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Review

Current status of the etiology of candidiasis in Mexico

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ABSTRACT

This study presents a systematic review of the literature on the etiology of superficial and invasive candidiasis in Mexico reported from 2005 to 2015. The data have shown that *Candida albicans* is the most prevalent species with an increasing tendency of the non-*C. albicans Candida* species, as reported in other countries. The use of phenotypical methods in the identification of the yeasts limits the identification at the species level, particularly in species that are part of complexes, this is important because the identification only at the genus level leads to inadequate treatment due to the different susceptibility to the antifungals among species. In addition, this finding reveals the need to implement in clinical laboratories the molecular methods for the correct identification of the species involved, and the antifungal susceptibility tests to prevent the etiological changes associated with a poor therapeutic management.

Estado actual de la etiología de la candidiasis en México

Palabras clave:
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Etiología
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RESUMEN

Este artículo presenta una revisión sistemática de la bibliografía sobre la etiología de las candidiasis superficiales e invasivas en México en los años 2005-2015. Los datos muestran que *Candida albicans* se sitúa en primer lugar, pero hay una tendencia al aumento de otras especies del género, como se reporta en otros países. El uso de métodos fenotípicos para la identificación de las levaduras limita la identificación de la especie, especialmente de aquellas que forman parte de complejos; esto es importante, ya que la identificación solo del género puede conducir a un tratamiento inadecuado por la diferente sensibilidad de las especies a los antifúngicos. Por ello es necesario implementar en los laboratorios clínicos los métodos moleculares necesarios para la completa identificación de las especies implicadas y las pruebas de sensibilidad a los antifúngicos, para evitar cambios etiológicos asociados con un manejo terapéutico inadecuado.

Over the past 30 years, the incidence of opportunistic fungal infections has increased significantly and consistently worldwide. These mycoses are caused by filamentous fungi and yeasts, most notably *Aspergillus* and *Candida*, respectively, which are the most frequent causative agents. However, *Candida* is the main cause of mycosis in the world, especially in neutropenic patients, patients with malignancies treated with immunosuppressants, patients undergoing surgery or organ transplantation, preterm infants, and HIV/AIDS patients.[37,66] Of the different clinical forms of candidiasis, the invasive variety is a persistent public health problem. The incidence and mortality rates associated with this infectious disease have remained unchanged for more than a decade despite major advances in the field of antifungal therapy; this persistence leads to an increase in the costs of hospital care.

The genus *Candida* includes more than 200 species, of which *Candida albicans*, *Candida glabrata*, *Candida parapsilosis*, *Candida*

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tropicalis, *Candida krusei*, *Candida guilliermondii* [current name *Meyerozyma guilliermondii* (Wick.)],[43] *Candida lusitaniae*, *Candida dubliniensis*, *Candida pelliculosa*, *Candida kefyr*, *Candida lipolytica*, *Candida famata* [current name *Debaryomyces hansenii* (Zopf)],[46] *Candida inconspicua*, *Candida rugosa* and *Candida norvegensis* are recognized as human pathogens more frequently.[75] Some of these species are found as commensals in humans but can become pathogenic as a result of alterations in their microenvironment or compromise of the host immune system, promoting proliferation of the fungus and producing endogenous candidiasis; by contrast, exogenous infections are transmitted through the hands of medical personnel or contaminated devices.[36]

C. albicans has been considered the predominant species found in bloodstream infections; however, in the last two decades, there has been a growing trend of infections caused by other species of the genus, such as *C. glabrata*, *C. parapsilosis*, *C. tropicalis*, *C. krusei*, *C. dubliniensis*, *C. kefyr*, *C. famata*, *C. lusitaniae*, *Candida lambica*, *Candida zeylanoides*, *Candida africana*, and *C. guilliermondii*.[11,57,61,62] In addition, within the *C. parapsilosis* complex, *Candida metapsilosis* and *Candida orthopsilosis* have been identified as infection-causing agents of superficial candidiasis; in the *C. glabrata* complex, *Candida nivariensis* and *Candida bracariensis* have been found in genital candidiasis.[29,47] However, it has been reported that *C. albicans* is still the most frequently isolated species among the different forms of candidiasis.[61–64]

This increase in non-*C. albicans Candida* species is attributed to the indiscriminate use of antifungals and increased use of implanted medical devices, organ transplantation and broad-spectrum antibacterial therapy.[7,17,20] The treatment of the infections caused by these species may be difficult due to the resistance to some antifungals. *C. krusei* (intrinsically resistant) and *C. glabrata* (dose-dependent)[9] are resistant to fluconazole, one of the most recommended antifungals in the treatment of invasive candidiasis, followed by echinocandins, voriconazole and amphotericin B.[42,48,59]

