Immunological Study About Blastomyces dermatitidis

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INTRODUCTION

Blastomycosis is a disease caused by a type of dimorphic fungus called Blastomyces dermatitidis, which is the fungus in which the Central United States is an endemic area.

These fungi multiply in nature in the form of filaments, or what is known as the filamentous form (Mold) and transform in the body from filamentous form to the yeasty form (yeast). The infection is transmitted by inhalation of conidia, or what is known as the external spores that the fungus produces, through the respiratory tract.

Healthy people who develop spondylosis recover automatically, and the spermatozoa breakfast has symptoms that include fever, in particular, feeling weak and losing weight. Sometimes, especially in people with impaired functioning of the immune system, systemic disease can develop - affecting many body systems and organs.

The most common symptom in these patients is pneumonia, followed by skin lesions (cauliflower or ulcerative lesions), osteomyelitis, prostatitis, or an infection of the central nervous system, some of those who breathe may have flu-like symptoms. in people with weakened immune systems, the infection can become severe, especially if it spreads from the lungs to other organs.

The infection may spread to the skin, bones, genital tract, urinary system and tissues that cover the brain, causing swelling, pain and other symptoms. doctors remove a sample from the infected sputum or tissue and send it to the laboratory for culture (Ibrahim et al., 2017).
Patients should take antifungal medications for months. Most cases of blastomycosis occur in areas of North America where fungi live in the soil near the river course (Bander et al., 2015); (Hussain et al., 2018); (Dahham et al., 2019). Ohio Valleys and the Mississippi River (extending to the states of the Middle Atlantic Region and southeastern states), Northwestern North New York State Southern infection occurs on rare occasions in the Middle East and Africa (Kaufman et al., 2001); (Green et al., 2002).

A Brief Summary of *Blastomyces dermatitidis*:

The inflamed skin bud was called the causative agent of blastomycosis in general. And over the years I think that this nomenclature from a taxonomic point of view is incorrect and I took many synonyms that were all rejected and kept the old designation. Dogs are the most vulnerable animal after humans (Bander et al., 2015); (Hussain et al., 2018). Therefore, it was used as a human substitute in epidemiological studies (Bander et al., 2015); (Hussain et al., 2018); (Dahham et al., 2019). As mentioned above, the lung is considered the gateway to infection for breakfast bud and there are no reports of transmission from person to person be the one that spoke about the transmission of the disease between two young people working in one place.

The possibility that the two young men have been exposed to the same pathogen is also possible.

It is also reported that two reports of the transmission of flanking inflamed skin via the placenta. As shown in Figure (2) Fungus under the microscope for the skin form of the disease as a result of accidental injection as a dog bite or the wrong exposure to the causative agent during autopsy (Hussain et al., 2018).
Blastomycosis was first described in 1894 by scientist Gilchrist in a patient with a skin injury. Initially, it was thought that it was the result of a tuberculosis-skin disease, as it was common at the time, but the histological examination showed the presence of multiple foreign bodies instead of the TB bacillus (Ibrahim et al., 2017). Then he published his second report in which he explained that the causative factor is the result of what is known as breakfast, which is a general term used at that time to refer to yeasts (Hussain et al., 2019).

Knowing the environment of the causative agent is very important in understanding the epidemiology of this fungus (Turner et al., 2016) ; (Young et al., 2000) (Furculow et al., 2011) . In spite of the early discovery of Burmese fungi, it was not specified the environments in which it could be found, the availability of some evidence and reports on it made it possible to determine its whereabouts of it (Klein et al., 2017).

In 1961, the bud, inflamed skin, was isolated from the soil and contained some tobacco and organic waste in America Denton / 1126. However, it was concluded that the place of isolation was an old house of a dog with Burmese breakfast (Abdulbaqi et al., 2018).

**The Identity of The Inflamed with The Skin, Is Determined Using Several Morphological Properties:**

1. **Transformation in Tubes:**

As these fungi are transformed from the filamentous form that is located at a temperature of 25-30 °C (on the subordinate glucose agar) to the fermented form on a nutrient medium (Klein et al, 2008) (such as the infusion of the brain and the heart containing 5% blood) at 37 °C within 3-5 days. It can also be transformed from the yeast shape to the filiform shape under the same conditions (Hussain et al., 2017).

2. **The Filiform Shape:**

In cultures: inflamed skin bud budsea colonies need 2-4 weeks at the 25th streptococcus degree to grow on the sabboard glucose agar culture when isolated from clinical samples. Typical colonies are white, airy, and cotton fungal strings (Klein et al., 2014). They are either flat or convex as a result of gathering fungal strings. Colonies are white at the beginning of growth and may later turn brown or light brown. He also sees concentrated episodes of growth in the old colonies (4-8 weeks) (Hussain et al., 2019).

Under the microscope: Both breakfast and non-sexual spores appear. The strings are 6-7 micrometers thin, transparent, branched, and partitioned. Creating new growths on top of the threads whereas,
single-cell spores are borne on thin, lateral, or direct stools on the strings. It also takes the oval to a circular shape with smooth walls and diameters of 3-7. m. It should be noted that the bud, inflamed skin, cannot be diagnosed only on the basis of the shape of the spores due to its similarity with many other types (Abdulbaqi et al., 2018)

3. Yeast Form:

Also known as the histological form, the colonies appear in the form of soft, brain-shaped colonies that look like butter and brown. Under the microscope: Yeasts appear 8-12 micro m in diameter and with a thick wall (often referred to as a double wall) with a single broad-based bud present when in contact with the parent cell. In an electron microscope, yeasts appear to contain 2-4 nuclei per cell, with a two-layer wall between them, with a transparent area as shown in Figure (3) A, B, and C Diagnosis of mycosis fungoides (Klein et al., 2013).

