DYSLIPIDEMIA AND ASSOCIATED FACTORS AMONG PATIENTS SUSPECTED FOR GASTRITIS AT JIMMA UNIVERSITY MEDICAL CENTER, JIMMA, SOUTHWEST ETHIOPIA.

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A THESIS TO BE SUBMITTED TO SCHOOL OF MEDICAL LABORATORY SCIENCE, FACULTY OF HEALTH SCIENCES, INSTITUTE OF HEALTH, JIMMA UNIVERSITY IN PARTIAL FULFILLMENT OF THE REQUIREMENT OF MASTER OF SCIENCE DEGREE IN CLINICAL LABORATORY SCIENCE SPECIALITY IN CLINICAL CHEMISTRY.

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SCHOOL OF MEDICAL LABORATORY SCIENCE

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ABSTRACT

Background: Dyslipidemia is a condition in which total cholesterol ≥200mg/dl, triglycerides ≥ 150mg/dl, low density lipoprotein cholesterol ≥130mg/dl, and/ or high-density lipoprotein cholesterol ≤ 40mg/dl. Colonization of the stomach by *Helicobacter pylori* causes chronic inflammation of the stomach wall which can change some biochemical parameters. On the association of *Helicobacter pylori* infection and its contributions to change in serum lipid profile, different studies reported inconsistent outcomes.

Objective: To assess prevalence of dyslipidemia and associated factors among patients suspected for gastritis at outpatient department of Jimma University medical center, Jimma, Ethiopia, from January 03 to April 05, 2019.

Method: A Hospital based cross- sectional study was conducted at Jimma University medical center on 369 gastritis suspected patients. The study subjects were selected by convenient sampling technique. Socio-demographic data were collected by structured questionnaire. 5ml of blood was collected from an overnight fasting subject. Data were edited, coded, and entered into Epidata version 3.1 and exported to statistical package for social sciences version 25 for analysis. Bivariate analysis was used to screen those variables which were candidates for multivariate analysis.

Result: From the total study subjects, 286 (77.5%) had at least one abnormality in lipid profile and 151/173(87.2%) of *H. pylori* positive patient had at least one abnormality in lipid profile. Our study demonstrated that there was significant increase of mean ± SD of TC, TG, and LDL-C in *H. pylori* positive patients than *H. pylori* negative patients (P-value < 0.05). After adjusting for traditional dyslipidemia risk factors, *H. pylori* infection was an independent predictor of dyslipidemia (AOR 2.628, 95%CI 1.477- 4.678, P = 0.001).

Conclusion and Recommendations: An increase in prevalence of dyslipidemia among *H. pylori* positive patients indicates *H. pylori* infected patients have a possibility of modified lipid profile, there for assessment of lipid profile in *H. pylori* infected patient is recommended.

Keywords *Helicobacter pylori* infection Lipid profile Ethiopia

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LIST OF ABBREVIATION AND ACRONYM

Abs :	Antibodies
ACD:	Acute Coronary Disease
AOR:	Adjusted Odds Ratio
BMI:	Body Mass Index
BP:	Blood Pressure
CagA:	Cytotoxin Associated geneA protein
CHD:	Coronary heart disease
COR:	Crude Odds Ratio
H. pylori:	*Helicobacter pylori*
H. pylori-NAP:	*H. pylori* neutrophil-activating protein
HC:	Hip circumference
HDL-C:	High Density Lipoprotein cholesterol
HTN:	Hypertension
IHD:	Ischemic Heart Disease
JUMC:	Jimma University Medical Center
LDL-C:	Low Density Lipoprotein cholesterol
LPS:	Lipopolysaccharide
PAP:	Para amino antipyrine Phenol
SOP:	Standard operating producers
SPSS:	Statistical Package for the Social Sciences
TC:	Total Cholesterol
TG :	Triglycerides
TLR:	Toll like receptors
VacA:	Vacuolating Associated Cytotoxin gene A
WC :	Waist circumference
WHO:	World Health Organization

OPERATIONAL DEFINITIONS

Adult: Person whose age is greater than or equals to 18 years.

Atherosclerosis: Formation of cholesterol deposit on the inner surfaces of arteries.

Coronary heart disease: A condition caused by atherosclerosis that reduces blood flow through the coronary arteries to the heart.

Dyslipidemia: One or more abnormalities in serum lipid profile level.

Dyspepsia: Chronic abdominal pain, discomfort, bloating, nausea, and vomiting.

***H. pylori* negative:** Subjects who are negative for *H. pylori* antibody test.

***H. pylori* positive:** Subjects who are positive for *H. pylori* antibody test.

High LDL-C: LDL-C $\geq$130 mg/dl.

Hypercholesterolemia: TC $\geq$200 mg/dl.

Hypertriglyceridemia: TG $\geq$150 mg/dl.

Lipid Profile: A panel of blood tests for measurement of lipids in serum, such as TC, TG, HDL-C, and LDL-C.

Low HDL-C: HDL-C $\leq$ 40 mg/dl.

OPD patient Patients who came to the outpatient department unit from referral site or for primary diagnosis.

CHAPTER ONE: INTRODUCTION

1.1 BACKGROUND

Dyslipidemia is abnormality in lipid profile in which (TC ≥ 200 mg/dl), (TG ≥ 150mg/dl, (LDL-C ≥130mg/l), and (HDL-C ≤ 40mg/dl) is one of the most important risk factors for the development of coronary heart disease (1). Lipids and lipoproteins are causes for coronary heart disease (CHD). It has been demonstrated that high levels of TC, TG, LDL-C, low HDL-C, and increased body mass index (BMI) are significantly associated with CHD (2).

Helicobacter pylori (H. pylori) is a spiral shaped, micro-aerophilic, gram-negative bacteria that was first isolated in 1982 from stomach biopsy specimens of patients with chronic gastritis. Approximately, half of the world's population is estimated to be infected with *H. pylori*, but the prevalence varies greatly among countries and the overall prevalence of *H. pylori* infection is strongly correlated with socioeconomic conditions (3). *H. pylori* infection is commonly acquired in early life via oral-oral or fecal-oral pathways and chronic infection is strongly linked to the development of gastric cancer and peptic ulcer disease (4).

Although *H. pylori* is the most predominant infection in the world, the epidemiologic link between the *H. pylori* infection and metabolic changes is a topic of debate and controversial (5). The colonization of the stomach by *H. pylori* causes chronic inflammation of the stomach wall which can change some biochemical parameters in the patient (6). *H. pylori* infection has linked to a variety of extra-gastric disorders, like coronary heart disease (CHD). The underlying possible mechanisms are chronic low-grade activation of the coagulation cascade, accelerating atherosclerosis, and antigenic mimicry between *H. pylori* and host epitopes leading to autoimmune disorders and lipid metabolism abnormality (7). Due to gastrointestinal inflammation caused by *H. pylori*, absorption of glucose and lipids can be decreased (8). An increase of TC, LDL-C and decrease in HDL-C levels in *H. pylori* infected people creates an atherogenic lipid profile which could promote atherosclerosis with its complications, myocardial infarction, stroke and peripheral vascular disease (9). This was indicated in an experimental investigation that interleukin-8 (IL-8), which is over expressed in *H. pylori* infected mucosa production is stimulated by oxidized LDL-C by monocytes and thus, this potent chemoattractant cytokine increases the recruitment of T lymphocytes and smooth muscle cells, contributing to atherosclerosis (10).

Now a day, infectious agents are being considered more frequently as causes of diseases that have been thought previously to be of non-infectious etiology like coronary heart disease. Additionally, lipopolysaccharide (LPS) affects circulating macrophages, and increase production of free radicals. Free radicals are known to oxidize LDL, the product of which (oxidized LDL), transform macrophages into foam cells, which are known to be important in the pathogenesis of atherosclerosis (11). Moreover, products of gram negative bacteria LPS, is recognized by toll like receptors(TLRs) on macrophages and other cells and these initiate marked changes in lipid and lipoprotein metabolism (12).

Infection with *H. pylori* triggers a chronic inflammatory state which along with other mechanisms such as dyslipidemia, hyper-homocysteinemia, hypercoagulability, impaired glucose metabolism and endothelial dysfunction, contribute in pathogenesis of atherosclerosis. Studies have shown a positive relation between cytotoxic associated gene-A (Cag A) positive strain of *H. pylori* and vascular diseases such as coronary artery disease (CAD) and stroke (13). The other mechanisms postulated to be the link between *H. pylori* infection and atherosclerosis are the activation of endothelial dysfunction by endotoxins released from virulent strains of *H. pylori*, the autoimmune response by secreting heat-shock proteins (14). Endothelial dysfunction leads to increased tension, vascular wall remodeling, vascular inflammation, increased platelet adhesion and aggregation. These processes play its role in the development of atherosclerosis (15). For example, *chronic chlamydia pneumoniae* infection seemed to be associated with an increase serum lipid profile considered to increase the risk of atherosclerosis, supporting the hypothesis that infections can play an indirect role in the pathogenesis of atherosclerosis (16).

Study has indicated that the presence of *H. pylori* in gastrointestinal ulcers results in change in lipid profile of serum including: cholesterol, TG, and LDL-C, HDL-C lipoproteins (17). But conflicting results also exist (18). Other study indicate *H. pylori* could play a role in the development of ischemic heart disease through different ways such as colonization of endothelial cells, changes in lipid profile, hyper coagulation, platelet aggregation, induction of molecular mimicry mechanisms, and progression of low-grade systemic inflammation (19).

Activation of human neutrophils, monocytes, and dendritic cells with *H. pylori* neutrophil-activating protein (*H. pylori*-NAP) strongly up regulates both interleukin 12 (IL-12) and interleukin 2 (IL-2) production, via toll like receptors and in the gastric mucosa of *H. pylori* infected patients, a

considerable proportion of T helper cells that are specific for different *H. pylori* antigens, including *H. pylori* -NAP, CagA, urease, vacuolating cytotoxin gene A (VacA) and heat shock proteins (10). Currently a growing evidence suggests that there are extra-intestinal manifestations of *H. pylori* infection (20). This research will help to assess the association of *H. pylori infection* and dyslipidemia.

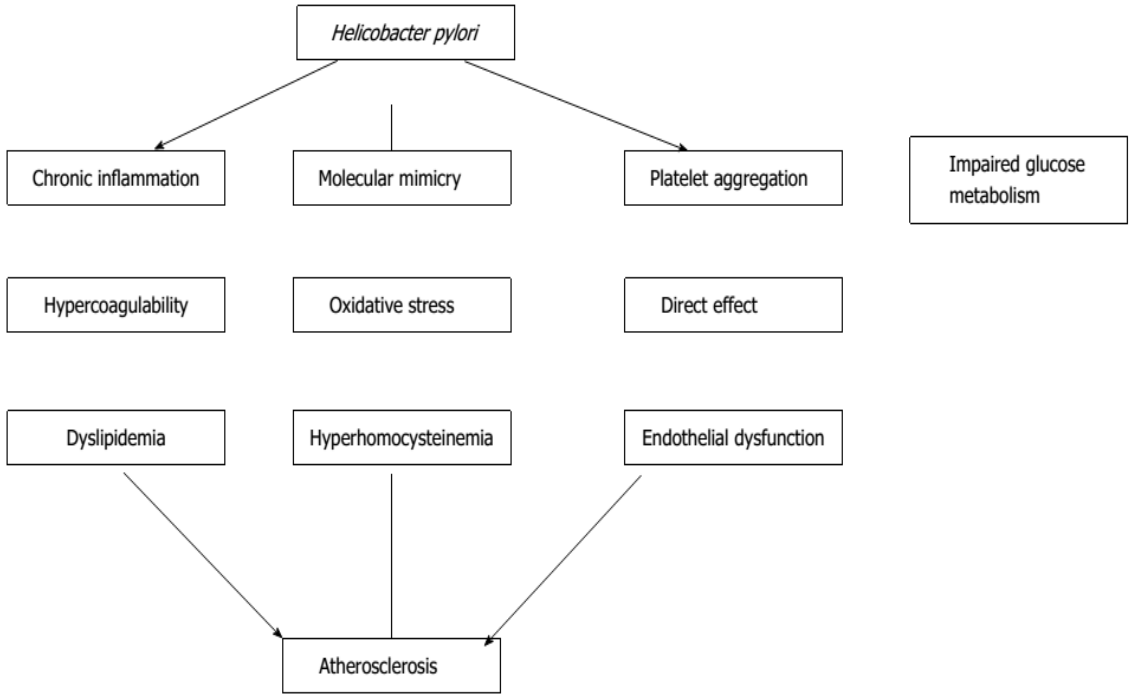


Figure 1: Theories of infection related atherosclerosis adapted from review on the role of *H. pylori* infection in pathogenesis of atherosclerosis (13).