The species *C. albicans* and *C. parapsilosis* are naturally susceptible to fluconazole; however, over time, the susceptibility of *C. albicans* to this antifungal agent has decreased, as observed in clinical isolates of *C. albicans*, subjected to prolonged treatment with azoles.[54,57,59] In addition, the susceptibility of *C. parapsilosis* and *C. glabrata* to fluconazole has changed discreetly and varies depending on the geographic region.[60,64,80] It is clear that no antifungal agent is exempt from the development of resistance, so it is essential that laboratories identify not only the genus, but the species of the clinical isolates of *Candida*, and consider performing *in vitro* susceptibility tests to guide the choice of treatment. The mortality attributable to invasive candidiasis remains high, mainly due to the delay in the administration of an appropriate antifungal therapy.[61] Therefore, this work presents the etiology of superficial and invasive candidiasis in Mexico, as well as an overview of the antifungal susceptibility of *Candida* species.

A systematic review of the medical literature published from 2005 to 2015 was carried out to know the prevalence of the etiology of superficial and invasive candidiasis in Mexico, as well as the susceptibility of the *Candida* species to antifungals. To achieve this objective, the selection of relevant articles based on their title and abstracts was carried out. The information was searched in the following databases: PubMed, Medline and Google Scholar (January 2005 to December 2015). The searches were performed using words present in articles, key words according to the database consulted; and synonyms extracted from the databases and publications available in both English and Spanish. The articles reviewed were those which included studies carried out in Mexico on superficial or invasive candidiasis with the *Candida* isolates identified at the species level by phenotypical or genotypical methods. Studies carried out in other countries; studies that reported the identification of *Candida* only at the genus level or that did not report the number of the isolates of *Candida* identified at the species level were not included.

The prevalence of the *Candida* species was estimated based on the total number of isolates. The prevalence of each type of candidiasis (oral, genital, cutaneous, onychomycosis and invasive) was estimated considering the number of the patients with each type of infection and the total population.

Etiology of superficial candidiasis

Twenty-one articles on superficial candidiasis were found published between 2005 and 2015, of which 18 were selected based on the inclusion criteria. Two thousand and ninety six patients out of 4103 patients with suspected superficial candidiasis suffered this mycosis, representing 51.1%. The data found in the literature showed that, in terms of superficial candidiasis, the most common clinical form is the genital one (47.81%),[32,47,72] followed by oral candidiasis (36.26%),[23,30,31,38,39,70,73,74] onychomycosis (14.65%)[1,27,49] and, finally, skin infections (1.29%)[13] (Table 1 and Fig. 1). These data correspond to the states of Ciudad de México, Puebla, Yucatán, San Luis Potosí, Nuevo León and Veracruz. The reports of genital candidiasis showed that *C. albicans* was the most common cause (71.36%), followed by *C. krusei* (13.77%), *C. tropicalis* (8.18%), *C. glabrata* complex (5.89%) and *C. parapsilosis* complex (0.8%)[4,14,32,47,70,77] (Table 1 and Fig. 2a). In 20 cases of balanitis and balanoposthitis, the most common etiologic agent was *C. albicans*, followed by *C. glabrata* complex. One case was reported as a mixed infection caused by *C. albicans* and *C. glabrata* complex.[4]

In oral candidiasis, the reports showed the effects on different populations,[39,73] including HIV and non-HIV children and adolescents,[31,74] patients with cancer,[70] diabetics,[23,30] malnourished children[31,38] and people of the Tarahumara community.[31] In this type of candidiasis, the most common etiologic agent was *C. albicans* (77.24%), followed by *C. glabrata* complex (13.16%), *C. tropicalis* (5.13%), *C. krusei* (3.29%), *C. parapsilosis* complex (0.79%), *C. kefyr* (0.26%), and *C. famata* (0.13%) (Table 1 and Fig. 2b).

In onychomycosis, the most common species in descending order were *C. albicans* (45.27%), *C. parapsilosis* complex (24.76%), *C. tropicalis* (7.17%), *C. guilliermondii* (6.84%), *C. glabrata* complex (5.21%), *C. famata* (3.58%), *C. lipolytica* (1.63%), *C. krusei* (1.63%), *C. zeylanoides* (0.98%), *C. dubliniensis* (0.65%), *C. lusitaniae* (0.65%),

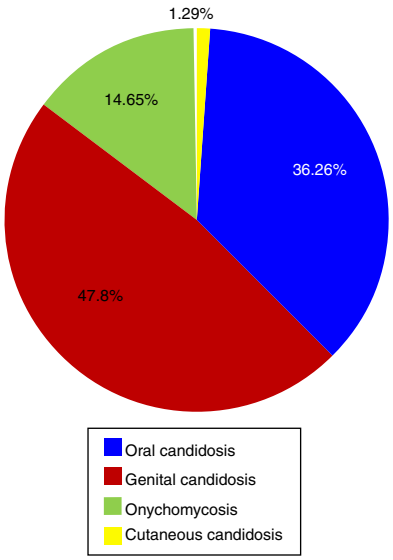

Fig. 1. Clinical manifestations of superficial candidiasis in patients from Mexico during the 2005–2015 period.

