RESEARCH / INVESTIGACIÓN

Prevalence of antibodies in Iraqi Urinary Tract Infection patients using radial immunodiffusion (RID) assay

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Abstract: Candida has different types that could cause bloodstream infections. A total number of 150 samples were collected from candidemia patients and examined. The *Candida spp.* Species isolated from blood samples were analysed. These were identified by culturing the species using different media, namely the chromogenic agar test. Then, the virulence factors of all samples were tested. *The Candida* glabrata isolates were tested with six commercial antifungal drugs. *C. glabrata* 67 (44.6%), *C. albicans* 34 (22.6%), *C. krusei* 18 (12%), *C. tropicalis* 17 (11.3%), and *C. parasilosis* 14 (9.3%). the production of phospholipase ranged between 0.63-0.99 mm. It was found that 96% of the species showed phospholipase activity in aerobic conditions. The protease activities of *Candida spp.* Isolates were experimentally tested by area of inhibition around the colonies, where 59.3% had the double (++) protease activity, 31.4% with (+) grade, and 9.3% had (–) grade or clear zone around the colony. The hemolytic capacity ranged from 0.69-0.89 in the optimum aerobic environments. Finally, 38.33% of the isolated *Candida spp.* were positive and 61.67% negative for biofilm formation. Out of the total positive *Candida spp.* for biofilm formation, 21.73% were strong biofilm producers, and 78.27% were weak. Minimum fungicidal concentration (MFC) of Fluconazole for *C. glabrata* isolates was not appropriate (NA) due to the occurrence of low inhibition tested for species. Micafungin exhibited the lowest fungicidal activity against *C. glabrata* ranging from 0.03 - 0.125, while Fluconazole showed the highest.

Key words: Candidemia, chromogenic agar, *Candida glabrata*, Antifungal.

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Introduction

Urinary tract infections (UTIs) are common bacterial infections, with nearly more than one hundred and fifty million individuals infected yearly[1]. In the United States, estimated 10.5 million clinic cases with UTI symptoms and[2–3] million cases visit the emergency department[2–4].

The urinary tract includes kidneys, ureters, bladder, and urethra. Several natural factors are secret with urine from the urinary tract for protecting against all infection agents; also, the anatomical and mechanical barriers have a significant role, such as the glycoprotein plaque uroplakins[5] and a layer of hydrated mucus[6]. Furthermore, the urinary tract lining is made by immune cells and epithelial cells, which have an important role in protecting against bacterial infection and preventing bacteria from getting into the urinary tract[6]. UTIs are most common in females more than males, and the incidence rate has increased during the last (30) years[6].

UTIs increase in the elderly due to the immune-compromised individuals, and the urinary catheter has an essential role in increasing the percentage of infection[7].

Immunity has two types innate immunity and adaptive immunity[8-10]. The individual with innate immunity has resistance against the infection. Innate immunity includes many chemical and cellular components. The infection gets in and spreads, but the person with high immunity has resistance against the bacteria[11]. Adaptive immunity has two types of humoral immunity, and cellular immunity includes all the naturally acquired immunity due to infection vaccination[12-15].

Features likely to contribute to disease of the human immune system involve the responses to antigens (B- and T-cell features, PMNs), the efficiency of bacterial destruction (e.g., lysozyme, complement), and types of antimicrobial substances produced (e.g., immunoglobulins, Cytokines)[16]. Immunoglobulins are a group of serum proteins with a crucial antimicrobial activity; IgM represents an indicator of recent infection, IgG levels increased in chronic infections[17].

Materials and methods

This study was conducted around UTI Iraqi patients during the period from November 2019 to April 2020. This study included 40 patients and 20 healthy controls.

Samples collection

Serum samples were collected from the patient in Al-Khalis General Hospital and National Laboratories in Al-Khalis District / Diyala Governorate (Iraq) from November 2019 to April 2020. After examining their urine samples by the microscope, they were divided into two groups first is patients with UTI, and the other group is healthy who does not suffer from UTI; this group is considered the control for comparison with the first group.

Estimation of antibodies

Using radial immunodiffusion test to detect levels of antibodies in serum is done by using (LTA, Italia). Normal values of antibodies were IgG (800-1800 mg/dl) and IgM (60-280 mg/dl).

Statistical Analysis

The Statistical Analysis System (SPSS) program was used to detect the effect of different factors in study parameters least significant difference, using T-test for comparing between the means value and the using of the Chi-Square test for comparing between the percentage.

Results and discussion

The patient group involved 40 patients, 8 males 20% and 32 females 80%, with a mean age of 40.60 ± 2.295 years. The healthy control group consisted of 20 subjects, 11 males (55%) and 9 females (45%), with a mean age of 36.80 ± 3.441 years, as seen in tables 1 and 2 explain the distribution of the two study groups according to gender and age.

