

STUDY EFFECT OF URINARY TRACT INFECTION ON SOME BIOCHEMICAL PROFILE AMONG PATIENTS IN AL- NAJAF GOVERNORATE, IRAQ

¹Saleem K. A. Al-Hadraawy, ²Maysoon K. A. Al-Hadraawy, ³Dr. Kareem Ghali Mohamed*

¹Department of Biology, Faculty of Science, University of Kufa, Iraq.

²Technical Institute, Kufa Al-Furat AL-Oust Technical University.

³College of Medicine, University of Kufa.

Article Received on
26 May 2015,

Revised on 19 June 2015,
Accepted on 12 July 2015

*Correspondence for
Author

Dr. Kareem Ghali
Mohamed

College of Medicine,
University of Kufa.

ABSTRACT

Urinary tract infection (UTI) is a worldwide health problem with an important health impacts on individual suffering from it in relation to effect on certain biochemical changes. **Aims of study:** The present study aims to find out the effect of UTI on some biochemical profile in patients and comparing that with normal individuals. **Methodology:** A cross-sectional study was conducted in Al- Najaf Province during the period between September 2014-March 2014, fulfilled by using different biochemical techniques for estimation of lipid and some other biochemical profile among randomly selected 120 patients with UTI and 150 healthy control individuals. **Results:** There were a significant

decrease in the level of serum Cholesterol, Triglyceride (TG), Low-density lipoprotein (LDL), Very low-density lipoprotein (VLDL), with significant increase of High-density lipoprotein (HDL), also there were significant increase of blood urea with slight increase in serum creatinin, all above changes were among those with UTI when compared with normal control group. **Conclusion:** Urinary tract infection has a significant impact on lipid profile and some other biochemical changes when compared with healthy control group. **Recommendations:** Measuring of certain cytokine changes among patient with UTI to know whether a relationship with changes in lipid Profile.

KEYWORD: Urinary tract infection (UTI), lipid profile.

INTRODUCTION

Urinary tract infections are infection that affects part of the urinary tract if affects the lower urinary tract it is known as a simple cystitis and if affects the upper urinary tract it is known as pyelonephritis . The most frequent healthcare-associated infection in the United States.^[1,2] and the second most frequent in Denmark.^[3] Notably, urinary tract infections have been reported as a frequent focus of infection for enterococcal bacteremia.^[2,4] As described earlier, human infections by enterococci have increased notably through the past two to three decades and this increase includes a rise in cases of urinary tract infections. It is reasonable to suspect the increase in enterococcal urinary tract infections to be partly responsible for the increase in enterococcal bacteremia due to the association of the two. A number of previous studies investigating enterococcal bacteremia, all found urinary tract infections as one of the dominant Sources of infection.^[4,5]

The majority of urinary tract infections are related to indwelling urinary catheters.^[6] Notably, patients with indwelling urinary catheters have been reported to have a high incidence of enterococcal urinary tract infections compared to patients receiving outpatient treatment.^[7] It was described by the Egyptians as "sending forth heat from the bladder."^[8] In young sexually active women, sexual activity is the cause of 75–90% of bladder infections, with the risk of infection related to the frequency of sex .^[9]

U.T.I. are important because they may involve the urethra, the bladder, urethras and kidneys,^[10] they are more common in females than males in ratio about 6:1 with exception of the neonatal period when the sexes are equally affected.^[11] UTI cause considerable discomfort and inconvenience to the patient and are occasionally responsible for protracted symptoms or more serious manifestation, such as sepsis and death.^[12]

MATERIALS AND METHODS

Study design and Patients: Samples were collected in the period from September 2014 until March 2015, 120 samples were randomly collected from patients with UTI and 150 healthy who attended the clinics AL-Sadder Teaching Hospital and AL-Zahra Hospital in AL-Najaf governorate.

