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Identification of *FKS 1* and *FKS 2* Gene Encoding Antifungal Resistance in Clinical Isolates of Candida Auris

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KEYWORDS

Antifungal Resistance, Clinical Isolates

ABSTRACT:

Background:

Candida auris was first isolated in Japan in 2009 from the external auditory canal of a patient. It has shown strong resistance to azole antifungal drugs and has caused severe nosocomial outbreaks and complicates the treatment of infectious diseases.

Material and Methods

:12 isolates of C.auris were identified from various clinical samples using morphological features on culture medium. Further identification and antifungal susceptibility testing was done using VITEK 2 compact system. Susceptibility was determined using an AST-YS09card which tests MIC of 6 antifungals, FLU, 5-FC, VRC, AMB, CAS and MFG. Isolates showing multi drug resistance were subjected to conventional PCR for the identification of FKS 1 and FKS 2 gene which is responsible for its multidrug resistance feature.

Result:

Of the 12 Candida auris isolates 8(67%) isolates showed multidrug resistance.6(75%)isolates were found to contain FKS1 gene and 5(41%) isolates contained FKS 2 gene that contributes in its antifungal resistance feature.

Conclusion:

The emergence of multidrug-resistant C. auris is a multifactorial phenomenon that raises many serious public health concerns. Our study documented 67% of resistance against C.auriswhich emphasizesthe rapid & accurate identification of C.auris for controlling its outbreaksand better treatment. The findings of this study also underline the importance of better routine diagnostic workflows for fungal infections particularly in regions where C. auris prevalence is increasing. A thorough understanding of the molecular insights into this organism is crucial for improved management which includes conductingC. auris surveillance and antifungal therapy in high-risk patients.

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Introduction: Over the last few years the incidence of mycotic infections has progressively increased. Fungi once considered as nonpathogenic or less virulent are now recognized as a primary cause of morbidity and mortality in immunocompromised and severely ill patients [1]. A wide range of clinical manifestations including bloodstream infections (BSIs), intra-abdominal candidiasis, deep-seated candidiasis and superficial infections are caused by*Candida spp.*[2]. Although *Candida albicans* is the most prevalent species, an epidemiological shift in *Candida* pathogens has been recently noted by the increasing number of infections caused by non-albicans Candida species (NAC) [3] [4]. *Candida auris* is a newly emerged member of the *Candida/Clavispora* clade first isolated in Japan in 2009 from the ear discharge of a female patient [5].*Candida auris*, an emerging multidrug-resistant human fungal pathogen that colonizes the skin and has been classified as an urgent threat by the US Centers for Disease Control and Prevention (CDC) Antibiotic Threats Report (2019) and ranked in the critical priority group by the World Health Organization (WHO) in a recently released list of fungal priority pathogens. [6,7]

It is a multidrug-resistant pathogenic yeast that causes invasive infections and outbreaks with high mortality rates in hospitalized patientsparticularly among patients with multiple comorbidities and who have been admitted to ICU or other special care facilities [8–11]. Due to its multidrug-resistant nature and extraordinary ability to spread rapidly in healthcare facilities causing outbreaks with associated high mortality rates [12-24]

Since *C. auris* isolates are usually misidentified as *Candida haemulonii, Candida duobushaemulonii,* or other *Candida* species and it has become important to efficiently & correctly identify *C. auris* in the capacity of hospital microbiology laboratory following the detection of positive cases, infection control measures which include alerting treating infectious disease specialists and notification to institutional authorities for setting up outbreak management teams should be done.

Aim: To identify and detect *FKS 1* and *FKS 2* gene in *Candida auris* isolates which might be responsible for its multidrug resistant feature.

Materials and Methods:

A study was conducted in the Department of Microbiology, Santosh Medical College and Hospital, Ghaziabad and atotal of 24 *Candida auris*isolates were isolated from the various clinical samples received in the lab whichwere identified by conventional methods. All the samples collected were cultured on SDA agar and incubated at 37 °C for 24 h. Further identification and antifungal susceptibility testing was done using VITEK 2 compact system. Susceptibility was determined using an AST-YS09card which tests the MIC of 6 antifungals, FLU, 5-FC, VRC, AMB, CAS, and MFG. Isolates showing multi drug resistance were subjected to conventional PCR & Gene detection of ITS region for confirmation of *Candida auris* and then detection of FKS1 and FKS 2 gene for evaluation of its multidrug resistance.

DNA Extraction

DNA extraction was carried out by using Silica membrane technology following the standard protocol using MN Bead Tube Type C. NucleoSpin® Microbial DNA, MACHEREY-NAGEL kit as per the manufacturer‘s instructions was used. The purity and quantification of the extracted DNA was evaluated by a NanoDrop-2000 spectrophotometer (Thermo Fisher Scientific, Wilmington, DE, USA) and the quality of the extracted DNA was estimated from the ratio of absorbance at 260 and 280 nm. An A260/A280 purity ratio of 1.8 was considered for the PCR assay.