1.2 STATEMENT OF THE PROBLEM

Dyslipidemia comprises a group of disorders in the metabolism of TC and TG, which has implications in the cardiovascular system, producing pathologies as vascular coronary disease and atherosclerosis (21). CAD are a global health problem. Its etiology is associated with various risk factors like hypertension, diabetes mellitus (DM) and dyslipidemia (22). The epidemiological distribution of *H. pylori* infection is worldwide, based on regional prevalence estimates, there were approximately 4.4 billion individuals with *H. pylori* infection worldwide in 2015, Africa had the highest prevalence (70.1%; 95% CI, 62.6-77.7), and Oceania had the lowest prevalence (24.4%; 95% CI,18.5-30.4) (23).

The burden of chronic diseases is increasing in low- and middle-income countries, where as it remains stable in high-income countries, developing countries are encountering a growing burden of chronic diseases, which constitute a double burden combined with infectious diseases and nutritional problems (24). Epidemiologic and clinical report suggest that *H. pylori* infection is a contributing factor in the progression of atherosclerosis. However, the specific cardiovascular disease risk factors associated with *H. pylori* remain unclear, and the infection remains a destructive, transmissible, with serious health consequences (25).

Some studies suggest a significant relationship between *H. pylori* infection and atherogenesis (26). *H.pylori* infection might play an important role in the pathogenesis of atherosclerosis (16). Due to accumulation of fatty substances, cholesterol, cellular waste products, calcium, and fibrin in the inner lining of the arterial wall cause the formation of atherosclerotic plaques (2).

The possible role of the *H. pylori* infection as a determinant of extra-gastric manifestations such as atherosclerotic processes and peripheral vascular disorders is matter of debate (27).

Currently, there is no convincing evidence supporting the presence of *H. pylori* within atherosclerotic plaques, and sero-epidemiological evidence is contradictory. Studies indicated that *H. pylori* infection bring to changes in serum lipid profile and may increase the risk of atherosclerosis (28). But there are studies, which do not support the idea of *H. pylori*-dependent dyslipidemia (29). This study was conducted to investigate the association between *H. pylori* infection and changes in serum lipid profile in gastritis suspected patients.

To the best of our knowledge, there is no study conducted in this area in particular and in Ethiopia in general, about the association between *H. pylori* infection, and dyslipidemia among adult gastritis suspected patients.

1.3 SIGNIFICANCE OF THE STUDY

- Early assessment of lipid profile in gastritis patient help patients to prevent dyslipidemia
- Assessment of lipid profile will benefit patients for early intervention in cost effective way
- This study will be helpful to assess the association of *H. pylori* infection and dyslipidemia.
- This study serves as a base for other study, and also can give information for policy makers
- Conducting this study will give direction for physician and other concerned bodies in *H. pylori* eradication therapy.

CHAPTER TWO: LITRATURE REVIEW

The world health organization (WHO) categorized *H. pylori* as a group I carcinogen, by stressing on its association with gastric cancer and causing several extra-gastric diseases like iron deficiency anemia, idiopathic thrombocytopenic purpura, and recently, studies demonstrated that *H. pylori* infection was also related to lipid and glucose metabolism abnormality (30).

Currently, evidence has come to the literature strongly suggesting causal link between *H. pylori* and extra gastric disorders. Cardiovascular system is one of the extra gastric organs that can be affected by *H. pylori* infection (31).

A study conducted in Korea to investigate that *H. pylori* is associated with dyslipidemia but not with other risk factors of cardiovascular disease revealed that *H. pylori* infection was significantly associated with higher TC level (r= 2.114, $p < 0.001$), higher LDL-C level (r = 3.339, $p < 0.001$), lower HDL-C level (r = −1.237, $p < 0.001$), and higher diastolic blood pressure (r = 0.539, p = 0.001. Similarly, a retrospective study done in Japan in 2002, to investigate the association between *H. pylori* infection and atherosclerosis in 1,165 residents, *H. pylori*-positive residents had significantly higher mean values or serum TC, LDL-C, and lower mean values for HDL-C ($p < 0.05$). LDL-C was significantly associated with *H.pylori* infection status ($p < 0.0088$, OR=1.01, 95% CI=1.00-1.01) while other factors were not significant (25.34, 35).

A study conducted in Turkey on 350 subjects on serum lipid profile in *H. pylori* infected patients, serum cholesterol concentrations were significantly higher in patients group when compared with healthy group (189.32±45.15 vs. 179.41±36.37 mg/dl ($p <0.05$), serum TG and TC/HDL-C concentrations also were significantly higher in patients group (169.46±68.53 vs. 135.67±94.35 mg/dl ($p<0.05$) and 3.93±1.23 vs. 3.51±1.62, ($p <0.05$) respectively) (33).

A hospital based cross sectional study conducted in Iran on dyslipidemia in 144 *H. pylori* infected patients showed that *H. pylori* infected patients are prone to acquire dyslipidemia, 60.4% prevalence of dyslipidemia with male predominance (58.6%)(34). Another study done in Iran on evaluation of serum lipid profiles among 58 subjects infected and 58 subjects non-infected with *H.pylori*, showed that infected groups have statistically significantly increased rate of serum lipid profiles,

including TC, TG, LDL-C and HDL-C compared with the non-infected groups (In all $p < 0.0001$) (17).

A study conducted in Iraq on 206 subjects in 2016 to determine lipid profile in patients with gastritis found that *H. pylori* was associated with increase in serum TC, TG and LDL-C and decrease in serum HDL-C and the positivity rate was 55.83%, which is also in line with a cross-sectional study conducted in Cameroon to investigate risk factors, uric acid and alanine aminotransferase and lipid profile in *H. pylori* positive and negative patients found that *H. pylori* infected patients had increased levels of uric acid ($p = 0.017$), total cholesterol ($p = 0.001$), LDL-cholesterol ($p = 0.021$) and TC/HDL-C ratio ($p = 0.046$) compared to the uninfected group (5,30).

A longitudinal prospective study conducted in Central Africa on 205 black urban Congolese individuals (128 with *H. pylori* positive and 77 negative patients for 10 years (1999–2008), showed that blood fibrinogen, and TC were higher in *H. pylori* infected patients than in the uninfected group; there was also a significant association among severity of *H. pylori* seropositivity and cardiovascular disease $p < 0.001$, likewise a prospective cohort study conducted in Egypt on the association of virulent *H.pylori* strain with CAD and coronary risk factors on 150 patients, there was a statistically significant positive association between *H. pylori* infection and dyslipidemia, with p value < 0.05 (39,40).

In a cross-sectional study conducted on association between *H. pylori* and CHD among patients, in Sudan, the serum TC were significantly increased among group which had *H. pylori* positive and coronary heart disease. while in the other group which had *H, pylori* positive without CHD the serum LDL-C was significantly increased (36).

However, contrary to the above concept, there are studies which do not support the idea of *H. pylori*-dependent dyslipidemia and no association between *H. pylori* infection and levels of TC and TG like, a prospective multicenter study, conducted in Spain on 830 patients on the influence of *H. pylori* infection and eradication on blood lipids and fibrinogen found that *H. pylori* has no influence on blood lipids or fibrinogen, another study conducted in Croatia to determine whether *H. pylori* infection is an independent risk factor for acute myocardial infarction (AMI) found out *H. pylori* infection is not an independent risk factor for acute myocardial infarction and a case control study done in Finland on *H. pylori* infection versus increased risk of CHD by modifying serum lipid concentrations, concluded that the impact of *H. pylori* infection as an independent risk factor for CHD seems to be minor. Similarly, a cross sectional study conducted on 363 subjects, on the association of *H. pylori* infection with

metabolic parameters and dietary habits among medical undergraduate students in south eastern of Iran showed that *H. pylori*-positive subjects had lower mean levels of TC and TG and higher levels of HDL-C compared to *H. pylori* negative subjects. In addition, lower levels of LDL-C (P = 0.044) were observed among subjects with positive *H. pylori* infection. (30, 31, 36, 37, 39).

Thus, the current study is trying to look at these contradictory concepts in this new study setting, in JUMC, Jimma, southwest Ethiopia.

CHAPTER THREE: OBJECTIVES

3.1 GENERAL OBJECTIVE

To assess prevalence of dyslipidemia and associated factors among patients suspected for gastritis at OPD of JUMC, Jimma, Ethiopia, from January 03 to April 05, 2019.

3.2 SPECIFIC OBJECTIVES

- To determine the prevalence of dyslipidemia on gastritis suspected patients visiting OPD at JUMC.
- To compare lipid profiles among *H. pylori* positive and negative adult patients visiting at OPD of JUMC.
- To assess the association of *H. pylori* infection with dyslipidemia among adult patients with a suspected case of gastritis at OPD of JUMC.

CHAPTER FOUR: METHODS AND MATERIALS

4.1 STUDY AREA AND PERIOD

The study was conducted at OPD of JUMC, which is one of the oldest public hospitals in Ethiopia. It was established in 1922. The hospital is located in Jimma city 352 km southwest of the capital Addis-Ababa. Cognizant of the fast-growing service and teaching role of the hospital, the federal government constructed a new and level best 600 bedded hospital and it is becoming one of the institutions with oncology center in Ethiopia. The hospital provide out patient service in the pediatric, internal medicine, surgery, and gynecology- obstetrics (38). Most of the cases are screened from the OPD to other wards and department. Like wises, gastritis suspected patients were screened in the adult cold OPD. The study was conducted from January 03, to April 05, 2019 G.C.

4.2 STUDY DESIGN

Hospital based cross sectional study design was used to conduct the study.

4.3 POPULATION

4.3.1 SOURCE POPULATION

All patients who were coming to JUMC adult OPD during the study period were used as source population.

4.3.2 STUDY POPULATION

Patients who were coming to adult cold OPD of JUMC with gastritis symptoms during the study periods was the study population.

4.3.3 STUDY SUBJECT

All individual patients suspected for *H. pylori* infection was taken as the study subjects.

4.3.4 SAMPLING TECHNIQUE

Consecutive convenient sampling technique was used until the required sample sizes was attained. All adult patients present with symptoms of dyspepsia, for more than a week duration were screened by physician at OPD and evaluated for *H. pylori* infection and dyslipidemia.

4.3.5 SAMPLE SIZE DETERMINATION

Sample size was calculated using single population proportion formula. Since the prevalence of dyslipidemia in *H. pylori* infected individual in Ethiopia was not obtained, prevalence of dyslipidemia in *H. pylori* infected from study conducted in Iran was 60.4% used (34). The expected margin of error (d) was taken 0.05 with the confidence interval level of 95%. The number of samples of gastritis suspected patients to be included in the study was calculated based on the following single population proportion formula.

$$N = \frac{Z_{\alpha/2}^2 P(1-P)}{d^2}$$

$$= \frac{1.96^2 0.604(1-0.604)}{0.05^2}$$

$$= 369$$

Where:

N = minimum sample size

$Z_{\alpha/2}$= 95% confidence interval (1.96)

P = Estimated prevalence rate (60.4%) = (0.60)

d = Marginal of sampling error

4.4 INCLUSION AND EXCLUSION CRITERIA

4.4.1 INCLUSION CRITERIA

- Adult patients suspected for *H. pylori* infection.
- Patients volunteer to participate in the study.

4.4.2 EXCLUSION CRITERIA

Study subjects who had received

- Lipid-lowering medication
- Antibiotics or proton-pump inhibitor within two weeks
- Pregnant women

4.5 STUDY VARIABLES

4.5.1 DEPENDENT VARIABLE

Dyslipidemia

4.5.2 INDEPENDENT VARIABLES

- Age
- Sex
- Occupational status
- Educational status
- Residence
- Marital status
- Duration of gastritis
- Physical exercise
- *H. pylori* status
- Khat chewing
- Alcohol consumption
- Cigarette Smoking
- Anthropometric indicator

4.6 DATA COLLECTION MATERIALS AND REAGENTS

- One step serum *H. pylori* rapid antibody test strip Wondfo (Guangzhou Wondfo Biotech Co., Ltd.)
- ABX Pentra 400 clinical chemistry analyzer (Horiba ABX SAS, Montpellier, France).
- Controls (both normal and pathological)
- HDL and LDL calibrator
- Multi-calibrator
- Plastic pipette
- Serum separator tube
- Cuvette
- Pen
- Pencil
- Pencil-sharpener
- Questionnaires
- A_4 paper
- Tape meter
- Digital BP apparatus(sphygmomanometer)
- Centrifuge
- 70% alcohol
- Disposable syringe
- Cotton
- Tourniquet
- Glove
- Weight scale
- Safety box

4.6.1 DATA COLLECTION PROCEDURE

After a brief explanation, the study participants were asked for their consent to be interviewed by clinical nurses to give fasting blood sample. Then five ml of blood was withdrawn by laboratory professionals from the study participants, who had fasted overnight (10-12 hours), for laboratory analysis. In addition, the questionnaire was filled by face to face interview and blood pressure were measured by clinical nurses.

