococal capcalical polysaccharides antigen in the spinal fluid is now an optional method for diagnosing cryptococal meningitis patients. In AIDS patients, cryptococcal antigencases can be detected in nearly 100% of cases in serum. However, non-AIDS patient antigen detection in the serum is less sensitive to that only about 60% of cryptococosis patients reported to be positive. Note, serum sample antigen detection should be pretreated with pronase to enhance detection and avoid false negative consequences. 5. Identification: Genuvecryptococcus is characterized by globos, which lengthen cells such as yeast or blastoconidia, reproduced by multilateral budding. Pseudo-pollinators are absent or rudimentary. Cultures in hard media are usually mucus or sticky on appearance. Red, orange or yellow carotenoid pigments can be produced, but young colonies of most species are usually pigment-free and creamy. Most strains encapsulate cells. The degree of capsule formation depending on the medium. Under certain conditions of growth, capsules may contain starch-like compounds that are released into the medium by many strains. Within the genus Cryptococcus, the fermentation of sugar is negative, the assimilation of nitrates is variable and the assimilation of inositol is positive. The genus cryptococcus is similar to the genus Rhodotolula. The unique difference between the two is the fairy tale of positive inositol in crypto-caucus. 6. Cause agent: Cryptococcus albidus, Cryptococcus laurentii, Cryptococcus neoformans, Cryptococcus gattii. Read more: Azelo L and R.J. Hay. 1997. Medical Mycological Vol 4 Topless && Wilson's microbiology and infectious infections. 9th edition, Arnold London.Ellis, D.H. and T.J. Piper. 1992. Crypto-Caucus Neoforman Ecology. Jour J. Fidemiol. 8:3 Ellis, D.H., D, Marriott, T. Sorrell. Candid and cryptococal infections. Interactive CD-ROM, Pfizer Australia.Kwon KJ and JE Bennett 1992. Medical Mystic Leah & Fevigher.Richardson MD and DW Warnock. 1993. Fungal infection: diagnosis and management. Blackwell Science Publications, London. Rippon JW. 1988. Medical Biology WB Saunders Co. Warnock DW and MD Richardson. 1991. Fungal infection in damaged patients. 2nd edition. John Wyle and sons. Aspergillosis Aspergilosis is a spectrum of diseases in humans and animals caused by members of the Aspergillus genus. This includes (1) bacillus due to the intake of contaminated foods; (2) allergies and aftereffects for the

presence of conidia or temporary growth of the organism in the body orifice; (3) colonization without prolonged in pre-formed cavities and debilitating tissues; (4) invasive, inflammatory, granulated, drug diseases and other organs of the lungs; Rarely (5) systemic and fatal prevalence diseases. The type of disease and severity depends on the physiological condition of the host and the species of Aspergillus associated. The e-eiteric agent is international and includes the Aspergillus Fumigatus complex, A. Flabus Complex, A. Niger Complex, A. Needulans and A. Therese Complex. Clinical Symptoms: 1. Pulmonary Asperyrostosis: Includes allergies, Aspergiloma and invasive aspergillosis. Clinical symptoms of pulmonary Aspersyrostosi range from harmless saprofitic colonies to acutely invasive diseases. Allergic Aspergilomyomyophilia is a continuum of clinical organs ranging from foreign asthma to external allergic pneumonia, allergic bronchial pneumonia Aspergilonosis (anaphylactic pneumonia), caused by the inhalation of Asperzzz Condidia. Features include asthma, intermittent or persistent lung penetration, peripheral eosinophils, positive skin tests on Asperzs anti-axextract extract, positive immuno-infusion precipia testing for Asperzls antibodies, high total IgE, high specific IgE for Aspergillus. There is also a history of chronic bronchitis with plug expectations. Symptoms may be mild without happiness, but recurrent episodes often progress with bronchial and fibrosis. Non-invasive Aspergilomydosis or Aspergiloma (fungal ball), caused by anti-inflammatory colonization of preformed cavities, usually secondary to tuberculosis or sarcoidosis. Features often include hemoptysis with blood-stained phlegm, a benign immune difusion precipitation test for antibodies to Aspergillus, certain IgE elevated against Aspergillus. However, in many cases it is asymptomacousand is usually found by