PCR Amplification of the ITS 1 Region

All the identified isolates were subjected to PCR with designed primers (Table 1,2& 3). For the amplification of the target region of the DNA strand PCR was carried out in total 20µL reaction volume containing DNA template 1.0 µL, 1.0µL of each forward and reverse primer, master mix 10 µL and nuclease free water 7.0 µL.

Table 1: OligonucleotidePrimers Used to sequence *ITS 1* gene

Gene	Primer	Sequence	Annealing Temp.
ITS	*ITS 1*	5'-TCCGTAGGTGAACCTGCGG-	60^0C

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1	*(F)*	3'
ITS 1	*ITS 4 (R)*	5'-TCCTCCGCTTATTGATATG-3'

Table 2: OligonucleotidePrimers Used to sequence *FKS 1*gene

Gene	Primer	Sequence	Annealing Temp.
FKS 1	*FKS 1 (F)*	5'-GAATCAACAACAGGGTGGGC-3'	60^0 C
FKS 1	*FKS 1 (R)*	5'-GAAGCACCTGAGGATCTGG-3'	

Table 3: OligonucleotidePrimers Used to sequence *FKS 2* gene

Gene	Primer	Sequence	Annealing Temp.
FKS 2	*FKS 2 (F)*	5'-CCAAATGGAAGGGTTGCACTTG-3'	60^0 C
FKS 2	*FKS 2 (R)*	5'-CCAGATAGATGCTCCAAGGTAG-3'	

Amplification was done using a BIO-RAD (T100) thermal cycler consisting of the following program: initial denaturation step at 94°C for 5mins, followed by 34 cycles of denaturation step at 94°C for 30 sec, annealing at 60°C for 30secand 72°C for 30 secand then final extension step at 72°C for 5mins. Amplified products were analysed by 1% agarose gel electrophoresis containing ethidium bromide (0.5 mg/mL) and visualized on a UV transilluminator. (10,17)

Results :

Of the 242 isolates of different *Candida species* collected 78 (32%)isolates were *Candida albicans* and 164 (68%)isolates were *Non Albicans Candida spp*. (Fig.1)

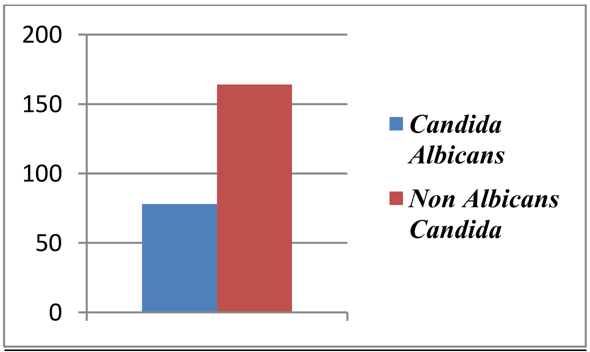

Fig.1 Distribution of *Candida* Species on the Basis of *Candida albicans* and *Non Albicans Candida*

Among the 242 different *Candida* species isolated from the clinical samples, the highest number of isolates were*Candida albicans* 78 (32%), followed by *C.tropicalis*46 (19%), *C.haemulonii*38 (15.7%), *C.krusei*30 (12.4%), *C.glabrata*20 (8.3%), *C.parapsilosis*18 (7.4%) and *C.auris*12 (5%) respectively.

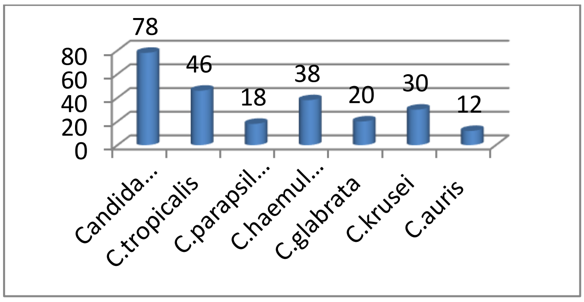

Fig.2 **Distribution of Different Isolates of *Candida* Species**

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Highest number of *Candida auris*islolates were obtained from blood(50%) followed by whichswabs (25%), sputum(16%), urine (8.3%) and pus(8.3%), respectively.

