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Dyslipidemia in Acute Renal Failure (ARF)

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Abstract

Introduction: Alteration of lipid levels in ARF patients fastens the atherosclerotic process which causes cardiovascular complications.(1) It is observed that levels of different lipoprotein fractions were changed in acute renal failure patients.

Materials and Method: This case control study includes total 100 subjects. Out of these 50 subjects were clinically diagnosed ARF patients having age more than 20 years and remaining 50 subjects were chosen from healthy controls not having any major medical problem. Fasting venous blood samples were collected and serum levels of Total cholesterol (TC), Triglycerides (TG), High density lipoprotein cholesterol (HDL-C), Low density lipoprotein cholesterol (LDL-C), Very low density lipoprotein cholesterol (VLDL-C) were measured.

Results: It is seen that Total cholesterol, Triglycerides, Very low density lipoprotein cholesterol and Total cholesterol to High density lipoprotein cholesterol ratio were significantly increased in ARF group as compared controls while mean value of High density lipoprotein cholesterol was significantly decreased in ARF as compared to control. The mean level of Low density lipoprotein cholesterol did not show any significant difference between these two groups i.e. controls & ARF.

Conclusion: The present study indicates that due to dyslipidemia in ARF, there is increased risk of cardiovascular complications.

Keywords: ARF, Lipid profile, Total cholesterol, HDL-C.

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Introduction

Acute Renal Failure (ARF) causes renal dysfunction which affects urinary excretion of waste products produced in human body.(2) The common causes for ARF are ischemic renal failure, acute gastroenteritis and hemolytic uremic syndrome.(3)

It is observed that incidence of severe ARF in the general population is approximately 70–140 per million of the population,(3) and around half of these will require dialysis.

Commonly, the etiological factors of ARF are classified by anatomical structure of kidney into pre-renal, renal and post renal causes. Pre-renal ARF is caused by decreased perfusion of normal kidneys, in which the EABV (Effective arteriolar blood volume) is reduced. Damage either to the kidney itself or the surrounding vasculature causes Intrinsic renal failure. Post-renal failure or obstructive nephropathy includes obstruction of urinary outflow which leads to increased pressure within the renal collecting systems and resulting in reduced GFR, reduced tubular reabsorption of sodium and water, and acquired renal tubular acidosis, phosphaturia and other abnormalities of tubular function.

Lipid abnormalities in ARF patients accelerate the process of atherosclerosis resulting in cardiovascular complications.(5)

Materials and Method

Present study was conducted in Department of Biochemistry, Government medical college, Nanded with the help of medicine & surgery Department. Institutional Ethics Committee for research work has given approval for research work. The selection of subjects is carried out from OPD & dialysis unit of Government Medical College, Nanded. A total number of 100 subjects were participated in the study, out of which 50 were clinically diagnosed ARF cases having age more than 20 years and 50 were healthy controls without any major medical illness were included. Patients with diabetes mellitus, hypertension, history of familial hyper lipoproteinemias, history of hepatic dysfunction, patients on hypolipidemic drugs were excluded from study. Following biochemical parameters were determined. Total cholesterol: CHOD PAP method (end point),(6) Triglycerides: GPO Trinder method (end point),(7) HDL: Direct method,(8) LDL: Direct method,(8) VLDL = TAG/5.

Observations and Results

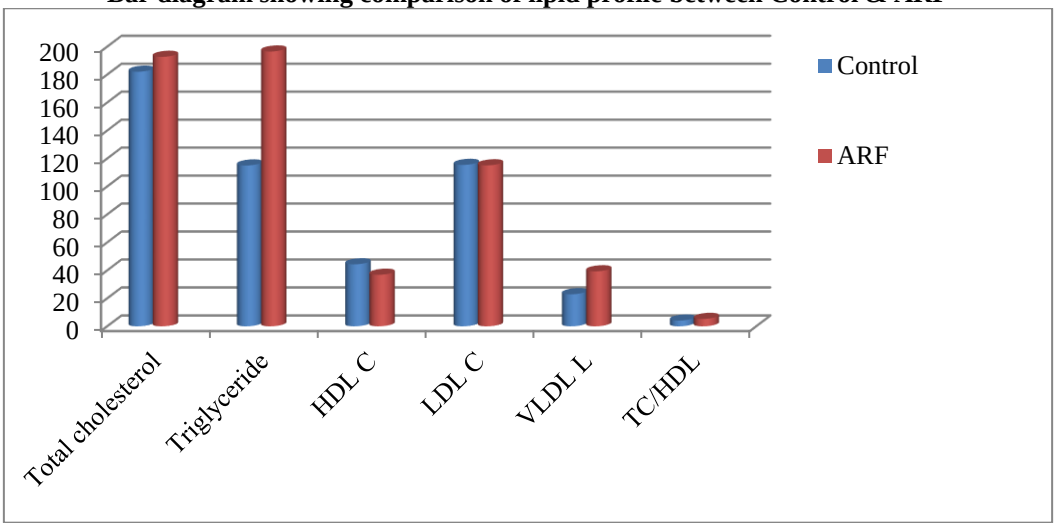
The concentrations of Total Cholesterol (TC), Triglycerides (TG), High Density Cholesterol (HDL-C), Low Density Cholesterol (LDL-C), and very low density cholesterol (VLDL-C) were analyzed and their results were shown in the following tables and graphs.