routine thoracic roentenogram. Acuteinvasive pulmonary Aspers gyropathy. Aging factors include long-term neutropenia, especially in leukemia patients or bone marrow transplant recipients, corticosteroid therapy, cytotoxic chemotherapy and patients with AIDS or chronic granulomatous disease. Clinical symptoms mimic acute bacterial pneumonia and can include bleeding infarction or drug bronchial pneumonia and fever, cough, pleural pain. Typical patients receive photospectrum antibiotics for granule cellosis and unexplained fever. Radiological features may be non-specific and the test for serum antibody precipitation is also generally negative. Clinical awareness is essential because this is the most common form of aspergillosis in immunosuppressed patients. Chronic drug Aspergilopathy is an unjust, slowly progressive, semi-invasive form of infection that can be seen in patients with mildly immunosuppressive diseases, especially in patients with a previous history of lung disease. Treatment with diabetes, sarcoidosis and low-dose glucocorticoids may be another predisposing factor. Common symptoms include fever, cough and phlegm production; Positive serum antibody precipitation can also be detected. 2. Disseminated Asper Gilroys: Hematomaic prevalence in other internal organs may occur, especially in patients with severe immunosuppression or intravenous drug poisoning. Abscesses can occur in the brain (cerebral asperitosis), kidneys (kidney asperisomes), heart, (endothetitis, myocarditis), bone (osteomyelitis), gastrointestinal tract. Eye lesions (myeloid keratitis, endothetitis and orbital Aspergiloma) can also occur, either as a result of prevalence or following local trauma or surgery. 3. Parabisle sinus aspergilosis: two types of paranassal sinus aspegilosis is generally recognized. (1) The non-invasive Aspergiloma form can be found mainly in non-immunosuppressive individuals. Aging factors include a history of chronic sinusitis and a history of draining the sinuses with excessive mucus. (2) Invasive forms are usually seen in immunosuppressive patients. This form has a clinical setting similar to the one seen in rhino zygomycosis; And symptoms include signs of fever, rhinitis and aggression in the track. 4. Added Aspergillrosis: Added Aspergillrosis is a rare symptom commonly spread in the primary lung infection, Patients. However, cases of primary wound aspergillosis also occur, usually as a result of trauma or colonization. The lesion refers to erythema spheres or macules with progressive central necrosis. Laboratory diagnosis: 1. Clinical substances: tissue biopsy in patients with organ aspiration and prevalent diseases in patients with sputum, bronchitis and pulmonary diseases. 2. Direct microscopy: (a) phlegm, washing and aspiration make a wet mount on 10% KOH and parker ink or calcoflur and/or gram stain. (b) The organization section It must be dyed with E, GMS and PAS digests. Note Aspergillus Hypnosis is h&m You may miss it in the E-dye section. Examine the specimens for disometric dust, purifying hypnosis. Interpretation: The presence of hyaline, which diverges the haemorrhoids consistent with aspergillis in any sample, should be considered important in patients who support clinical symptoms. Biopsies and evidence of tissue aggression are especially important. Remember that direct microscopy or histopathology does not provide specific identification of the cause agent. 3. Culture: Clinical specimens must be inoculated in primary isolation media, such as Saburaud's Dextros cloth. The colony is growing rapidly and can turn from white, yellow, yellow brown, brown to black or green. Interpretation: Benign cultures of non-aching specimens such as phlegm are not evidence of infection because the Aspergillus species is well recognized as a common environmental pollutant. However, the detection of Aspergillus (especially A. fumigatus and A. flavus) in phlegm culture is likely to be a critical and empirical antifungal therapy of diagnosis from patients with proper predisposition conditions. Unfortunately, patients with invasive pulmonary Aspers, often have a negative phlegm culture, which makes lung biopsy as a prerequisite for a conclusive diagnosis of lung biopsy. 