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Faculity of medicine

The relationship between the biochemical diagnostic biomarkers of coronary artery disease with the clinical presentation and electrocardiographic findings

BY

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بسم الله الرحمن الرحيمِ

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Certificate

I certify that this research was prepared under my supervision at the Faculty of medicine, University of diyala

Signature……

Dr. Suad Muslih Aldeen Abdalmajeed

Supervisor

In view of the available recommendations, I forward this research for debate by the examining committee

Signature……

Committee Certification

We, the examining committee, after reading this research and examining the students in its content find it an adequate as research project. In medicine science.

Signature…….

…….

Dedication

To all the illuminating stars who have led me here and will continue to do so, to my family, my backbone in this life (My Father, My Mother, Sisters, Brothers, and Enas), to my best friend, my sister from another mother (Rose) I dedicate this work to all of them, who would not have thrived without .

I dedicate this work to those who value its meaning and creativity. I express my heartfelt gratitude and thanks to those who contributed to the success of my study and those who stood by me in the most difficult circumstances and motivated me.

Acknowledgments

Above all, we give thanks to Allah, the Most Glorious, the Most Merciful, and the Most Compassionate.

THANK YOU TO MY SUPERVISOR, DR. SUAD MUSLIH ALDEEN ABDALMAJEED.

For her assistance and supervision of my research. We pray to our God to keep her in good health and to help her progress in all aspects of life.

We want to express our heartfelt gratitude to everyone who helped us fill out the questionnaire.

Abstract

Coronary artery disease is a major cause of morbidity and mortality around the world. Noninvasive imaging tests are important in diagnosing coronary artery disease, risk stratification and revascularization guidance.

The electrocardiograph is widely used. This research review accuracy and clinical significance of these methods for diagnosing and managing coronary artery disease in this study.

In addition, review diagnostic biomarkers for coronary artery disease, compare the utility of other noninvasive tests—coronary CT angiography, and cardiac MRI—in assessing ischemia and myocardial infarction.

And demonstrate the effect of blood glucose, cholesterol, TG, and HDL levels on the incidence of coronary artery disease.

This research was conducted at Baquba teaching hospital . information were collected for about 100 patients admitted to coronary care unit using questionnaires.

The data were analysed statistically according to chi -square for independence

Test using SPSS software version 26

Aim

To expect people how are at risk of coronary artery disease

And early detectation by use non invasive tests and proper management and determine if the patient need medical or surgical intervention

Abbreviations

- CABG =coronary artery bypass graft
- CCTA =coronary computed tomography angiography
- CHD=coronary heart disease
- CHF=congestive heart failure
- CT= computed tomography
- DM=diabetes mellitus
- ECG=electrocardiogram
- HDL=high density lipoprotein
- HT=hypertension
- MI =myocardial infarction
- MRI=manetic resonance imaging
- NSTEMI=non st elevation myocardial infarction
- RV=right ventricule
- RBG= random blood glucose
- SIHD=stable ischemic heart disease
- STEMI=st elevation myocardial infarction
- TG =triglyceride

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Chapter 1

1.1 Overview

Numerous clinical trials indicate that we have reached a plateau in ability to reduce the incidence of coronary heart disease and cardiovascular disease

using traditional diagnostic evaluation, prevention, and treatment strategies for the top cardiovascular risk factors of hypertension, diabetes, dyslipidemia, obesity, and smoking.

Optimal nutrition, optimal exercise, optimal weight and body composition, moderate alcohol consumption, and no smoking can prevent approximately 80% of heart disease (heart attacks, angina, coronary heart disease, and CHF). Statistics show that despite currently defined 'normal' levels of the five risk factors listed above, approximately 50% of patients continue to have CHD or myocardial infarction (MI).

The 'CHD gap' is a term used to describe this situation

More precise definitions and assessments of these top risk factors are INDECATED, including 24 hours blood pressure results, lipid profiles, fasting and 2 hour dysglycemia parameters, visceral obesity and body composition, and the effects of adipokines on cardiovascular risk. There are numerous traumatic factors from the environment that damage the cardiovascular system but there are only three finite vascular endothelial responses, which are inflammation, oxidative stress and immune vascular event. Furthermore, in order to correlate the incidence of CHD risk factors to the presence or absence of functional or structural damage to the vascular system, preclinical and clinical CHD, This is possible through the use of advanced and updated CV risk scoring systems, new and CV risk factors and biomarkers, genetics, nutrigenomics, metabolomics micronutrient testing, cardiovascular, genetic expression testing, and noninvasive cardiovascular testing.