Table 1
Distribution of *Candida* species associated with superficial mycosis in Mexico during the 2005–2015 period.

Number and characteristics of the population studied	Number of patients with *Candida*	Frequency of the species identified (%)	Reference
Oral candidiasis			
111 patients with HIV/AIDS	72	*C. albicans* (90.27), *C. glabrata* complex (5.55), *C. tropicalis* (2.78), *C. parapsilosis* complex (1.39)	74
201 HIV-uninfected patients	112	*C. albicans* (63.39), *C. glabrata* complex (27.68), *C. tropicalis* (8.03), *C. parapsilosis* complex (0.89)	
67 children malnourished	21	*C. albicans* (100.0)	38
35 patients without symptoms of *Candida* infection	33	*C. albicans* (47.0), *C. tropicalis* (23.6), *C. krusei* (17.6), *C. glabrata* complex (11.8)	39
151 patients with different oncologic pathologies	21	*C. albicans* (100.0)	70
74 HIV-infected and HIV-uninfected patients	74 with 184 isolations	*C. albicans* (73.91), *C. glabrata* complex (19.02), *C. tropicalis* (5.98), *C. parapsilosis* complex (1.1)	73
60 children with HIV/AIDS and HAART therapy	36	*C. albicans* (83.3), *C. krusei* (5.56), *C. tropicalis* (11.1)	
55 children with malnutrition	26	*C. albicans* (61.53), *C. krusei* (30.77), *C. tropicalis* (3.85), *C. glabrata* complex (3.85)	31
57 Tarahumara patients	10	*C. albicans* (80.0), *C. krusei* (20.0)	
56 patients with DM	37	*C. albicans* (59.46), *C. glabrata* complex (18.92), *C. tropicalis* (16.22), *C. kefyr* (2.7), *C. famata*[a] (2.7)	23
80 individuals without DM and chronic dialysis	34	*C. albicans* (64.7), *C. glabrata* complex (17.65), *C. tropicalis* (8.82), *C. kefyr* (2.94), *C. parapsilosis* complex (5.88)	
141 patients with type 2 DM	56	*C. albicans* (85.71), *C. glabrata* complex (8.93), *C. krusei* (5.36)	30
Genital candidiasis			
468 gynecological patients	325	*C. albicans* (67.7), *C. tropicalis* (16.0), *C. glabrata* complex (12.6), *C. krusei* (2.5), *C. parapsilosis* complex (1.2)	32
818 gynecological patients	230	*C. albicans* (100.0)	72
300 gynecological patients	300	*C. albicans* (53.0), *C. krusei* (41.0), *C. tropicalis* (6.0)	47
110 gynecological patients	110	*C. albicans* (71.3), *C. glabrata* complex (11.7), *C. tropicalis* (8.5), *C. krusei* (5.3), *C. parapsilosis* complex (3.1)	14
150 gynecological patients	21	*C. albicans* (42.8), *C. krusei* (23.8), *C. glabrata* complex (19.0), *C. tropicalis* (14.3)	77
20 patients with balanitis or balanoposthitis	20	*C. albicans* (95.0), *C. glabrata* complex (5.0)	4
Onychomycosis			
936 patients with suggestive signs of onychomycosis	152	*C. parapsilosis* complex (34.86), *C. albicans* (24.34), *C. guilliermondii*[b] (13.81), *C. famata*[a] (7.23), *C. tropicalis* (4.60), *C. lipolytica* (3.28), *C. glabrata* complex (2.63), *C. zeylanoides* (1.97), *C. dubliniensis* (1.32), *C. lusitaniae* (1.32), *C. krusei* (1.32), *C. kefyr* (1.32), *C. lambica* (1.32), *C. intermedia* (0.66)	49
185 patients with candidal onychomycosis	153	*C. albicans* (66.3), *C. parapsilosis* complex (14.3), *C. tropicalis* (9.1), *C. glabrata* complex (8.0), *C. krusei* (2.0)	1
One patient with hand onychomycosis	1	*C. albicans* (33.3), *C. parapsilosis* complex (33.3), *C. tropicalis* (33.3)	27
Cutaneous candidiasis			
27 patients with diaper dermatitis	27	*C. albicans* (88.89), *C. parapsilosis* complex (7.41), *C. glabrata* complex (3.70)	13