Collection of Samples

Urine samples: Patients were supposed to have UTI after performing urinalysis and then confirmed with a urine culture, midstream urine collection is done to minimize sample

contamination. High colony count of one type of bacterium will be present if a person has a urinary tract infection, if there are three or more types of bacteria present, the sample is considered to be contaminated and discarded.

Blood samples: were also drawn from the patients by vein-puncture into specimen tubes and remains for 30 minutes at room temperature. After that the samples were centrifuged at 3000 rpm for 5 minutes (Backman /counter, Germany) to separate the serum and collected in other sterile tubes and kept in deep freeze at -20C° till used for the determination of Lipid profile, urea, creatinine and uric acid.

SEROLOGICAL DIAGNOSIS

Estimation of Total Serum Cholesterol

Measurement of serum cholesterol by dependent on enzymatic method Where cholesterol esterat lysis to cholesterol and fatty acid by cholesterol esterase.^[13]

Estimation of Total Serum glycerides (TG)

Measurement of TG in the serum by used enzymatic and colorimetric method, the Tri-glycerides in serum lysis enzymatically to Glycerol Phosphate and fatty acid by Lipase.^[14]

Estimation of Total Serum High Density Lipoprotein (HDL)

Measurement of (HDL) in the serum by used sedimentation Lipoproteins are found with HDL, its include (LDL, VLDL) by used phosphotungstic acid solution with found Mg⁺.^[15] The Very Low Density Lipoprotein concentration was calculated by using the following formula^[16] VLDL.

Cholesterol (mg/100 ml) = Tri- glycerides/5.

The Low Density Lipoprotein Concentration was calculated by using the following formula Low density lipoprotein =Serum cholesterol - (VLDL+HDL).^[17]

Statistical analysis

Data were analyzed using the software packages Graph pad prism for Windows (5.04, Graph pad software Inc. USA), Data are presented as the mean ± standard error (SE). The comparison between the patients and healthy groups were analyzed by one-way ANOVA. A p-value < 0.05 was considered significant.

RESULT

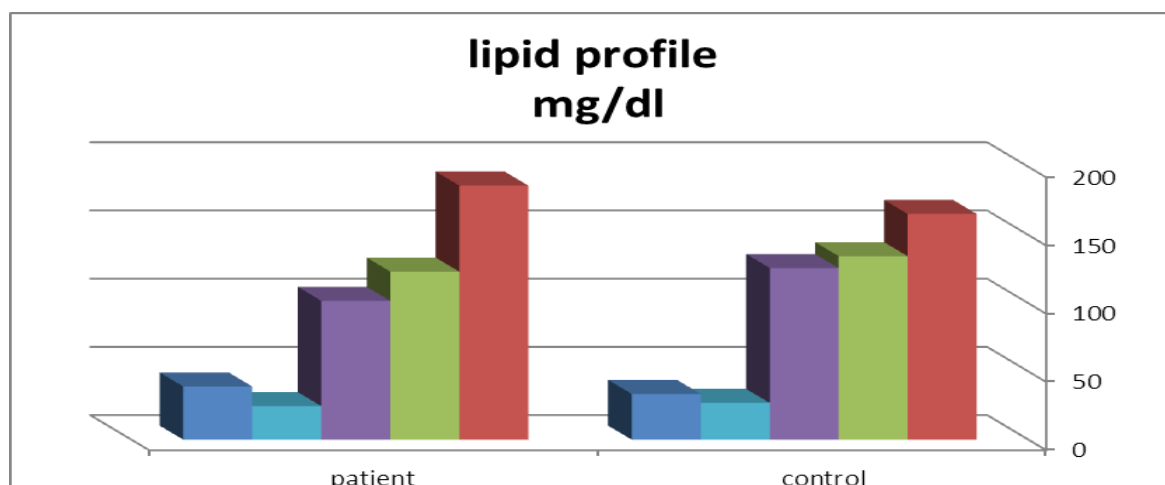
The present study recorded significant decrease in the level of cholesterol and Triglyceride, LDL and VLDL in patients (165.47 ± 0.601 mg/dl; 123.2 ± 0.552 ; 101.83 ± 0.328 ; 24.64 ± 0.702) compared to healthy (186.3 mg/dl; 134.9 mg/dl; 125.82 ; 26.98) respectively. While the HDL was significantly increase (39.0 ± 0.065 mg/dl) compared to control group (33.5 ± 0.321 mg/dl). as showed in figuer (1); table(1), also this study recorded a non significant increase in level of serum uric acid but significant increase in level blood urea (28.6 ± 0.032 mg/dl) compared with control group (25.1 ± 0.811 mg/dl) as showed figuer (2); table(2).

