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Erosio interdigitalis blastomycetica: A review of interdigital candidiasis

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Abstract

Erosio interdigitalis blastomycetica (EIB) is a *Candida* infection affecting the third web space, between the third and fourth fingers. In 1915, Gougerot and Goncea first described saccharomycetic organisms isolated from the hands and feet. Johannes Fabry later named it in 1917, well before the genus *Candida* was introduced in 1923. EIB is most common among those who work with their hands frequently in water, such as dishwashers, launderers, bartenders, and homemakers. Clinical presentation most commonly consists of a central erythematous erosion surrounded by a rim of white macerated skin involving at least one interdigital web space. The differential diagnosis is narrow, consisting of irritant contact dermatitis (ICD), erythrasma, inverse psoriasis, and bacterial infection (i.e. impetigo). The diagnosis is made by clinical examination in addition to fungal culture and KOH testing. The prognosis is good and treatment options include avoidance of frequent water immersion and topical or oral antifungal agents. Suspicion for secondary infections such as erysipelas and cellulitis should remain high until lesions have resolved. This review aims to address the history, epidemiology, pathophysiology, histopathology, clinical presentation, differential diagnoses, diagnosis, prognosis, and management of EIB. It also suggests an alternative name in place of the current misnomer.

Keywords: *erosio interdigitalis blastomycetica*, *candida* infection

Introduction

Erosio interdigitalis blastomycetica is not well represented in current literature. In fact, studies that investigated the condition's incidence have been scarce. However, there have been two studies in particular that gave us a good estimation of what fraction of cutaneous *Candida* is represented by EIB [1]. First, a 1990 study of 150 patients with cutaneous *Candida* infections revealed that EIB represented only 14 of the patients, a mere 9.3% [2]. A second study performed in 1997, showed similar results to this, finding that out of 714 participants diagnosed with cutaneous *Candida*, just 15% of them had EIB [3].

It is well known that diabetes mellitus patients are frequently affected by cutaneous *Candida* infections; thus, it follows that many patients affected with EIB have a history of diabetes [1, 4, 5]. However, there is an even more strikingly unifying characteristic amongst those affected by EIB: exposure of the hands to water [1, 6, 7]. There are common careers and lifestyles amongst patients diagnosed with EIB. These patients are often dishwashers, launderers, bartenders, farmers, poultry slaughterhouse workers, and homemakers [4, 6, 8, 9]. Additionally, patients that are immunocompromised, either because of a disease state or corticosteroid use, are particularly represented in the patient population affected by EIB [4, 5]. The condition is almost exclusively seen in adults and does not usually affect children,[10].

This up-to-date review serves to expand the available knowledge regarding EIB and its history, causative factors, differential diagnosis, and treatment. PubMed, MEDLINE, and Web of Science databases were searched up to June 2017 using the terms “Erosio Interdigitalis Blastomycetica,” “Interdigital Candida,” “Interdigital Candidiasis,” and “Superficial Candidiasis”.

History and Background

Cutaneous infections caused by *Candida species* (*Candida spp.*) present in a multitude of fashions, including paronychia, onychomycoses, folliculitis, and various forms of intertrigo (inflammation involving skin folds) including diaper candidiasis and erosio interdigitalis blastomycetica (EIB). EIB is a form of intertrigo specifically involving the interdigital webs, most commonly the 3rd interdigital space (area between the 3rd and 4th finger or toe), but also seen in the 4th interdigital web [4].

The condition was first studied in 1915 by Gougerot and Goncea, who isolated a saccharomycetic organism from the hands and feet [10]. Johannes Fabry described EIB again in 1917 in a series of cases of the condition. The infections described may have been secondary to routine use of washing powders throughout the day for religious purposes [10, 11].

It was Fabry who gave the condition the name erosio interdigitalis blastomycetica, which may be a confusing term in modern times as the causative organism is not *Blastomyces dermatitidis*, the

causative organism of blastomycosis. This name probably resulted because *Candida* is in the class *Blastomycetes*, and the genus *Candida* was not introduced until 1923 [6, 10, 11].

The first case report in the United States was described by Mitchell at a Chicago Dermatological Society meeting in 1922 [11]. This case report described a housewife who had the infection consistently for 6 years, only rarely undergoing remission for a few days [11].