AIDS, Acquired Immunodeficiency Syndrome; DM, diabetes mellitus; HAART, Highly Active Antiretroviral Therapy; HIV, Human Immunodeficiency Virus.
[a] Current name: *Debaryomyces hansenii* (Zopf).[48]
[b] Current name: *Meyerozyma guilliermondii* (Wick.).[45]

C. kefyr (0.65%), *C. lambica* (0.65%) and *C. intermedia* (0.33%) (Table 1 and Fig. 2c). Also, a case of a mixed infection caused by *C. albicans*, *C. parapsilosis* complex, and *C. tropicalis* was reported.[27] Onychomycosis was mainly reported in the fingernails of adult women and was associated with diabetes, injuries, circulatory disorders, the use of corticosteroids and the use of fake nails,[1,27,49] in agreement with the data reported by other countries.[78] *C. parapsilosis* complex predominated in toenails, while the most common fungus in fingernails was *C. albicans*.[49]

Regarding cutaneous candidiasis, only diaper rash reports were found during the period 2005–2015, where *C. albicans* was the most frequent species (88.89%), followed by *C. parapsilosis* complex (7.41%) and *C. glabrata* complex (3.70%)[13] (Table 1 and Fig. 2d).

In superficial candidiasis, the *Candida* isolates were identified by conventional methods: culture,[1,4,13,14,23,32,72,74] KOH (10–20%) test,[1,4,13,14,27,49] germ tube,[1,39,70,73,74] API 20C[1,13,14,23,32,74] and ID32[49,73] systems, CHROMagar[4,13,27,30,31,38,39,47,49,70,73,77] chlamydoconidia formation,[1,14,49,73] and growth at 45 °C.[49]

Susceptibility to antifungal agents in superficial candidiasis

There are few studies on superficial candidiasis where antifungal susceptibility data are reported. The determination of the susceptibility to antifungals was performed using the reference procedures of broth microdilution (M27-A2 of the CLSI)[32,49] and disc diffusion method (M44-A),[73] as well as commercial systems such as Sensi-Disk,[32] and Fungitest®,[74] which produce results that, in general, correlate with the reference procedures.[22] Based on these data, in general, oral candidiasis species showed <11% resistance to azoles.[73,74] In vaginal candidiasis, *C. glabrata* showed >50% resistance to fluconazole.[32] In onychomycosis, low resistance to azoles was reported in *C. albicans* and *C. parapsilosis* complex, high resistance to itraconazole was reported in *C. guilliermondii* and *C. famata*, and resistance was also reported to ketoconazole, itraconazole and fluconazole in four *C. glabrata* complex isolates.[49] In cutaneous candidiasis, susceptibility to 2% sertaconazole was reported in *C. albicans*, *C. parapsilosis* complex and *C. glabrata* complex.[13]