Blood Pressure measurement: An automatic digital sphygmomanometer (OMRON HEALTH CARE Co., Ltd. Kyoto, Japan) was used to measure patients blood pressure. The patient was seated upright with their upper arm positioned on the bench and excess clothing was removed that might interfere with the BP cuff or constrict blood flow in the arm (39).

4.7 ANTHROPOMETRIC MEASUREMENT

Waist circumference measurement: Participants waist circumference was measured at the level of the iliac crest and the umbilicus with a tape measure to evaluate abdominal obesity (40).

Height measurement: Height was measured using height measure scale (Infiniti Med Lab Pvt. Ltd., India). Participants stand erect on the floor board of the stadiometer with their back to the vertical backboard of the stadiometer. The heels of the feet are placed together with both heels touching the base of the vertical board. Feet were pointed slightly outward at a 60-degree angle. The buttocks, scapulae, and head are positioned in contact with the vertical backboard. During the height measurement, the participant's shoes and hats were removed. The height measurement was recorded to the nearest 0.1 cm (41).

Weight measurement: First the weight scale (Infiniti Med Lab Pvt. Ltd., India) was turn to zero then participants were asked to remove extra layers of clothing, shoes, jewelry, and any items in their pockets. Next the participant were asked to step on the scale backwards then body weight was evenly distributed between both feet, arms hang freely by the sides of the body, palms toward thighs and head is up and facing straight ahead then weight is recorded to nearest 0.1 kg (100 gm).

Body Mass Index (BMI): After measuring participant's height and weight, the BMI was calculated by dividing weight in (kg) by height squared (m^2). The WHO definition of obesity is based on various categorical cut-points based on the body mass index (BMI) of weight-for-height: underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), and obesity ($\geq$ 30 kg/m^2) (42).

4.7.1 BLOOD COLLECTION AND LABORATORY INVESTIGATION

After obtaining consent from the study subjects by giving detail information about the study objective, venous blood was selected preferably at the antecubital area by applying a tourniquet, puncture area of the vein was disinfected by 70% alcohol. Five ml of blood was withdrawn aseptically from the antecubital vein from fasting individuals using serum separator tube. Following collection, specimens was transported to clinical chemistry unit of JUMC laboratory for analysis. The collected blood sample was left for more than 30 minutes at room temperature for clotting. Then the clotted blood samples were centrifuged for 5 minutes at 4000 revolutions per minutes to separate serum from formed elements. All these procedures were done by the principal investigator. The extracted serum was kept in Nunc tube under -20^{o}c deep freeze until laboratory analysis. These frozen sera were later analyzed for the biochemical parameters. TG, HDL-C, and TC were measured in serum by using ABX Pentra 400 clinical chemistry analyzer (Horiba ABX SAS, Montpellier, France) fully automated auto analyzer by the direct end point enzymatic method.

4.7.2 LIPID PROFILE TESTS

4.7.2.1 TOTAL CHOLESTEROL (CHOD-PAP METHOD)

Cholesterol was measured by CHOD-PAP, enzymatic photometric method. Absorbance is measured at 500 nm. The reaction sequence is as follows:

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{CHE}} \text{Cholesterol} + \text{Fatty acid}$$

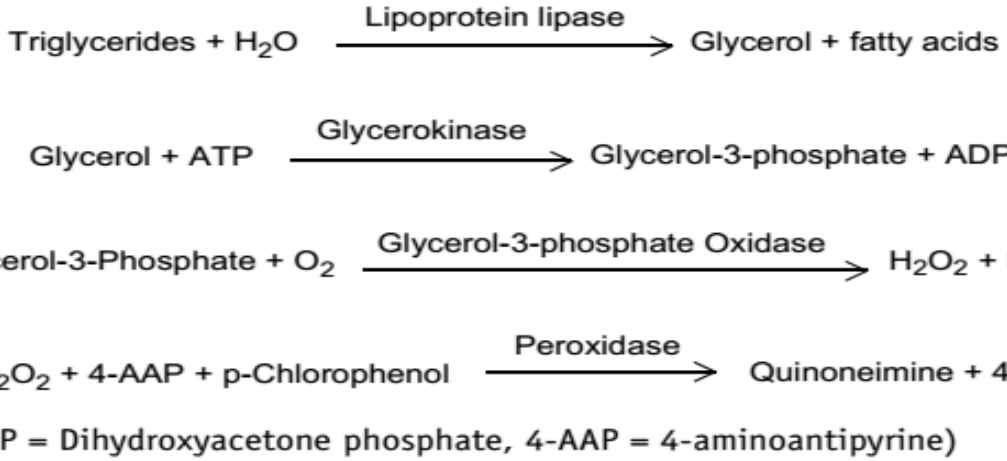
$$\text{Cholesterol} + O_2 \xrightarrow{\text{CHO}} \text{Cholesterol-3-one} + H_2O_2$$

$$2H_2O_2 + \text{4-Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{POD}} \text{Quinoneime} + 4H_2O$$

(CHE = Cholesterol Esterase, CHO = Cholesterol oxydase, POD = Peroxidase)

4.7.2.2 TRIGLYCERIDES TEST (GPO-PAP METHOD)

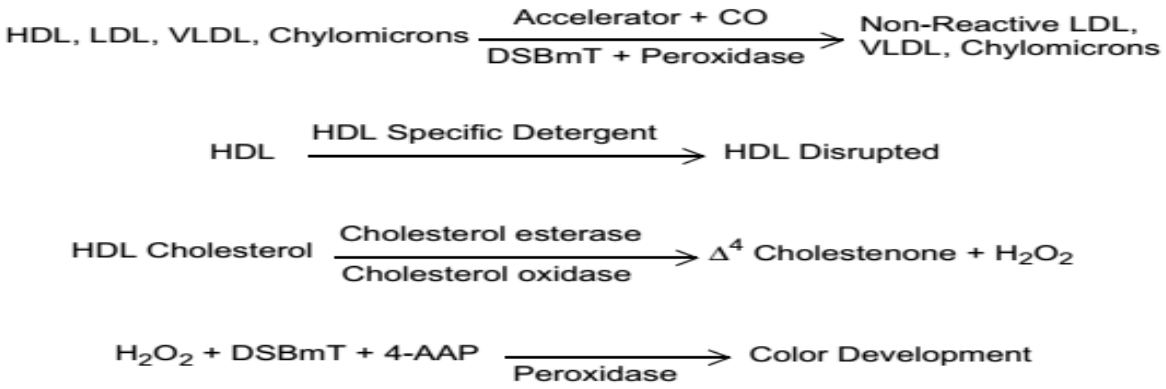
A series of coupled reactions in which triglycerides are hydrolyzed to produce glycerol. Absorbance is measured at 500 nm. The reaction sequence is as follows:



(DHAP = Dihydroxyacetone phosphate, 4-AAP = 4-aminoantipyrine)

4.7.2.3 HIGH DENSITY LIPOPROTEIN CHOLESTEROL (HDL-C)

HDL-C is measured by direct assay in a homogeneous method for directly HDL-C levels in serum without the need for any off-line pretreatment or centrifugation steps. The reactions are as follows:



(4-AAP = 4-Aminoantipyrine, CO = Cholesterol Oxidase, DSBmT = N,N-bis(4-sulphobutyl)-m-toluidine-disodium)

4.7.2.4 LOW DENSITY LIPOPROTEIN CHOLESTEROL (LDL-C)

LDL-C was estimated from Friedwald formula (43). LDL-cholesterol was calculated from measured values of TC, TG and HDL-C according to the relationship: [LDL-C] = [TC] - [HDL-C] - [TG/5] mg/dl. The detail SOP for lipid profile is indicated in annex (XII).

4.7.3 DETERMINATION OF *H. PYLORI* STATUS

Study participants *H. pylori* status were determined by rapid antibody test strip Wondfo (one step *H. pylori* serum/plasma test), because this rapid test can be used for screening purpose and diagnosis test in an endemic area where the prevalence of *H. pylori* infection is greater than 30%. The performance of Wondfo (Guangzhou Wondfo Biotech Co., Ltd) one step *H. pylori* serum/plasma test had sensitivity of 99.0% and specificity of 99.2% when compared with *H. pylori* ELISA kit, ELISA kit has 99.5% sensitivity and 99.6%

specificity to detect *H. pylori*. Test principle and procedure of serum rapid *H. pylori* test is indicated in (Annex XIII).

4.8 DATA QUALITY ASSURANCE AND MANAGEMENT

Data collection was carried out after approval of the research proposal. After completion of each questionnaire, cross checking was done between data collector and principal investigator to assure the completeness of the information gathered. The label on the test tube and subject's unique identification number on questionnaire was checked for similarity.

After checking the expiry date of the reagents and control, ABX Pentra 400 clinical chemistry analyzer (Horiba ABX SAS, Montpellier, France), was checked for delivering correct result by using normal and pathological controls. Before any patient sample processed, dual quality controls (normal and pathological) was performed and the patient result was taken after the controls passed. All necessary procedures and steps were followed based on the manufacture instructions. Collected results were checked for completeness on a daily basis by the principal investigator.

4.9 STATISTICAL ANALYSIS

Information from the questionnaires and laboratory analysis was entered into Epidata version 3.1, and exported to SPSS version 25 software (IBM Corporation, USA). Lipid profile result was expressed as mean ± SD. Descriptive statistics were used to analyze the data. For categorical variables, percentage and frequencies were used, whereas mean, standard deviation and range were used for continuous variables. Tables were used to assist data presentation and independent sample t-test was used to compare the different studied parameters between groups of *H. pylori* positive and negative subjects. Bivariate logistic regression analysis was conducted to see the existence of crude association and to select candidate variables (with P value below 0.25) to multivariable logistic regression. Logistic regression analysis was done to control possible confounders and to determine factors that may be significantly associated with dyslipidemia. P value ≤ 0.05 was considered as a cut point for statistical significance in the final model. Fitness of goodness of the final model was checked by Hosmer and Lemeshow.

4.10 ETHICAL CONSIDERATION

Data collection were carried out after approval of the research proposal by the institutional review board (IRB) of Jimma university, institute of health with letter protocol number **IHRPG/567/2018,** and support letter from post graduate program directorate was submitted to clinical director of the JUMC (Ethical approval and support letters are attached in annex part at of this document). After getting all permission from all responsible body, the data collector informs the patients by reading or giving to read the information sheet which is translated to patients' language about the objectives of the study. Confidentiality was maintained by using of identification numbers instead of individual names. Finally, patient's laboratory result was printed and submitted to cold OPD for appropriate intervention and awareness creation on dyslipidemia to study subjects.

4.11 PLAN FOR UTILIZATION AND DISSEMINATION OF RESULT

The finding of this study will be submitted to Jimma university, institute of health, faculty of health sciences, school of medical laboratory science, and JUMC. The findings of this study will be also published in peer reviewed scientific journals. It will also be presented on different scientific forums.

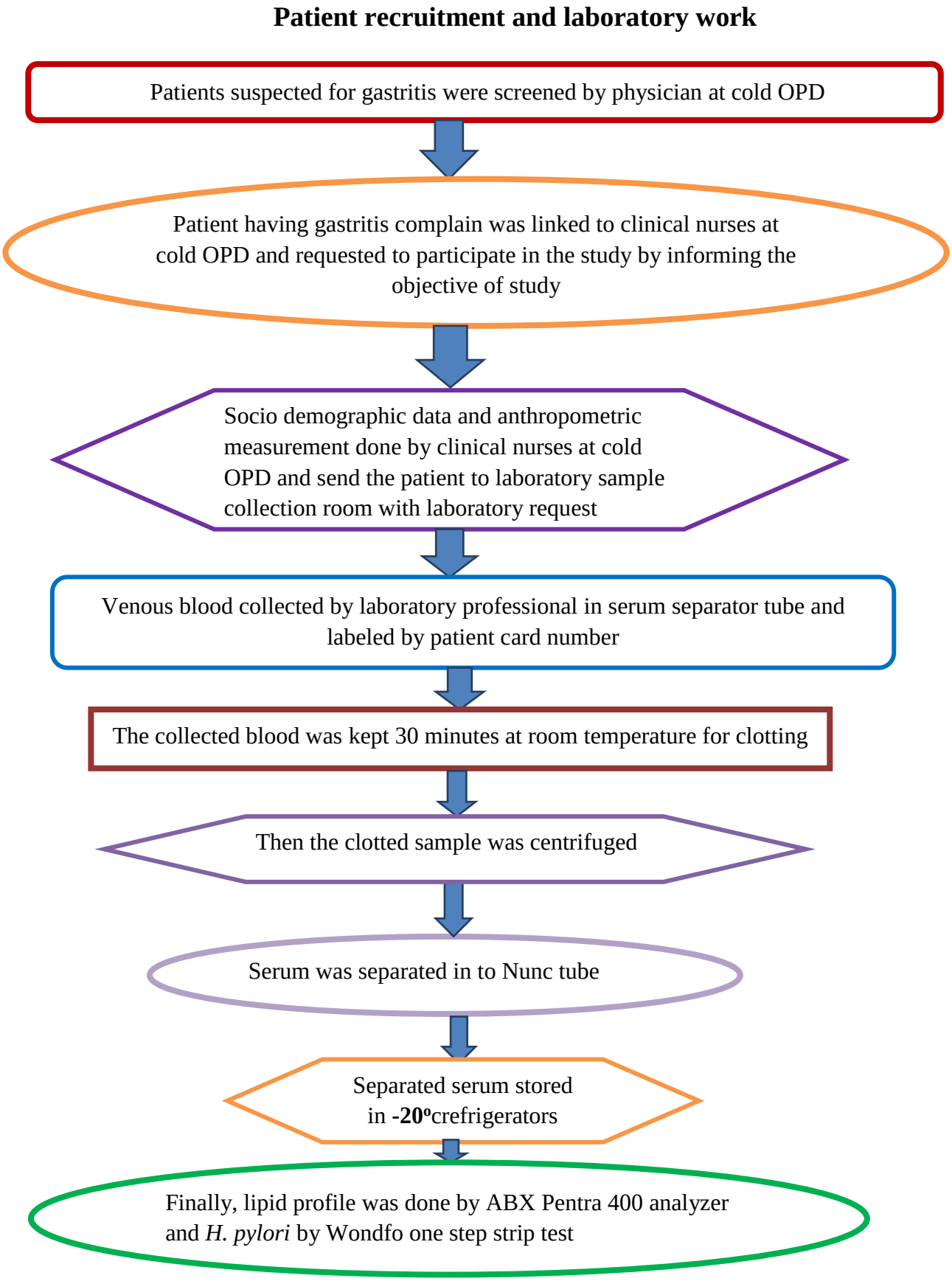


Figure 2. Flow chart of patient recruitment and the laboratory work for the study.