1.2 Literature reviews :

Hayato Tada *et al* 2018

Result /

“accumulated evidence, including infantile cases exhibiting progression/regression of systemic xanthomas associated with LDL cholesterol levels, have shown that the elevation of LDL cholesterol seems to be the major cause of development of atherosclerosis”[1]

Ahmed AlBadri *et al* 2019.

Result/

Approximately 50% of patients with signs and symptoms of ischemia who undergo elective coronary angiography have no obstructive coronary epicardial atherosclerotic disease (CAD) . However, two-thirds of these patients have evidence of coronary microvascular dysfunction (CMD) . CMD has been implicated as the primary cause of angina in these patients and has been associated with increased risk of adverse cardiovascular events . While more than 90% of patients with normal coronary angiography have evidence of atherosclerosis by intravascular ultrasound (IVUS)[2]

Godo S, *et al* 2021

Result/

accumulating evidence has shown that structural and functional abnormalities of coronary microvasculature are highly prevalent, associated with adverse clinical outcomes in patients with various cardiovascular diseases[3]

Chapter 2

2.1 Introduction

clinical practice strategies for ischemic heart disease prevention and treatment have evolved in recent years, this condition a significant burden on mortality and morbidity [4].

At the moment clinical data have provided a wealth of information about the causes of myocardial ischemia [5,6,7,8,9,10,11].

However, clinical, angiographic all point to a more complex factors beyond role of atherosclerosis.

It is critical to examine the proposed IHD samples to better understand this complex disease and provide best management.

more than one regulatory mechanism must fail simultaneously to cause an event.

2.2 Pathophysiology

The proximal section of the coronary tree is represented by epicardial coronary arteries with diameters ranging from 250 m to 2-5 mm [12].

these vessels contribute only a small amount to coronary vascular resistance. Epicardial arteries are responsive to flow dependent dilatation and are subjected to shear stress that vary every heartbeat, during the phasicity of coronary blood flow. Shear stress's vasodilatory effects are mediated by endothelial-dependent vasodilatation.

2.3 Definition

Coronary artery disease is a common condition characterized by the formation of atherosclerotic plaques in the coronary vessel .

This reduces blood flow and oxygen delivery to the myocardium.

It is a major cause of morbidity and mortality in the world.

To avoid the high morbidity and mortality associated with this condition, it must be diagnosed and treated as soon as possible.

This activity depicts the evaluation, diagnosis, and management of coronary artery disease, as well as the role of the healthcare team in assessing and treating patients with this condition.

2.4 Coronary artery disease is typically classified as follows:

Stable Ischemic heart disease

Acute coronary syndrome (nonstable)

ST-elevation (STEMI)

Non ST elevation (NSTEMI)[13-16].

2.5 Myocardial infarction due to causes other than atherosclerosis

MI is caused by factors other than atherosclerosis. There are several different etiologies among it.

A rare cause is coronary artery embolization. The emboli can form in left atrium as a result of atrial fibrillation, or in left ventricle as a result of ventricular aneurysm or severely impaired left ventricle systolic function, or in prosthetic or infected native heart valves. Systemic hypotension, as a result of any etiology of shock, can result in myocardial ischemia and subsequent infarction. Increased oxygen demand (like severe anemia, tachyarrhythmias, or hyperthyroidism), significant ischemia and subsequent infarction can occur, particularly in patients with moderate epicardial coronary artery stenosis.

coronary artery dissection is becoming widely accepted cause of AMI. This happens when there is sudden tear in the wall of the coronary artery, due to hematoma formation, thrombus formation resulting in decreased blood flow distally . It is more common in younger patients and it is more common during pregnancy. drug-induced, such as cocaine use, contributes to MI by reducing blood supply to the heart muscle,Takayasu's arteritis and giant cell arteritis have been linked to MI and idiopathic Coronary spasm

.

2.6 Clinical presentation and electrocardiograph

Coronary artery disease can be manifested as either stable ischemia or acute coronary syndrome .

The treatment is determined by the type of disease.

We will discuss each subtype's management separately:

2.6.1.Stable Ischemic Heart Disease

The most common symptom of stable ischemic heart disease is stable angina. Stable angina is present as substernal chest pain or tightness that worsens with emotional stress or exertion relieved by rest or nitroglycerin and lasting for at least two months.

It is important to understand that classic anginal symptoms may be absent and may present atypical symptoms and exertional dyspnea in certain groups such as old age, diabetic patients and women.

2.6.2.Acute Coronary Syndrome

characterized by sudden onset substernal chest pain or tightness that typically radiates to the neck and left arm accompanied by dyspnea , dizziness, syncope, cardiac arrest, palpitations, or new-onset congestive heart failure. ECG is required to assess for STEMI, and is typically performed by emergency medical services . STEMI is identified by the presence of 1 mm ST elevation in contiguous limb or precordial leads .

with the exception of V2 and V3

To be diagnosed with STEMI in V2 and V3, men must have 2 mm elevations. STEMI include new-onset left bundle branch block . If STEMI is present, emergency PCI

within 2 hours is required. If the nearest PCI-capable facility is more than 2 hours away, intravenous thrombolytic therapy is recommended after ruling out any contraindications.

