بسم الله الرحمن الرحيم

Sudan University of Science and Technology

College of Graduate Studies

Assessment of Lipids Profile among Sudanese Patients With Chronic Renal Failure Under Heamodialysis in North Kordfan State

تقييم مستوى الدهون في السودانين المرضى بالفشل الكلوي ويتعرضون للغسيل الدموي في ولاية شمال كردفان

A Dissertation submitted in partial Fulfillment for the requirement of M.Sc Degree Medical Laboratory science(Clinical Chemistry)

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Dedication

To the one who is dearer to me than myself:

Mohammad Basher

To me he is continuous source of help and strength ….

To my father

To my sister and brother

To my children

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first and foremost, thanks to Allah, forgive me strength and patience

to carry out this work.

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Finally, Yet importantly ,I would like to express my heartfelt thanks to my beloved parents for their blessings,my friends for their help ,and everyone who helped and encourage me in this work.

Abstract

Background:. Renal failure is a condition in which the kidneys fail to remove metabolic end-products from the blood and regulate the fluid, electrolyte, and pH balance of the extracellular fluids. Dyslipidemias is a very common complication of Chronic Renal Failure (CRF).

Objective: This study done to assess the lipid profile among end stage renal disease under heamodialysis.

Material and method: This study include 100 participants (50 patients on heamodialysis and 50 controls) was carried out in Elobeid Teaching Hospital Aljmeih Renal center) during September 2019 to December 2019.

Three ml of blood was taken from each volunteerson lithium heparin to prepare plasma,the plasma was used to measure total cholesterol,triglycerides,high density lipoprotein and low density lipoprotein, using spectrophotometer (semi automation instrument),and results were analyzed using statistical package for social science (SPSSversion21).

Result: The result shows significant increase of total cholesterol, LDL-C and Triglyceride in pre heamodialysis versus control group while HDL-C (mean±SD): insignificantly decrease

159.52±44.62versus 127.00±38.78mg/dl, P- value=0.000(TC)

78.02±32.64 versus 61.20±20.51mg/dl and p- value 0.003(LDL-C)

151.16±49.73 versus106±25.88,P-value 0.000(TG) .

42.92±9.74 versus 47.18±12.30mg/dl,P value0.004, (HDL-C),

Also The result show significant increase of total cholesterol, LDL-C and Triglyceride in postheamodialysis versus control group while HDL-C (mean±SD): insignificantly decrease

(159.52±38.75 versus127.0025.28 mg/dl ,p-value0.000 (TC)

.(71.38±31.85 versus 61.20±20.51mg/dl, p-value 0.060)LDL-C

,(196.54±72.63 versus 106.56±25.8mg\dl8, p –value0.000).TG

(45.74±12.85, versus47.18±12.30 mg/dl P- value0.005)HDL-C

Also result show there was significant increase in TG and effect in HDL-C , LDL-c and cholesterol in postheamodialysis compare to pre heamodialysis mean ±SD

,(42.92±9.74 versus 45.74±12.85 mg\dl,and p-value0.029) (HDL-C

,(151.16+-49.73 versus196.54 ±72.63 mg\dl ,p value=0.000).TG

(159.52±44.64 versus 159.52±38.78mg/dl ,p-value1.000)(TC)

,(78.02±32.64 versus71.3831.85 mg\dl ,p-value =0.138) LDL-C

Result also showed no correlations between T.cholesterol, HDL-C, LDL-C and Triglyceride in preheamodialysis and duration of disease.

(r=0.032, p=0.2). TC

(r=-0.08 ,p=0.5). HDL-C

(r=0.15 , p=0.29). LDL-C

(r=-0.06 ,p=0.60). TG

Result also Shows no correlations between T.cholesterol, HDL-C, LDL-C and Triglyceride in post heamodialysis and duration of disease

(r=0.1, p=0.40). TC

(r=-0.22 ,p=0.4). HDL-C

(r=0.25 , p=0.07). LDL -C

(r=-0.13 ,p=0.36).TG

Result also showed significant increase in TG\HDL-c ratio in casegroup compare to control group and before and after dialysis.

3.85±2.53 p-value 0.04 (pre heamodialysis)

4.64±2.38 p-value0.001 (post heamdialysis)

3.8±2.53 ,4.6±3.38 .P-value= 0.00). in post heamdialysis compared to pre heamodialysis.

Conclusion: From this study it can be concluded that plasma (TC, LDL and TG were significant rise in pre and post heamodialysis patients compared to control

Also showed TG\HDL-C ratio increasedin case group compare to control group and postheamodialysis compared to pre heamodialysis patients.

ملخص البحث :

المقدمة :

الفشل الكلوي هوحالةتفشل فيهاالكليتان في إزالةالمنتجات النهائيةالأيضيةمن الدم وتنظيم توازن السوائل ودرجةالحموضة في السوائل خارج الخلية. خلل نسبة الدهون في الدم احد مضاعفات الفشل الكلوي المزمن.

الهدف:أجريت هذه الدراسة لتقييم مستوي الدهون بين مرضي الفشل الكلوى اللذين يخضعون للغسيل الدموي .

الموادوالاساليب:شملت هذه الدراسة 100 مشارك (50 مريضاعلى الغسيل الدموي50عنصر تحكم) تم إجراؤها في مستشفى الابيض التعليمي في مركزالجميح للغسيل الكلوي خلال الفترةمن سبتمبر 2019 إلى ديسمبر 2019.

تم أخذ 3 مل من الدم من كل متطوع لإعداد البلازما،وتم استخدام البلازما لقياس الكولسترول الكلي والبروتين الدهني منخفض الكثافة وثلاثي الجلسريدالبروتين الدهني عالي الكثافة،باستخدام مقياس اسبكترو فوتو ميتر (أداةشبه كاملة الآلية) ،وتم تحليل النتائج باستخدام برنامج التحليل الاحصائي .

النتائج :تشير نتائج هذه الدراسة الي زياده ذو دلالة احصائيه في مستوي الكولسترول الكلي والبروتين الدهني منخفض الكثافة وثلاثي الجلسريد بين اللذين يعانون من الفشل الكلوي قبل الغسيل الدموي مقارنة بالوضع الطبيعي .بينما نقصان ليس ذو دلالة احصائية في مستوي البروتين الدهني عالي الكثافة (المتوسط الانحرافي المعياري)

159.52±44.62(مقابل , 127.00±38.78mg/dl القيمة الاحتمالية 0.000=(الكلسترول الكلي)

78.02±32.64مقابل 61.20±20.51mg/dl and القيمة الاحتمالية 0.003 (البروتين الدهني منخفض الكثافة)

49.73±16.مقابل, 106±25.88 القيمة الاحتمالية . 1510.000 (ثلاثي الجليسريد)

42.92±9.74مقابل,47.18±12.30mg/dl القيمة الاحتمالية ,058.(بروتين دهني عالي الكثافة)

وايضا اظهرت الدراسة الي زياده ذو دلالة احصائية في مستوي الكولسترول الكلي والبروتين الدهني منخفض الكثافه وثلاثي الجلسريد بين اللذين يعانون من الفشل الكلوي بعد الغسيل الدموي مقارنة بالوضع الطبيعي .بينما نقصان ليس ذو دلالة إحصائية في مستوي البروتين الدهني عالي الكثافة (المتوسط الانحرافي المعياري)

159.52±38.75)مقابل,127.0025.28 mg/dl القيمة الاحتمالية000. (الكولسترول الكلي 31.85±71.38).(مقابل ,61.20±20.51mg/dl القيمة الاحتمالية 0.060 (البروتين الدهني منخفض الكثافة)

106.56±25.8mg\dl8, p –value0.000).TGمقابل,(196.54±72.63

,45.74±12.85)مقابل 47.18±12.30 mg/dlالقيمة الاحتمالية 0.05 (بروتين دهني عالي الكثافة)

وايضا اظهرت الدراسة زياده ذو دلالة احصائية في مستوي ثلاثي الجليسريد وايضا يوجد تاثير علي مستوي مستوي الكولسترول الكليوالبروتين الدهني منخفض الكثافة مستوي البروتين الدهني عالي الكثافة (المتوسط الانحرافي المعياري)

42.92±9.74),مقابل ,45.74±12.85 mg\dl القيمة الاحتمالية (0.029بروتين دهني عالي الكثافة)

151.16+-49.73), مقابل 196.54 ±72.63 mg\dlالقيمة الاحتمالية .(0.000=ثلاثي الجلسريد)

159.52±44.64) مقابل, 159.52±38.78mg/dl القيمة الاحتمالية (1.000الكولسترول الكلي)

78. 02±32.64),مقابل, 71.3831.85 mg\dl القيمة الاحتمالية (0.138= البروتين الدهني منخفض الكثافة)

وايضا اظهرت النتائج الي زياده ذو دلالة احصائية في نسبة ثلاثي الجليسريد الي البروتين الدهني مرتفع الكثافة في الاشخاص المرضي (قبل وبعد الغسيل)مقارنة بعنصر التحكم

3.85±2.53القيمة الاحتمالية 0.04 (قبل الغسيل الدموي)

4.64±2.38القيمة الاحتمالية 0.001 (بعد الغسيل الدموي)

وايضا اظهرت النتائج الى زيادة ذو دلالة احصائية في بالنسبة ثلاثي الجلسريد الى البروتين الدهني في الاشخاص المرضى (قبل وبعد الغسيل الدموي) .

3.8±2.53, 4.6±3.38. القيمة الاحتمالية 0.00 (قبل الغسيل الدموي مقارنة مع بعد الغسيل الدموي)

الخلاصة : من خلال هذه الدراسة تبين ان هناك زياده ذات دلالة احصائيةفي مستوي الكولسترول الكلي والبروتين الدهني منخفض الكثافة وثلاثي الجلسريد بين اللزين يعانون من الفشل الكلوي قبل وبعد الغسيل الدموي مقارنة بالوضع الطبيعي

وايضا اظهرت النتائج الي زياده ذو دلالة احصائية في نسبه ثلاثي الجليسريد الي البروتين الدهني مرتفع الكثافة في الاشخاص المرضي (قبل وبعد الغسيل)مقارنة بعنصر التحكم وبعد الغسيل الدموي مقارنة بقبل الغسيل الدموي .

