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Investigations on Serum Lipid Profile in Patients With Urinary Tract Infections

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ABSTRACT

To determine the effect of urinary tract infections On serum lipid profile(Total cholesterol, Triglyceride, High density lipoprotein and Low density lipoprotein) Materials and methods: A cross-sectional study was conducted involving 130 patients with urinary tract infections attending General Hospital Owerri and 130 healthy individuals as controls. Both groups are within the age of 45 to 60years. The serum lipid profile was measured in all subjects. The statistical analysis was done using student t-test at $P < 0.05$. Results: The levels of lipid profile was decreased. However, the level of cholesterol and high density lipoprotein was significantly depleted when compared with the control at $P < 0.05$. Conclusion; These observation shows that low levels of lipid profile particularly total cholesterol and high density lipoprotein may be probably be associated with urinary tract infections. Hence, the decreased levels of cholesterol and HDL could probably serve as indicators of urinary tract infection.

Keywords: *Urinary tract infection, Lipid profile.*

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INTRODUCTION

Urinary tract infection is a bacterial infection that affects part of the urinary tract. It affects the lower urinary tract as well as upper urinary tract. It is characterized by painful urination, urge to urinate or sometimes frequent urination. Other symptoms include fever in addition to flank pains.

The bacteria that are commonly implicated are *Echerichia coli*. However, other bacteria, viruses and fungi can be the cause in rare cases. The bacteria that cause urinary infections mainly enter the bladder through the urethra. Also, infection occur through the blood or lymph. It is pertinent to note that bacteria are usually transmitted to the urethra from the bowel, with females at greater risk due to their anatomy. Hence, urinary tract infection is most common among women after gaining entry to the bladder. Bacteria infections cause a variety of changes in lipid profile concentrations by generating free radicals. Free radicals are reactive species generated by biochemical redox interactions that occur as a part of normal cell metabolism. These free radicals may cause lipid peroxidation and damage cellular structures of organism particularly erythrocytes and endothelium (Nnodim et al., 2012). Lipid peroxidation is a free radical mediated chain of reaction that, once initiated, results in oxidative deterioration of polyunsaturated lipids. The most common targets are components of biological membrane. When propagated in biological membranes, these reaction can be initiated or enhanced by a number of toxic products, including endoperoxides and aldehydes (Barja, 2012; Machlin and Bandich, 2010). The injury mediated by free radicals can be measured by conjugated dienes, malondialdehyde, 4-hydroxy nonetal, and others. Normally, free radical formation is

controlled naturally by various beneficial components known as antioxidants. When there is a deficiency of these antioxidants, damages due to free radicals can become cumulative and debilitating (Sies and Helmut, 2010; Stadtman, 2012).

Lipid profile is a group of tests that are often ordered together to determine risk of coronary heart disease. It is a good indicator of whether someone will have a heart attack or stroke caused by blockage of blood vessels or hardening of arteries (Nnodim et al., 2011). The principal lipids found in the human plasma are fatty acids, triglycerides, cholesterol and phospholipids (Zhang et al., 2007 and Nwanjo, 2005). However, this study is design to evaluate the concentrations of total cholesterol, triglycerides, high density lipoprotein and low density lipoprotein in patients with urinary tract infection.

MATERIALS AND METHODS

Subjects:130 patients with urinary tract infections attending General Hospital Owerri and 130 healthy individuals as controls were involved. Both groups are within the age of 45 to 60years. Patients with past history of systemic disease like diabetes, hyperlipidaemia and hypertension were excluded from the study. Their consent was obtained as ethical approval from the ethical committee of the hospital.

Blood collection: In all subjects,5ml of fasting veinous blood was collected into plain bottles. The serum was separated by centrifuging the whole blood in a westerfuge (model 684) centrifuge at 5000g for 5minutes.

Estimation of biochemical assay: The serum lipid profile was measured by the colorimetric method using Biosystem Kits. Cholesterol (COD11505), triglyceride (COD11528) and HDL-C (COD 11523) and LDL-Cholesterol(COD11579).

Statistical analysis: The results were expressed as mean \pm standard deviation. The statistical evaluation of data was performed by using Studet t-test

RESULTS AND DISCUSSION

Results:

Table 1. The level of lipid profile in patients with urinary tract infection and control group.