Table 1: The level of lipid profile in patients with UTI and control group

Parameters	Paients with UTI	Control
Cholestrol (mg/dl)	165.47 ± 0.60 *	186.3 ± 1.24
Triglyceride (mg/dl)	123.2 ± 0.552 *	134.9 ± 1.65
LDL (mg/dl)	101.83 ± 0.328 *	125.82 ± 0.62
VLDL (mg/dl)	24.64 ± 0.702 *	26.98 ± 0.54
HDL (mg/dl)	39.0 ± 0.06 **	33.5 ± 0.3

*Significantly lower than control at $p < 0.05$.

**significantly higher than control at $p < 0.05$.

**Figure 1: Lipid profile serum concentration (mg/dl) Comparison between Patients Suffering from urinary tract Infection and Control Group.****Table 2: The level of blood urea and in patients with UTI and control group**

Parameters	Paients with UTI	Control
Blood urea (mg/dl)	28.6 ± 0.032 *	25.1 ± 0.811

*significantly higher than control at $p < 0.05$

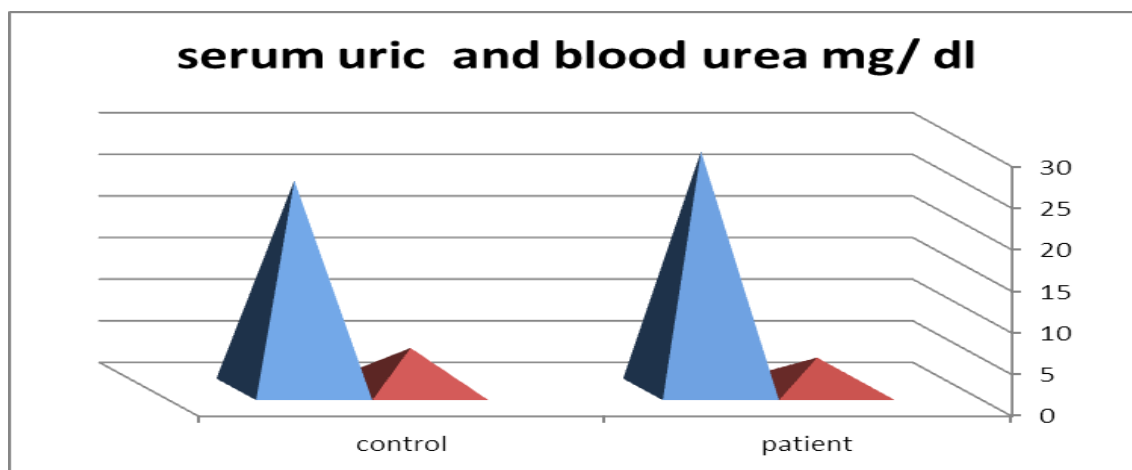


Figure 2: Serum uric acid and urea (mg/dl) Comparison between Patients Suffering from urinary tract Infection and Control Group.