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List of Abbreviations

ASCVD	Atherosclerotic Cardiovascular Disease
CVD	Cardiovascular disease
CKD	Chronic Kidney Disease
CRF	Chronic Renal Failure
CT	Computed Tomograghy
CHD	Continuous Heamodialysis
ESRD	End Stage Renal Disease
HD	Heamodialysis
HDL	High Density Lipoprotein
IgA	Immunoglobulin A
LP	Lipoprotein
MHD	Maintenance Heamodialysis
TC	Total Cholesterol
VLDL	Very Low Density Lipoprotein

CHAPTER ONE
INTRODUCTION

Rationale

Objective

INTRODUCTION

1.1 introduction

Renal failure is a condition in which the kidneys fail to remove metabolic end-products from the blood and regulate the fluid, electrolyte, and pH balance of the extracellular fluids. Chronic renal failure commonly defined as a sustained reduction in kidney function or evidence of kidney damage present for 3 months or longer(William , Stephen ,2008)

The underlying cause may be renal disease, systemic disease, or urologic defects of non renal origin. Renal failure can occur as an acute or a chronic disorder. Acute renal failure is abrupt in onset and often is reversible if recognized early and treated appropriately. In contrast, chronic renal failure is the end result of irreversible damage to the kidneys. It develops slowly, usually over the course of a number of years. Chronic renal failure is a devasting disease with clinical, economic and ethical dimensions and is emerging as a major public health problem globally(William , Stephen ,2008) .

Dyslipidemias is a very common complication of Chronic Renal Failure (CRF). Disturbances in lipoprotein metabolism are evident even at the early stages of CRF and usually follow a downhill course that parallels the deterioration in renal function. Recently published studies indicate that dyslipidemias in these patients may actively participate in the pathogenesis of Cardiovascular disease (CVD) as well as in the deterioration of renal function(Vaziri 2003). The characteristic lipid abnormalities seen in CRF patients are elevated triglycerides, normal/reduced total cholesterol (TC), decreased High Density Lipoprotein (HDL), normal Low Density Lipoprotein (LDL). Progressive (Amin, *etl* 2006). CRF not only leads to End stage renal disease (ESRD),

but it is associated with high cardiovascular morbidity & mortality. In fact, patients with CRF are much more likely to die because of dyslipidemias than to progress to ESRD (Brosnahan, and Fraer.2010) .With the implication of plasma lipids in the pathogenesis of atherosclerosis and ischemic heart disease, it becomes worthwhile to study the behavior of various lipid fractions in CRF patientsCVD constitutes the major cause of death in patients with ESRD and it is still higher in hemodialysis patients than in post transplantation patients.ESRD Patients on hemodialysis have abnormalities in lipoprotein structure and metabolism and have a high incidence of cardiovascular diseases(Ravichandran, *et al*1983).

1.2 Rationale

Chronic renal failure patient is at risk of CVD due to elevation of various form of lipids.CRF is complicated bycharacteristic dyslipidemias.Cardiovascular disease (CVD) is the leading cause of death among patients with chronic and end-stage renal disease. This problem has been rather poorly studied in various cardiovascular studies, particularly the pattern of dyslipidemia and the factors contributing to these abnormalities are different among patients with renal insufficiency, severe renal failure, those on hemodialysis.therefore clarify the impact of chronic renal failure in lipid profile and the benefits of establishing appropriate lipid-lowering therapy. This study was done to know the burden and the type of lipid dysfunction in our Maintenance Heamodialysis MHD patients to adopt appropriate measures todecrease CVD mortality in this population.In Sudan to my knowledge There is no published study about lipid profile in CRF patients.

1.3Objectives.

1.3.1genral objective

- To Assess plasma lipid profile among end stage renal disease under heamodialysis

1.3.2spesific objective

- To measure and compare total plasma cholesterol, high density lipoprotein, low density lipoprotein and triglyceride in pre and post heamodialysis patients and control
- To study the effect of haemodialysis on total plasma cholesterol, high density lipoprotein, low density lipoprotein and Triglycerides.
- To correlate between total plasma cholesterol, high density lipoprotein and low density lipoprotein,triglyceride and duration of disease.
- To compare the TG/HDL-C ratio pre and post heamodialysis

Literature review

2.1Renal

Located on the left and right in the kidneys are two bean-shaped organs found in vertebrates. They are retroperitoneal space, and in adult humans are about 11 centimeters (4.3 in) in length. They receive blood from the paired renal arteries; blood exits into the paired renal veins. Each kidney is attached to a ureter, a tube that carries excreted urine to the bladder.(Cotran,*et al*2005).

The nephron is the structural and functional unit of the kidney. Each human adult kidney contains around 1 million nephrons, while a mouse kidney contains only about 12,500 nephrons. The kidney participates in the control of the volume of various body fluid compartments, fluid osmolality, balance, various electrolyte concentrations, and removal of toxins. Filtration occurs in the glomerulus: one-fifth of the blood volume that enters the kidneys is filtered. Examples of substances reabsorbed are solute free water, sodium, bicarbonate, glucose, and amino acids. Examples of substances secreted are hydrogen, ammonium, potassium and uric acid. The kidneys also carry out functions independent of the nephron. For example, they convert a precursor of vitamin D to its active form, calcitriol; and synthesize thehormones erythropoietin and renin(Cotran,et al2005).

Renal physiology is the study of kidney function. Nephrology is the medical specialty which addresses diseases of kidney *function*: these include chronic kidney disease, nephritic and nephrotic syndromes, acute kidney injury, and pyelonephritis. Urology addresses diseases of kidney (and urinary tract) *anatomy* these include cancer, renal cysts, kidney stones and ureteral stones, and urinary tract obstruction.(Cotran,*et al*2005

2.1.1 Epidemiology of ESRD in the Developing

The exact number of patients with chronic renal failure requiring RRT in the developing world is not known. Unlike the developed world, most developing countries lack renal registries. Therefore, the exact incidence and prevalence of ESRD in the population, its burden on the health care system, and the outcome of these patients are not known Glomerulo nephritis is the most common cause of ESRD in India and Pakistan. (Barsoum,2002). It is similar in countries within the same World Bank Low- economy category and less prominent in countries in the Medium-economy category, ranging from about 11% in Egypt and Argentina to 28% in Saudi Arabia. Proliferative glomerulonephritis constituted the major bulk of primary etiology in contrast to its remarkably low frequency in the developed world. Focal and segmental glomerulosclerosis is the second most common lesion in Africa. Amyloidosis was reported as a fairly common glomerulopathy with a prevalence ranging from 6.1% to 10.3% of all glomerular lesions. (Barsoum ,2002). İt may be attributed to chronic infection as tuberculosis in India, schistosomiasis in Egypt, or to familial Mediterranean fever in North Africa. Interstitial nephritis accounted for 2.4% to 20% of reported ESRD: the highest prevalence being encountered in India and Pakistan (Barsoum ,2002) In the developing world, diabetic nephropathy that is increasing over the years, constitutes more than 25% of the dialysis population. It appears to be higher in Latin America and India than in Africa. Diabetes ranged from 9.1% inEgypt to 29.9% in Thailand. Hypertensive nephrosclerosis accounted for 13% to 21% of reported ESRD(Barsoum 2002). But the range is much wider in other reports, spanning between 4% in the Sudan, and 43% in Nigeria(Ojogwu ,1990). The mean age of ESRD patients requiring dialysis in most developing

countries is much lower, 32 to 42 years, than that in the developed world, 60 to 63 years. Among the reasons for this difference are the delay in detecting renal disease and the failure to institute controlling and preventive measures in patients with progressive renal failure, both of which result in faster deterioration of renal function and progression to ESRD. Patients aged under age 18 years constitute about 3% in Egypt and Thailand, 12% in Mexico and South Africa. There is a significant male preponderance in developing countries that varying from about 52% in Thailand to 68% in Egypt and up to 80% in India.

In sudan the causes of ESRD in the presents are hypertension, obstructive nephropathy, chronic glomerulonephritis, analgesic nephropathy, polycystic kidney disease and reno vascular disease, while Osman *et al* and Abboud *et al* reported in 1987 and 1989, respectively, that the causes of most of the CKD in Sudan are chronic glomerulonephritis and renal calculi. Both studies were performed in Khartoum. Osman *et al* reported that only one-fifth of the patients in Sudan have controlled blood pressure; this may explain, possibly, you finding of hypertensive nephropathy as the leading cause of ESRD. In other study, also found that obstructive nephropathy was the second most common cause of ESRD(Mudawi, 2008), explained by the delay in the diagnosis and management. The prevalence of infection with *S.mansoni* in endemic areas such as the Gezira regions may reach up to 70%. Mustafa et al stated that the incidence of malaria in the Gezira regions (Elhosh) is episodes per 1000 population.The high incidence of schistosomiasis and malaria may contribute to the high prevalence of chronic glomerulonephritis. Because of the late presentation of patients when ESRD has already developed resulting in the inability to diagnose the cause, they found 53.57% of the cases of unknown etiology; this may

reflect the lack of awareness of medical problems, lack of medical facilities in rural areas and/or delay in referral before arriving to the specialist physician.

2.1.2 Epidemiology of ESRD in the Developed World

The largest group of patients falls in the 45 to 64- year age group. The disease was more common in men than women. Black race constituted 30% to 32% of treated ESRD patients in contrast to 12.6% of the US population. Japan and the USA recorded higher rates than European countries, The incidence and prevalence of RRT for ESRD has continued to increase through the world, but at rates that vary considerably between countries. Reasons for this increase are likely to be an actual increase in the Occurance of chronic kidney disease improved survival from other diseases (so called competitive risk) and wider acceptance criteria for RRT, more elderly patients, patients with diabetes, and patients with several other co-morbidities (malignancies, systemic diseases etc) requiring (Locatelli*et al*,2002) The most obvious trends are the rapid increase in RRT incidence in the older patients and in patients with diabetes mellitus. These changes are due in large part to an increase in these population groups in the community. However, improved access to RRT will also have contributed to the increase in incidence observed. This increase was greater in men than in women, 5.2 vs 4.0 %/year. In most countries, the incidence rate remained stable for those younger than 45 years, rose by 2.2% on average in the 45-64 years' age group and by 7.0% among those 65- 74 years and tripled over the decade in those 75 years.