CHAPTER FIVE: RESULTS

5.1 SOCIO-DEMOGRAPHIC AND CLINICAL CHARACTERISTICS

A total of 369 patients suspected for gastritis were included in the present study. Among the study subjects 194 (52.6%) were females and 175 (47.4%) were males aged from 20 years to 74 years with the mean ± SD age of 41.03 ± 13.55 years. Majority of the study participants 182 (49.3%), were Muslim by religion and 211(57.2%), were Oromo by ethnicity. Occupational status and smoking were significantly associated to *H. pylori (*p<0.05).

Regarding life style condition of study participants, majority 344 (93.2%) were nonsmoker, 329 (89.2%) did not drink alcohol, 248 (67.2%) did not chew khat, and 313 (84.8%) did not have regular physical exercise. Majority of study participant 265 (71.8%) had normal (18.5-25 kg/m^2), 55 (14.9%) had overweight (25-29.9 kg/m^2), and 49 (13.3%) underweight (<18.5kg/m^2) had BMI values respectively. There was no statistically significant association with *H. pylori* infection to known history of diabetes (P=0.876), hypertension (P=0.168), renal disease (P=0.078), khat chewing (P=0.10), and alcohol drinking (P=0.676) as indicated in table 1.

Table 1: Socio-demographic characteristics, and *H. pylori* antibody status with its association among adult OPD patients at JUMC, Jimma Ethiopia (N=369).

Variables	Category	*H. pylori* Ab positive No (%)	*H. pylori* Ab negative No (%)	P.value
Age in years		42.68 ± 13.66	39.58 ± 13.32	0.028
Sex	Male	89 (24.1)	86 (23.3)	
	Female	84 (22.8)	110 (29.8)	0.146
Marital status	Single	36(9.8)	44(11.9)	
	Married	114(30.9)	128(34.7)	0.972
	Divorced	8(2.2)	9(2.4)	
	Widowed	15(4.1)	15(4.1)	
Educational status	Illiterate	54 (14.6)	53(14.4)	0.625
	Read and write	24(6.5)	27(7.3)	
	Elementary	38(10.3)	54(14.6)	
	High school and above	57(15.4)	62(16.8)	
Residence	Urban	107 (29.0)	106 (28.7)	0.132
	Rural	66 (17.9)	90 (24.4)	
Occupational Status	Civil servant	37 (10)	30(8.1)	
	House wife	27(7.3)	60(16.3)	0.014
	Merchant	10(2.7)	5(1.4)	

	Private worker	23(6.2)	29(7.9)	
	Farmer	45(12.2)	42(11.4)	
	Other	31(8.4)	30(8.1)	
Smoking	Yes	19 (5.1)	6(1.6)	0.003*
	No	154 (41.7)	190(51.5)	
Physical Exercise	Yes	28 (7.6)	28 (7.6)	0.612
	No	145 (39.3)	168 (45.5)	
History of DM	Yes	14 (3.8)	15 (4.1)	0.876
	No	159(43.1)	181(49.1)	
History of hypertension	Yes	31(8.4)	25(6.8)	0.168
	No	142(38.5)	171(46.3)	
History of renal disease	Yes	24(6.5)	16(4.3)	0.078
	No	149(40.4)	180(48.8)	
khat chewing	Yes	64(17.3)	57(15.4)	0.10
	No	109(29.5)	139(37.7)	
Drinking alcohol	Yes	20(5.4)	20(5.4)	0.676
	No	153(41.5)	176(47.7)	

*Note: p value by Pearson chi-square for categorical variable, and by independent t-test for continuous variable, SD= Standard Deviation, other: student, * significant p<0.005.*

5. 2 Prevalence of dyslipidemia among *H. pylori* positive and negative subjects

In this study we found an overall prevalence of 286/369 (77.5 %) for dyslipidemia in at least one of the four lipid profiles. 151 (87.2%) *H. pylori* positive had dyslipidemia in at least one of the four lipid profiles. Individual who had abnormality in all of the four-lipid profile were 25 (6.8%) as indicated in figure 3. From the total study subjects 154 (41.7%) had low HDL-C, 99 (26.8%) had high LDL-C, 130 (35.2%) had high TC and 196 (53.1%) had high TG. The distribution of abnormal lipid profile among *H. pylori* positive subjects were 89 (51.4%), 116 (67.05%), 66 (38.1%) and 68 (39.3%) by serum TC, TG, LDL-C and HDL-C respectively, whereas the prevalence of dyslipidemia among *H. pylori* negative individual were 41 (20.9%), 80 (40.8%), 33 (16.8%) and 86 (43.8%) by serum TC, TG, LDL-C and HDL- C respectively. Statistically significant mean value was observed in TC ($p < 0.001$), LDL-C ($p< 0.001$) and TG ($p< 0.001$) in *H. pylori* positive subjects.

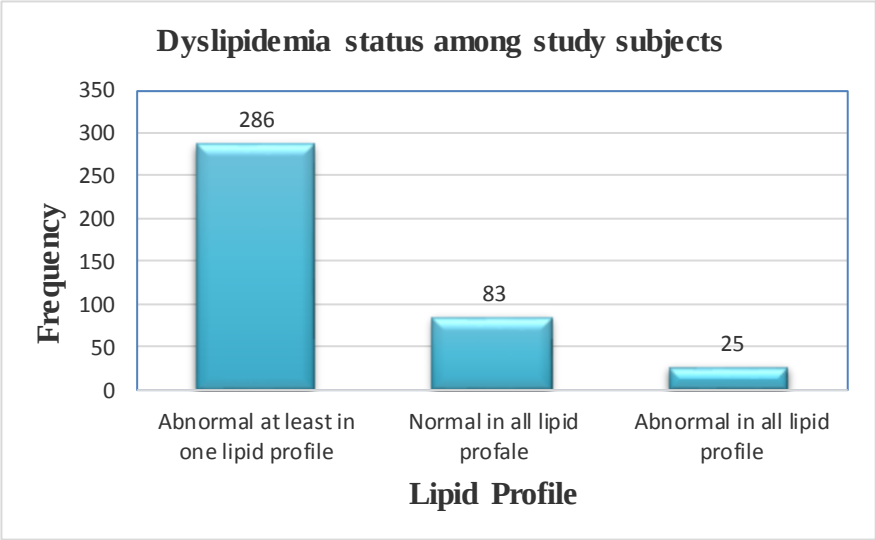


Figure 3: Prevalence dyslipidemia among study subjects at JUMC, Jimma, Ethiopia (N=369).

5.3 Association between *H. pylori* infection and lipid profile among study participants.

The mean level of serum TC, TG, LDL- C was significantly higher and serum HDL-C was not significant between *H. pylori* positive and *H. pylori* negative patients ($p < 0.001$) as shown in table 2.

Table 2: Mean serum value of lipid profile among *H. pylori* positive and negative adult patients at JUMC, Jimma, Ethiopia (N=369).

Variable	*H. pylori* status		P. value
	Positive (Mean ± SD)	Negative (Mean ± SD)	
TC	200.80± 43.48	173.67± 42.41	<0.001
TG	185.61± 74.82	138.18± 60.17	<0.001
HDL-C	41.79± 8.70	41.91± 9.87	0.900
LDL-C	122.00± 37.00	104.27± 34.71	<0.001
Age	42.68 ± 13.66	39.58 ± 13.32	0.280
SBP	120.24 ± 15.82	116.89 ± 14.28	0.033
DBP	78.13 ± 9.21	76.00 ± 8.35	0.021
WC	79.38 ± 9.40	77.79 ± 10.51	0.129
HC	90.43 ± 10.38	87.84± 10.85	0.020

Note; TC- total cholesterol, TG- triglycerides, HDL- C- high density lipoprotein cholesterol, LDL- C- low density lipoprotein cholesterol, SBP- systolic blood pressure, DBP- diastolic blood pressure, WC -waist circumference, HC- hip circumference, CI - confidence interval, and p.value- done by independent sample t-test.

5.4 Correlation between *H. pylori* infection and lipid profile

There was statistically positive correlation between serum TC with weight (r=0.399, p<0.001), BMI (r=0.432, p<0.001), and WC (r=0.320, p<0.001) whereas there was no statistically significant correlation between serum TC with age (r=0.044, p=0.566), SBP (r=0.029, p=0.706, and DBP (r=0.102, p=0.183).

Table 3: Correlation analysis of lipid profile with predictors in *H. pylori* positive subjects (N =173 (46.9%)) among adult patients at JUMC, Jimma, Ethiopia.

Predictors	TC		TG		HDL-C		LDL-C	
	r	p	r	p	r	p	r	P
Age	0.044	0.566	0.068	0.374	-0.155	0.131	0.049	0.520
Sex	-0.006	0.939	-0.064	0.404	0.108	0.158	-0.003	0.967
Physical exercise	-0.091	0.234	-0.131	0.085	-0.081	0.290	-0.033	0.664
Weight	0.399	0.000	0.396	0.000	0.024	0.756	0.304	0.000
BMI	0.432	0.000	0.382	0.000	0.012	0.875	0.351	0.000
WC	0.320	0.000	0.254	0.001	0.089	0.245	0.253	0.001
Smoking	-0.054	0.483	0.029	0.704	0.099	0.196	-0.097	0.204
Khat chewing	-0.016	0.837	0.024	0.752	0.084	0.274	-0.046	0.552
Alcohol drinking	-0.092	0.229	-0.024	0.752	-0.010	0.898	-0.095	0.214
SBP	0.029	0.706	-0.104	0.174	0.019	0.802	0.067	0.378
DBP	0.102	0.183	0.082	0.282	0.100	0.192	0.059	0.444
Duration of gastritis	-0.106	0.164	-0.073	0.338	-0.125	0.100	-0.063	0.411

Note: r = Pearson correlation coefficient, p = p value for correlation, TC =Total Cholesterol, TG= Triglyceride, LDL-C = Low Density Lipoprotein-Cholesterol, HDL-C= High Density Lipoprotein Cholesterol

Regarding to the magnitude of lipid profile among study subjects 89 (46.9%) *H. pylori* positive and 41 (11.1%) *H.* pylori negative had TC greater than 200mg/dl. The frequency and percentage of lipid profiles are indicated in (Table 4).

Table 4: Lipid profile versus *H. pylori* status among adult patients at JUMC, Jimma, Ethiopia (N=369).

Lipid profile		***H. pylori* (+)**	***H.* pylori (-)**	**COR**	**95% CI**	
		No (%)	**No (%)**		**Lower**	**Upper**
TC	< 200 mg/dl	84 (22.8)	155 (42.0)			
	≥ 200 mg/dl	89 (46.9)	41 (11.1)	0.250	0.158	0.394
HDL-C	> 40 mg/dl	105 (28.3)	110 (29.9)			
	≤ 40 mg/dl	68 (18.5)	86 (23.4)	0.279	0.181	0.430
LDL-C	< 130 mg/dl	117 (31.7)	177 (48.0)			
	≥ 130 mg/dl	56 (15.2)	19 (5.1)	0.224	0.552	1.268
TG	< 150 mg/dl	70 (19.0)	139 (37.7)			
	≥ 150 mg/dl	103 (27.9)	57 (15.4)	0.279	0.127	0.397

Note: TC =Total Cholesterol, TG= Triglyceride, LDL-C= Low Density Lipoprotein-Cholesterol, HDL-C= High Density Lipoprotein Cholesterol

5.5 Bivariate and multivariate analysis of factors associated with total cholesterol

On multivariate analysis, having BMI > 25kg/m^2 (AOR 0.243, 95% CI, 0.096-0.617, P= 0.003), and having weight >56kg (AOR 0.403,95%CI, 0.220- 0.737, P=0.003) were independently associated with serum cholesterol concentration. However, being female sex (AOR 1.254, 95% CI, 0681- 2.309, P=0.467), was not associated with serum cholesterol concentration as shown table 5.