4. Cerro: An immuno-injection test for the detection of antibodies to aspergillus species has been proven to be valuable in the diagnosis of allergic, Aspergiloma, and invasive Aspergiloma. However, they should not be used alone, and should be correlated with other clinical and diagnostic data. Several antigen tests for the detection of Aspergillus in blood, urine and CFS are now available. (1→3) -β-D- glucan test detects a variety of fungal pathogens, including Aspergillus, Candida, Pusarium, Tricosforone and a number of commercial kits (FungiTec G, Fungitell). But the most widely used system is the Aspergillus Gallatman ELISA test (Plathria® Aspergillus ELISA kit. PPV of 26-53% (meta-analysis 27 research piper et al. CID 2006). However, as galatomann is rapidly removed from the blood, it is recommended to check twice a week for optimal diagnosis. 5. Identification: Aspergillus Colony It consists of fast-growing white, yellow, yellow brown, brown to green or green shades, most of which consist of a dense feel of an upright pony. Conidiophores end up in a vesicles covered with layers of body cells (metulae) that bear a combination cell (uniseriate) or a small amniotic fluid (so-called biseriate structure) such as a single palcade. Parcels, salids, metula (if present) and conidia form a stubborn head. Conidia is a 1-cell, smooth or rough wall, hyaline or pigment and forms a long dry chain that is vasocanate and can be aggregated into different (radiation) or small columnsar. Some species may produce Hülle cells or sclerosis. 6. Cause Agent: Aspergillus Soup. Aspergillus Flabus Complex, Aspergillus Fumigatus Complex, Aspergillus Nidulans Complex, Aspergillus Niger Complex, Aspergillus Terero Complex. Read more: Chandler FW., W. Kaplan and L. Azelo. 1980. Color Atlas and textbooks of histopathology of fungal diseases. Wolf Medical Publications, London.Kwon KJ and JE Bennett 1992. Medical Mystic Leah & Fevigher.Richardson MD and DW Warnock. 1993. Fungal infection: diagnosis and management. Blackwell Science Publications, London. Rippon JW. 1988. Medical Biology WB Saunders Co. Warnock DW and MD Richardson. 1991. Fungal infection in damaged patients. 2nd edition. John Wyle and sons. Scedosporiosis (Pseudoescheria) has a spectrum of similar diseases in terms of varying and severity as scedosporium and romentospora infections are caused by AsperGilles. The majority of infections include mycetomas, the rest of the infection of the eye, the ear, the central nervous system, the internal organs and more commonly the lungs. Etioric agent scedosporium apiospermum, Scedosporium aurantiacum, Scedosporium Boydi and Romenaspora are prolific. Clinical symptoms: 1. Scedosporium apiospermum, Scedosporium boydii and Scedosporium aurantiacum infections: non-invasive colonies are similar to those formed by properly draining bronchial or paravirivers and bear spherical cavities. Invasive infections in normal patients are usually caused by traumatic transplantation. Micetoma, where fungi are present in tissues with resistant microcolonies or grains, is the most common infection in normal patients. This is behind penetrating joint injuries to the knee, especially as a result of arthritis and osteomyelitis. Other manifestations include non-mysticism like polycystic keratinitis and non-strain infections. Invasive infections have also been reported in patients treated with corticosteroids and immunosuppressive therapy for organ transplants, leukemia, lymphoma, systemic lupus erythema. Disease. Infections include invasive sinusitis, pneumonia, arthritis with osteomyelitis, multiple and subcutaneous granulatitis, brain abscess, internal infections and spread systemic diseases. 2. Romenaspora prolific infection: The spectrum of clinical symptoms is similar to the one described above for Scedosporium. The disease was reported in immunosuppressive patients, especially in patients treated with long-term neutropenia and post-transplant ation. Colonization of the outer ear, parabisle sinuses and lungs, including fungal balls, has been reported. Cases of onichomycosis and fungal keratitis are also documented. However, sepsis arthritis and osteomyelitis, based on topical invasive infections, especially injuries that penetrate the joints, are a new clinical problem that now accounts for 80% of reported cases. Culture identification is important because these fungi are often resistant to antifungal therapy and treatment may require surgical intervention. Laboratory diagnosis: 1. Clinical substances: sputum, bronchial washing and tissue biopsy from patients with organ inhalation and subcutaneous and prevalence diseases from patients with bronchial and pulmonary diseases. 