The major causes of ESRD in developed countries are diabetes and hypertension, which together account for almost 60% of dialysis patients., followed by, glomerulonephritis, others (interstitial nephritis, vasculitis,

eg), unknown, cystic diseases and urological diseases (Am J Kidney RDS 1998).

2.1.3chronic renal failure

Unlike acute renal failure, chronic renal failure represents progressive and irreversible destruction of kidney structures. As recently as 1965, many patients with chronic renal failure pro- gressed to the final stages of the disease and then died(Daelemans ,*et al* 2001).

Chronic renal failure can result from a number of conditions that cause permanent loss of nephrons, including diabetes, hypertension, glomerulonephritis, and polycystic kidney disease.Typically, the signs and symptoms of renal failure occur gradually and do not become evident until the disease is far advanced. This is because of the amazing compensatory ability of the kidneys. As kidney structures are destroyed, the remaining nephrons undergo structural and functional hypertrophy,each increasing its function as a means of compensating for those that have been lost. It is only when the few remaining nephrons are destroyed that the manifestations of renal failure become evident.

Symptoms of ChronicKidney Disease include: Fluid retention causing breathlessness and swelling of ankles and feet, Headaches Poor memory and concentration IrritabilitySleep disturbances,restless legs Itchiness Loss of appetite and nausea. Weight loss(Daelemans ,*et al* 2001)

2.1.3.1Laboratory investigation of renal failure:

-Urine analysis

-BUN

- Serum creatinine

- Creatinine clearance

-cystatin C

- Electrolyte

-total protein and albumin

-Protein/creatinine ratio

-Albumin/creatinine ratio

A range of imaging modalities including ultrasound,computed tomography (CT), magnetic resonance imaging (MRI) and isotope scanning can detect a number of structural abnormalities including polycystic kidney disease, reflux nephropathy, chronic pyelonephritis and Reno vascular disease. Renal biopsy histopathology is most useful in defining underlying glomerular disease such as immunoglobulin A (IgA) nephropathy or focal glomerulosclerosis.

2.1.3.2 Treatment of chronic renal failure:

The Greek word "dialysis" means "dissolution". The word "dia" means "through" and the word "lysis" means "loosening or splitting". Dialysis is the process for removing waste and excess water from blood, and is used primarily to provide an artificial replacement for the lost kidney function in people with renal failure. 2Dialysis works on the principles of diffusion (waste removal) and ultrafiltration (fluid removal) across a semipermeable membrane.

2.1.3.3.Hemodialysis:

Maintenance hemodialysis is the support of treatment for the patients with CRF who are waiting for, or who are not suitable to undergo renal. transplantation. Adequate dialytic treatment has prolonged the survival of patients with quality of life. This improved quality and extended duration

of life, by hemodialysis treatment. (Daelemans .,*et al* 2001).

2.1.3.4peritoneal dialysis: In contrast to hemodialysis, peritoneal dialysis, filters the blood inside the body. It uses the peritoneum or peritoneal membrane as the filter for dialysis. The peritoneum is a

membrane that lines the wall of the abdomen and covers the abdominal organs

2.1.3.5kidney transplant: kidney transplantation the treatment of choice for many patients with chronic renal failure. The availability of donor organs continues to limit the number of transplantations performed each year. Donor organs are obtained from cadavers and living related donors (*e.g.*, parent, sibling). The success of transplantation depends primarily on the degree of histocompatibility, adequate organ preservation, and immunologic management(Saland,Ginsberg,2007).

2.2plasma Lipids:

Lipids are hydrophobic molecules with low solubility in water,while lipoproteins are afamily of structurally similar particles,(Saland,Ginsberg,2007).

consisting of large spherical complexes that function as transport vehicles for lipids in blood.(Saland,Ginsberg,2007).(Rader,Hobbs,2007).These substances also carry fat-soluble vitamins,drugs,andantioxidantenzymes(Mahley,Weisgraber,Bersot,2008). They have a central hydrophobic core, hydrophilic surface monolayer consisting of proteins, free cholesterol and phospholipids. (AttmanPO,SamuelssonO 2009). High densitylipo- protein (HDL) are the smallest and densest lipoproteins while the chylomicrons and very low-density lipoproteins (VLDL) are the largest and leastdense. Their density is inversely related to size (Malloy,Kane,2006). Most triglycerides are transported in chylomicrons and VLDL, while most cholesterol is transported in the form of esterified cholesterol in low- density lipoprotein(LDL) and HDL.(Malloy,Kane2006),(Kwan *et al* 2007). About 70% of total plasma cholesterol is inLDL-

C.(Mahley,2008).Lipoproteins are synthesised mainly in the liver and intestines.(prop)

Function of lipids: -serving as hormone to facilitate communication. (e. g cholesterol), energy source (e.g. triglycerides) act as structural component of cell and cell membranes. (phospholipids and cholesterol,act as vitamins to regulate physiological process in the body(vit.D3).(Mahley,2008).

2.2.1Lipid profile.

The lipid profile is group of tests that are often ordered together to determine risk of coronary heart disease. They are tests that have been shown to be good indicators of whether someone is likely to have a heart attack or stroke caused by blockage of blood vessels or hardening of the arteries.The lipid profile typically includes:(Total cholesterol, High density lipoprotein, Low density lipoprotein, Triglyceride, very low-density lipoproteins)

Cholestrol is vital substance that your body uses to produce such things as digestion –aiding material, hormones and cell membrane. it is both produce by the body and absorbed from some of the foods you eat.cholestrol and triglycerides are transported in the blood by combination of lipids and proteins called lipoproteins.HDLs ,the so called good or healthy cholesterol, are lipoproteins made mostly of protein and little cholesterol .HDLs can help to clear cholesterol deposits in blood vessels left by another blood component called low density lipoprotein or LDLs. Low density lipoprotein and very-low density lipoproteins VLDls are the so called bad cholesterol.unlike HDLs ,LDLs and VLDLs are high-cholestrol particles .While cholesterol is necessary for various(Malloy,Kane2006). bodily functions,too much cholesterol is

harmful, since,excess cholesterol can be deposited in blood vessel walls. These fat deposits can lead to atherosclerosis, Atherosclerosis is the buildup of fatty deposits called plaque on the inside of walls of arteries .Arteries are blood vessel that carry oxygen and blood to the heart , brain , and other part of the body .as plaque builds up in Arteries , the arteries gradually narrows and become clogged. as an artery becomes more and more narrowed,less blood can flow through .the artery may also become less elastic (hardening of arteries) .atherosclerosis is the main cause of a group of disease called cardiovascular disease of the heart and blood vessel. Or hardening of the arteries. High levels of triglycerides are also associated with an increased risk of heart disease(Malloy,Kane2006).

2.3Association of lipid with renal disease

Dyslipidemias in the CRF patients may actively participate in the pathogenesis of cardiovascular disease (CVD) as well as in the deterioration of renal function. (Vaziri, 2003).

The characteristic of lipid abnormalities seen in CRF patients are elevated triglycerides, normal/reduced total cholesterol (TC), decreased High Density Lipoprotein (HDL), normal Low Density Lipoprotein (LDL) ,(Brosnahan, and Fraer,2010) .

Progressive CRF not only leads to End stage renal disease (ESRD), but it is associated with high cardiovascular morbidity & mortality. In fact, patients with CRF are much more likely to die because of dyslipidemias than to progress to ESRD, (Brosnahan, and Fraer, 2010) With the implication of plasma lipids in the pathogenesis of atherosclerosis and ischemic heart disease, it becomes worthwhile to study the behavior of various lipid fractions in CRF patients(Ravichandran, *et al* 1983).CVD constitutes the major cause of death in patients with ESRD and it is still higher in hemodialysis patients than in post transplantation patients,

(Fauci ,1983).Patients on hemodialysis have abnormalities in lipoprotein structure and metabolism and have a high incidence of cardiovascular diseases,(Cressman,1992).

The lipid abnormalities are very common complication of Chronic Renal Failure (CRF). Disturbances in lipoprotein metabolism are evident even at the early stages of CRF and usually follow a downhill course that parallels the deterioration in renal function.

The main lipid metabolism abnormalities seen in renal patients in these stages are hypertriglyceridemia, a rise in triglyceride remnant-rich lipoproteins and lipoprotein a (Lp (a)) levels, and a decline in HDL-cholesterol levels. Moreover, of Total (T) cholesterol and LDL-cholesterol levels are generally at normal limits in stage 1-4 CKD patients, rise in LDL-cholesterol levels has been determined in patients with nephrotic syndrome reported by(Vaziri, 2006),(Tsimihodimos *et al*.2008).

The characteristic dyslipidemias observed in CRF patients also reported by Amin *et al*(K Amin, *et al* 2006), Vaziri, (Vaziri *et al* 2006)and Saland*etal* ,(Saland &Ginsberg2007).Hypertriglyceridemia is the most common plasma lipid abnormality in patients with renal failure, coexisting with cholesterol levels within the normal range. Tanaka S J et al. who observed a decrease in total cholesterol and in HDL-cholesterol and LDL-cholesterol in patients dialysed using polysulphone or cellulose membranes. TC and LDL-C was significantly lower in MHD patients as compared to healthy controls (p=0.0001)(Tanka *et al* 2000).

Abrass CKalso observed low total cholesterol, LDL-C and HDL-Cl in MHD patients compared to healthy controls.

Abrass CKhowed dyslipidaemia is highly prevalent in patients on maintenance haemodialysis (MHD), with predominance of the

atherogenic triad, i.e., hypertriglyceridemia, elevated VLDL and reduced HDLThis mimics the lipid abnormalities of metabolic syndrome, which accelerate the progression of atherosclerosis and increase the risk for cardiovascular mortality,(Abrass,2006).