Parameters	patients with urinary tract infection	control
Cholesterol(mg/dl)	122.4 \pm 5.93 *	160.32 \pm 6.01
Triglyceride(mg/dl)	110.5 \pm 10.70*	148.58 \pm 10.32
HDL(mg/dl)	37.41 \pm 1.92*	43.61 \pm 1.49
LDL(mg/dl)	72.88 \pm 8.31*	95.47 \pm 8.53

*Significantly different from control at P<0.05

Discussion

Lipids are carried in lipoproteins that transport the lipid to various tissues for steroid hormone formation, energy usage, bile acid formation as well as lipid deposition(Farmer, 2008 and Goldenberg et al 2009).

In this study,it was observed that patients with urinary tract infections had significantly decreased level of total cholesterol when compared with the control. This could probably be associated with synthesis and utilization of plasma lipids as well as the interactions of cytokines which are produced during urinary tract inflammation. This is in line with the work of Vanleeuwen et al., 2003 which stated that increased cytokines caused decreased level of cholesterol in acute illness. In the same vein, the level of triglyceride was significantly increased in urinary tract infection when compared with the control. This is in line with the work of Gordon et al., 2001. Urinary tract infections produce alterations in the functions and compositions of lipoproteins.

Furthermore, it was observed that the levels of HDL and LDL were decreased when compared with the control. This is consistent with the work of Alvarez and Ramos, 1986 in which apoprotein and HDL decreased during sepsis. Vermont et al., 2005 also reported the decreased levels of HDL in children with severe meningococcal disease.

Finally, the levels of LDL was significantl reduced when compared with the control. This could be associated with the host response to urinary tract infection which could induce LDL oxidation leading to reduced LDL. It could be suggested that decreased plasma lipid profile may serve as markers of urinary tract infections

REFERENCES

- Alvarez C and Ramos A. 1986. Lipids, lipoproteins and apoproteins in serum during infections. Clin Chem32:142-145.
Barja G. 2012. "Free Radicals and Ageing". *Tends Neuroscience* 257:1-6.

- Farmer JA. 2008. Diabetic dyslipidemia and atherosclerosis: evidence from clinical trials. *Curr Diab Rep* ; 8: 71–7.
- Goldenberg I, Benderly M, Sidi R, Boyko V, Tenenbaum A and Tanne D. 2009. 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* . 1; 103: 41–5.
- Gordon BR, Parker TS, Levine DM, Saal SD, Wang JC and Sloan BJ. 2001. Relationship of hypolipidaemia to cytokine concentrations and out comes in critically ill patients. *Crit Care Med*:29:1563-1568.
- Machlin LJ and Bandich A. 2010. “Free Radical Tissue Damage”. Protective Role of Antioxidant Nutrient. *Journal of Biochemistry* 1: 41-445.
- Nnodim JK, Emejulu A and Nwadike CN. 2011. Hypolipidaemic Effects of Aqueous Extract of *Acalypha capitata* Leaves in Rats Fed on High Cholesterol Diet. *Asian Pacific Journal of Tropical Biomedicine* S183-185.
- Nnodim JK, Ihim A and Uduji HI. 2012 . Attenuation of chloroquine-induced hepatotoxicity and renal damage by *Gnetum bucholzianum* leaf extract. *New Zealand Journal of medical Laboratory Science*.66:46-47.
- Nwanjo HU. 2005. Efficacy of aqueous leaf extract of *Vernonia amygdalina* on plasma lipoprotein and oxidative status in diabetic rats models. *Nig.J.physiol sci*.20:39-42.
- Sies H and Helmut. 2010. “Oxidative stress: Oxidant and antioxidants”. *Experimental physiology* 82: 291-295.
- Stadtman ER. 2012. “Protein Oxidation and Ageing”. *Science Journals* 257: 220-225.
- Vanleeuwen HJ, Heezius EC, Dallinga GM, Vanstrijp JA, Verhoef J and Vankessel KP. 2003. Lipoprotein metabolism in patients with severe sepsis. *Crit Care Med*. 31:1359-1366.
- Vermont CL, Denbrinker M, Kakeci N, Dekleijn ED, Derijke YB and Joosten KF. 2005. Serum lipids and disease severity in children with severe meningococcal sepsis. *Crit Care Med* ;33:1610-1615.
- Zhang HW, Zhang YH and Lu MJ. 2007. Comparison of hypertension, dyslipidaemia and hyperglycemia between buckwheat seed-consuming Mongolia Chinese population in inner Mongolia china. *Clin Exp pharmacol physiol* 34:838-344.