2. Direct microscopy: (a) phlegm, washing and aspiration make a wet mount on 10% KOH and parker ink or calcoflur and/or gram stain. (b) The organization section It must be dyed with E, GMS and PAS digests. The hypnotic elements of Scedosporium Boydi and Scedosporium prolificans are indistinguishable from those of Aspergillus hypnosis and h&amp You may miss it in the E-dye section. Branch, examine the specimen for hypnosis. Interpretation: The presence of haemorrhoids in any specimen from patients supporting clinical symptoms should be considered significant. Biopsies and evidence of tissue aggression are especially important. Remember that a culture is needed for the specific identification of causal agents. 3. Culture: The colony is growing rapidly and is gray-white, with a surface texture such as suede from olive gray to black. Interpretation: S. apiosperm, S. boydii, S. aurantiacum and L. prolificans are common soil fungi, and therefore benign cultures from non-acorn specimens such as phlegm or skin, need to be directly supported by microscopic evidence in order to be considered important. A biopsy of the sterile site or benign culture from aspiration saperation should be considered important. Cultural identification is the only reliable means of distinguishing these fungi from the AsperGillus species. 4. Cerro: Aspergilomyosis immunochloride blood injection test has become valuable in the diagnosis of caustic sutosis. However, the current reagent is not commercially available and antigen extract should be made in the laboratory. 5. Identification: Cultural characteristics and microscopic forms are especially important in the easy form, the arrangement of Conidia for conscientious. and the form of a large-resistant cells, in this case Annelid. 6. Cause formulations: Scedosporium apiospermum, Scedosporium aurantiacum, Scedosporium Boydi, Romenaspora fertility. Read more: Azelo L and R.J. Hay. 1997. Medical Mycological Vol 4 Topless && Wilson's microbiology and infectious infections. 9th edition, Arnold London.Roadside Road, F., J. Cano, J. Jean, J. Guaro. 2005. Molecular phyloscience of the Shudalesharia Boidi species complex: the proposal of two new species. Jay Clean. Microorganisms. 43:4930-4942.Kwon KJ and Zebenet 1992. Medical Mystic Leah & Fevigher.Richardson MD and DW Warnock. 1993. Fungal infection: diagnosis and management. Blackwell Science Publications, London. Rippon JW. 1988. Medical Biology WB Saunders Co. Warnock DW and MD Richardson. 1991. Fungal infection in damaged patients. 2nd edition. John Wyle and sons. Zygomycosis (mucosis) the term zygomycosis describes the infection in the broadest sense because it is a member of Zygomymycetes. These are primitive, fast-growing, terrestrial, and saffromatic fungi with a largely international distribution. To date, some 665 species have been described although infections in humans and animals are generally rare. Medically important sequences include Genera: 1. Mukallares and Mortierela, Subcutaneous and systemic zygomycosis causing (Mucormycosis) - Rizophopus, Lichteimia, Rikomukor, Mukor, Cunningnella, Sasenaa, Apophehisomes, Kokeromys. 2. Mymorphitoles, subcutaneous zagomicosis (Entomophthoromycosis) - Conidiobolus and Basidiobolus. Hyalohyphomycosis fungal infections of humans or animals caused by the number of hyaline (non-dematiaceous) hyphomycetes, where the tissue form of the phosphorus is a fungus of the organism. This separates the cause from the brown pigment, the bacterium. Hyalohyphomycosis is a common term used to group infections together caused by unusual hyaline fungal pathogens, not agents of otherwise named infections. Like Aspergilosis. The pathology agents include species of penicillium, Paecilomyces, Acremonium, Beaubeia, Fusarium and Spleuriosis. Clinical symptoms: Clinical symptoms of hyalohyphomycosis range from harmless sofafitic colonies to acutely invasive diseases. Aging factors include prolonged neutropenia, especially in leukemia patients or bone marrow transplant recipients, corticosteroid therapy, cytotoxic chemotherapy and fewer patients with AIDS. Typical patients receive photospectrum