Most of the knowledge that we have about the disease comes from experimental research trials done on human subjects. In 1922, an article by Koetter demonstrated successful inoculation of **volunteer medical students' finger webs with the organism** and production of the typical lesion [6]. Another study in 1973 by Rebora, Marples, and Kligman recruited a cohort of 53 male prison volunteers (18 black, 35 white) who were inoculated with *Candida albicans* (*C. albicans*), allowing the field of dermatology to gain further insight into the condition [7]. Table 1 depicts all studies of EIB existing in the literature.

Clinical Presentation

The lesions of EIB have very characteristic features. To begin, the lesion is always seen in the areas of friction between the fingers, never extending to the dorsal or palmar surfaces of the hand [10]. The area



Figure 1. A white collarette surrounded by erythematous tissue



Figure 2. A round to oval focus of white, macerated tissue separated from the erythematous layer beneath it. Reprinted with permission from www.dermnetnz.org.

of infection is usually a round to oval focus with white, macerated tissue that becomes separated from the layer beneath it, forming a halo around the underlying tissue. This is commonly referred to as the

Table 1. Cases of *Erosio Interdigitalis Blastomycetic* reported in the literature.

Number of Cases	Demographics	Year	Occupation/Exposure/ Medical Problems	Treatment	Reference
NM	NM	1915	NM	NM	[3]
NM	NM	1917	NM	NM	[5]
NM	NM	1918	NM	NM	[5]
NM	NM	1918	NM	NM	[5]
45	38 F, 7 M	1921	NM	NM	[5]
7	NM	1922	Experimental inoculation	NM	[2]
3	NM	1922	Clothing washer	Whitfield's ointment^a , chrysarobin in chloroform and petroleum, radiotherapy, weak tincture of iodine, salicylic acid powder, discontinuation of washing clothes ^b	[2]
13	12 F, 1 M	1928	Dishwasher (1); "washerwomen"; experimental inoculation of a medical student	Whitfield's ointment^a , chrysarobin, combination of salicylic acid and ammoniated mercury, radiotherapy ^c , avoidance of soapy water ^b , application of 2% copper sulphate aqueous solution ^b	[5]
135	115 F, 20 M	1989	Poultry slaughterhouse workers	Handwashing with medicated soap, polyethylene or cotton gloves	[13]
94	NM	1990	Chronic water exposure	NM	[8]
53	M	1973	Experimental inoculation of prisoners	Removal of occlusive dressing ^b	[6]
108	82 F, 26 M	1997	NM	NM	[9]
1	M	2002	Diabetic	NM	[10]
2	NM	2005	Farmers	NM	[12]
1	F	2011	NM	Oral cephalosporins, azithromycin, topical dexamethasone cream, triamcortisone ointment, mupirocin ointment, Itraconazole and topical bifonazole cream	[30]

NM = not mentioned; ^aWhitfield's ointment is a combination of half strength benzoic and salicylic acid ointment; ^bsuccessful treatment regimen; ^cunfiltered X-ray and Kromeyer light.

“white collarette” of epidermis [6, 7]. The underlying tissue is intensely red, shiny, raw, and eroded (Figures 1, 2), [6, 7]. There is often erythema surrounding the lesion that can extend down the sides of the fingers. Also, it is not uncommon to have satellite lesions extending down the inner sides of the fingers as well [7]. The most commonly affected interdigital space is the third web, between the middle and ring fingers [12].

The infection of the interdigital spaces of the toes present similarly to the fingers. However, the white uppermost epidermis may not separate from the bottom layer as easily because the glabrous skin of the feet are thicker and retain integrity, despite a moist state [12]. Since the pathognomonic beefy red layer is not seen in EIB of the webs of the feet, it can be easily mistaken for tinea pedis [12].

The symptom that patients with EIB most commonly report is pruritus [6]. Patients may also complain of pain, although less commonly, and they will most likely experience this discomfort with attempts to remove the overlying white tissue from the underlying red tissue [6]. Otherwise, patients with the condition often tolerate it very well [7].

Differential Diagnosis

The differential diagnosis of erosio interdigitalis blastomycetica includes irritant contact dermatitis (ICD), erythrasma, psoriasis (i.e. inverse), and bacterial infection (i.e. impetigo), [1, 4]. Irritant contact dermatitis usually presents as a diffuse dermatitis, most often localizing to the site of contact, which would usually include the dorsal hand in addition to the web spaces. Erythema, scaling, hyperkeratotic plaques, and fissures are not uncommon in ICD [1, 13].