The incidence of cardio vascular disease (CVD) is high in patients on hemodialysis, (Gowdak , 2007).

Cardiovascular disease is the leading cause of death in hemodialysis patients accounting for almost. Many atherosclerotic cardio vascular disease (ASCVD) risk factors are more prevalent in end stage renal disease (ESRD) than in the general population. Of the traditional risk factors for ASCVD in patients with ESRD, dyslipidemias may play a major role. Control of these risk factors may have a substantial impact in reducing the excessburden of CHD.(Muntner *et al*,2006).

During dialysis, the high flux biocompatible membrane may be responsible to remove cholesterol in post HD samples(Sperschneider *et al*1997) , The Post Hemodialytic total cholesterol levels are still high as compared to that of controls. Few factors are responsible to elevate the mean level of

serum total cholesterol. Among them upregulation of hepatic enzymes Hydroxy-3-Methylglutaryl- CoA reductase and cholesterol 7_ hydroxylase is important.Heavy proteinuria in CRF patients can lead to upregulation of HMG CoA reductase. In addition LDL receptor deficiency, may play a central role in the genesis of the associated hypercholesterolemia in CRF patients Shah B, Nair (Shah *et al*,1994) . Other common lipid abnormality observed in haemodialysis patients was low HDL-C levels. International Journal of Scientific and Research Publications, Volume 3, Issue 7, July 2013 1 ISSN 2250-315,TG is increase ,and normal TC and LDL, low HDL. Results of earlier studies show that plasma total cholesterol is usually normal or reduced and

occasionally elevated in patients with CRF (Vaziri ,2006). Elevation of serum total cholesterol in CRF patients. Post hemodialytic samples of the CRF patients show a fall in serum total cholesterol, as compared to Pre hemodialytic samples. During dialysis, the high flux biocompatible membrane may be responsible to remove cholesterol in post HD samples (Sperschneider , 1997) The Post Hemodialytic total cholesterol levels are still high as compared to that of controls.One of the principal characteristics of lipid metabolism alterations in CRF patients is hypertriglycerdemia, which reflect the balance between removal and production of triglyceride .

Lipoprotein lipase bound to the endothelium may be released by heparin which is given during HD process, and high flux biocompatible dialysis membrane may be responsible for increasing the lipoprotein lipase activity during dialysis (Sperschneider , 1997).

The CRF associated HDL abnormalities are marked by reduction of plasma HDL cholesterol. Impaired maturation of cholesterol ester-poor HDL-3 to cholesterol ester rich cardioprotective HDL-2.(Vaziri , Liang 2004) And other factors may be responsible for progressive decline in the level of HDL in CRF. Among them deficiency of lecithin cholesterol acytransferase (LCAT), hepatic lipase, potential increase of Cholesterol ester transfer protein (CETP) and acyl-CoA: cholesterol acyltransferase (ACAT) activity may contribute for diminished plasma HDL and HDL maturation in CRF,(Klin,1996), (Ayub ,1999). ApoA-I and ApoA-II constitute main structural constitutes of HDL. In addition, apoA- serves as the LCAT activator, whereas apoA-II hepatic lipase activator. The reduction in plasma concentration of these important constituents can, therefore, contribute to both diminished HDLC concentration and impaired HDLC function in CRF, (Painter,2005).

study concentration of HDLC is significantly reduced in prehemodialytic samples. After hemodialysis, we found an elevation in HDLC levels. Dialysis characteristics and heparin dose could be responsible factors for effect on HDLC level in hemodialysis,(Sperschneider , 1997).

, Journal of Scientific and Research Publications, Volume 3, page 560-570 July 2010 the pattern of dyslipedemia in ESRD patients on MHD showed hypertriglyceridemia, elevated LP-a and reduced HDL-c.

The serum triglyceride levels were found to be significantly higher in MHD patients as compared to control group. Similar hyper-triglyceridemia was also observed in several other studies including the Shah B, etal 1994), (Gomez Dumm ,*et al* 2001),(Pennell , *et al* 2006), (Longenecker *et al*,2002),The second most common lipid abnormality in our study was low HDL-c level as compared to healthy volunteers. HDL-C was similarly found to be low in MHD patients by Pennell P et al(Pennell , et al 2006) . Piperi C et al (Piperi *et al*, 2004), also reported significantly low HDL-c level in their study. Total cholesterol, LDL-c, VLDL-c and chylomicrons were not significantly different between the patient and controlgroups.

Routine counseling and encouraging physical activity in MHD patients has potential to improve physical functioning, optimizing the quality of life,(Painter,2005) and possibly improving the plasma lipids and lipoprotein pattern. this study indicates that if we apply regular exercise program in our dialysis patients, we can achieve improvement in lipid andlipoprotein level,(Painter,2005).

CHAPTER THREE

MATERIALS AND METHODS

3-Materials and Methods

3.1Materials

3.1.1 Study design:

descriptive cross-sectional study.

3.1.2 Study area:

Elobeid Teaching Hospital during September 2019 to December 2019.

3.1.3Study Population:

All patients admitted to Aljmeih Renal center in Elobeid suffering from chronic renal failure on hemodailysis

3.1.4 Sample size:

One hundred ,50 patients on HD and 50 controls

3.1.5 Inclusion criteria

Patients with end stage renal disease on heamodialysis and healthy individual were included.

3.1.6 Excluding Criteria:

Exclude patients with factor that might affect blood lipids such as hepatic disease lipid lowering drug oral contraceptive DMs,Hypertensive patients,hypothyroidism ,alcoholism , and smoker.

3.1.7 Ethical consideration:

Samples will be collect from patients after telling them by the importance of research.

3.1.8 Data Collection:

Data were collected using structural interviewing Questionnaire which was designed to collect and maintain all valuable information concerning each case examined and laboratory data

3.1.9 Specimen collection:

Clean the area of collection by skin Cleansing Alcohol Wipe then insert the new syring in to vein then collected four ml from blood and transfer the blood in to plain container then centrifuge to obtain plasma

3.2 methodology

3.2.1Estimation of Total cholesterol:

Total serum cholesterol estimated by enzymatic spectrophotometric method (using Biosystem reagents)

3.2.1.1principles of T cholesterol

a) Cholestreol ester+ H2O in the present of cholesterol esterase→ cholesterol +fatty acid
b) Cholestreol+O_2+H2O in the present of cholestreol oxidase→cholestenone +H_2O_2
c) H_2O_2+4-Aminoantipyrine+phenol in the present of peroxidase →Quinoneimine +4-H2O

3.2.1.2. Procedure of T cholesterol : Appendix II

3.2.2 Estimation of HDL-C

High density lipoprotein was estimated by precipitation enzymatic spectrophotometric method (using Biosystem reagents)

3.2.2.1principle of **HDL-C:** Very low density lipoprotein and low density lipoprotein in the sample precipitate with phosphotungstate and magnesium ion. The supernatant contains high density lipoprotein, the HDL cholesterol is then measure by mean of coupled reaction describe above:

3.2.2.2 procedure of HDL:Appendix III

3.2.3Estimation of lowdensity lipoprotein:Appendix III

3.2.3.1principle of Low density lipoprotein :-

Low density lipoprotein was estimated by precipitation enzymatic spectrophotometric method (using Biosystem reagents)

Low density lipoproteins LDL in sample precipitate with polyvinyl sulphate their concentration is calculated from the difference between the serum total cholestreol and the cholestreol in the supernatant after centrifugation the cholesterol is spectrophotometrically measured by means of the coupled reaction described below

a) Cholestreol ester+ H_2O in the present of cholestreol esterase→ cholesterol +fatty acid
b) Cholestreol+O_2 +H_2O in the present of cholestreol oxidase→cholestenone +H_2O_2
c) H_2O_2+4-Aminoantipyrine+phenol in the present of peroxidase → Quinoneimine +4-H_2O

3.2.3.2 Procedure of LDL-C:appendix IV

3.2.4 Estimation Total Triglyceride

3.2.4.1Principle of Triglyceride

a) Triglyceride+H_2O in the present of lipase enzyme→glycerol +fatty acid
b) Glycerol +ATP in the present of glycerol kinase → glycerol-3-p+ADP
c) glycerol-3-p+O_2 in the present of G-3-p-oxidase →Dihidroxyacitone-p+H_2O_2
d) H_2O_2+4-Aminoantipyrine+phenol in the present of peroxidase →Quinoneimine +4-H_2O

3.2.4.2 procedure of triglyceride :Appendix V

3.3 quality control method

The precision and accuracy of all methods used in this study were checked by commercially prepared control sample before it is application for the measurement of test control samples.

3.4Statistical analysis

Data obtained from this study was analyzed using statistical package for the social science(SPSS version 21).paired sample T-test was used for comparison.

CHAPTER FOUR

RESULTS

RESULTS

The results obtained were illustrated in figure and tables as below:

Figure (4.1): Show gender distribution among end stage renal disease patients group.

Table (4.1):Show significant increase of total cholesterol, LDL-C and Triglyceride in preheamodialysis versus control group while HDL-C insignificantly decrease(mean ±SD) 159.52±44.62versus127.00±38.78mg/dl, P- value=0.000,78.02±32.64 versus 61.20±20.51mg/dl and p-value 0.003,151.16±49.73 versus25.88 ,P-value 0.000 and 42.92±9.74 versus 47.18±12.30mg/dl,P value0.058 respectively .

Table(4.2): Represent The comparison mean±SD of T.cholesterol, HDL-C, LDL-C and Triglyceride in post heamodialysis versus control group. The result showed (159.52±38.75 versus 127.00±25.28 mg/dl ,p-value0.000) ,(45.74±12.85, versus47.18±12.30 mg/dl P- value0.568).(71.38±31.85 versus 61.20±20.51mg/dl, p-value 0.060),(196.54±72.63 versus 106.56±25.8mg\dl8, p –value0.000). respectively.