Table 5: Associated factor for serum total cholesterol concentration among adult cold OPD patients at JUMC, Jimma, Ethiopia (N=369).

Variable	Category	Total cholesterol		Bivariate analysis		Multivariate analysis	
		<200mg/dl No (%)	≥200mg/dl No (%)	COR 95% CI	P-value	AOR 95% CI	P-value
Age	< 40 year	146 (39.6)	51 (13.8)	1	<0.001	1	0.005*
	≥40 year	93 (25.2)	79 (21.4)	0.411(0.265- 0.637)		0.467(0.276 - 0.790)	
Sex	Female	133(36.0)	61(16.5)	0.705(0.459 -1.082)	0.109	1.254(0.681- 2.309)	
	Male	106 (28.7)	69(18.7)	1		1	0.467
H. pylori	Neg.	155(42.0)	41(11.1)	1	<0.001	1	
	Pos.	84(22.8)	89(24.8)	4.006(2.540- 6.316)		0.273(0.158 - 0.470)	<0.001*
BMI	< 25kg/m^2	228(61.8)	86(23.3)	1	0.000	1	

	≥ 25kg/m^2	11(3.0)	44(11.9)	0.094(0.047- 0.191)		0.243(0.096-0.617)	0.003*
DBP	< 90mmHg	228 (61.8)	117 (31.7)	1	0.050	1	
	≥ 90mmHg	11 (3.0)	13 (3.5)	0.434(0.189- 0.999)		0.703(0.266-1.855)	0.476
WC	< 94cm	229 (62.1)	106 (28.7)	1	<0.001	1	
	≥ 94cm	10 (2.7)	24 (6.5)	5.185(2.394-11.230)		0.669(0.198-2.264)	0.518
Weight	< 56kg	139(37.7)	29(7.9)	1	<0.001	1	
	≥ 56kg	100(27.1)	101(27.4)	0.207(0.127- 0.336)		0.403(0.220- 0.737)	0.003*
Alcohol	No	220(59.6)	109(29.5)	1		1	
drinking	Yes	19(5.1)	21(5.7)	2.231(1.151- 4.323)	0.017	1.192(0.499-2.846)	0.692
Residence	Rural	118(32.0)	38 (10.3)	1	<0.001	1	0.001*
	Urban	121(32.8)	92(24.9)	2.361(1.498-3.722)		0.357(0.192-0.662)	
HC	< 102cm	225 (61.0)	108 (29.3)	1	0.001	1	0.377
	≥102cm	14 (3.8)	22 (6.0)	0.305(0.150-0.620)		0.649(0.248-1.695)	
Diet	Vegetable	181 (49.1)	81(22.0)	1	0.007	1	0.768
	Meat	58(15.7)	49(13.3)	0.530(0.334-0.841)		0914(0.503-1.660)	
Khat	No	169(45.8)	79(21.4)	1	0.053	1	0.011*
chewing	Yes	70(19.0)	51(13.8)	1.559(0.995-2.442)		0.434(0.227-0.828)	

*1 = Reference category, COR = Crud Odds Ratio, AOR = Adjusted Odds Ratio, WC= waist circumference, HC=hip circumference, H. pylori=helicobacter pylori, BMI=body mass index DBP= diastolic blood pressure, kg=kilogram, cm=centimeter, mmHg=millimeter mercury, *significant p<0.005*

5.6 Bivariate and multivariate analysis of factors associated with dyslipidemia

On multivariate analysis of dyslipidemia (at least one abnormality in one of lipid profile), After adjusting for traditional dyslipidemia risk factors, *H. pylori i*nfection was the only independent predictor of dyslipidemia (AOR 2.628, 95%CI 1.477-4.678, p =0.001). However, other risk factors were not associated with dyslipidemia by multivariate analysis (p > 0.05) table 6.

Table 6: Associated factor for dyslipidemia among adult cold OPD patients at JUMC, Jimma, Ethiopia (N=369).

Variable	Category	Dyslipidemia		COR (95%CI)	P-value	AOR (95%CI)	P-value
		Yes No (%)	No No (%)				
Age	< 40	144(39.0%)	53(14.4%)	1	0.031*	1	
in year	≥ 40	142(38.5%)	30 (8.1%)	0.574(0.347-0.950)		0.696(0.391-1.240)	0.219
Sex	Female	141(38.2)	53(14.4)	1		1	
	Male	145(39.3)	30(8.1)	1.817(1.097-3.008)	0.020*	0.851(0.436-1.659)	0.635
Residence	Rural	110(29.8)	46(12.5)	1		1	
	Urban	185(50.1)	37(10.0)	0.503(0.307-0.824)	0.006*	1.803(0.989-3.287)	0.055
Occupation	Un employed	106(28.7)	42(11.4)	1		1	
al status	employed	180(48.8)	41(11.4)	1.740(1.063-2.847)	0.028*	1.107(0.606-2.024)	0.741
Physical	Yes	48(13.0)	8(2.2))	1		1	
exercise	No	238(64.5)	75(20.3	1.891(0.856-4.175)	0.115	0.709(0.286-1.758)	0.458

History of HTN	No	239(64.8)	74(20.1)	1		1	
	Yes	47(12.7)	9(2.4)	1.617(0.757-3.455)	0.215	0.998(0.427-2.335)	0.997
Smoking	No	264(71.5)	80(21.7)	1		1	
	Yes	22(6.0)	3(0.8)	2.222(0.648-7.617)	0.204	1.183(0.297-4.704)	0.812
Khat	No	187(50.7)	61(16.5)	1		1	
	Yes	99(26.8)	22(6.0)	1.468(0.851-2.531)	0.167	1.477(0.747-2.921)	0.262
Regular diet	Vegetable	198(53.7)	64(17.3)	1		1	
	Meat	88(23.8)	19(5.1)	1.497(0.846-2.648)	0.166	1.061(0.560-2.013)	0.855
weight	< 56kg	113(30.6)	55(14.9)	1		1	
	≥ 56kg	173(46.9)	28(7.6)	0.333(0.199-0.555)	<0.001*	0.612(0.328-1.139)	0.121
WC	< 94cm	253(68.6)	82(22.2)	1		1	
	≥ 94cm	33(8.9)	1(0.3)	0.093(0.013-0.694)	0.210	0.148(0.016-1.377)	0.093
HC	< 102cm	255(69.1)	78(21.1)	1		1	
	≥ 102cm	31(8.4)	5(1.4)	0.527(0198-1.402)	0.200	1.453(0.454-4.655)	0.529
H. pylori	Negative	151(46.9)	22(6.0)	1		1	
	Positive	135(36.6)	61(16.5)	0.322(0.185-0.553)	<0.001*	2.628(1.477-4.678)	**0.001***
BMI	< 24.99kg/m^2	233(63.1)	81(22.0)	1		1	
	≥ 24.99kg/m^2	53(14.4)	2(0.5)	0.109(0.026-0.456)	0.002*	0.345(0.070-1.696)	0.190

*1 = Reference category, COR = Crud Odds Ratio, AOR = Adjusted Odds Ratio, WC= waist circumference, HC=hip circumference, H. pylori=helicobacter pylori, BMI=body mass index DBP= diastolic blood pressure, kg=kilogram, cm=centimeter, mmHg=millimeter mercury, *significant p<0.005*

CHAPTER SIX: DISCUSSION

H. pylori infection is a causative agent for the development of peptic ulcer and gastric cancer (44). There are evidence that indicate the role of *H. pylori* infection had pathogenesis of various extra gastric diseases (45). In the present study we found that, the prevalence of dyslipidemia, at least in one of the four lipid profile, in *H. pylori* positive patients is 151/173 (87.2%). Our result is higher than the study done in Iran with a prevalence of dyslipidemia in *H. pylori* infected individual of 60.4% (34). The probable reason of variation of the result might be due to geographical area, source population, sample size and the way they define dyslipidemia.

Our result is comparable with study done in Iran on evaluation of serum lipid profiles among *H. pylori* infected and non-infected. The study of the lipid profile showed that infected groups have statistically significantly increased rate of TC, TG, LDL-C and HDL-C compared with the non-infected groups in all $p < 0.001$ (17). The only difference from our study was with HDL-C which was not significant in *H.pylori* positive patients, the association might be due to the effect of *H.pylori* infection on lipid metabolism (46).

The current result is in agreement with a study done in Finland which states that *H. pylori* infection might modify the serum lipid concentrations in a way that could increase the risk of CHD. *H. pylori* positive had significantly ($p = 0.03$) higher concentrations of serum TG than those who were *H. pylori* negatives (47). Our results were also in consistent with the study conducted in Korea to investigate whether *H. pylori* is associated with dyslipidemia revealed that *H. pylori* infection was significantly associated with higher TC level ($r = 2.114$, $p < 0.001$), higher LDL-C level ($r = 3.339$, $p < 0.001$), lower HDL-C level ($r= -1.237$, $p < 0.001$), and higher DBP ($r = 0.539$, $p = 0.001$) (48). The result of serum lipid profile such as LDL-C, TG, and TC obtained in the present study were higher in *H. pylori* positive individuals when compared to *H. pylori* negative individuals.

Our result is also similar to a study conducted in Iraq determine lipid profile in patients with gastritis found that *H. pylori* was associated with increase in serum cholesterol, TG and LDL-C and decrease in serum HDL-C and the positivity rate was 55.83% (28).

In our study the mean ± SD of *H. pylori* positive patients were higher than *H. pylori* negative patients for TC, TG, and LDL-C (200.80 ± 43.48 mg/dl vs 173.67 ± 42.41), (185.61 ± 74.82mg/dl vs 138.18± 60.17), and (185.61 ± 74.82mg/dl vs104.27 ± 34.71) respectively with ($p < 0.05$). These findings are also supported with the results of a study conducted in Turkey on serum lipid profile in *H. pylori* infected patients, serum cholesterol concentrations were significantly higher in patients group when compared with healthy group (189.32±45.15 vs. 179.41±36.37 mg/dl ($p < 0.05$), serum TG and TC/HDL-C concentrations also were significantly higher in patients group (169.46±68.53 vs. 135.67±94.35 mg/dl ($p < 0.05$) and 3.93±1.23 vs. 3.51±1.62, ($p <0.05$) respectively (33).

The result of the present study also indicates that *H. pylori infection* increases TC, LDL-C and TG level of infected subjects compared to the negative subjects. It might be, due to LPS present in the cell walls of gram-negative bacteria *H. pylori*, there is stimulation of large quantities of cytokines (TNF-α andIL-6) which inhibit lipoprotein lipase activity. The consequence being mobilization of lipid tissue through an increase in serum TG level and in contrast, a decrease in serum HDL cholesterol level (49). Abnormal high increase of TC ($P < 0.001$) and LDL-C ($P < 0.001$) and TG level ($p<0.001$) were observed in the *H. pylori* positive patients compared to *H. pylori* negative patients. Our results were in agreement with the study done by Kim et al who showed that *H.pylori* is independently associated to increased level of TC, and LDL-C (9).

Our result was in contrast to a study conducted in Croatia, and Spain the result indicate that *H. pylori* infection was not an independent risk factor for acute myocardial infarction this is due to difference in the study design, source population and study subjects they use prospective where as our study is simple cross sectional (37).

Our study revealed that *H. pylori* was an independent risk factor with a significant positive association to TC, TG, LDL-C but *H. pylori* was inversely related with HDL-C. This findings is comparable with other study conducted in Korea (48).

CHAPTER SEVEN: CONCLUSION AND RECOMENDATION

7.1 CONCLUSION

- *H. pylori* infected patients could have a possibility to develop dyslipidemia.
- The study provides knowledge to physicians to investigate and treat dyslipidemia in context to *H. pylori* infection.
- An increase in prevalence of dyslipidemia among *H. pylori* positive patients indicates the possible modification of serum lipid profile. So, it is possible to conclude that *H. pylori* positive patients are more likely to have modified lipid profile than *H. pylori* negative patients.