Erythrasma is a chronic cutaneous infection caused by the gram-positive rod *Corynebacterium minutissimum*. Classically, this condition presents as erythematous, brown patches with scale and maceration involving intertriginous areas, such as the groin, axillae, or interdigital regions of the hands or feet. Lesions may be asymptomatic or patients may present with the complaint of pain or pruritus. Coral-red fluorescence under Wood's light in the

affected area further differentiates the lesion from EIB [1, 14].

Inverse psoriasis may also present within flexural surfaces and interdigital spaces. However, the pruritic plaques are thin, often lack scale, and tend to appear symmetrical [1]. Additionally, a full physical examination may reveal other characteristic findings of psoriasis such as nail pitting, gluteal cleft pinking, and scalp psoriasis [1].

Impetigo is a cutaneous infection that also exists in the differential diagnosis of EIB. The causative organism is usually *Staphylococci* or *Streptococci*. Skin trauma or breaks (i.e. scratch, wound, ulcer, and insect bite) allow for infection to commence, producing a focal, erythematous and possibly tender area of skin. Lesions may progress to superficial erosions with serous exudates, which dry into a characteristic “honey yellow crust.” Pustules, blisters, and bullae may also occur [1].

Histopathology

If the lesion is biopsied, hematoxylin-eosin stain will show loss of the epidermis with necrosis and an inflammatory infiltrate including lymphocytes and neutrophils in the necrotic tissue with or without fungal spores and pseudohyphae. Periodic acid Schiff (PAS) stain and fungal silver impregnation stains (Grocott or Gomori methenamine silver [GMS]) can also be performed to identify fungal elements [15, 16].

Diagnosis

Thorough clinical examination alone is sufficient to diagnose EIB, but ancillary methods are often helpful. For example, direct swabbing of the interdigital space with direct examination and soaking of the macerated epidermis in KOH will reveal pseudohyphae and budding yeast [1, 6]. Fungal culture is often necessary to diagnose EIB, whereas bacterial culture with gram stain of the exudate is useful to rule out impetigo [1, 4, 12]. Skin biopsy of the tissue is another consideration, though less commonly performed [15]. Depending on additional symptoms present, tests to rule out immunodeficiencies or endocrine disorders may be necessary. This includes serum glucose, HIV

antibodies, purified protein derivative (PPD) skin test and/or chest X-ray, and serum immunoglobulins, specifically IgA [15].

Pathophysiology

The pathophysiology of EIB, and infections by *Candida* in general, involves multiple interacting factors including organisms, host factors, endocrine factors, immunological factors, and immunosuppressive conditions such as HIV/AIDS.

Endocrine Factors

Endocrine disorders such as diabetes mellitus enhances susceptibility to infection by *Candida*. Ueta et al. suggest that elevated glucose suppresses PMN activity, which prevents the immune system from killing the yeast and leads to increased prevalence and therefore enhanced risk for the development of cutaneous *Candida* infections such as EIB [17].

Additional endocrinopathies associated with T-cell impairment, specifically T_H17 cells, reduce the **immune system's ability to combat the dimorphic fungi** resulting in opportunistic infections such as chronic mucocutaneous candidiasis and possibly EIB. This is exemplified in patients with Autoimmune Polyglandular Syndrome Type 1, which is also known as Autoimmune Polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED), a disorder in which a mutation in the autoimmune regulator (AIRE) gene results in a triad of hypoparathyroidism, Addison disease, and chronic mucocutaneous candidiasis (CMC), [18, 19]. Though the mucosa is primarily affected in this disorder, candidal onychomycosis and paronychia can also occur [20]. Additionally, this condition is more common among children than adults. Cushing syndrome, spontaneous and iatrogenic, is another endocrine disorder resulting in a suppressed immune system that allows opportunistic infections such as candidiasis to thrive. This is through direct suppression of T-lymphocytes by endogenous or exogenous corticosteroid [21].

Host Factors

Macroscopically, the necessary environment for the disease to occur includes three components: moisture, maceration, and heat. As already **discussed, moisture is provided by the patient's** workplace or lifestyle, or as the result of occlusion

[22]. The requirement for moisture is illustrated by a 1973 study on prisoners in which the infection would only persist if the interdigital space was kept occluded (the fingers were bound together), thus allowing the area to remain moist. This moisture is the reason why the second necessary component occurs: the skin macerates [7]. As maceration occurs, *Candida*, **a normal inhabitant of the skin's** microbiome, becomes a pathogen [12]. Once the *Candida spp.* creates a suitable inflammatory environment, gram-negative rods are thought to inhabit the macerated lesion and play a role in the chronicity of the lesion through the production of proteases [7].