Table (4.3): Represent The comparison mean ±SD of T.cholesterol, HDL-C, LDL-C and Triglyceride in preheamodialysis versus post heamodialysis The result showed (159.52±44.64 versus 159.52±38.78 mg/dl,p-value1.000) ,(42.92±9.74 versus 45.74±12.85 mg\dl ,and p-value0.029),(78.02±32.64 versus71.38±31.85 mg\dl ,p-value =0.138) ,(151.16±49.73 versus196.54±-72.63 mg\dl ,p value=0.000) respectively.

Table(4.4): shows correlations between T.cholesterol, HDL-C, LDL-C and Triglyceride in preheamodialysis and duration of disease

Correlation between T.cholesterol level and duration(r=0.032, p=0.2)

Correlation between HDL –C level and duration (r=-0.08,p=0.5) Correlation between LDL-C level and duration (r=0.15 , p=0.29)

Correlation betweenTriglyceride level and duration (r=-0.06 ,p=0.60)

Table (4.5): Shows correlations between T.cholesterol, HDL-C, LDL-C and Triglyceride in post heamodialysis and duration of disease

Correlation between T.cholesterol level and duration(r=0.1, p=0.40)

Correlation between HDL –C level and duration (r=-0.22,p=0.4)

Correlation between LDL-C level and duration (r=0.25 , p=0.07)

Correlation betweenTriglyceride level and duration (r=-0.13,p=0.36).

Table (4.6): Shows Triglyceride/HDL-c ratio between control and case group(pre heamodialysis) 2.65±2.56 ,3.85±2.53 ,p-value0.04

Table (4.7) : Shows mean Triglyceride/HDL-c ratio between control and case group (post heamodialysis) 2.65±2.56 ,4.64±2.38 ,p- value 0.001 .

Table (4.8): Represent The comparison mean±SD of TG\HDL-C ratio pre and versus post heamodialysis 3.8±2.53 ,4.6±3.38 .P-value= 0.00) respectively.

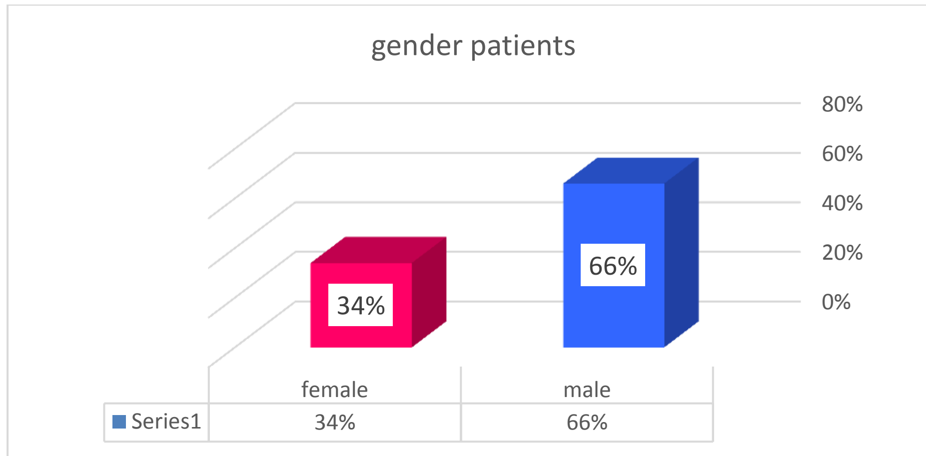

Figure (4.1): Gender distribution among end stage renal disease patients group

Table 4.1: Comparison between total cholesterol,HDL-C , LDL-C and TG in patients with renal failure (Preheamodialysis) with that of the control group:

Biochemical Parameter	**Pre HDN=50**	**Control N=50**	**P .value**
Total cholesterol mg//dl	159.52±44.62	127.00±25.28	.000
Serum HDLC mg/d	42.92±9.7	47.18±12.3	.004
Serum LDLC mg/dl	78.02±32.6	61.2±20.5	.003
Serum TG mg/dl	151.16±49.7	106.56±25.88	.000

The table show the mean±SD and p-Value .

p-Value ≤ 0.05 consider significant

Paired sample T-test used .

Table 4.2: Comparison between total cholesterol ,HDL-C , LDL-C and TG in patients with renal failure (Post heamodialysis) with that of the control group:

Biochemical Parameter	PostHD N=50 Mean±SD	Control N=50 Mean±SD	P .v
Total cholesterol mg//dl	159.52 ±38.78	127 ±25.2	.000
Serum HDLC mg/d	45.74 ±12.85	47.18 ±12.30	.005
Serum LDLC mg/dl	71.38 ±31.85	**61.2 ±20.51**	.060
Serum TG mg/dl	196.54 ±72.63	106.56 ±25.88	.000

The table show the mean±SD and p-Value .

p-Value ≤ 0.05 consider significant

Paired sample T-test used .

Table 4.3: Comparison between total cholesterol ,HDL-C , LDL-C and TG in patients with renal failure (pre and Post heamodialysis) with that of the control group:

Biochemical Parameter	**Pre HD** **N=50** **Mean±SD**	**Post HD** **N=50** **Mean±SD**	**P .v**
Total cholesterol mg//dL	159.52±44.62	159.52±38.785	1.000
Serum HDLC mg/d**L**	42.92±9.74	45.74±12.85	.029
Serum LDLC mg/dL	78.02±32.64	71.38±31.85	.138
Serum TG mg/dL	151.16±49.73	196.54±72.63	.000

The table show the mean±SD and p-Value .

p-Value ≤ 0.05 consider significant

Paired sample T- test used .

Table 4.4:correlations between total cholesterol ,HDL, LDL and triglyceride and duration of disease (preheamodialysis)

Variable	Coefficient	T.Cholesterol	HDL	LDL	Triglyceride
Duration of disease	R	0.032	-0.08	0.15	-0.06
	P	.20	0.5	0.29	0.60

Pearson correlation test was used

Is the strength of the correlation

P: Is the significance of the correlation(Values considered significant if≤.05)

Table 4.5:correlations between total cholesterol,HDL,LDL,and triglycrride and duration of disease(post heamodialysis)

Variable	**Coefficient**	**T.Cholesterol**	**HDL**	**LDL**	**Triglyceride**
Duration of disease	R	0.1	-0.22	0.25	-0.13
	P	.040	0.11	0.07	0.36

Pearson correlation test was used

R: Is the strength of the correlation

P: Is the significance of the correlation(Values consider≤0.05)

Table (4.6) : Shows Triglyceride/HDL-c ratio between control and case group(pre heamodialysis)

Biochemical Parameter	Pre HD Mean±SD	Control Mean±SD	P .v
Triglyceride/HDL-C ratio	3.85±2.53	2.65±2.56	0.04

Result given mean±SD ,P-valueindependent sample T-test was used for comparison. (p-Values considered significant if≤.05)

Table (4.7) : Shows mean Triglyceride/HDL-c ratio between control and case group(post heamodialysis)

Biochemical Parameter	Post HD Mean±SD	Control Mean±SD	P .v
Triglyceride/HDL-C ratio	4.64±2.38	2.65±2.56	0.001

Result given mean±SD ,P-valueindependent sample T-test was used for comparison. (P-Values considered significant if≤.05)

Table (4.8) : Shows mean Triglyceride/HDL-c ratio pre and post heamodialysis

Biochemical Parameter	Pre HD Mean±SD	Post HD Mean±SD	P .v
Triglyceride/HDL-C ratio	3.85±2.53	4.64±3.38	0.000

Result given mean±SD ,P-valueindependent sample T-test was used for comparison. (Values considered significant if≤0.05)

Chapter Five

Discussion, Conclusion and Recommendation

5.1 Discussion:

End stage renal disease patient are at risk for CVD due to elevation of various form of lipids.

This descriptive study was include 100 participants (50 patients with CRF, beside 50 healthy individuals as control) conducted in Aljmeh Renal center in Elobied during the period from September 2019 to December 2019 to estimate lipid profile .

In this study the mean levels of serum total cholesterol,and , TG and LDL-C were significantly increased in patients with CRF pre and post heamodilysis as compared to those of controls (p- value ≤ 0.05) this agree with studies done by (Amin *et al* 2006)**,** Vaziri *et al* (2006)and Saland *et al*2007) they said Hypertriglyceridemia is the most common plasma lipid abnormality in patients with renal failure, coexisting with cholesterol levels elevated. While value of HDL-C was found to be significantly reduced in pre and posthemodialytic samples as compared with controls with p value (P=0.005) .These finding supported the study conducted *by (Al Ameen Charitable Fund Trust, Bangalore* 2011)

But these increased levels of TG and LDL-C total cholesterol and degrease of HDL-c still lie in the reference range of the parameters because the CRF patients may under program by taking low fat diets advised by the clinician.

To study the effect dialysis on the level of lipids in the present study post hemodialytic samples mean value of TG significantly increasecompare with prehemodialytic samples (p value=0.000 this finding not support the study by. (Al Ameen 2011) also disagree with study by Tanaka et al and. Kalantar-Zadeh etJ Ayub Med Coll Abbottabad 2007;19(4)who observed a decrease in total cholesterol, HDL-cholesterol and LDL-cholesterol in

patients dialysed using polysulphone or cellulose membranes. TC and LDL-C was significantly lower.

The duration of dialysis has no effect on lipids profile in this study but the ratio of TG/HDL-c was significantly increase (p- value $\leq$ 0.05 and the mean ($\geq$ 3)in pre and postheamodialysis compare to control group and preheamodialysis compare with postheamodialysis this indicate the patients in this study risk for cardiovascular disease , this agree with (Reaven, M. D et al 2001) who propose that high TG:HDL, especially >3, indicates significant risk cardiovascular disease and TG:HDL ratios (>3) had the highest incidence of heart attacks and strokes, compared to those with the lowest ratio (<1.1), and the incidence of events was linear with the TG:HDL ratio also (Jeppesen , et al 1998).study showed triglycerides on their own to be another strong risk factor, but it found that stratifying triglyceride levels by HDL-c levels led to more accurate detection of increased risk of coronary disease.