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Results in Tables 1 and 2 show UTI infections occur in females more than males; the mean age is 40.60 ± 2.295 years. The higher incidence of UTI in females than males might be due to a variety of factors, such as the proximity of the female urethral meatus to the anus[18]. Also, most school girls are avoiding urinable in school, which is accompanied by urinary infection[19]. Alternatively, vaginal flora has a vital role vaginal infection with coliforms which leads to urinary tract infection[20]. The female anatomic feature also contributes to higher prevalence among the female subject. This finding is agreed with earlier studies[21-22].

There was a significant increase in the mean concentration of IgG in sera of patients (854.82±138.79) mg/dl compared with the mean concentration of IgG in sera of the control group (616.4 ± 67.65) mg/dl. These results show high significance between two groups UTI patients and control group according to IgG while non-significant to IgM show table 3. This finding is agreed with AlSaadawi and Alkhaled (2015)[23]. Elevation in serum concentration of IgG in patients suffering from UTI may

be the reason for the significant increases in the concentration of complement components in patients suffering from UTI24. Higher values IgG undergo increases to protect and combat infection[24].

Table 4 shows the non-significant difference in gender distribution between two groups according to IgG and IgM. IgG and IgM prevalence in UTI patients was (838.82 ± 121.49) mg/dl and (70.23± 8.48) mg/dl for male IgG and IgM respectively, while in females was (858.82 ± 171.63) mg/dl and (77.28 ± 7.62) mg/dl for IgG and IgM respectively, this similar with El Mashad et al.2017[25].

Non-significant between two groups (with RBC and without RBC) according to IgG and IgM. With RBC group was (823.85 ± 152.75) mg/dl and (82.01± 15.78) mg/dl for IgG and IgM respectively, while in without RBC group was (871.49 ± 199.17) mg/dl and (72.57 ± 4.93) mg/dl for IgG and IgM respectively show table 5.

	Patient		Control	
Group	Number	%	Number	%
Male	8	20	11	55
Female	32	80	9	45
Total	40	100 %	20	100 %
Chi-Square (χ²)	7.548**			
Sig.	.006			
(P<0. 05).				

Table 1. Distribution of the two-studied group according to gender.

Study group	**N**	**Minimum**	**Maximum**	**Mean**	**Std. Error**
Patient	40	4	75	40.60	2.295
Control	20	3	67	36.80	3.441
Total	60	3	75	39.33	1.909
T	0. 937*				
Sig.	0.981				
(P<0. 05).					

Table 2. Distribution of the two-studied group according to age.

			IgG		IgM	
Groups	**N**	**%**	**Mean**	**Std. Error**	**Mean**	**Std. Error**
Patients	40	33.3	854.82	138.79	75.87	6.30
Control	20	66.7	616.4	67.65	87.94	15.15
T			1.176**		-.868	
Sig.			.069		.029 NS	
(P<0. 05).						

Table 3. APrevalence of IgG and IgM among UTI patients and control group.

			IgG		IgM	
Groups	**N**	**%**	**Mean**	**Std. Error**	**Mean**	**Std. Error**
Male	8	20	838.82	121.49	70.23	8.48
Female	32	80	858.82	171.63	77.28	7.62
T			-0.057		-0.443	
Sig.			0.210		0.365	
(P<0. 05).						

Table 4. Effect of gender on IgG and IgM prevalence in UTI patients.

Groups	N	%	IgG		IgM	
			Mean	Std. Error	Mean	Std. Error
With RBC	14	35	823.85	152.75	82.01	15.78
Without RBC	26	65	871.49	199.17	72.57	4.93
T			-0.162		0.710	
Sig.			0.623		0 .015 NS	
(P<0. 05).						

Table 5. Effect of present RBC on IgG and IgM prevalence in UTI patients.

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Conclusions

We conclude that UTI infection occurs in females more than males; the mean age is 40 years. In all patients with UTI compared to control, the IgG serum level increases and IgM serum level decreases. Serum IgG and IgM showed high significance between two groups of UTI patients and the control group according to IgG while IgM showed non-significant and serum IgG and IgM showed no significant difference in UTI patients and RBC groups.

Bibliographic references

1. Stamm WE, Norrby SR. Urinary tract infections: disease panorama and challenges. The Journal of infectious diseases. 2001 Mar 1;183(Supplement_1):S1-4.

2. Schappert SM, Rechtsteiner EA. Ambulatory medical care utilization estimates for 2007. Vital and Health Statistics. Series 13, Data from the National Health Survey. 2011 1 April (169):1-38.

3. Foxman B. Urinary tract infection syndromes: occurrence, recurrence, bacteriology, risk factors, and disease burden. Infectious Disease Clinics. 2014 Mar 1;28(1):1-3.