On a microscopic level, phagocytosis is another important host element that both combats fungal growth, but also enhances recurrent *Candida* infection. Phagocytosis of the yeast by polymorphonuclear neutrophils (PMN) leads to its destruction by free radical formation. Epithelial and endothelial cells are also capable of engulfing the yeast; however, they are less equipped with mechanisms to destroy the yeast [23, 24]. Thus, the epithelial and endothelial cells protect the internalized yeast from the destructive effects of **antifungals and the host's immune system** [23].

Immunological Factors

There are many immunological components involved in preventing or combating infection by *Candida* (i.e. lymphocytes, PMNs, cytokines). Thus, patients with defective or suppressed lymphocytes, neutrophils, macrophages, and specific cytokines (interleukins-12, -23, -27, and -35) are at increased risk of infection by *Candida*. This includes defects in any part of the interactive pathways involved in killing the yeast, such as gene expression or peptide production leading to cytokine expression [18, 19]. Examples include severe combined immunodeficiency syndrome (SCID), hyper-IgE syndrome, ICAM-1 deficiency, dectin-1 deficiency, CARD9 deficiency, interleukin IL-17RA and IL-17F gene deficiency [18, 19]. Although these immunodeficiencies are more often associated with CMC, fungal skin and nail infections also exist in the literature.

Corticosteroid use is another avenue of immune suppression associated with candidiasis. Both systemic (i.e. oral and intravenous) and local (i.e. topical) applications of corticosteroid are demonstrated in association with cutaneous *Candida* overgrowth. Topical application of corticosteroid reduces the local inflammatory response and cell-mediated immunity, allowing for *Candida* to thrive and cause infection [15, 25, 26]. Systemic corticosteroids also suppress innate and adaptive immune pathways on a much broader scale, also leading to cutaneous *Candida*.

Immunosuppressive Factors

Candida is considered an opportunistic organism owing to its natural role in the normal mucosa of about 50% of immunocompetent individuals and its ability to cause disease in the immunocompromised [27, 28]. This endangers individuals with immunosuppressive diseases such as HIV/AIDS. Although oral candidiasis is one of the most common dermatological diagnoses in AIDS patients, non-mucosal candidiasis may also occur [29, 30]. For example, Muñoz-Pérez et al. evaluated dermatological conditions in 1161 patients with HIV-1 and found 28 patients with candidal folliculitis, 16 with candidal intertrigo, and 9 with candidal nail infection [29]. These conditions, more specifically oral candidiasis, are believed to arise specifically through an immune deficiency in IL-17 produced by Th17 cells in patients with HIV-1 and expressing AIDS-like disease [31].

Organisms

There are many subspecies of *Candida*; however, *C. albicans* is one of the more frequently encountered and more virulent organisms. Within the *C. albicans* species itself, there are certain strains more likely to cause disease than others. These virulent strains are polymorphic, have specific adhesin and invasin proteins, possess the ability to form biofilms, contain contact sensing capabilities and thigmotropism, and secrete certain hydrolases (i.e. protease, phospholipase, and lipase) in addition to many other pathogenic characteristics [23, 32].

With respect to polymorphism, *C. albicans* can grow as a budding yeast (i.e. in disseminated candidiasis), as ellipsoid cells with pseudohyphae, or as true

hyphae (i.e. invasive *Candida*) [32]. Ability to produce hyphae is suspected to contribute to virulence as this form is most often found on histopathology examination of invasive *Candida*. There is also data demonstrating decreased virulence in strains lacking hyphal-associated gene products [33]. Hyphal growth is influenced by host environment, with induced growth occurring with a pH >7, starvation, physiological temperature and carbon dioxide changes, and low cell densities (versus high cell density inducing yeast growth), [32]. *Candida* may also undergo phenotypic switching in addition to morphogenesis, which may also contribute to virulence [32].

Adhesin proteins, specifically agglutinin-like sequence (ALS), are equally important for *C. albicans* virulence as it mediates the ability of the yeast to attach to other microorganisms or host endothelial cells [32]. Invasins, such as Als3 (also an adhesin) and Ssa1 (cell surface heat shock protein), are responsible for fungal induced endocytosis and active penetration, both essential for virulence [23, 32, 34].