7.2 RECOMMENDATIONS

Based on the above research finding the following recommendation are forwarded

- Lipid profile abnormalities in *H. pylori* infected patients could happen and may be used as valuable aids in patient's clinical management.
- Monitoring and evaluation of TC, TG, LDL-C, and HDL-C in *H. pylori* infected patient is important.
- Further studies should be conducted with larger sample size using prospective study design and advanced test for *H. pylori* determination to investigate the effect of *H. pylori* infection on serum lipid profiles.

LIMITATION OF THE STUDY

- As the study was carried out among patients attending OPD of JUMC, results cannot be generalized to general population.
- Lack of related literatures particularly in Ethiopia and across the world generally to compare and discuss with our findings.
- Being a cross-sectional study by design it cannot associate causal relationships between the factors under study.
- The test methodology used to determine the prevalence of *H. pylori* is simple qualitative rapid serum antibody test.

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ANNEX I: PARTICIPANT INFORMATION SHEET

TITLE OF THE RESEARCH: DYSLIPIDEMIA AND RISK FACTORS AMONG GASTRITIS SUSPECTED PATIENTS IN THE OUT PATIENT DEPARTMENT OF JIMMA UNIVERSITY MEDICAL CENTER, JIMMA, SOUTH WEST ETHIOPIA.

Name of Principal Investigator: Ahmedmenewer Abdu Seid

ADVISORS: 1. AKLILU GETACHEW

2. WAQTOLA CHENEKE

Name of the Organization: Jimma University, Institute of Health, Faculty of Health Sciences, School of Medical Laboratory Sciences.

Introduction: This information sheet was prepared for the aim of explaining the research that you are asked to join with research participant. This information Sheet describes about the research.

Aim of the study: To assess dyslipidemia and associated risk factor among gastritis suspected patients in the OPD of JUMC, Jimma, south west Ethiopia from January 03 to April 05, 2019.

Procedure: If the patient was agreed to take part in the study, clinical nurse was given verbal and/or written information about the study and patient was signed on consent form. Patients were kindly requested to give the correct information about themselves and the necessary measurements were performed by the assigned nurse. Then 5ml of an overnight (10-12) hour fasting blood samples for lipid profile was collected.

Risk and discomfort: Participating in this research will not cause more discomfort than is required you could go through for routine examination. If there is any discomfort, we shall offer you necessary medical treatment freely. The amount of blood taken from each volunteer throughout the study period is 5ml which will not affect your health.

Benefits: If you are participating in this study, there may not be direct benefit to you but your participation is likely to help us an important input to find the association of *H. pylori* infection with lipid profile and if the medical examination reveals any abnormalities that need immediate treatment, your doctor will be notified about the result.

Incentives and payment for participating in the study: You will not be provided with any direct incentives for your participation in this study. But the cost for your medical examination will be covered.

Confidentiality: All information about the patients will be kept confidential. The name of participant will be coded. The information sheet that links the coded number to patient name will be locked inside a computer and it will not be revealed to anyone except your physician and the principal investigator.

Right to refuse or withdraw: You have full right to withdraw from participating in the study at any time before and after consent without explaining the reason and not respond to some or all the questions. Your decision will not affect your right to get health service you are supposed to get otherwise.

Contact Address

If you have any question or concern, you can contact Ahmedmenewer Abdu at any time using the following address:

Ahmedmenewer Abdu, MSc student in Jimma University, Institute of Health, Faculty of Health Sciences, school of Medical Laboratory Sciences.

Tel: 09-22-61-89-55

Email: menewer59@gmail.com

Jimma, Ethiopia

UUNKAA ODEEFFANNOO HIRMAATAA (AFAAN OROMOO VERSION)

Mata duree qorannichaa: Faffaca'iinsa Helicoobaacter paayiloorii fi dhiibbaa inni 'lipid profile' irraan gahu shakkamtoota dhukkuba garaachaa kutaa yaala deddeebii giddu gala yuuniversiitii Jimmaa, Jimma, Itiyoophiyaa.

Maqaa qorataa olaanaa: Ahmedmenewer Abdu Seid

Gorsitoota: 1. Aklilu Getachew

2. Waqtola Cheneke

Maqaa dhaabbatichaa: Yuuniversiitii Jimmaa, Institiyuutii Fayyaa, Faakaaltii Saayinsii Fayyaa, Iskuulii Saayinsii Meedikaal Laaboraatorii.

Seensa: uunki odeeffannoo kun kan qophaa'eef waa'ee qorannoo ati akka irratti hirmaattuf gaafatamte ibsuufi. Innis waa'ee qorannoo kanaati ifa godha.

Kaayyoo qorannoo kanaa: Faffaca'iinsa Helicoobaacter paayiloorii fi dhiibbaa inni 'lipid profile' irraan gahu shakkamtoota dhukkuba garaachaa kutaa yaala deddeebii giddu gala yuuniversiitii Jimmaa (JUMC), Jimma, Ethiopia.

Adeemsa: yoo qorannoo kana irratti hirmaachuuf waliigalte, qorattoota keessaa namni tokko ykn narsiin waa'ee qorannichaa afaanin ykn barreeffamaan siif kennu; akkasumas uunkaa waliigaltee mallatteessitu siif kennu. Waa'ee kee odeeffannoo sirrii akka nuuf kennitu kabajaan sigaafachaa, safarri adda addaa narsiidhan siif godhama. Qorannoo keessatti waan hammatamtu yoo ta'e gaditeechumni qorannoo *H. pylori* dhaaf fi dhiigni miliilitirri 5 qorannoo 'lipid profile' dhaf sirraa fuudhama.

Hubaatii: qorannoo kana irratti hirmaachuun hubaatii qorannoo yaalaaf godhamuun ol hin fidu. Yoo waan sitti hin tolle sitti dhagahame yaalli fayyaa bilisaan siif godhama. Hammi dhiiga nama qorannoo kana irratti hirmaatu hnda irraa fuudhamu mililiitira 5qofa kan fayyaa namaa hin miine dha.

Bu'aa: qorannoo kana irratti hirmaachuun bu'aa kallattiin siif kennuu dhabuus garuu walitti dhufeenya h. payilori fi 'lipid profile' argachuuf kan fayyadu fi yoo qorannoo yaalaa siif godhame rakkoo fayyaa agarsiise doktorri kee waa'ee firii qorannoo keetii itti himama.

Onnachiiftuu fi kanfaltii: hirmaannaa keef onnachiiftuun kallattii godhamu hin jiru. Garuu kanfaltiin qorannoo siif godhamuu qorannichaan ni uwwifama.

Iccitii: odeeffannoon waa'ee dhukkubsattootaa hundi iccitiin qabama. Maqaan dhukkubsataa koodiidhaan qabama. Barreeffamni koodiin fi maqaa dhukkubsataa walqabsiisu kompiyuutara keessatti itti cufama. Innis qorataa olaanaa fi ogeessa si yaaleen ala eenyumattu hin kennamu.

Mirga diduu ykn kutanii keessaa bahuu: qorannicha keessaa yeroo kamittu waliigalteen duraa fi booda sababa ibsuun osoo sirraa hin eegamin keessaa bahuu dandeessa. Keessaa bahuun kee tajaajila siif kennamu irratti miidhaa hin qabu.

Teessoo

Gaafii yoo qabaattan obboo Ahmedmenewer Abdu yeroo barbaaddan teessoo kanaa gadiin gaafachuu dandeessu:

Ahmedmenewer Abdu, barataa MSc yuuniversitii Jimmaa, Institiyuutii Fayyaa, Faakaaltii Saayinsii Fayyaa, Iskuulii meedikaal laaboraatorii saayinsii.

Bilbila: 09-22-61-89-55

Email: menewer59@gmail.com

Jimma, Itiyoophiyaa

ለጥናቱ ተሳታፊዎች የሚሰጥ መረጃ (AMHARIC VERSIONINFORMATION SHEET)

የጥናቱ ርዕስ: የደም ስብ ልኬት ችግሮች (Dyslipidemia)ና ችግሩን የሚያመጡ ተያያዥ ሁኔታዎችን በጅማ ዩኒቨርስቲ ህክምና ማዕከል በተመላላሽ የጨጓራ ህሙማን ላይ ማጥናት ጅማ ፣ ኢትዮጵያ፡፡

ተመራማሪ፡ አህመደመነወር አብዱ ሰዒድ

አማካሪ፡ 1. **አክሊሉ ጌታቸው**

2. ዋቅቶላ ጨነቀ

የተቋሙ ስም፡-ጅማ ዩኒቨርሲቲ፣ ጤና ኢንስቲትዩት ፣ሜዲካል ላቦራቶሪ ትምህርት ክፍል

ወጪውን የሚሸፍነው ተቋም፡- ጅማ ዩኒቨርሲቲ

መግቢያ፡-ይህ የማብራሪያ ቅፅ አሁን እርስዎ እንዲሳተፉ የምንጠይቀዎትን ምርምር ጥናት የሚያብራራ ነው፡፡በዚህ ጥናት ለመሰሳተፍ ከመወሰንዎ በፊት ይህንን ቅፅ መረጃ ሰብሳቢዎቹ በሚያነቡበት ጊዜ በጥሞና በማድመጥ ጥያቄ ካለዎት በመጠየቅ ትክክለኛዉን መልስ ይመልሱ፡፡ በዚህ ጥናት መሳተፍ ከጀመሩ በኃላ በማንኛውም ጊዜ ጥያቄ ካለዎት መጠየቅ ይችላሉ፡፡

የጥናቱ ዓላማ፡ የደም ስብ ልኬት ችግሮች (Dyslipidemia)ና ችግሩን የሚያመጡ ተያያዥ ሁኔታዎችን በጅማ ዩኒቨርስቲ ህክምና ማዕከል በተመላላሽ የጨጓራ ህሙማን ላይ ማጥናት ፣ጅማ ፣ ደቡብ ምዕራብ ኢትዮጵያ፡፡

የጥናቱ ሂደት፡ይህን ጥናት ለማካሄድ የደም ናሙና በመውሰድ የላብራቶሪ ምርመራ ማደርግ ነው፡፡

ከጥናቱ ጋር የተያያዘ ጉዳት/አለመመቸት፡ እርስዎ በዚህ ጥናት ውስጥ በመሳተፈዎ ለከፋጉዳት የሚጋለጡበት ሁኔታ አይኖርም፡፡ደም በሚወሰድበት ወቅት አነስተኛ ህመም ሊሰማወት ይችላል፡፡እንዲሁም የመቅላት፣እና የማበጥ ሁኔታ ደም ከተወሰደበት ቦታ ላይ ሊታይ ይችላል፡፡ነገርግን እነዚህ ሁኔታዎች የከፋጉዳት የሚያስከትሉ አይደሉም፡፡

በጥናቱ የመሳተፍ ጥቅም፡ እርስዎ በዚህ ጥናት ላይ በመሳተፍዎ ነፃ የኤች ፓይሎሪና የደም ስብልኬት (lipid profile) ምርመራ ያገኛሉ፡

የጥናቱ ተሳታፊ ድርሻ፡ በዚህ ጥናት ለመሳተፍ ፍቃደኛ ከሆኑ ከጤናዎ ሁኔታ ጋር የተያያዙና ሌሎች የግል መረጃዎችን እንዲሰጡ ይጠየቃሉ፡፡ በመቀጠልም የሰዉነት ክብደተዎን እና የደም ግፊተዎን እንዲለኩና 5 ሚሊ መጠን ያለው የደም ናሙና ለተጠቀሰዉ ዓላማ እንድንወስድ ይጠየቃሉ፡፡

የጥናቱ ተሳታፊዉ መብት፡ በጥናቱ ላይ ለመሳተፍ ባይስማሙ ምንም አይነት ቅጣት የማያስከትል ሲሆን ማንኛውም እርስዎ ሊያገኙ የሚገባውን ህክምናና ተያያዥ መብት የማያሳጣ መሆኑን እናረጋግጣለን፡፡

የጥናቱ መረጃዎች ምስጥራዊነት፡ እርሰዎን በተመለከተ የምንናገኘውን መረጃ በጥናቱ ወቅትም ሆነ ከዚያ በኃላ ባሉት ጊዜያት እንዲሁም ከጥናቱ የተገኘው መረጃ ሚስጥራዊነት የሚጠበቅ ሲሆን መረጃዎቹም የሚያዙት በስም ሳይሆን በልዩ ኮድ ነው፡፡ይሽው መረጃ በጥንቃቄ የሚያዝና የተፈቀደለት ተመራማሪ እና ለህክምና ባለሙያዉ ብቻ ይህም እጅግ አስፈላጊ በሆነ ጊዜ ብቻ ካልሆነ በስተቀር ለሌላ ለማንም ሰው አይሰጥም፡፡ማንኛውም ከርስዎ ጋር የተያያዘ ውጤት በልዩ ኮድ ብቻ የሚያዝ ሲሆን ውጤቱም ለሳይንሳዊ ዓላማ ብቻ ስም በማይገልፅ ሁኔታ እንዲታተም ይደረጋል፡፡

ስለጥናቱ መረጃ ማግኘት ቢፈልጉ፡ ጥናቱን በተመለከተ ግልጽ ያልሆነ ማንኛዉንም ጥያቄ ካለዎት ነፃ ሆነው ከዚህ በታች ባለው አድራሻ መጠየቅ ይችላሉ፡፡

አህመድመነወር አብዱ ሰዒድ

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Jimma, Ethiopia

በጣም እናመሰግናለን!!