Table 1: Tukeys multiple comparison of lipid profile between control & ARF

Parameter	Controls	ARF	p-value
Total Cholesterol (mg/dl)	186.10 ± 20.3	192.7 ±11.02	< 0.05
Triglyceride (mg/dl)	122. ± 28.2	196.5 ± 23.1	< 0.05
HDL-C (mg/dl)	40.23 ± 6.0	36.84 ± 3.19	< 0.05
LDL-C (mg/dl)	119.51 ± 22.7	119.16	> 0.05
VLDL-C (mg/dl)	23.8 ± 6.39	40.7 ± 5.44	< 0.05
TC/HDL (mg/dl)	4.62 ± 0.70	5.23 ± 0.58	< 0.05

P < 0.05 - significant
P >0.05 - not significant

Bar diagram showing comparison of lipid profile between Control & ARF



Above Table and graph shows that ARF group shows significantly increased level of total cholesterol, triglyceride, VLDL-C and total cholesterol to HDL-C ratio as compared controls while mean value of HDL-C was significantly decreased in ARF as compared to control. The mean level of LDL did not show any significant difference between control & ARF.

Discussion

Disorders of lipoprotein metabolism during uremia is an important mechanism of atherogenesis in ARF. The percentage of cholesterol in nervous cells in more. It is a important constituent of the cell membrane and of plasma lipoproteins. Serum cholesterol is positively correlated with the incidence of atherosclerosis and coronary vascular disease.[9]

ARF is associated increased serum cholesterol which is due to associated proteinuria and renal insufficiency per se. Hypercholesterolemia is a result of alteration in gene expression for HMG-CoA reductase due to proteinuria which results in increased activity of HMG-CoA reductase. Cholesterol balance in the body is well regulated by LDL receptor mediated cholesterol uptake.[10]

Increased TG level is a commonly observed in ARF. It may be due to decreased clearance of triglyceride rich lipoprotein and their atherogenic remnants.[11] The main reason of dyslipidemia in ARF is abnormal lipolysis. The activities of lipolytic systems, peripheral lipoprotein lipase and hepatic triglyceride lipase are decreased in patients with ARF to less than 50% of normal.[12]

Reverse cholesterol transport is carried out by HDL-C i.e. transportation of cholesterol from extrahepatic tissues to liver for its degradation which is important for cellular cholesterol balance and prevention from atherosclerosis. HDL-C also inhibits inflammation, platelet adhesion and LDL oxidation.[10] Decrease in HDL cholesterol level in ARF shows deregulation of HDL cholesterol metabolism. This may be due to the alterations in the composition and anti oxidant and anti inflammatory functions of HDL cholesterol.[11] Also there is decreased activity of lipoprotein lipase and hepatic lipase in ARF.[12]

ARF associated with decreased clearance of VLDL and chylomicrons. This is due to dysregulation of LPL, hepatic lipase, VLDL receptor, hepatic ACAT and LRP expression/ activities and impaired HDL metabolism leading to increased level of VLDL-C.

Conclusion

This study demonstrates that there is significant alteration found in all lipid profile parameters in cases as compared with control except LDL-C. The altered concentration of serum lipoproteins leads to accelerated atherosclerosis in ARF patients. Hence by correcting the dyslipidemia, associated complications can be avoided.

References

1. Siems W, Quast S, Carluccio F, Wiswedel I, Hirsch D, Augustin W, Hampi H, Riehle M, Sommerburg O Oxidative stress in chronic renal failure as a cardio vascular risk factor. Clin. Nephrol. 58 (1) S1 2-9(2002).
2. Srisawat N. Hoste EE, Kellum JA. Modern classification of acute kidney injury. Bloodpurif. 29:300-7(2010).
3. Bhuyan UN, Bagga A Thrombo. Occulusive nephro antiopathy in children with acute renal failure and hyper tension. Indian J Nephrol 1:137-138, (1991).
4. Feest TG, Round A, Hamad S. Incidence of severe acute renal failure in adults: Results of a community based study. BMJ 1993; 306, 481–483.
5. Siems W, Quast S, Carluccio F, Wiswedel I, Hirsch D, Augustin W, Hampi H, Riehle M, Sommerburg O Oxidative stress in chronic renal failure as a cardio vascular risk factor. Clin Nephrol. 58 (1) S1 2-9(2002).
6. Myers GL et al. A reference method laboratory network for cholesterol: a model for standardization and improvement of clinical laboratory measurement. Clinical Chemistry 2000;46:1762-72.
7. Henry JB. Clinical diagnosis and management of laboratory methods. 18th edition. W. B. Saunders Philadelphia:204-11.
8. Miller WG et al. Seven Direct Methods for Measuring HDL and LDL Cholesterol Compared with Ultracentrifugation Reference Measurement Procedures. Clinical Chemistry 2010;56:977-86.
9. Mayes PA. Lipid transport and storage. In: Murray PK, Granner DK, Mayes PA and Rodwell VW, eds. Harper's Biochemistry, 25th edn. Stampford: Appleton and lange Co.,2000;268-270.
10. Vaziri ND. Dyslipidemia of chronic renal failure: The nature, mechanisms and potential consequences. Am J Physiol Renal Physiol 2006;290:262-272.
11. Nosratola D. Vaziri. Causes of dysregulation of lipid metabolism in chronic renal failure. Semin Dial 22(6):644-651,(2009).
12. Druml W, Zechner R, Magometschnigg D, et al.: Post-heparin lipolytic activity in acute renal failure. Clin Nephrol 1985, 23:289–293.