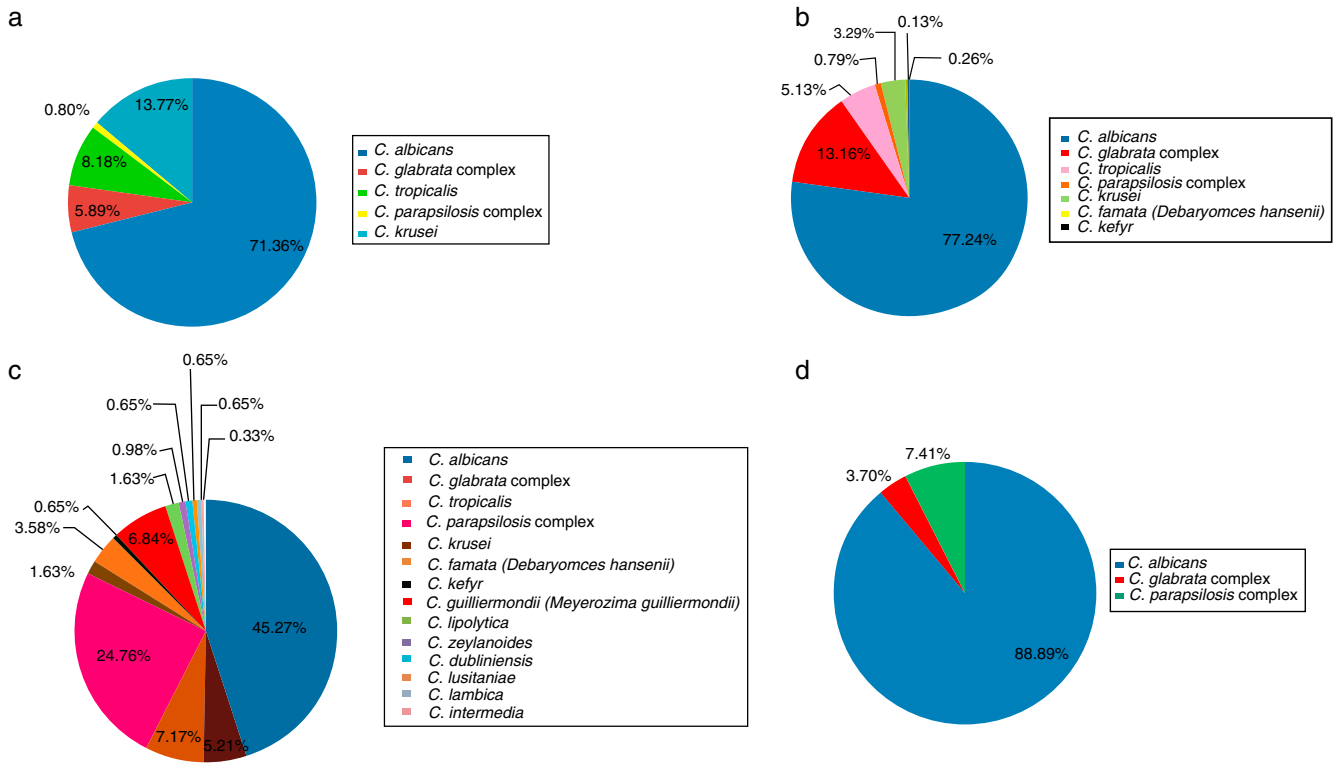

Fig. 2. Frequency distribution of *Candida* species causing superficial candidiasis in Mexico during the 2005–2015 period. (a) Oral candidiasis. (b) Genital candidiasis. (c) Onychomycosis. (d) Cutaneous candidiasis.

Table 2
Distribution of *Candida* species associated with invasive infection in Mexico during 2005–2015 period.

Number and characteristics of the population studied	Number of patients with *Candida*	Frequency of the species identified (%)	Reference
1221 neonates with candidiasis	25	*C. albicans* (56.0), *C. parapsilosis* complex (32.0), *C. glabrata* complex (8.0), *C. guilliermondii*[b] (4.0)	66
567 patients hospitalized (148 patients with sepsis)	18	*C. albicans* (55.55), *C. tropicalis* (11.11), *C. glabrata* complex (33.33)	18
107 pediatric patients with fever and neutropenia	31	*C. albicans* (61.29), *C. glabrata* complex (19.35), *C. tropicalis* (12.90), *C. krusei* (3.22), *C. parapsilosis* complex (3.22)	5
398 patients with nosocomial bloodstream infection	398	*C. parapsilosis* complex (37.69), *C. albicans* (31.9), *C. tropicalis* (14.82), *C. glabrata* complex (8.04), *C. krusei* (2.8), *C. guilliermondii*[b] (1.26), *C. zeylanoides* (0.5), *C. utilis* (0.5), *C. famata*[a] (1.0), *C. rugosa* (0.5), *C. lusitaniae* (0.5), *C. boidinii* (0.3)	34
Three neonates with nosocomial candidiasis	3	*C. parapsilosis* complex (100)	37
52 organ transplant patients	52	*C. albicans* (100)	67
104 patients with fever of unknown origin and immnunosupression	7	*C. albicans* (71.42), *C. tropicalis* (14.3), *C. guilliermondii*[b] (14.3)	52
326 pediatric patients with candidemia	358 (some patients had more than one yeast isolate)	*C. albicans* (38.95), *C. tropicalis* (22.08), *C. parapsilosis* complex (38.95) of which *C. parapsilosis sensu stricto* (95.3), *C. orthopsilosis* (3.1), *C. metapsilosis* (1.6)	35
15 patients with candidemia	12	*C. parapsilosis* complex (75.0), *C. glabrata* complex (16.67), *C. albicans* (8.33)	56
24 patients with blood infection	24	*C. albicans* (45.83), *C. tropicalis* (25.0), *C. glabrata* complex (12.5), *C. parapsilosis* complex (4.16), *C. krusei* (4.16), *C. guilliermondii*[b] (4.16), *C. lipolytica* (4.16)	21
18 patients with candidemia	11	*C. albicans* (45.45), *C. tropicalis* (27.27), *C. glabrata* complex (18.18), *C. famata*[a] (9.1)	24
One patient with community-acquired pneumonia	1	*C. lusitaniae* (100)	50
30 patients with candidemia	9	*C. albicans* (77.78), *C. parapsilosis* complex (22.22)	51
116 patients with candidiasis	86	*C. albicans* (71.59), *C. parapsilosis* complex (17.04), *C. tropicalis* (11.36)	33
1225 patients with candidiasis	1171	*C. albicans* (39.0), *C. glabrata* complex (21.0), *C. tropicalis* (18.0), *C. krusei* (11.0), *C. parapsilosis* complex (7.0), *C. dubliniensis* (2.0), *C. guilliermondii*[b] (1.0), *C. zeylanoides* (1.0)	69