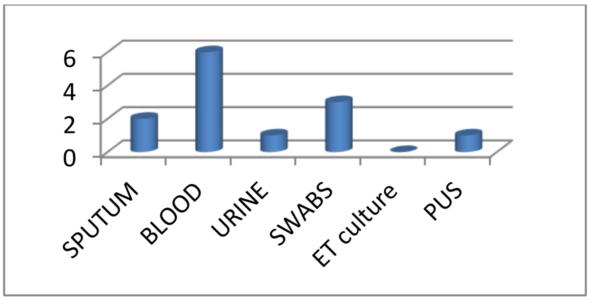

Fig.3 Distribution of *Candida auris* Isolates From Different Clinical Sample

Of the 12 *Candida auris* isolates 8 (67%) isolates showed multidrug resistance along with significant resistance against Fluconazole and Voriconazole (Table 4 & Fig. 5)

Organism	Antifungals					
Candida Auris	Fluconazole	Voriconazole	Caspofungin	Micafungin	Amphotericin B	Flucytosine
Sensitive	0	0	1	2	2	0
Resistant	12	12	11	10	10	12

Table 4. Antifungal Susceptibility Pattern of *Candida auris* Isolates

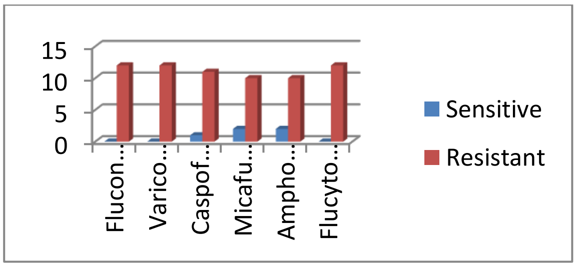

Fig.4 Antifungal Susceptibility Pattern of *Candida auris*Isolates

12 Candida auris isolates were subjected to conventional PCR for detection of ITS 1 gene for its confirmation (Fig.2)using ITS 1 gene-specific primers in PCR (Table1). An approximatelengthof 162 bp was obtained in 9(75%) out of 12 isolates of *Candida auris*isolates. The corresponding amplicon images are depicted below in Fig.5

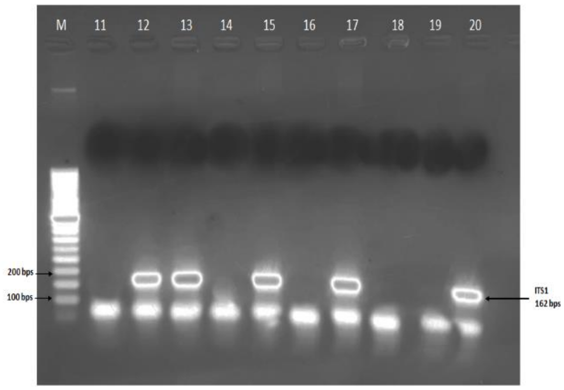

Fig.5 Gel Electrophoresis of DNA Fragments generated by PCR Amplification showing DNAladder;L-12, L-13, L-15, L-17, L-20 ITS 1 Positive Result of the Test Isolates and L-11, L-14, L-16, L-18, L-19 Negative Results with no ITS 1 gene expression

8(67%)multidrug resistant isolates of *Candida auris* were then subjected to PCR for detection of *FKS1* using primers specific for *FKS 1* gene (Table 2) andan approximate length of 320 bp was obtained in 6 (75%) out of 8 isolates of candida auris isolates(Fig 6) .

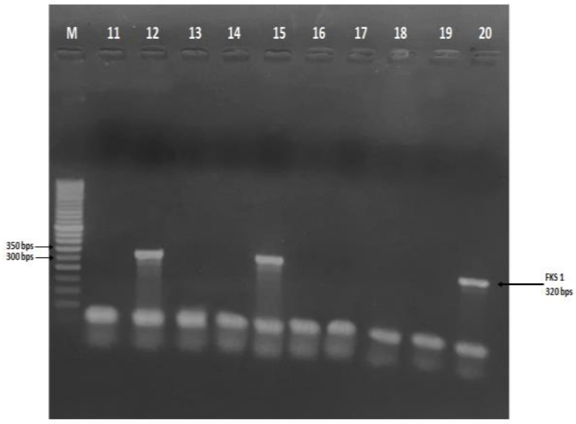

Fig.6Gel Electrophoresis of DNA Fragments generated by PCR Amplification showing DNAladder;L-12, L-15, L-20 FKS1 Positive Result of the test isolates,L-15, L-

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13, L-14, L-16, L-17, L-18, L-19 Negative Results with no FKS1 gene expression

*FKS 2*gene detection was carried out through PCR using specific *FKS 2* gene primers (Table 3). Of the8 multidrug resistant isolates of *Candida auris*5(41%) isolates contains *FKS 2* gene (Fig.7) which could be responsible for its antifungal resistance feature