Biofilm formation on both abiotic (i.e. catheter) and biotic (i.e. cell surface) substrates is another contributing factor in determining virulence of *C. albicans*. More specifically, increased expression of the major heat shock protein (Hsp) Hsp90 plays a key role in regulating biofilm formation in addition to dispersion from the mature biofilm, another key virulence factor [35]. Within the biofilm, increased expression leading to the development of drug efflux pumps and metabolic plasticity allow the yeast to resist the effects of antifungals [32, 36]. Furthermore, β -glucans within the biofilm extracellular matrix itself also protect *C. albicans* from attack by neutrophils through the prevention of reactive oxygen species production, an innate host defense mechanism [32, 37].

Another feature of *C. albicans* is its ability to morphologically switch from yeast to hyphae upon contact with a surface, which is a characteristic shared by only *C. albicans*, *Malssezia*, and dermatophytes [38]. Hyphal growth can then be directional (thigmotropism) and invade gaps between cells or surfaces through contact sensing capabilities, allowing the organism to enter into host

tissue while also forming mature biofilms. This feature is essential for *C. albicans* virulence [23, 32].

Research has demonstrated that more virulent strains of *C. albicans* are capable of secreting aspartyl protease (SAP), [23, 32]. These hydrolases enhance **the organism's ability to penetrate host epithelial cells and extract nutrients** [32].

Lastly, the presence of gram-negative bacteria may also act as a co-pathogen with *C. albicans*, enhancing its pathogenicity as opposed to competing with the yeast [39]. This is especially true in EIB as *C. albicans* is thought to initiate disease, which is then maintained by the presence of gram-negative bacteria in a collaborative effort [22].

Management

Removal of contributing factors, antifungal therapy, or a combination of the two are effective in managing patients with EIB. In terms of contributing factors, preventing further occlusion of the affected site, drying of the hands, or avoidance of chemical or repetitive water exposure may be enough for resolution of the condition [1, 4, 6, 7, 12]. The use of topical antifungal agents directed at *Candida* such as imidazoles (clotrimazole, nystatin, or bifonazole) have been reported as successful treatments. If lesions fail to regress with topical therapy alone, oral fluconazole or itraconazole may also be used [1, 12, 15]. Applying filter paper soaked with Castellani paint (phenol, acetone, purified water, resorcinol, and alcohol) may also achieve therapeutic success [12]. If secondary infection exists, that should be treated as well. In patients with diabetes, blood glucose should be controlled; in patients with HIV or AIDS, CD4 cell count should be monitored and HAART therapy begun.

Complications and Prognosis

Prognosis for patients with EIB is generally favorable with treatment, but persistence of the lesion increases risk for secondary infection. Superficial and deep skin infections such as erysipelas and cellulitis

are of greatest concern when skin breaks exist (i.e. EIB) [40, 41]. Dupuy et al. demonstrated that disruption of the cutaneous barrier (fissured-toe web intertrigo, pressure ulcer, wound), in addition to lymphedema, was one of the main risk factors in the development of erysipelas of the leg [40].

Time for a Name Appraisal

The name, *erosio interdigitalis blastomycetica* has been upheld since it was given by Fabry in 1917. Though the name is partially descriptive of the condition, it is not all encompassing and is considered a misnomer. Since the discovery of the genus *Candida* in 1923, it only seems appropriate that this term should be acknowledged in the name. Thus, modernization of the name for the condition involving the moist interdigital area of the hands **seems appropriate. We suggest that "interdigital candidiasis (IC)" be used in place of EIB.**

Conclusion

Since 1915, few reports of this unique presentation of interdigital infection by *Candida* exist in the literature. This may relate to the unusual nature of the disease, but also because of underreporting. More detailed reports of the condition will enhance our current understanding of the disease process and the patient population primarily affected. For now, our understanding of EIB is that it primarily affects patients who frequently work with their hands in water, commonly related to an occupation. There is no exact data demonstrating a distinct presence of EIB in patients with HIV/AIDS and much of what we know about EIB is extracted from other conditions caused by *Candida*. Thus, the need for further reports regarding this entity is crucial to further characterize this condition. Additionally, we **propose that "interdigital candidiasis" replace EIB** moving forward as it is a more descriptive and appropriate name for the condition.

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