[a]Current name: *Debaryomyces hansenii* (Zopf).[48]
[b] Current name: *Meyerozyma guilliermondii* (Wick.).[45]

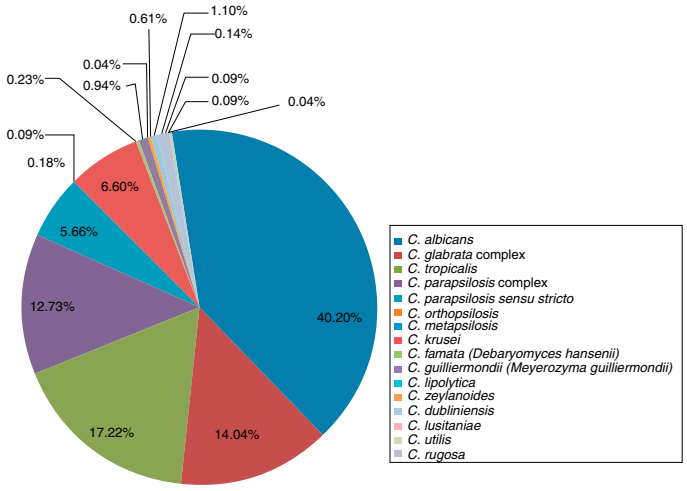

Fig. 3. Frequency distribution of *Candida* species causing invasive candidiasis in Mexico during the 2005–2015 period.

Etiology of invasive candidiasis

Regarding invasive candidiasis, 15 publications reported 2184 cases (Table 2). The affected populations included neonates[56,66] and pediatric patients,[35,51] transplant patients,[52,67] patients with fever of unknown origin,[24,51,52] patients with neutropenia,[5] patients with a bloodstream infection,[21,24,34,35,37,51,56] patients in intensive care units[18,24,37,51,56,66] and patients hospitalized in hematology unit,[24,52] infectious unit, internal medicine,[24,51,52,56] rheumatology,[52] emergency,[56] neurosurgery,[24] neurology,[24] pulmonology units,[24] and other patients hospitalized in non-specified services.[21,33,34,50,69] The reports found were from Nuevo León, Guadalajara, Durango, Morelia, Sonora, Sinaloa, Coahuila, Estado de México, Ciudad de México, and Tamaulipas.

The main species associated with invasive candidiasis were *C. albicans* (40.2%), *C. tropicalis* (17.22%), *C. glabrata* complex (14.04%), *C. parapsilosis* complex (12.73%), *C. krusei* (6.6%), *C. parapsilosis sensu stricto* (5.66%), *C. guilliermondii* (0.94%), *C. zeylanoides* (0.61%), *C. famata* (0.23%), *C. orthopsilosis* (0.18%), *C. lusitaniae* (0.14%), *Candida utilis* (0.09%), *C. metapsilosis* (0.09%), *C. rugosa* (0.09%), *C. lipolytica* (0.04%), and *Candida boidinii* (0.04%)[5,18,21,24,33–35,37,50–52,56,66,67,69] (Table 2 and Fig. 3).

In invasive candidiasis, *Candida* isolates were identified by conventional and molecular methods: culture,[5,18,21,24,34,37,50,51,56,66,67,69] KOH (10–20%) test,[52,69] germ tube production,[5,21,35,69] API 20C,[5,34,35,52,69] chlamydoconidia formation,[33,35,52,69] CHROMagar,[33,50,51,56,69] MicroScan[33,37,51] and Vitek YBC[21] automated systems, PCR,[5,35] and RFLP.[50]

Susceptibility to antifungal agents in invasive candidiasis

Of the 15 studies found in the literature on invasive candidiasis, only six[21,24,34,35,50,56] included reports on antifungal susceptibility tests showing resistance to itraconazole in *C. tropicalis*, and to amphotericin B, fluconazole and itraconazole in *C. glabrata* complex.[24,34,35,50] Susceptibility tests to antifungals were performed using the broth microdilution (M27-A2[34] and M27-A3[21,35,56] of the CLSI) reference procedures and the Fungitest® commercial test.[50]

Discussion

Candida is the main causative agent of mycosis in humans, but the epidemiology of candidiasis varies according to the geographical region.[8,16,81] For this reason, it is important to conduct studies to determine the incidence of mycosis, the distribution of causative species, and their antifungal susceptibility profiles.