5:2 Conclusion

From this study it can be concluded that plasma (TC and LDL and TG were rise in pre and post heamodialysis patients compared to control . And plasma TG and HDL-C were significant increase in post heamodialysis compare to pre heamodialysis patients .

Also the study show no correlation between T.cholesterol ,LDL-C,HDL-C , Triglyceride and duration of disease while TG\HDL-C ratio was significant increase in case group compare to control group.

Also showed TG\HDL-C ratio increased in post heamodialysis compared to pre heamodialysis patients.

5:3Recommendations:

- Laboratory investigation panel should include lipid profile for CRF patients.
- Dietary education and exercise program were importance for MHD patients, to prevent CVD mortality.
- Further study to investigate lipoproteins fractions should be doneto clarify there assoaciation with risk of CVD.

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Sudan University

Faculty of medical laboratory

Department of clinical chemistry

Questionnaire Assessment of lipids profile among Sudanese patients with chronic renal failure under heamodialysis in north kordofan state

1) Age :…………………………….

2) Gender :

   a- Male ☐ b- female ☐

3) Complaning of :

   a- hypertensive ☐ b- DM ☐

4) duration of disease………………………………..years

   a- two ☐ b- Three ☐

5) family history of dyslipidemias : a- Yes ☐ b- No ☐

6) Use of lipid lowering drug : a- Yes ☐ b- No ☐

8) Laboratory investigation

Total cholesterol :………………………………………………. mg/dl

Triglyceride:…………………………………………………….. mg/dl

HDL –C:…………………………………………………………….. mg/dl

LDL – C :…………………………………………………………….. mg/dl

Appendix (I)

Sudan University

Faculty of medical laboratory

Department of clinical chemistry

Questionnaire about effect of heamodialysis on lipid profile among chronic reanal failure

1) Age : ....

2) Gender :

a- Male ☐ b- femal ☐

3) Residence : a- Urban ☐ b- Rural ☐

4) job : ....

5) chronic disease :

a- hypertensive ☐ b- DM ☐

6) date of disease : ....

7) duration of dialysis perweek :

a- two ☐ b- Three ☐

8) family history of dyslipidemias : a- Yes ☐ b- No ☐

9) Use of lipid lowering drug : a- Yes ☐ b- No ☐

Total cholesterol :..................................

Triglyceride:..................................

HDL –C:..................................

LDL – C :..................................

Appendix (II)

COD 11805 1 x 50 mL	COD 11505 1 x 200 mL	COD 11506 1 x 500 mL	COD 11539 1 x 1 L
STORE AT 2-8ºC			
Reagents for measurement of cholesterol concentration Only for *in vitro* use in the clinical laboratory			

CHOLESTEROL

BioSystems
REAGENTS & INSTRUMENTS

CE

CHOLESTEROL
CHOLESTEROL OXIDASE/PEROXIDASE

PRINCIPLE OF THE METHOD

Free and esterified cholesterol in the sample originates, by means of the coupled reactions described below, a coloured complex that can be measured by spectrophotometry[1,2].

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{chol. esterase}} \text{Cholesterol} + \text{Fatty acid}$$

$$\text{Cholesterol} + \tfrac{1}{2}\,O_2 + H_2O \xrightarrow{\text{chol. oxidase}} \text{Cholestenone} + H_2O_2$$

$$2\,H_2O_2 + 4-\text{Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{peroxidase}} \text{Quinoneimine} + 4\,H_2O$$

CONTENTS

	COD 11805	COD 11505	COD 11506	COD 11539
A. Reagent S. Standard	1 x 50 mL 1 x 5 mL	1 x 200 mL 1 x 5 mL	1 x 500 mL 1 x 5 mL	1 x 1 L 1 x 5 mL

COMPOSITION

A. Reagent. Pipes 35 mmol/L, sodium cholate 0.5 mmol/L, phenol 28 mmol/L, cholesterol esterase > 0.2 U/mL, cholesterol oxidase > 0.1 U/mL, peroxidase > 0.8 U/mL, 4-aminoantipyrine 0.5 mmol/L, pH 7.0.

S. Cholesterol Standard. Cholesterol 200 mg/dL (5,18 mmol/L). Aqueous primary standard.

STORAGE

Store at 2-8ºC.

Reagent and Standard are stable until the expiry date shown on the label when stored tightly closed and if contaminations are prevented during their use.

Indications of deterioration:

- Reagent: Presence of particulate material, turbidity, absorbance of the blank over 0.200 at 500 nm (1 cm cuvette).
- Standard: Presence of particulate material, turbidity.

REAGENT PREPARATION

Reagent and Standard are provided ready to use.

ADDITIONAL EQUIPMENT

- Thermostatic water bath at 37ºC
- Analyzer, spectrophotometer or photometer able to read at 500 ± 20 nm

SAMPLES

Serum or plasma collected by standard procedures.

Cholesterol is stable for 7 days at 2-8ºC. Heparin, EDTA, oxalate and fluoride may be used as anticoagulants.

PROCEDURE

1. Bring the Reagent to room temperature.
2. Pipette into labelled test tubes: (Note 1)

	Blank	Standard	Sample
Cholesterol Standard (S)	—	10 µL	—
Sample	—	—	10 µL
Reagent (A)	1.0 mL	1.0 mL	1.0 mL

3. Mix thoroughly and incubate the tubes for 10 minutes at room temperature (16-25ºC) or for 5 minutes at 37ºC.
4. Measure the absorbance (A) of the Standard and Sample at 500 nm against the Blank. The colour is stable for at least 2 hours.

CALCULATIONS

The cholesterol concentration in the sample is calculated using the following general formula:

$$\frac{A_{Sample}}{A_{Standard}} \times C_{Standard} = C_{Sample}$$

If the Cholesterol Standard provided has been used to calibrate (Note 2):

$\frac{A_{Sample}}{A_{Standard}}$	x 200 = mg/dL cholesterol x 5.18 = mmol/L cholesterol

REFERENCE VALUES

The following uniform cut-off points have been established by the US National Cholesterol Education Program and have also been adopted in many other countries for the evaluation of coronary artery disease risk[3].

Up to 200 mg/dL = 5.2 mmol/L	Desirable
200-239 mg/dL = 5.2-6.21 mmol/L	Borderline High
> 240 mg/dL = > 6.24 mmol/L	High

QUALITY CONTROL

It is recommended to use the Biochemistry Control Serum level I (cod. 18005, 18009 and 18042) and II (cod. 18007, 18010 and 18043) to verify the performance of the measurement procedure.

Each laboratory should establish its own internal Quality Control scheme and procedures for corrective action if controls do not recover within the acceptable tolerances.

METROLOGICAL CHARACTERISTICS

- Detection limit: 3.3 mg/dL = 0.086 mmol/L
- Linearity limit: 1000 mg/dL = 25 mmol/L. For higher values dilute sample 1/2 with distilled water and repeat measurement.
- Repeatibility (within run):

Mean Concentration	CV	n
121 mg/dL = 3.13 mmol/L	1.1 %	20
257 mg/dL = 6.66 mmol/L	0.9 %	20

- Reproducibility (run to run):

Mean Concentration	CV	n
121 mg/dL = 3.13 mmol/L	1.9 %	25
257 mg/dL = 6.66 mmol/L	1.2 %	25

- Trueness: Results obtained with this reagent did not show systematic differences when compared with reference reagents (Note 2). Details of the comparison experiments are available on request.
- Interferences: Lipemia (triglycerides 10 g/L) does not interfere. Bilirubin (>10 mg/dL) and hemoglobin (>5 g/L) may affect the results. Other drugs and substances may interfere[4].

These metrological characteristics have been obtained using an analyzer. Results may vary if a different instrument or a manual procedure are used.

DIAGNOSTIC CHARACTERISTICS

Cholesterol is a steroid of high molecular weight and possesses the cyclopentanoperhydrophenanthrene skeleton. Dietary cholesterol is partially absorbed and it is also synthesized by the liver and other tissues. Cholesterol is transported in plasma by lipoproteins. It is excreted unchanged into bile or after transformation to bile acids.

Increased total cholesterol values are associated with a progressively escalating risk of atherosclerosis and coronary artery disease[5,6].

Clinical diagnosis should not be made on the findings of a single test result, but should integrate both clinical and laboratory data.

NOTES

1. This reagent may be used in several automatic analysers. Instructions for many of them are available on request.
2. Calibration with the provided aqueous standard may cause a matrix related bias, specially in some analyzers. In these cases, it is recommended to calibrate using a serum based standard (Biochemistry Calibrator, cod. 18011 and 18044).

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M11505i-20

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04/2011

Appendix (III)

COD 11579 20 mL

CHOLESTEROL LDL
PRECIPITATING REAGENT

BioSystems

COD 11648 50 mL
STORE AT 2-8ºC
Reagents for measurement of HDL cholesterol concentration Only for *in vitro* use in the clinical laboratory

CHOLESTEROL HDL
PRECIPITATING REAGENT

BioSystems
REAGENTS & INSTRUMENTS

CE

CHOLESTEROL HDL
PRECIPITATING REAGENT

PRINCIPLE OF THE METHOD

Very low density lipoproteins (VLDL) and low density lipoproteins (LDL) in the sample precipitate with phosphotungstate and magnesium ions. The supernatant contains high density lipoproteins (HDL). The HDL cholesterol is then spectrophotometrically measured by means of the coupled reactions described below[1,2].

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{chol. esterase}} \text{Cholesterol} + \text{Fatty acid}$$

$$\text{Cholesterol} + \tfrac{1}{2} O_2 + H_2O \xrightarrow{\text{chol. oxidase}} \text{Cholestenone} + H_2O_2$$

$$2\,H_2O_2 + 4-\text{Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{peroxidase}} \text{Quinoneimine} + 4\,H_2O$$

CONTENTS AND COMPOSITION

A. Reagent: 1 x 50 mL. Phosphotungstate 0.4 mmol/L, magnesium chloride 20 mmol/L.

S. HDL Cholesterol Standard: 1 x 5 mL. Cholesterol 15 mg/dL. Aqueous primary standard.

STORAGE

Store at 2-8ºC.