Diagnosis of Macrophages:

Blastomycosis is easily diagnosed by extracting samples from the affected organ and examining it with a microscope or by culture.

Fig. 3: A, B, C Diagnosis of mycosis fungoides (Ibrahim et al., 2017).
Serologic tests are not effective for the diagnosis of blastomycosis and are used primarily for epidemiological reasons. The doctor diagnoses blastomycosis by sending a sample of infected sputum or tissue to the laboratory for examination under a microscope and culture, and the chest x-ray is imaged to check for signs of infection in the lungs (Cox et al., 2014).

Doctors may also examine a sample of urine to check for proteins (antigens) released by fungi.

**Blastomyces dermatitidis Treatment:**

If the *Blastomyces Dermatitidis* is severe, it is treated with intravenous amphotericin B, and if the condition ranges from mild to moderate, then oral itraconazole is administered as shown in Table (1) Types of fungi and treatments.

The treatment improves the patient's condition rather quickly, but the treatment period should last for 6 to 12 months. Untreated, mycosis fungi slowly worsen, and in rare cases can be fatal (Hall et al., 2001; Deighton et al., 2005).

The main drug for treating mycosis is Itraconazole, which belongs to the Azoles family (Dheeb et al., 2019).

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathogen</th>
<th>Signs and Symptoms</th>
<th>Transmission</th>
<th>Antimicrobial Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspergillosis (cutaneous)</td>
<td><em>Aspergillus fumigatus, Aspergillus flavus</em></td>
<td>Distinctive eschars at site(s) of infection</td>
<td>Entry via wound (primary cutaneous aspergillosis) or via the respiratory system (secondary cutaneous aspergillosis); commonly a hospital-acquired infection</td>
<td>Itraconazole, voriconazole, amphotericin B</td>
</tr>
<tr>
<td>Candidiasis (cutaneous)</td>
<td><em>Candida albicans</em></td>
<td>Intertigo, localized rash, yellowing of nails</td>
<td>Overgrowth of normal skin microbiota, especially in moist, dark areas</td>
<td>Azoles</td>
</tr>
<tr>
<td>Sporotrichosis (rose gardener's disease)</td>
<td><em>Sporothrix schenckii</em></td>
<td>Subcutaneous ulcers and abscesses; may spread to a large area, e.g., hand or arm</td>
<td>Entry via thorn prick or other wound</td>
<td>Itraconazole</td>
</tr>
<tr>
<td>Tineas</td>
<td><em>Trichophyton spp., Epidermophyton spp., Microsporum spp.</em></td>
<td>Itchy, ring-like lesions (ringworm) at sites of infection</td>
<td>Contact with dermatophytic fungi, especially in warm, moist environments conducive to fungal growth</td>
<td>Terbinafine, miconazole, clotrimazole, griseofulvin</td>
</tr>
</tbody>
</table>

In the event that there is a risk to the patient's life, mycosis is treated with amphotericin B. Fungi are eukaryotic organisms belonging to the fungi kingdom, which require interaction with another organism to survive, the risk factors for this infection are humidity, the presence of fats in the body and the immune system. Caused by Blastomyces dermatitidis Systemic diphosphate (Klein et al., 2008).

**Body Immunity:**

Most fungi are highly immunogenic and generate significant antibody and T cell-mediated immune responses (Cox et al., 2011), which may be identified by serology and delayed-type (type IV) hypersensitivity
skin reactions. T cell-mediated immunity is essential for fungus resistance (Klein et al., 2009).

TH1 T cells and macrophage activation, rather than antibody-mediated responses, are thought to have the most important protective function (Hall et al., 2001; Deighton et al., 2005). Type 1 (T1) reliant cell-mediated immunity, which can be mediated by CD4 and CD8 T cells, is required for protection against infections with dimorphic fungi such Blastomyces dermatitidis (Cox et al., 2017). Resistance by both T cell subsets requires the production of T1 cytokines, particularly IFN-γ and TNF-α. IL-12 induces IFN-γ production from T and NK cells, promotes the differentiation of T1 cells from naive T cells, and therefore regulates cell-mediated immunity against fungal and parasitic diseases (Cox et al., 2011).

Resistance to most pathogenic fungi and most systemic mycoses including B. dermatitidis is clearly dependent upon T cell-mediated immunity, particularly CD4 TH1 cells secreting IFNγ, dendritic cells are necessary for this response and produce IL-12, after engulfing fungi (Hussain et al., 2017).

CONCLUSION
Clinically, dermatophytes may be confused with spinal keratoma, squamous cell carcinoma, tuberculosis, tertiary syphilis, leprosy, or bacterial pyoderma. Skin symptoms occur in about 80% of people with fungi. They look like warts and may vary from gray to purple, and blisters that blister bleeds easily, over time these lesions can lead to scarring and loss of skin color.

Through a review conducted of several reports about the occurrence of blastomycosis infection, it was found that there was no correlation between the occurrence of the disease and the seasonal period. Also, arriving at a correct judgment of the relationship between time and infection is very difficult, perhaps due to the skin lesions that can appear at different times after the initial infection (Lancaster et al., 2004). Also, the pulmonary infection can be invisible or not distinguished, and it can be long before the diagnosis.

It was concluded that there was no relationship between infection, profession, or hobby of a person with Burma's disease. But it can be said that the relationship is between infection and a person's presence in a possible environment for infection (Bradsher et al., 2003) (Lancaster et al., 2004). There is no relationship between age, gender, or race, and the incidence of infection (Thurmond et al., 2019). But considering that men's occupations are more likely to be present in infection-causing places, the percentage of men's infections is more than for women.

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