antibiotics for granule cellosis and unexplained fever. Laboratory diagnosis: 1. Clinical substances: skin and nail scratching; urine, phlegm and bronchial washing; Cerebrospinal fluid, pleural fluid and blood; Tissue biopsy from various visceral organs and residential catheter tips. 2. Direct microscopy: (a) skin and nail scratches, phlegm, washing and aspiration should be examined using 10% KOH Ink or calcofluor white mount; (b) Exec date and fluids must be centrifuged and sediment must be inspected using 10% KOH and parker ink or calcofluer white mount, and (c) tissue sections must be stained using PAS digest, grocote's metenamine (GMS) or gram stain. Note that hypnosis elements are often H&Amp E staining is difficult to detect in the section. Interpretation: The presence of hyalin to branch any specimen similar to aspergillis should be considered significant from patients supporting clinical symptoms. Biopsies and evidence of tissue aggression are especially important. Remember that direct microscopy or histopathology does not provide specific identification of the cause agent. 3. Culture: Clinical specimens must be inoculated in primary isolation media, such as Saburaud's Dextros cloth. Interpretation: The hyaline hyphomycetes involved are well recognized as common environmental air pollutants, and therefore a positive culture from non-sterile specimens such as phlegm or skin, need to be supported directly by microscopic evidence to be considered important. It is also helpful to support clinical hisores in patients with a proper predisposition history. Cultural identification is the only reliable means of distinguishing these fungi. 4. Cerro: There are currently no commercially available serological procedures for the diagnosis of infections classified under the term hyalohyphomycosis. 5. Identification: Culture characteristics and microscopic forms are especially important in the form of organization, conidia arrangement for the eccentricity cells, and the form of the eccentric ity cells. 6. Cause Disciple:Acremonium sp., Beauberia Soup., Pusarium Soup., Paecilomyces sp., penicillium soup., Scopulariopsis sp. Read more: Azzelo L and R.J. Hay. 1997. Medical Mycological Vol 4 Topless && Wilson's microbiology and infectious infections. 9th Edition, Arnold London Booth, C. 1977. Fusarium: A laboratory guide to the identification of major species. Commonwealth Institute of Mystics, Kew, Surrey, England.Burgess, L.W., C.M. Riddell. 1983. Laboratory manual for hussarium research. Hussarium Institute, Department of Plant Pathology and Agricultural Entomology. University of Sydney, Australia. Domsch, Gams, Anderson. 1980. Compensation of soil fungus volume 1. Academic Press.Hog de GS and J Guadro. 1994. The Atlas of Clinical Fungus in Centraalcountry voor Schimmelcultures, Barnes, Netherlands. CBS publications are available for order from Tinke van den-Berg-Visor, Centraal Country voor Schimmelcultures, PO Box 273, 3740 AG Baarn, Dutch FAX + 31 2154 16142. Medical Mystic Leah & Fevigher.Richardson MD and DW Warnock. 1993. Fungal infection: diagnosis and management. Blackwell Scientific Publications, London.Warnock DW and MD Richardson. 1991. Fungal infection in damaged patients. 2nd edition. John Wyle and sons. Myrcan infection of humans and lower animals, caused by various dematiaceous (brown pigment) fungi, where the tissue form of causal organisms is a fungus. This separates it from other clinical types of the disease, which contain brown pigment fungus, in which the tissue form of the organism is grains (myeloid myeloid) or curable (cromomocytomy). Etiology agents include exofiala, fialalala, bipolar, execehelium, cladopialala, berukonis, aurenovasium, chleosporium, kerbularia and alteraria in particular dematiaceous hyphomycetes species. Agelo (1986) listed 71 species in the 39th generation as the causative agent for peoyu pomicos. Clinical symptoms: The clinical forms of paohisomikosis range from topical infections of the astrocytore (tinea nigra) to subcutaneous cysts to brain aggression. 1. Subcutaneous pomycosis: Subcutaneous infections usually occur worldwide, depending on the traumatic transplantation of fungal elements from contaminated soil, thorns or tree fragments. Exofiala jeans Elmei and Wangiela dermatitis are the most common preparations in adults, and cystic lesions occur most often. Sometimes, overly verrucous lesions are formed especially in immunosuppressed patients. 