ANNEX II- CONSENT FORM

I confirm that, as I give consent to participate in the study, it is with a clear understanding of the objectives and conditions of the study and with recognition of my right to withdraw from the study if I change my idea. I have been given the necessary information about the research. I have also been assured that I can withdraw my consent at any time without penalty or loss of benefits. The proposal is explained to me in the appropriate language I understand. I_____ do here by give consent to Dr. /Mr. /Mrs. /Miss _____to include me in the proposed research.

Participant code _____

Participant (signature) _____date_____

Name of the data collector_____

Data collector (signature) _____date_____

GUCA WALIIGALTEE (AFAAN OROMOO VERSION)

Qorannicha irratti hirmaachuuf waliigaluun koo kaayyoo fi haala qorannichaa haala ifaa ta'een hubachuu fi yaada kiyya yoon jijjiire mirga addaan kutuu qabaachuu koo hubachuu nan mirkaneessa, odeeffannoo waa'ee qorannichaa ilaalchisee naaf kennamee jira. Yeroon barbaade adabbii fi tajaajila dhabuu tokko malee qorannicha keessaa bahuu akkan dandahu naaf himameera, qorannichi afaanin ani hubadhuun naaf ibsame.

Ani _____ Dr. /Mr. /Mrs. /Miss _____qorannoo yaadame keessatti hirmaachuf waliigaleera.

Koodii hirmaataa _____

Mallatoo hirmaataa _____guyyaa_____

Maqaa nama raga funaanuu_____

Mallatoo nama raga funaanuu _____guyyaa_____

Consent Form (Amharic Version)

ከላይ የተፃፈውን የመረጃ ቅፅ አንብቤ የጥናቱ ዓላማና ጥቅም በግልጽ ተረድቻለሁ በማንኛውም ጊዜ ከጥናቱ ያለምንም ችግርና መንገላታት መውጣት እንደምንችል ተገልፆልኛል፡፡ከዚህም በተጨማሪም የጥናቱን ዓላማ በሚገባኝ ቋንቋ ተረድቻለሁ፡፡በዚህ መሠረት ያለጥናት ቡድኑ አባላት ተፅዕኖ በሙሉ ፈቃደኝነት በዚህ ጥናት ውስጥ በመሣተፍ የሚጠበቅብኝን አስተዋፅዎ ለማበርከት በፊርማዬ አረጋግጣለሁ፡፡

የተሳታፊው የሚስጥር ቁጥር ----------

የተሳታፊው ፊርማ ----------ቀን ----------

የመረጃ ሰብሳቢው ስም ----------

የመረጃ ሰብሳቢው ፊርማ ----------ቀን ----------

ANNEX III: QUESTIONNAIRE

JIMMA UNIVERSITY, INSTITUTE OF HEALTH, FACULTY OF HEALTH SCIENCES, SCHOOL OF MEDICAL LABORATORY SCIENCE.

INSTRUCTIONS: This questionnaire contains a question, which are pertinent to the research objectives. You are kindly requested to answer all as much as possible and carefully by filling the blank spaces and encircling one appropriate choice from the alternatives given.

Participant Identification

Participant serial number _____ Identification code_____

Participant address_____ Phone number_____

PART I. Socio-Demographic Characteristics

S.NO	Questions	Alternatives	Comments
01	Age	----------in year	
02	Sex	1. Male 2. Female	
03	Religion	1. Muslim 2. Orthodox 3. Protestant 4. Catholic 5. Others (specify)_____	
04	Educational status	1. Illiterate 2. Read and write 3. Elementary 4. High school and above	
05	Ethnicity	1. Oromo 2. SNNP 3. Amhara 4. Tigray 5. Other specify_____	
06	Marital status	1. Single 2. Married 3. Divorced 4. Widowed	
07	Residence	1. Urban 2. Rural	

08	Occupational status	1. Civil servant 2. Housewife 3. Merchant 4. Private worker 6. Farmer 7. Other specify----------	
PART II ASSOCIATED RISK FACTORS			
10	Duration of gastritis	1. In this week 2. Before a month 3. More than 6 months 4. More than a year	
11	Do you have regular physical activity	1.Yes 2. No	
13	Do you have known	1. DM 2. Hypertension 3. Renal diseases 4. Liver disease 5. Other, specify	
14	Have you taken any medications recently?	1. Yes 2. No If yes, specify	
15	Would you drink alcohol	1. Yes 2. No	
16	Would you smoke cigarette	1. Yes 2. No	
17	Would you chew khat	1. Yes 2. No	
18	What is your regular food used	1.Injera and wott(shiroo) 2.Meat product 3.vegetables 4. Other specify-----	
PART III: Anthropometric Measurements			
19	Height(m)		
20	Body weight (Kg)		
21	Body mass index (Kg/m^2)		
22	Waist circumference		
23	Hip circumference		
Part IV Blood Pressure Measurement			
24	Systolic pressure (mmHg)		
25	Diastolic pressure (mmHg**)**		
26	Blood pressure (mmHg)		

Data collector name _____ Date _____ Signature

Part V: laboratory test result		Patients code_____	
27	*H. pylori* rapid Antibody test result	1.Positive 2. Negative	
28	HDL-C (mg/dl)		
29	TG (mg/dl)		
30	TC (mg/dl)		
31	LDL- C (mg/dl)		

Name of laboratory investigator ---------- Signature----------date----------

AFAAN OROMOO VERSION QUESTIONNAIRE

Yuuniversitii Jimmaa, Institiyuutii Fayyaa, Faakaaltii Saayinsii Fayyaa, Iskuulii Meedikaal Laaboraatorii Saayinsii.

Qajeelfama: uunkaan kuni gaafilee qorannicha waliin hidhata qaban of keessaa qaba. Gaafilee hunda hamma danda'ametti akka deebistu kabajaan gaafachaa akkasumas bakka duwwaa guutaa fi filannoo ta'u tokkotti maruun deebisaa.

Ibsa hirmaataa

Tartiiba lakkoofsa hirmaataa _____ koodii_____

Teessoo hirmaataa_____ lakk bilbilaa_____

Maqaa odeeffannoo guuraa _____ guyyaa _____ mallatoo

PART I. ibsa eenyummaa

S.NO	Questions	Alternatives	Comments
01	Umrii	Baraan	
02	Saala	1. Dhiira 2. Dhalaa	
03	Amantii	1. Muslima 2. Ortodoksii 3. Protestanti 4. Catolikii 5. Kan biraa (ibsi)_____	
04	Sadarkaa barumsaa	1. Kan hin baranne 2. Dubbisuu fi barreessuu 3. sadarkaa tokkoffaa 4. Sadarkaa lammaffaa fi isaa ol	
05	Sabummaa	1. Oromo 2. Uummattoota kibbaa 3. Amaara 4. Tigree 5. Kan biraa ibsi_____	
06	Haala gaa'ilaa	1.Kan hin heerumne/fuune 2. Kan heerumte/fuudhe 3. Kan adda bahan 4. Gursummaa	
07	Bakka jireenyaa	1. Magaala 2. Baadiyyaa	

08	Haala hojii	1. Hojjataa/ttuu mootummaa 2. Haadha manaa 3. daldalaa 4. Hojjataa dhuunfaa 5. Qotee bulaa 6. Kan biraa ibsi_	
09	Galii ji'aa (ETBirr)	1. <500 2. 501-1000 3. 1001-2000 4. 2001-4000 5. > 4000	
Kutaa II: Sababoota Walqabatan			
10	Erga dhukkubni garaachaa si eegalee hagami?	1. Torbee kana keessa 2. Ji'a tokko dura 3. Ji'a 6 oli 4. Waggaan tokkoon ol	
11	Sochii qaamaa yeroo maraa gootaa:	1. Eeyyee 2. Lakki	
12	Deebiin kee gaafii lakk 11 eeyyee yoo ta'e torbeetti si'a meeqa?	1. Guyyaa guyyaan 2. Al lama 3. Al sadii 4. Al afur	
13	Dhukkuboota kana dhukkubsattee beektaa?	1. dhibee sukkaara 2. dhiibbaa dhiigaa 3. dhukkuba kalee 4. dhukkuba tiruu 5. kan biro/ibsi	
14	Yeroo dhiyootti qoricchoota fudhatte qabdaa?	Eeyyee yoo ta'e ibsi	
15	Dhugaatii alkoolii dhugdaa?	1. Eeyyee 2. lakki	
16	Tamboo fayyadamtaa?	1. Eeyyee 2. lakki	
17	Jimaa fayyadamtaa?	1. Eeyyee 2. lakki	
18	Nyaatni yeroo mara fayyadamtu kamii ?	1. buddeena fi ittoo 2. bu'aa foonii 3. kuduraa 4. kan biraa, ibsi-----	

Kutaa III: Safara Qaamaa			
19	Dheerina (m)		
20	Ulfaatina Qaamaa (Kg)		
21	“Body mass index” (Kg/m^2)		
22	Safara mundhii(cm)		
23	'Safara 'hip(cm)		
Kutaa IV: Safara dhiibbaa dhiigaa			
24	‘Systolic pressure’ (mmHg)		
25	‘Diastolic pressure’ (mmHg**)**		
26	Dhiibbaa Dhiigaa(mmHg)		

Maqaa supparvaayizaraa---------- mallattoo ----------guyyaa----------

Waan hirmaattaniif galatoomaa!

AMHARIC VERSION QUESTIONNAIRE

ጅማ ዩኒቨርሲቲ፣ ጤና ኢንስቲትዩት፣ ጤና ሳይንስ ፋካሊቲ፣ ሜዲካል ላቦራቶሪ ትምህርት ክፍል

መመሪያ፡ ይህ መጠይቅ በውሰጡ ከጥናቱ ዓላማ ጋር የተያያዙ ጥያቄዎችን ይዟል እርሰዎም ትክክለኛዉን መልስ እንድሰጡን በትህትና እነጠይቆታልን በታቻለዎ መጠን በጥንቃቄ ባዶ ቦታዎችን በመሙላት ወይም ከተሰጡት አማራጮች ውስጥ ተገቢ የሆኑትን መልሶችን ያክብቡ ካለሆነ ጥያቄው ሲነበብለዎት ትክክለኛዉን መልሰ ይናገሩ፡፡

የተሳታፊ መለያ

የተሳታፊ ተራ ቁጥር_____

የተሳታፊ መለያ ኮድ----------አድራሻ---------- ስልክ ቁጥር----------

ክፍል1፡የማህበራዊእናስነ-ህዝብባህሪያት

ተ. ቁ	መጠይቅ	አማራጮች	አስተያየት
01	ዕድሜ	በዓምት	
02	ፆታ	1. ወንድ 2. ሴት	
03	ሀይማኖት	1.ሙስሊም 2.ኦርቶዶክስ 3.ፕሮቴስታንት 4. ካቶሊክ 5.ሌላካለይግለፅ_____	
04	የትምህርት ደረጃ	1. ምንምያልተማረ/ች 2. ማንበብናመጻፍ 3. አንደኛደረጃ 4. ሁለተኛደረጃናከዚያበላይ	
05	ብሄር	ነ. ኦሮሞ 2. ደቡብ ብሄር ብሄረሰቦች 3. አማራ 4.ትግራይ 5. ሌላ ከሆነ ይጥቀሱ----------	
06	የጋብቻ ሁኔታ	1. ያላገባ/ች 2. ያገባ/ች 3. የተፋቱ 4. በሞትየተለየ/ች	
07	የሚኖሩበት አካባቢ	1.ገጠር 2. ከተማ	
08	የሚተዳደሩበት የስራ አይነት	1. የመንግስት ሰራተኛ 2. የቤት እመቤት 3. ነጋዴ 4. የግል ሰራተኛ 5. ገበሬ 6. ሌላ ከሆነይጥቀሱ	

ክፍል 2 ፡ከጨጓራ ህመም እና የደም ሥብ ልኬት ጋ ተያያዥ መጠይቆች			
10	የጨጓራ ህመም ከጀመሮት ምን ያህል ግዜ ሆነው	1. በዚህ ሳምንት 2. ከወራት በፊት 3. ከ 6 ወር በፊት 4. ከአንድ ዓመት በፊት	
11	የአካል ብቃት እንቅስቃሴ ያደርጋሉ	1.አዎ 2.አላደርግም	
13	**ከዚህ ቀደም** በ ሐኪም የታዎቀ ህመም ዓላቸው **ለምሳሌ**	1. **ሰኳር** 2. **የደም ግፊት** 3. **ኩላሊት** 4. **ጉበት** 5. **ሌላ ካለ ይጠቀስ----**	
14	**በቅርብ የወሰዱት መድሀኒት**	**ካለ ሰሙ ይጠቀስ**	
15	**አልኮል ይጠጣሉ**	1. **አዎ** 2. **አልጠጣም**	
16	**ሲጋራ ያጨሳሉ**	1. **አዎ** 2. **አላጨሰም**	
17	**ጫት ይቅማሉ**	1. **አዎ** 2. **አልቅምም**	
18	**በብዛት አዘውትረው የሚጠቀሙት ምግብ ምንድን ነው**	1. **እንጀራና ወጥ** 2. **ስጋ ነክ መገቦች** 3. **አተክልት** 4. **ሌላ ካለ ይጠቀስ-----**	
ክፍል 3 ፡ የሰዉነት ልኬት			
19	**ቁመት**(ሜ.)		
20	**ክብደት**(ኪ.ግ)		
21	የሰዉነት አቋም መጠን (ኪ.ግ/ሜ^2)		
22	Waist circumference		
23	Hip circumference		
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24	**ሲስቶሊክ የደም ግፊት**(mmHg)		
25	**ዳያስቶሊክ የደም ግፊት**(mmHg)		
26	የደም ግፊት መጠን (mmHg)		

Data collector name _____ Date _____ Signature

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27	H. pylori rapid Antibody test result	1. Positive 2. Negative	
28	HDL-C (mg/dl)		
29	TG (mg/dl)		
30	TC (mg/dl)		
31	LDL-C (mg/dl)		

Name of laboratory investigator ---------- Signature----------date----------

ANNEX XII: ABX Pentra 400 clinical chemistry analyzer (SOP for lipid profile).