Reagent and Standard are stable until the expiry date shown on the label when stored tightly closed and if contaminations are prevented during their use.

Indications of deterioration:
- Reagent: Presence of particulate material, turbidity.
- Standard: Presence of particulate material, turbidity.

ADDITIONAL REAGENTS

These auxiliary reagents are to be used together with the Cholesterol Reagent contained in any of the BioSystems Cholesterol kits (cod. 11805, 11505, 11506, 11539).

REAGENT PREPARATION

Reagent and Standard are provided ready to use.

ADDITIONAL EQUIPMENT

- Desktop centrifuge.
- Thermostatic water bath at 37ºC.
- Analyzer, spectrophotometer or photometer able to read at 500 ± 20 nm.

SAMPLES

Serum or plasma collected by standard procedures.
HDL cholesterol in serum or plasma is stable for 7 days at 2-8ºC. Heparin, EDTA, oxalate and fluoride may be used as anticoagulants.

PROCEDURE

Precipitation

1. Pipette into labelled centrifuge tubes (Note 1):

Sample	0.2 mL
Reagent (A) (Cholesterol HDL kit)	0.5 mL

2. Mix thoroughly and let stand for 10 minutes at room temperature.
3. Centrifuge at a minimum of 4000 r.p.m. for 10 minutes.
4. Carefully collect the supernatant (Note 2).

Colorimetry

5. Bring the Reagent (Cholesterol kit) to room temperature.
6. Pipette into labelled test tubes: (Note 3)

	Blank	Standard	Sample
Distilled water	100 µL	—	—
HDL Cholesterol Standard (S)	—	100 µL	—
Sample supernatant	—	—	100 µL
Reagent (A) (Cholesterol kit)	1.0 mL	1.0 mL	1.0 mL

7. Mix thoroughly and incubate the tubes for 30 minutes at room temperature (16-25ºC) or for 10 minutes at 37ºC.
8. Measure the absorbance (A) of the Standard and Sample at 500 nm against the Blank. The colour is stable for at least 30 minutes.

CALCULATIONS

The HDL cholesterol concentration in the sample is calculated using the following general formula:

$$\frac{A_{Sample}}{A_{Standard}} \times C_{Standard} \times \text{Sample dilution factor} = C_{Sample}$$

If the HDL Cholesterol Standard provided has been used to calibrate (Note 4):

$\frac{A_{Sample}}{A_{Standard}}$	x 52.5 = mg/dL HDL cholesterol x 1.36 = mmol/L HDL cholesterol

REFERENCE VALUES

HDL cholesterol concentrations vary considerably with age and sex. The following cut-off point has been recommended for identifying individuals at high risk of coronary artery disease[3].

Up to 35 mg/dL = 0.91 mmol/L	High
> 60 mg/dL = > 1.56 mmol/L	Low

QUALITY CONTROL

It is recommended to use the Biochemistry Control Serum level I (cod. 18005 and 18009) to verify the performance of the measurement procedure.

Each laboratory should establish its own internal Quality Control scheme and procedures for corrective action if controls do not recover within the acceptable tolerances.

METROLOGICAL CHARACTERISTICS

- Detection limit: 3.0 mg/dL = 0.078 mmol/L.
- Linearity limit: 150 mg/dL = 3.9 mmol/L.
- Repeatability (within run):

Mean Concentration	CV	n
30 mg/dL = 0.78 mmol/L	3.3 %	20
55 mg/dL = 1.42 mmol/L	2.0 %	20

- Reproducibility (run to run):

Mean Concentration	CV	n
30 mg/dL = 0.78 mmol/L	4.2 %	20
55 mg/dL = 1.42 mmol/L	3.2 %	20

- Trueness: Results obtained with this reagent did not show systematic differences when compared with reference reagents (Note 4). Details of the comparison experiments are available on request.
- Interferences: Lipemia (triglycerides 10 g/L) does not interfere. Bilirubin (10 mg/dL) and hemoglobin (5 g/L) may intefere. Other drugs and substances may interfere[4].

These metrological characteristics have been obtained using an analyzer. Results may vary if a different instrument or a manual procedure are used.

DIAGNOSTIC CHARACTERISTICS

HDL play an important part in the removal of cholesterol from tissues and its transportation to the liver for removal as bile acids.

Decreased plasma HDL-cholesterol concentrations are positively correlated with the incidence of atherosclerosic diseases, basis of myocardial infarction and cerebrovascular accidents[5,6].

There are several disease states or environmental influences associated with reduced levels of HDL: acute or chronic hepatocellular diseases, intravenous hyperalimentation, severe malnutrition, diabetes, chronic anemia, myeloproliferative disorders, Tangier disease, analphalipopro-teinemia, acute stress, some drugs and smoking[5,6].

Clinical diagnosis should not be made on the findings of a single test result, but should integrate both clinical and laboratory data.

NOTES

1. Sample and Reagent A volumes may be varied as long as the same ratio is maintained.
2. Supernatant must be clear. When supernatant is turbid or the pellet floats, add again 0.5 mL of Reagent A, mix thoroughly and centrifuge. Multiply the obtained concentration by 1.7 (dilution).
3. These reagents may be used in several automatic analysers. Instructions for many of them are available on request.
4. Calibration with the provided aqueous standard may cause a matrix related bias, specially in some analyzers. In these cases, it is recommended to calibrate using a serum based standard (Biochemistry Calibrator, cod. 18011 and 18044).

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M11648i-21

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10/2013

Appendix (IV)

COD 11579 20 mL
STORE AT 2-8°C
Reagents for measurement of LDL cholesterol concentration Only for *in vitro* use in the clinical laboratory

CHOLESTEROL LDL
PRECIPITATING REAGENT

BioSystems
REAGENTS & INSTRUMENTS

CE

CHOLESTEROL LDL PRECIPITATING REAGENT

POLYVINYL SULPHATE / POLYETHYLENEGLYCOL

PRINCIPLE OF THE METHOD

Low density lipoproteins (LDL) in the sample precipitate with polyvinyl sulphate. Their concentration is calculated from the difference between the serum total cholesterol and the cholesterol in the supernatant after centrifugation[1]. The cholesterol is spectrophotometrically measured by means of the coupled reactions described below.

$$\text{Cholesterol esters} + H_2O \xrightarrow{\text{chol. esterase}} \text{Cholesterol} + \text{Fatty acid}$$

$$\text{Cholesterol} + \tfrac{1}{2} O_2 + H_2O \xrightarrow{\text{col. oxidasa}} \text{Cholestenone} + H_2O_2$$

$$2\,H_2O_2 + 4-\text{Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{peroxidasa}} \text{Quinoneimine} + 4\,H_2O$$

CONTENTS AND COMPOSITION

A. Reagent. 1 x 20 mL. Polyvinyl sulphate 3 g/L, polyethyleneglycol 327 g/L.

STORAGE

Store at 2-8°C.

Reagent is stable until the expiry date shown on the label when stored tightly closed and if contaminations are prevented during their use.

Indications of deterioration:

– Reagent: Presence of particulate material, turbidity.

ADDITIONAL REAGENTS

This precipitating reagent is to be used together with the Cholesterol Reagent contained in any of the BioSystems Cholesterol kits (cod. 11805, 11505, 11506, 11539).

REAGENT PREPARATION

Reagent is provided ready to use.

ADDITIONAL EQUIPMENT

– Desktop centrifuge

– Thermostatic water bath at 37°C

– Analyzer, spectrophotometer or photometer able to read at 500 ± 20 nm

SAMPLES

Serum collected by standard procedures.

LDL cholesterol in serum is stable for 24 hours at 2-8°C.

PROCEDURE

Precipitation

1. Pipette into labelled centrifuge tubes (Note 1):

Sample	0.2 mL
Reagent (A) (Cholesterol LDL kit)	0.2 mL

2. Mix thoroughly and let stand for 15 minutes at room temperature.
3. Centrifuge at a minimum of 4000 r.p.m. for 15 minutes.
4. Carefully collect the supernatant (Note 2).

Colorimetry

5. Bring the Reagent (Cholesterol kit) to room temperature.
6. Pipette into labelled test tubes: (Note 3)

	Blank	Standard	Sample
Distilled water	20 µL	—	—
Cholesterol Standard (S)	—	20 µL	—
Sample supernatant	—	—	20 µL
Reagent (A) (Cholesterol kit)	1.0 mL	1.0 mL	1.0 mL

7. Mix thoroughly and incubate the tubes for 30 minutes at room temperature (16-25°C) or for 10 minutes at 37°C.
8. Measure the absorbance (A) of the Standard and Sample at 500 nm against the Blank. The colour is stable for at least 30 minutes.

CALCULATIONS

The cholesterol concentration in the supernatant is calculated using the following general formula:

$$\frac{A_{\text{Sample}}}{A_{\text{Standard}}} \times C_{\text{Standard}} \times \text{Sample dilution factor} = C_{\text{Supernatant}}$$

If the Cholesterol Standard, provided in the Cholesterol kit, has been used to calibrate (Note 4):

$\frac{A_{\text{Sample}}}{A_{\text{Standard}}}$	x 200 x 2 = mg/dL cholesterol in supernatant x 5.18 x 2 = mmol/L cholesterol in supernatant

The LDL cholesterol concentration in the sample is calculated as follows:

LDL cholesterol = total cholesterol – cholesterol in supernatant

REFERENCE VALUES

The following uniform cut-off points have been established by the US National Cholesterol Education Program and have also been adopted in many other countries for the evaluation of coronary artery disease risk[2].

Up to 100 mg/dL = 2.59 mmol/L	Optimal
100-129 mg/dL = 2.59-3.34 mmol/L	Near optimal/above optimal
130-159 mg/dL = 3.37-4.12 mmol/L	Borderline High
160-189 mg/dL = 4.14 –4.90-mmol/L	High
> 190 mg/dL = 4.92 mmol/L	Very High

QUALITY CONTROL

It is recommended to use the Biochemistry Control Serum level I (cod. 18005, 18009 and 18042) and II (cod. 18007, 18010 and 18043) to verify the performance of the colorimetry with the cholesterol reagent.