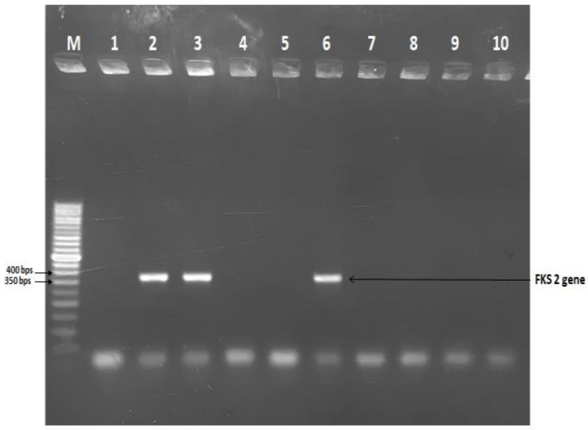

Fig.7 Gel Electrophoresis of DNA Fragments Generated by PCR Amplification showing DNAladder;L-2, L-3, L-6 FKS 2 *positive result* of the test isolates,L-1, L-4, L-5, L-7, L-8, L-9, L-10 Negative Results with no FKS 2 gene expression

Discussion:

Of the total 242 isolates of different *Candida* species the predominance of *Non albicans candida* was reported more in comparison to *Candida albicans* that corresponds with the study done by Bhattacharjee P et al. The present study focuses on the multidrug resistance pattern of *C. auris*isolates and the results of antifungal susceptibility are similar to a study done by V Mirabet et al as the majority of clinical *C. auris* isolates display resistance to main classes of antifungals (azoles or echinocandins) and there is pan-drug resistance to more than two antifungal classes [13].

The present study is also based on its molecular identification and detection of gene which are reported as one of the factor responsible for its resistance. Genetic studies of Candida auris have shown expansions of genes linked to drug resistance and multidrug efflux.[26] FKS 1 gene is targeted to study its resistance against echinocandins. In the present study 75% of isolates were found to contain FKS 1 gene. Echinocandins inhibit the activity of 1,3-β-D-glucan synthase, an enzyme encoded by the FKS1 and FKS2genes, which is important for synthesis of a primary fungal cell wall polymerTheechinocandin resistance in another study consists of C. auris collection characterized by 3.8%prevalence and the mechanism of FKS1 HS1 mutations [34]. Resistance to azoles and echinocandins is mediated by inhibiting the activity of lanosterol 14-α-demethylase enzyme; prevent converting lanosterol to ergosterol leading to damaging integrity of cell membrane and drug target 1,3-beta-glucan synthase (FSK1) respectively[27].Echinocandin resistance was linked to a novel mutation S639F in FKS1 hot spot region I [29].

The development of echinocandin resistance has been documented in *C. auris* isolates recovered from patients treated with these ... multiple geographic areas. This is partic FKS 2 380bps vorrisome, given the recommendation f ... of echinocandins as an empirical treatmer ... *uris* infection prior to the availability of specific susceptibility testing results. The development of echinocandin resistance has been documented in C. aurisisolates recovered from patients treated with these drugs in multiple geographic areas.FKS 1 gene is detected in the present study as another study reported *C. auris* echinocandin resistance is directly related to mutations in *FKS*1 (40).

In the present study 62% of isolates were found to contain FKS 2 gene. Hot-spot mutation in FKS1 gene associated with S639Y, S639P and S639Y regions and FKS2. Azole resistance has been demonstrated in association with amino acid substitutions in ERG11. The primary factor contributing to its high mortality rates is its ability to develop resistance to multiple antifungal agents [38]. Detection of likely echinocandin resistance than for azole resistance, as few isolates with azole resistance had detectable mutations in the ERG11 gene, reflecting the multi faceted nature of the mechanisms of azole resistance [28]. FKS1 genes among C. auris isolates clearly confer drug resistant characteristics of the isolates as reported in other studies as well. Hot-spot mutation in FKS1 gene associated with S639Y, S639P and S639Y regions and FKS2 [29].

Conclusion:*Candida auris* is an emerging pathogenic yeast of increasing global concern. In just over a decade since its discovery, *Candida auris*emerged as a significant nosocomial pathogen around the globe. The emergence of multidrug-resistant *Candida auris* is a multifactorial phenomenon that raises many serious public health concerns.This study focused on the molecular characterization of *Candida auris* isolates, with a particular emphasis on the detection of the *ITS1* (for identification), *FKS1*, and *FKS2* genes. The successful identification of these genes provides critical insights into the genetic determinants associated with antifungal resistance. The ITS1 gene, a well-established molecular marker for fungal species identification, was detected in isolates, confirming the presence of *Candida auris*and supporting its utility in accurate, species-level diagnostics.

This study reinforces the growing concern surrounding antifungal resistance in *Candida auris* and emphasizes the value of molecular diagnostics in enhancing our ability and need to institute rapid genomic screens for drug-resistant markers as a corollary to susceptibility testing to track, manage, and ultimately combat this emerging global health threat There is Finally new drugs and combinations directed against *Candida auris*and other drug-resistant pathogens must always remain a high priority.

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