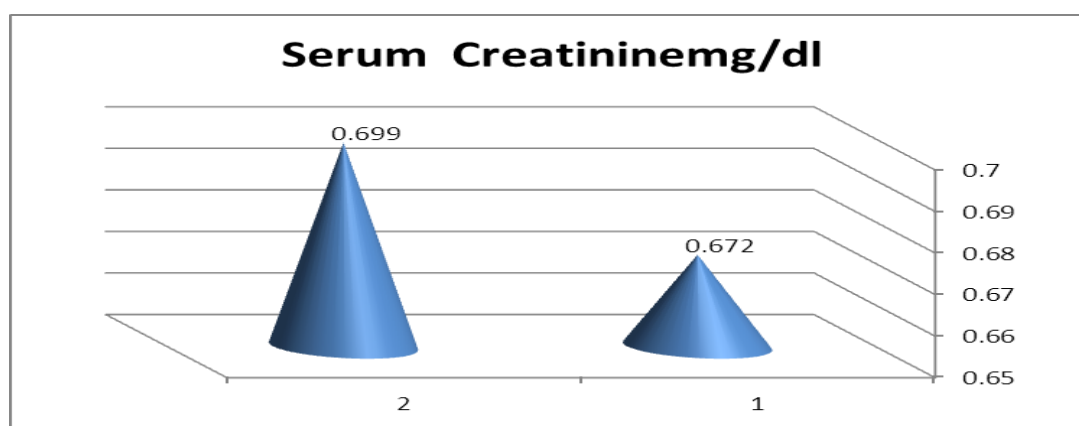


Figure 3: Creatinine serum concentration (mg/dl) Comparison between Patients Suffering from urinary tract Infection and Control Group.

DISCUSSION

In the present study the patients with urinary tract infection had significantly decreased level of total cholesterol when compared with the control, this go with finding of vanleuwen *et al.*, 2003 (18) who stated that increased cytokines caused decreased level of cholesterol in acute illness. The interaction of increased level cytokines which are produced during urinary tract infection probably play a role to decrease the level of low density lipoprotein (LDL), or the level of (LDL) decreased due to the host immune response toward microorganism causing urinary tract infection which could induce (LDL) oxidation leading to reduced level of (LDL).^[19]

It was stated that bacterial infection cause profound effect on different types of lipid concentration via formation of free radicals, these radicals will cause lipid peroxidation,^[20]

consequently lipid peroxidation result in oxidative deterioration of polyunsaturated lipid,^[19] moreover lipid are carried by lipoproteins, which is responsible for transportation of lipid to different tissue,^[21,22] when an individual have UTI the function of these lipoprotein will be affected thereby the level lipid profile will be changed as a net result. The level of (HDL) will increase when compared with controlled healthy group this inconsistent with finding of Alvarez and Romas, 1986^[23] who stated that (HDL) will decrease during sepsis.

Regarding triglyceride (TG) the current study revealed that TG was significantly decreased as compared with the healthy controlled group due to the alteration in the function and composition of the lipoprotein ,the present result was in agreement with gordon *et al.*, 2001.^[23]

CONCLUSIONS

In the present study patients with UTI have significant effect on the lipid profile in that there were significantly decreased in serum Cholesterol, TG, LDL, VLDL among patients with UTI when compared with health controlled group.

REFERENCES

1. Chang R. Epidemiology of hospital-acquired urinary tract-related bloodstream infection at a university hospital. *Infect. Control Hosp. Epidemiol.* 2011; 32: 1127–9.
2. Klevens R M .Dialysis Surveillance Report: National Healthcare Safety Network (NHSN)-data summary for 2006. *Semin. Dial.* 2006; 21: 24–8.
3. Landsprævalensundersøgelsen foråret 2013; 1–10.
4. Graninger W & Ragette R .Nosocomial bacteremia due to *Enterococcus faecalis* without endocarditis. *Clin. Infect. Dis.* 1992; 15: 49–57.
5. Maki D G & Agger W A. Enterococcal bacteremia: clinical features, the risk of endocarditis, and management. *Medicine (Baltimore)*. 1988; 67: 248–269. 26 17. Greene, M. T. et al. Predictors of hospi.
6. Burton D C., Edwards J. R, SrinivasanA , Fridkin SK & Gould, C. V. Trends in catheter associated urinary tract infections in adult intensive care units-United States, 1990-2007. *Infect. Control Hosp. Epidemiol.* 2011; 32: 748–56.
7. Warren J W, Tenney J. H., Hoopes J. M., Muncie, H. L. & Anthony, W. C. A Prospective microbiologic study of bacteriuria in patients with chronic indwelling urethral catheters. *J. Infect. Dis.* 1982; 146: 719–723.