Each laboratory should establish its own internal Quality Control scheme and procedures for corrective action if controls do not recover within the acceptable tolerances.

METROLOGICAL CHARACTERISTICS

– Detection limit: 0.45 mg/dL = 0.01 mmol/L

– Linearity limit: 1000 mg/dL = 26 mmol/L

– Repeatibility (within run):

Mean Concentration	CV	n
120 mg/dL = 3.11 mmol/L	1.6 %	20
200 mg/dL = 5.18 mmol/L	1.4 %	20

– Reproducibility (run to run):

Mean Concentration	CV	n
120 mg/dL = 3.11 mmol/L	2.8 %	25
200 mg/dL = 5.18 mmol/L	1.5 %	25

– Trueness: Results obtained with this reagent did not show systematic differences when compared with reference reagents (Note 4). Details of the comparison experiments are available on request.

– Interferences: Lipemia (triglycerides 10 g/L) does not interfere. Bilirubin (10 mg/dL) and hemoglobin (5 g/L) may intefere. Other drugs and substances may interfere[3].

These metrological characteristics have been obtained using an analyzer. Results may vary if a different instrument or a manual procedure are used.

DIAGNOSTIC CHARACTERISTICS

LDL is the main lipoprotein transporting cholesterol from liver to tissues.

Increased plasma LDL-cholesterol concentrations are positively correlated with the incidence of atherosclerosic diseases, basis of myocardial infarction and cerebrovascular accidents[4,5].

There are several disease states or environmental influences associated with increased levels of LDL-cholesterol: nephrosis, diabetes, obesity, some drugs and smoking[4,5].

Clinical diagnosis should not be made on the findings of a single test result, but should integrate both clinical and laboratory data.

NOTES

1. Sample and Reagent volumes may be varied as long as the same ratio is maintained.
2. Supernatant must be clear. When supernatant is turbid or the pellet floats, add again 0.2 mL of Reagent, mix thoroughly and centrifuge. Multiply the obtained concentration by 1.5 (dilution).
3. These reagents may be used in several automatic analysers. Instructions for many of them are available on request.
4. Calibration with the provided aqueous standard may cause a matrix related bias, specially in some analyzers. In these cases, it is recommended to calibrate using a serum based standard (Biochemistry Calibrator, cod. 18011 and 18044).

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M11579i-17

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08/2017

Appendix (V)

Réactifs pour mesurer la concentration de triglycérides
A utiliser uniquement *in vitro* dans les laboratoires cliniques

CE

TRIGLYCERIDES
GLYCEROL PHOSPHATE OXYDASE/PEROXYDASE

PRINCIPE DE LA METHODE

Les triglycérides présents dans l'échantillon donnent, selon les réactions décrites ci-dessous, un complexe coloré quantifiable par spectrophotométrie.[1,2]

$$\text{Triglycérides} + H_2O \xrightarrow{\text{lipase}} \text{Glycérol} + \text{Ac. gras}$$

$$\text{Glycérol} + ATP \xrightarrow{\text{glycérol kinase}} \text{Glycérol} - 3 - P + ADP$$

$$\text{Glycérol} - 3 - P + O_2 \xrightarrow{\text{G-3-P-oxidase}} \text{Dihydroxyacétone} - P + H_2O_2$$

$$2\,H_2O_2 + 4 - \text{Aminoantipyrine} + 4 - \text{Chlorophénol} \xrightarrow{\text{péroxidase}} \text{Quinoneimine} + 4\,H_2O$$

CONTENU

	CODE 11828	CODE 11528	CODE 11529
A Réactif S Etalon	1 x 50 mL 1 x 5 mL	4 x 50 mL 1 x 5 mL	2 x 250 mL 1 x 5 mL

COMPOSITION

A. Réactif: Pipes 45 mmol/L, acetate de magnésium 5 mmol/L, 4-chlorophénol 6 mmol/L, lipase > 100 U/mL, glycérol kinase > 1,5 U/mL, glycérol-3-phosphate oxydase > 4 U/mL, péroxydase > 0,8 U/mL,4-aminoantipyrine 0,75 mmol/L, ATP 0,9 mmol/L, pH 7,0.

S. Etalon de Triglycérides: Glycerol équivalent à 200 mg/dL (2,26 mmol/L) de trioléine. Etalon primaire en solution acqueuse.

CONSERVATION

Les réactifs et étalon doivent être conservés à 2-8°C. Bien refermer les flacons et éviter toute contamination lors de l'utilisation. Dans ces conditions ils resteront stables jusqu'à la date indiquée sur l'étiquette.

Indications de dégradation:

- Réactifs: Présence de particules, turbidité, absorbance du blanc supérieure à 0,150 à 500 nm (cuvette de 1 cm).
- Etalon: Présence de particules, turbidité.

PREPARATION DES REACTIFS

Réactif (A) et Etalon (S) sont prêts à l'emploi.

EQUIPEMENT SUPPLEMENTAIRE

- Bain thermostaté à 37°C.
- Analyseur, Spectrophotomètre ou photomètre pour lectures à 500 ± 20 nm.

ECHANTILLONS

Sérum ou plasma collecté par procédures normalisées.
Les triglycerides dans le sérum ou plasma sont stables 5 jours à 2-8ºC. L'héparine , l'EDTA, l'oxalate et le fluorure peuvent être utilisés comme anticoagulants.

PROCEDURE

1. Placer les réactifs à température ambiante.
2. Pipeter dans des tubes à essais: (Note 1)

	Blanc	Etalon	Echantillon
Etalon de Triglycérides (S)	—	10 µL	—
Echantillon	—	—	10 µL
Réactif (A)	1,0 mL	1,0 mL	1,0 mL

3. Bien agiter et incuber les tubes pendant 15 minutes à température ambiante (16-25°C) ou pendant 5 minutes à 37°C.
4. Lire l'absorbance (A) de l'Etalon et de l'Echantillon face au Blanc à 500 nm. La couleur est stable au moins 2 heures.

CALCULS

La concentration en triglycérides de l'échantillon est calculée selon la formule suivante:

$$\frac{A_{\text{Echantillon}}}{A_{\text{Etalon}}} \times C_{\text{Etalon}} = C_{\text{Echantillon}}$$

Si l'étalon Triglycérides du kit est utilisé pour étalonner (Note 2):

$\frac{A_{\text{Echantillon}}}{A_{\text{Etalon}}}$	x 200 = mg/dL triglycerides x 2,26 = mmol/L triglycerides

VALEURS DE REFERENCE

Les fourchettes de valeurs données ci-dessous ont été établies par l'US National Institutes of Health et ont été adoptées par plusieurs autres pays pour l'évaluation du risque[3].

Jusqu'à 150 mg/dL = 1,7 mmol/L	Normal
150-199 mg/dL = 1,70 – 2,25 mmol/L	Douteux
200 – 499 mg/dL = 2,26 – 5,64 mmol/L	Elevé
> 500 mg/dL = > 5,65 mmol/L	Très élevé

CONTRÔLE DE QUALITE

Il est recommandé d'utiliser les Sérums Contrôles de Biochimie niveau I (Code 18005, 18009 ou 18042) et II (Code 18007, 18010 ou 18043) pour vérifier la qualité de la méthodologie.

Chaque laboratoire doit établir ses propres protocoles et méthodes de Contrôle de Qualité interne afin d'apporter les modifications nécessaires en cas de dépassement des tolérances.

CARACTERISTIQUES METROLOGIQUES

- Limite de détection: 1,6 mg/dL = 0,018 mmol/L
- Limite de linéarité: 600 mg/dL = 6,78 mmol/L. Pour des valeurs supérieures diluer l'échantillon au 1/4 en eau distillée et répéter l'essai.
- Répétabilité (intrasérielle):

Concentration moyenne	CV	n
100 mg/dL = 1,13 mmol/L	1,7 %	20
245 mg/dL = 2,77 mmol/L	0,7 %	20

- Reproductibilité (intersérielle):

Concentration moyenne	CV	n
100 mg/dL = 1,13 mmol/L	2,6 %	25
245 mg/dL = 2,77 mmol/L	1,7 %	25

- Justesse: Les résultats obtenus avec ce réactif n'ont pas montrés de différences systématiques significatives par rapport aux réactifs de référence (Note 2). Les détails des études comparatives sont disponibles sur demande.
- Interférences: L'hémoglobine (10 g/L) n'interfère pas. La bilirubine (2,5 mg/dL) interfère. Certains médicaments et substances peuvent interférer[4].

Ces données ont été obtenues en utilisant un analyseur. Les résultats peuvent varier d'un instrument à l'autre ou en utilisant une technique manuelle.

CARACTERISTIQUES DIAGNOSTIQUES

Les triglycérides sont des esters de glycérol et des acides gras qui viennent du régime ou sont synthétisés par le foie. Les triglycérides sont transportés dans le plasma par les lipoprotéines et servent aux tissus adipeux, muscles et autres. Leur première fonction est de fournir de l'énergie aux cellules.

Les concentrations élevées de triglycérides peuvent être dues à une altération hepathique, diabète Mellitus, néphrose, hypothyroïde, alcoolisme, hyperlipoprotéinémie familiale IV, V ou autres.[3,5]

Le diagnostic clinique ne doit pas être basé sur les conclusions d'un test unique mais il doit intégrer l'ensemble des données cliniques et de laboratoire.

NOTES

1. Ces réactifs peuvent être utilisés dans la plupart des analyseurs automatiques. Demandez les information à votre distributeur.
2. L'étalonnage avec l'étalon acqueux fourni, peut entrainer des biais sur certains analyseurs. Dans ce cas il est recommandé d'étalonner l'appareil avec un sérum étalon (Calibrateur Biochimique, Code. 18011 ou 18044).

BIBLIOGRAPHIE

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4. Young DS. Effects of drugs on clinical laboratory tests, 5th ed. AACC Press, 2000.
5. Friedman and Young. Effects of disease on clinical laboratory tests, 4th ed. AACC Press, 2001.

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