2. Paranasl sinus fiopiamikosis: Empirical fungi, especially bipolar, xerohilium, Curvularia and sinusitis by alternariasis are increasingly reported in patients with a history of allergic rhinitis or immunosuppression. 3. Cerebral peofamycosis: cerebral paoifamycosis is rarely infected, mainly occurs in immunosuppressive patients after conia inhalation. However, cerebral infections caused by Cladophialophora bantiana have been reported in a number of patients without any apparent indisposition. This fungus is rare lysitating on sites other than neuronutrition and CNS. Laboratory diagnosis: 1. Clinical substances: skin scratches and / or biopsy; Sputum and bronchial washing; Cerebrospinal fluid, pleural fluid and blood; Tissue biopsy from various visceral organs and residential catheter tips. 2. Direct microscopy: (a) skin scratches, phlegm, bronchitis and aspiration should be examined using 10% KOH and parka ink or calcofluor white mount. (b) Exadate and fluids must be centrifuged and 10% to be inspected for sediment using 10% KOH and parker ink or calcofluer white mounts, and (c) tissue section It must be dyed using metenamine (GMS) from E, PAS Digest and Grocote. Analysis: brown pigment, branch edifying presence in any sample, should be considered important from patients supporting clinical symptoms. Biopsies and evidence of tissue aggression are especially important. Remember that direct microscopy or histopathology does not provide specific identification of the cause agent. Note: Direct microscopy of the tissue is required. Between cromomocytes, characterized in that the tissue form of chromosomes is present in the tissues of the fungus brown pigment, flat division, round hardening body and symptomatic. 3. Culture: Clinical specimens must be inoculated in primary isolation media, such as Saburaud's Dextros cloth. Interpretation: The associated dematiaceous hyphomycetes are well recognized as common environmental air pollutants, and therefore have a positive culture in non-sterile specimens, such as sputum or skin, which need to be supported by direct microscopic evidence in order to be considered important. It is also helpful to support clinical hisores in patients with a proper predisposition history. Cultural identification is the only reliable means of distinguishing these fungi. 4. Seroji: There are currently no commercially available serological procedures for the diagnosis of infections classified under the term peoipomikos. 5. Identification: Culture characteristics and microscopic forms are especially important in the form of organization, conidia arrangement for the eccentricity cells, and the form of the eccentric ity cells. We recommend preparing cellotape flags and/or slide cultures. 6. Causal agents: Alteranaria soup., aureobadid pululans, bipolar soup., cladopialalofora bantiana, berluconis gallofaba, kerbularia soup., exofiala soup., Exo-Pyla soup., Pialova Berukosa. Read more: Azelo L and R.J. Hay. 1997. Medical Mycological Vol 4 Topless && Wilson's microbiology and infectious infections. 9th edition, Arnold London.Ellis MB. 1971 and 1976. Dematiaceous Hyphomycetes and more Dematiaceous Hyphomycetes. Institute of International Mysticism. Hoog de GS et al. 1977. High-formeise of black yeast and alliance. Research research N0. 15 From Centraal country voor Schimmelcultures, Right, Netherlands. CBS publications are available for order from Tinke van den Berg-Visor, Centraal Bureau voor Schimmelcultures, PO Box 273, 3740 AG Barn, Dutch FAX + 31 2154 16142.Hoog de GS and J Guarro. 1994. The Atlas of Clinical Fungus in Centraalcountry voor Schimmelcultures, Barnes, Netherlands. CBS publications are available for order from Tinke van den-Berg-Visor, Centraal Country voor Schimmelcultures, PO Box 273, 3740 AG Baarn, Dutch FAX + 31 2154 16142. Medical Mystic Leah & Fevigher.McGuinness MR. 1980. Laboratory Handbook of Medical Mystics. Academic Press [This is not printed, but a copy will be a valuable argument]. Richardson MD and DW Warnock. 1993. Fungal infection: diagnosis and management. Blackwell Science Publications, London. Rippon JW. 1988. Medical Biology WB Saunders Co. Warnock DW and MD Richardson. 1991. Fungal infection in damaged patients. 2nd edition. John Wyle and sons. Son.