4. Foxman B. The epidemiology of urinary tract infection. Nature Reviews Urology. 2010 Dec;7(12):653-60.

5. Wu XR, Kong XP, Pellicer A, Kreibich G, Sun TT. Uroplakins in urothelial biology, function, and disease. Kidney international. 2009 1 June;75(11):1153-65.

6. Grist M, Chakraborty J. Identification of a mucin layerin the urinary bladder. Urology. 1994 1 July;44(1):26-33.

7. Nicolle LE. Urinary tract pathogens in complicated infection and elderly individuals. The Journal of infectious diseases. 2001 1 March;183(Supplement_1): S5-8.

8. Becknell B, Schwaderer A, Hains DS, Spencer JD. Amplifying renal immunity: the role of antimicrobial peptides in pyelonephritis. Nature Reviews Nephrology. 2015 Nov;11(11):642-55.

9. Hains DS, Schwaderer AL. Genetic variations in vesicoureteral reflux sequelae. Pathogens. 2016 Mar;5(1):14.

10. Kucheria R, Dasgupta P, Sacks S, Khan M, Sheerin N. Urinary tract infections: new insights into a common problem. Postgraduate medical journal. 2005 Feb;81(952):83.

11. Schilling JD, Mulvey MA, Hultgren SJ. Dynamic interactions between host and pathogen during acute urinary tract infections. Urology. 2001 1 June;57(6):56-61.

12. Cheng CH, Lee YS, Chang CJ, Lin JC, Lin TY. Genetic polymorphisms in inflammasome-dependent innate immunity among pediatric patients with severe renal parenchymal infections. PloS one. 2015 Oct 7;10(10):e0140128.

13. Lüthje P, Brauner A. Novel strategies in the prevention and treatment of urinary tract infections. Pathogens. 2016 Mar;5(1):13.

14. Mak RH, Kuo HJ. Pathogenesis of urinary tract infection: an update. Current opinion in pediatrics. 2006 Apr 1;18(2):148-52.

15. Ulett GC, Totsika M, Schaale K, Carey AJ, Sweet MJ, Schembri MA. Uropathogenic Escherichia coli virulence and innate immune responses during urinary tract infection. Current opinion in microbiology. 2013 Feb 1;16(1):100-7.

16. Akhavan Sepahi M, Hosseini R, Akhavan Sepahi A. The innate immune response during acute urinary tract infections; an overview. Caspian Journal of Pediatrics. 2018 Mar 10;4(1):282-9.

17. Al-khafaf DM. Study of some microbial and Immunological parameter in patients with Urinary Tract Infection in Al-Diwanyia city. Al-Qadisiyah Medical Journal. 2012;8(13):28-38.

18. Goldman M, Lahat E, Strauss S, Reisler G, Livne A, Gordin L, Aladjem M. Imaging after urinary tract infection in male neonates. Pediatrics. 2000 1 June;105(6):1232-5.

19. Schneede P, Hofstetter AG, Naber KG, Vahlensieck W, Ludwig M, Bach D, Bauer HW, Beyaert G, Blenk H, Bootz T, Friesen A. European Association of Urology guidelines on urinary and male genital tract infections. Der Urologe. Ausg. A. 2003 Jan 1;42(1):104-12.

20. Newman TB, Bernzweig JA, Takayama JI, Finch SA, Wasserman RC, Pantell RH. Urine testing and urinary tract infections in febrile infants seen in office settings: the Pediatric Research in Office Settings' Febrile Infant Study. Archives of pediatrics & adolescent medicine. 2002 1 January;156(1):44-54.

21. Oluwafemi TT, Akinbodewa AA, Ogunleye A, Adejumo OA. Urinary tract infections and antibiotic sensitivity pattern of uropathogens in a tertiary hospital in South West, Nigeria. Sahel Medical Journal. 2018 1 January;21(1):18.

22. AlSaadawi MA. Prevalence of toxoplasmosis in renal infections patients in Al-Muthanna province/Iraq. Al-Qadisiyah Journal of Veterinary Medicine Sciences. 2015 30 June;14(1):58-60.

23. NAJDAT B MAHDI SK. Relationship among Some Immunoglobulins, Complement Protein C3 and Urinary Tract Infection of Women Caused by Gram Negative Bacilli in Kirkuk, Iraq. International Journal of Current Research and Academic Review. 2016;4:99-106.

24. Weichhart T, Haidinger M, Hörl WH, Säemann MD. Current concepts of molecular defence mechanisms operative during urinary tract infection. European journal of clinical investigation. 2008 Oct;38:29-38.

25. El Mashad GM, Ibrahim SA, Abdelnaby SA. Immunoglobulin G and M levels in childhood nephrotic syndrome: two centers Egyptian study. Electronic physician. 2017 Feb;9(2):3728.