In Mexico, according to the morbidity yearbook published by the General Epidemiology Directorate (Dirección General de Epidemiología – http://www.epidemiologia.salud.gob.mx/anuario/html/morbilidad_nacional.html), urogenital candidiasis has remained among the 20 leading causes of illness nationwide, ranked between the 11th and 15th places. This is the only mycosis registered in the yearbook from 1988 to date. However, these data do not agree with the findings of the literature review, in which besides genital candidiasis other clinical forms of superficial candidiasis are also frequent. This discrepancy is due to the fact that fungal infections are not notifiable diseases. The results of this review also show that, between 2005 and 2015, the number of reports of superficial candidiasis was higher (18 publications) than that of invasive candidiasis (15 publications). The size of the population with suspected superficial candidiasis was 4103, with a positive frequency of 51.1%. In invasive candidiasis the suspected population suffering it was 4387 patients with a positivity of 49.8%. These data reveal that, in Mexico, the prevalence of superficial candidiasis is slightly greater than that of the invasive one, probably because factors predisposing to the superficial form (humidity, inadequate hygiene, pediatric age, pregnancy, hypothyroidism and type 2 diabetes mellitus) are more frequent than those predisposing to the invasive form.[3] It is important to mention that, in Mexico, type 2 diabetes is one of the most prevalent chronic diseases (http://www.epidemiologia.salud.gob.mx/anuario/html/morbilidad_nacional.html), which may be one of the most important predisposing factors to acquire superficial candidiasis compared with predisposing factors such as neutropenia, central parenteral nutrition, central venous catheter use, abdominal surgery, broad-spectrum antimicrobial use and severe immunocompromise.[79] This may also be due to, as in other countries, the incidence of invasive candidiasis being stable or decreased at certain institutions[58] or to a greater interest in invasive candidiasis because of the more frequent mortality.

Regarding the etiology of superficial candidiasis, especially the oral form, there is agreement within the international scientific literature, which reports *C. albicans* as the predominant species, followed in a different order, depending on the clinical form, by *C. krusei*, *C. tropicalis*, and *C. parapsilosis* complex. In contrast, other species such as *C. kefyr* and *C. famata* are rare.[65] Among the patients with oral candidiasis, there is a greater prevalence of *C. albicans* in those with HIV/AIDS than in those without HIV/AIDS.[31,74]

In vaginal candidiasis, the diversity of species reported in Mexico was low as only *C. albicans*, *C. tropicalis*, *C. krusei*, *C. glabrata* complex and *C. parapsilosis* complex have been reported; however, in other countries, and during the same period, *C. dubliniensis*, *C. famata*, *C. lusitaniae*, *C. africana*, *C. guilliermondii*, *C. lambica*, *C. kefyr*, *C. zeylanoides*, *C. nivariensis* and *C. bracariensis* were reported.[44,76] In balanitis and balanoposthitis, *C. albicans* and *C. glabrata* complex were found as causative agents, but these data cannot be compared because there are no studies on the distribution of *Candida* species in these mycoses. They are considered sexually transmitted diseases; therefore, it is thought that their distribution must be similar to that of vaginal candidiasis.[2]

For onychomycosis, we found that *C. parapsilosis* complex is the most frequently isolated yeast, and *C. albicans* is ranked third. The diversity of species isolated in Mexico is higher; in other countries, up to four species have been reported[40] and in Mexico, up to 14 species have been isolated.[48] This may be due to the geographical variation in the distribution of species.[78] In cutaneous candidiasis (diaper rash), we also noted that *C. albicans* is the most prevalent species,[13] as in other parts of the world.[55]

The etiological diversity of invasive candidiasis in Mexico coincide with that in reports from other countries, where *C. albicans*, *C. glabrata* complex, *C. krusei*, *C. tropicalis*, and *C. parapsilosis* complex cause more than 90% of cases; the isolation of *C. guilliermondii*, *C. lipolytica*, *C. famata*, *C. zeylanoides*, *C. utilis*, *C. rugosa*, and *C. boidinii* is rare.[7,16]