8. Wilson. *Topley and Wilson's Principles of bacteriology, virology and immunity: in 4 volumes* (8. ed. ed.). London: Arnold. 1990; 198. ISBN 0-7131-4591-9.
9. Nicolle LE "Uncomplicated urinary tract infection in adults including uncomplicated pyelonephritis". *Urol Clin North Am*, 2008; 35 (1): 1–12. v.doi:10.1016/j.ucl.2008.09.004. PMID 18061019.
10. Tortora G.J., Funke B R.,Case CH. L.. *Microbiology An Introduction* .Fifth Edition 1995; 588-611.
11. Prescott L M, Harley J P, Klein D. A. *Microbiology* .Fifth edition. 2002; 899-940.
12. Levinson W, Jawetz E, *Medical- Microbiology & Immunology*. Sixth Edition. 2000; 49: 107,194.
13. Allain AC; Poon LS and Chan CSG." Enzymatic determination of total serum cholesterol ". *Clin. Cham*. 1974; 20- 470.
14. Fossati P. and Prencipe. L. Serum TG determination colorimetrically with on enzyme that produces inflammatory reaction" *.Am. j. pathol*. 1982; 107-397.
15. Kaplan L and Pesce AJ *Clinical chemistry Theory Analysis and Correlation 2nd ed*. Mosby Company United State of America 1989.
16. Friedewald W .Levy RJ and Frederickson DS Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparation ultracentrifuge". *Cline Chem*. 1972; 18: 499-502.
17. Wilson PW "Why treated dislipidemia". *Saudi med .J.*; 1998; 19(4): 3776- 381.
18. Vanleeuwen HJ, Heezius EC, Dallinga GM, Vanstrijp JA, Verhoef J and Vankessel KP. Lipoprotein metabolism in patients with severe sepsis. *Crit Care Med*, 2003; 31: 1359-1366.
19. Nnodim Johnkennedy, Obi Patrick Chinedu, Nwacha Richard, Ihim Augustine Chinedu, Osuoha Chinyere and and Edward Ukamaka Investigations on Serum Lipid Profile in Patients With Urinary Tract Infections. *Global Journal of Scientific Researches*. 2013; 1(3): 68-70.
20. Nnodim JK, Ihim A and Uduji HI.. Attenuation of chloroquine-induced hepatotoxicity and renal damage by Gnetum bucholzianum leaf extract. *New Zealand Journal of medical Laboratory Science*, 2012; 66: 46-47.
21. Farmer JA. Diabetic dyslipidemia and atherosclerosis: evidence from clinical trials. *Curr Diab Rep* 2008; 8: 71–7.
22. Goldenberg I, Benderly M, Sidi R, Boyko V, Tenenbaum A and Tanne D. Relation of clinical benefit of raising highdensity lipoprotein cholesterol to serum levels of low-

- density lipoprotein cholesterol in patients with coronary heart disease. *Am Cardiol* . 2009; 103: 41–5.
23. Alvarez C and Ramos A. Lipids, lipoproteins and apoproteins in serum during infections. *Clin Chem* 32:142 Barja G. 2012. “Free Radicals and Ageing”. *Tends Neuroscience* 1986; 257: 1-6.
24. Gordon BR, Parker TS, Levine DM, Saal SD, Wang JC and Sloan BJ.. Relationship of hypolipidaemia to cytokine concentrations and out comes in critically ill patients. *Crit Care Med*: 2001; 29: 1563-1568.