Introduction: ABX Pentra 400 clinical chemistry analyzer is an instrument that automatically performs clinical chemistry tests by mixing samples and reagents then measuring their absorbance. It has been designed as a processing and reading unit, connected to an internal built computer where the application runs.

SOP FOR TOTAL CHOLESTEROL

Introduction: Cholesterol is a component of cell membranes and a precursor for steroid hormones and bile acids synthesized by body cells and absorbed with food. The determination of the individual TC level is used for screening purposes while for a better risk assessment it is necessary to measure additionally HDL-C and LDL-C.

Method: The cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase. In the presence of cholesterol esterase, the cholesterol ester in the sample is hydrolyzed to cholesterol and free fatty acids. Produced cholesterol oxidized by cholesterol oxides to cholesterene and hydrogen peroxide. Hydrogen peroxides are detected by chromogen oxygen accepter phenol -4-aminophenazo in the presence of peroxides, then red quenoid is formed, this is proportional to cholesterol present in the sample

Specimen: Serum

Equipment

- Centrifuge
- Automated clinical chemistry analyzer: ABX PENTRA 400
- Calibrator: ABX Pentra Multical
- Controls: ABX Pentra N Control, and ABX Pentra P Control
- Sample cup
- Pipettes
- Reaction cuvette

Reagents

- ABX Pentra Cholesterol CP is ready-to-use reagent
- **Reagent contents**
  - Good's buffer pH 6.7 50 mmol/l
  - Phenol 5 mmol/l
  - 4-Aminoantipyrine 0.3 mmol/l
  - Cholesterol esterase (CHE) ≥ 200 U/l
  - Cholesterol oxidase (CHO) ≥ 50 U/l
  - Peroxidase (POD) ≥ 3 kU/l
  - Sodium azide 0.95 g/l

SOP FOR TRIGLYCERIDES

Introduction: Triglycerides constitute 95% of fat stocked in tissues and their main role is to provide energy to cells. They are synthesized on one hand in the intestine from fat brought by food and on the other hand in liver from ingested saccharides, and are then transported in the blood by chylomicrons and very low-density lipoproteins (VLDL). High levels of triglycerides are associated with important risks of atherosclerosis.

Method: Triglycerides are determined after enzymatic hydrolysis with lipase. indicator is quinoneimine formed from hydrogen peroxide, 4-aminoantipyrin and 4-chlorophenol under the catalytic influence of peroxidase.

$$\text{Triglycerides} + H_2O \xrightarrow{\text{Lipoprotein lipase}} \text{Glycerol} + \text{fatty acids}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{Glycerokinase}} \text{Glycerol-3-phosphate} + \text{ADP}$$

$$\text{Glycerol-3-Phosphate} + O_2 \xrightarrow{\text{Glycerol-3-phosphate Oxidase}} H_2O_2 + \text{DHAP}$$

$$2H_2O_2 + \text{4-AAP} + \text{p-Chlorophenol} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + 4H_2O$$

(DHAP = Dihydroxyacetone phosphate, 4-AAP = 4-aminoantipyrine)

Specimen: Serum

Equipment

- Automated clinical chemistry analyzer: ABX PENTRA 400
- Centrifuge
- Calibrator: ABX Pentra HDL Cal
- Controls: ABX Pentra N Control, and ABX Pentra P Control
- Cleaning solution:
- ABX Pentra Clean-Chem
- Sample cup
- Pipettes
- Reaction cuvette

Reagents

ABX Pentra Triglycerides CP is ready-to-use.

Reagent contents:

- Pipes free acid -----50 mmol/l
- Sodium hydroxide -----3.36 g/l
- Triton X-100---------- 1 ml/l
- Magnesium salt --------14.8 mmol/l
- p-chlorophenol---------- 2.7 mmol/l
- ATP---------- 3.15 mmol/l
- Sodium azide------- 7.99 mmol/l
- Potassium ferrocyanide------- 10 µmol/l
- 4-aminoantipyrine---------- 0.31 mmol/l
- Lipoprotein lipase-------- ≥2000 U/l
- Glycerokinase----------≥500 U/l
- Glycerol phosphate Oxidase------≥ 4000 U/l
- Peroxidase---------- ≥500 U/l

SOP FOR (HDL- C)

Introduction: The principle role of HDL in lipid metabolism is the uptake and transport of cholesterol from peripheral tissues to the liver through a process known as reverse cholesterol transport (a proposed cardioprotective mechanism)

Method: ABX Pentra 400 HDL Direct CP assay is a homogeneous method for directly measuring HDL-C levels in serum or plasma without the need for any off-line pretreatment or centrifugation steps.

Specimen: Serum

Equipment

- Automated clinical chemistry analyzer: ABX PENTRA 400
- Calibrator: ABX Pentra HDL Cal.
- Controls: ABX Pentra N Control and ABX Pentra P Control
- Sample cup
- Pipettes
- Reaction corvette
- Centrifuge

Reagents

ABX Pentra HDL Direct CP is ready-to-use.

Reagent 1:	Good's Buffer	
	Cholesterol oxidase	< 1000 U/l
	Peroxidase	< 1300 ppg U/l
	N,N-bis(4-sulphobutyl)- m-toluidine-disodium (DSBmT)	< 1 mM
	Accelerator	< 1 mM
	Preservative	< 0.06 %
Reagent 2:	Good's Buffer	
	Cholesterol esterase	< 1500 U/l
	4-Aminoantipyrine (4-AAP)	< 1mM
	Detergent	< 2 %
	Restrainer	< 0.15%
	Preservative	< 0.06 %
	Ascorbic acid oxidase	< 3000 U/l

SOP FOR LOW DENSITY LIPOPROTIEN (LDL-C)

LDL was derived by subtracting the HDL-C plus one fifth of the triglycerides from the total cholesterol

$$LDL = \text{Total Cholesterol} - (HDL + \text{Triglyceride} /5)$$

ANNEX XIII: SOP FOR DETERMINATION OF *H. PYLORI*

Introduction: *H. pylori* is a small spiral shaped bacterium that lives in the surface of the stomach and duodenum. It is implicated in the etiology of a variety of gastrointestinal diseases, including duodenal and gastric ulcer, non- ulcer dyspepsia and active and chronic gastritis.

The Wondfo (Guangzhou Wondfo Biotech Co., Ltd.) One Step serum /plasma *H. pylori* test is a simple test that utilizes a combination of *H. Pylori* antigen coated particles and anti-human IgG to qualitatively and selectively detect *H. pylori* antibodies in serum or plasma in just minutes.

Test Principle: Wondfo (one step *H. pylori* serum/plasma test) is a qualitative test based on immunoassay for the detection of *H. pylori* antibodies (Abs) in serum or plasma. In this test procedure, *H. pylori* antigen is immobilized in the test line region of the device. When the absorbent end of the test devise is immersed in to the specimen sample, the specimen is absorbed in to the devise by capillary action, mixes with the antigen-dye conjugate, and flows across the pre-coated membrane. When the antibodies to *H. pylori* levels are at or above the target cutoff (the detection limit of the test), *H. pylori* antibodies in the specimen bind to the antigen-dye conjugate and are captured by the antigen immobilized in the test region (T) of the device. This produces a colored test band and indicate a positive result. When the *H. pylori* antibodies levels of the specimen are zero or below the target cutoff. There is no visible colored band in the test region (T) of the device. This indicate a negative result. To serve as a procedure control, a colored will appear at the control region(C), if the test has been performed properly.

Test Procedure

Allow the device and specimen to equilibrate to room temperature (10-30^{o}C) prior to testing.

1. Remove the test strip from the foil pouch by tearing at the notch and place it on a level surface.
2. Holding a sample dropper vertically, add three drops of patients' serum(60-80**µ**l) of the serum or plasma into the sample pad.
3. Wait for 15 minutes and read results. do not read results after 30 minutes.

INTERPRETATION OF RESULTS

Positive (+)

Rose pink bands are visible both in the control region and the test region. A positive result indicate the presence of *H. pylori* antibody is equal to or higher than the detection limit of the test**.**

Negative (-)

Rose pink bands are visible in the control region. No color band appear in the test region. Negative results indicate the presence of *H. pylori* antibody is lower than the detection limit of the test**.**

Invalid

No visible band at all, or there is visible band in the only in the test region but not in the control region

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ቁጥር Ref. No IHRPGD/567/2018
ቀን Date 14/12/2018

Institutional Review Board (IRB)
Institute of Health
Jimma University
Tel: +251471120945
E-mail: zeleke.mekonnen@ju.edu.et

To: Ahmedmenewr Abdu

Subject: Ethical approval of research protocol

The IRB of institute of health has reviewed your research project entitled:

"Dyslipidemia and associated risk factors among suspects of gastritis in the outpatient department of Jimma University Medical Center, Jimma, south west Ethiopia"

This is to notify that this research protocol as presented to the IRB meets the ethical and scientific standards outlined in national and international guidelines. Hence, we are pleased to inform you that your protocol is ethically cleared.

We strongly recommended that any significant deviation from the methodological details indicated in the approved protocol must be communicated to the IRB before they are implemented.

With regards!

Zeleke Mekonnen (PhD)
Associate Professor, Health
Research and Postgraduate
Director

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Date 18/04/2011

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ጉዳዩ:-ትብብር ስለመጠየቅ::

በዩኒቨርስቲያችን ውስጥ ከሚካሄዱ ጥናቶች መካከል **"Dyslipidemia and associated risk factors among suspects of gastritis in the outpatient department of Jimma University Medical Center, Jimma, south west Ethiopia"** በሚል ርዕስ ምርምር ጥናታቸው የሚሰሩ መሆኑን እየገለጽን ለተመራማሪዋ **አህመድመነወር አብዱ** እና ለመረጃ ሰብሳቢዎቻቸው አስፈላጊው ትብብር እንዲደረግላቸው በትህትና እንጠይቃለን::

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School of Medical Laboratory Sciences

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Date 11/07/2011

ለሚመለከተው ሁሉ

ጉዳዩ፡-<u>ትብብር እንዲደረግላቸዉ ስለመጠየቅ</u>

ከላይ በርሱ ለመግለፅ እንደተሞከረዉ በጅማ ዩኒቨርስቲ፡ የድህረ ምረቃ ት/ቤት በክሊኒካል ኬሚስትሪ የትምርት ዓይነት ሁለተኛ ዲግሪያቸዉን እየሰሩ ያሉት ተማሪ አህመድመነወር አብዱ **Dyslipidemia and associated risk factors among gastritis suspected patients in the outpatient department of Jimma university medical center, Jimma, south west Ethiopia."** በሚል ርዕስ ላይ ጥናት ስለሚያደርጉ ለዚህ ጥናት የሚያስፈልጉ እቃዎችን ከዚህ ደብዳቤ ዝርዝር መሰረት በእናንተ በኩል የበላቦራቶሪ ሪኤጀንት 1ገጽ አባሪ ያደረግን ስለሆነ አስፈላጊዉን ትብብር ሁሉ እንድታደርጉላቸዉ እንጠይቃለን፡፡

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DECLARATION

I, the undersigned, declare that this thesis paper is my original work and has not been presented for degree in this or any other university and all source of materials used for this thesis has been fully acknowledge.

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This paper has been submitted with my approval as internal examiner

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