Thus, a review of the studies conducted in Mexico shows that the etiology of candidiasis in our environment is similar to that of other countries, where *C. albicans* remains the most isolated species, both in surface and invasive infections, but other species of the genus are becoming more frequent. The phenotypic methods used in the identification of *Candida* species were direct examination to observe the micromorphology, filamentation on serum to discriminate *C. albicans* from other species, culture in CHROMagar Candida to visualize the use of chromogenic substrates characteristic of *C. albicans*, *C. tropicalis* and *C. krusei*, production and clustering of chlamydoconidia to differentiate *C. albicans* from *C. dubliniensis*, and biochemical tests using the VITEK 2, API 20C, or API 32C systems. In most cases, these methods do not allow to achieve the correct identification of the species, especially in the case of infrequently isolated yeasts and those forming species complexes. For example, with filamentation on serum, other species, in addition to *C. albicans*, can also produce germ tubes.[41] In the case of CHROMagar Candida, which is one of the most widely used methods in clinical laboratories, there have been reports indicating that the color intensity of the colonies is not restrictive for *C. albicans* and *C. dubliniensis* species. In addition, it has been observed that the color of the colonies is gradually lost with subculturing or storage of the strains,[28] leading to misidentifications. However, it has been demonstrated that systems designed to identify multiple species (VITEK 2, API 20C and API 32C) are less accurate for the classification of unusual species, as reported by Desnos-Ollivier et al.[26] These investigators found that several isolates identified by API 32C as *C. famata* corresponded to *C. guilliermondii*, *Candida haemulonii*, *C. lusitaniae* and *Candida palmioleophila*, according to sequence analysis of gene segments. *C. parapsilosis* was also erroneously identified as *C. famata* with this method.[15] Another disadvantage of these identification systems is that they fail to discriminate species with similar biochemical profiles that are genetically distinct, as in the case of the species complexes *C. glabrata sensu stricto*, *C. nivariensis* and *C. bracariensis*; *C. parapsilosis sensu stricto*, *C. metapsilosis* and *C.orthopsilosis*; and *C. guilliermondii sensu stricto*, *Candida fermentati*, *Candida carpophila* and *Candida xestobii*, which can only be differentiated by molecular methods.[29,44] Due to the clinical importance of identifying the species of *Candida* involved, several researchers have proposed the use of molecular techniques. These include RAPD (Random Amplified Polymorphic DNA),[80] PCR-RFLP (Restriction Fragment Length Polymorphism)[53] and AFLP (Amplified Fragment Length Polymorphism),[6] PCR amplification of segments of specific genes, such as MP,[63] which encodes a mannoprotein of 65 kDa associated with morphogenesis and pathogenicity in *Candida*,[12] pulsed-field gel electrophoresis (PFGE),[45] sequencing of ITS (internal transcribed spacer) regions[25] and microsatellites[71] and multilocus sequence typing (MLST).[68] These techniques have been widely used in phylogenetic, taxonomic, and population structure studies and are particularly useful for the delimitation of *Candida* species.[19] However, the sequencing of ITS rDNA regions is the most widely used marker for the rapid, accurate identification of *Candida* species.[25] The importance of discriminating the species within a complex is that they differ in their susceptibility to antifungal agents, virulence and biofilm formation, so it is important for clinical laboratories to conduct antifungal susceptibility tests to administer the proper treatment. However, this review found that only 33.3% of the selected publications show antifungal susceptibility results,[13,21,24,32,34,35,49,50,56,73,74] which is a reflection that these tests are not routinely performed. This situation represents an additional problem to the lack of specific identification methods because antifungal resistance may depend on the *Candida* species; thus, it is essential to perform this identification for the proper management of the patients. However, in the few reports that were found, we observed that *C. glabrata* complex showed higher resistance to azoles and amphotericin B in cases of both superficial and invasive mycosis.[32,34,49,56,74] This information is relevant because *C.glabrata* complex is one of the most common species in all forms of candidiasis. In addition, because discrimination between the members of *C. glabrata* complex is not reported, it is likely that a portion of the resistant isolates correspond to *C. nivariensis*.[10]

It is worth noting that the available epidemiological data in Mexico is limited, and the publications found corresponding to the 2005–2015 period only reflect the situation in the larger states of the country. However, these data facilitate predictions of what may be occurring in other states.

Conclusions

The etiological behavior of candidiasis in Mexico is similar to that in other countries. However, it is important that both molecular tests for the accurate identification of yeasts and antifungal susceptibility tests are implemented and used routinely in healthcare facilities to ensure a specific diagnosis and the selection of appropriate therapeutic strategies. In addition, this strategy may help to prevent changes in the etiology of candidiasis as a result of the improper use of antifungals.

Authors' contributions

MGFDL, MRRM, EDE, EMH, and GAA performed literature research and wrote the paper. All authors read and approved the final version.

Conflicts of interest

The authors declare that they have no competing interests.

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