It is critical to distinguish a true STEMI from other conditions like STEMI on ECG, such as acute pericarditis.

2.7 Cardiac markers for assessing the acute coronary syndromes

Markers of myocardial injury play important role in the assessment and management of patients presenting with an acute coronary disease, a term that refers to acute myocardial ischemia ranging from angina to Q-wave myocardial infarction.

Markers of plaque inflammation , platelet reactivity, and thrombosis can all be used to detect coronary artery lesion instability. Several markers are released from the damaged myocyte when there is severe impairment of coronary blood flow due to myocardial injury. Creatine kinase-MB isoenzyme has long been used as a standard marker for myocardial infarction.

creatine kinase-MB remains an important component in determining re-infarction, as well as monitoring reperfusion after thrombolytic therapy and asess re infarction .

although there are many cardiac markers for myocardial infartion , cardiac troponins have the highest sensitivity and specificity for detecting myocardial damage.

Troponin can detect minor myocardial damage , infarct size, detection of perioperative myocardial infarction, risk stratification, and therapeutic decision making in patients with acute coronary syndromes.

2.8 MRI Technique

Cine balanced steady-state free precession (bSSFP) imaging is used to assess myocardial function because it provides optimal contrast between the blood pool (bright signal) and myocardium (intermediate-low signal).

Cine MRI can assess ventricular and valvular function

Transverse cine images provide impression of left ventricular function, regional function (scar, aneurysm), and valvular function (stenosis , regurgitation , valve morphology). Short-axis cine images used to calculate ventricular volume

(ejection fraction is very important for clinical decision-making and prognostication). LGE-MRI is standard for detecting myocardial scar.

Using inversion recovery methods, a distinction is made between normal gadolinium-free intact cells and scar myocardium white due to gadolinium accumulation).

2.9 Ct technique

CCTA noninvasive and diagnostic tool for a evaluation patients with suspected or kown case CAD and stable angina .

there is large evidence demonstrating sensitivity ranging from 94 to 99% and a specificity of 64-83% for the detection of significant coronary stenoses [14]. CCTA is a reliable diagnostic imaging tool for excluding obstructive CAD, particularly in low- to intermediate-risk settings [17-19].

trials and large-scale registries demonstrated that CCTA is a valuable alternative to invasive diagnostic testing [17-19],

for asymptomatic, younger patient, and less extensive coronary artery disease.

2.10 Management

2.10.1.Management of SIHD

This include Non-pharmacologic and pharmacologic interventions .

Smoking cessation, regular exercise, good diabetes and hypertension control, weight loss, and a healthy diet.

Cardioprotective and antianginal medications are examples of pharmacologic interventions.

Every patient should receive medical therapy , which includes low-dose aspirin, beta-blockers, as-needed nitroglycerin, and statins.

If this does not control the symptoms, beta-blocker therapy should be increased and the addition of long-acting nitrates and calcium channel blockers [20].

To treat refractory anginal symptoms, ranolazine could be added. If maximal medical treatment fails to relieve angina, cardiac catheterization should be performed and a decision for percutaneous coronary intervention or coronary artery bypass graft (CABG) should be made [21].

2.10.2.Management of acute coronary syndrome

Upon presentation, all patients should receive sublingual aspirin (324 mg). After ensuring that there are no contraindications to nitrates such as hypotension, RV failure, or consumption of phosphodiesterase inhibitors in the previous hours, nitrates should be administered for pain relief. High-dose statin therapy and beta-blockers should also be started as soon as possible. Based on the patient's profile, P2Y12 inhibitors like prasugrel, ticagrelor should be started.

Patients with NSTE ACS should be treated with anticoagulants such as heparin or enoxaparin. For patients with intermediate to high TIMI scores early invasive therapy within 24 hours is recommended [22,23].

Follow up with cardiologists and family doctor are essential for effective long-term management of coronary artery disease.

lifestyle modification and Medications are critical.

2.11. Complications

The most common complications associated with coronary artery disease are arrhythmias, congestive heart failure, mitral regurgitation, ventricular free wall rupture, pericarditis , aneurysm formation, and mural thrombi.[24,25,26]

Chapter3

methodology

This research was conducted at Baquba teaching hospital . information were collected for about 100 patients admitted to coronary care unit using questionnaires designed accurately.

And these patients undergo investigation tests like

ECG,ECO,RBG,TROPONIN, CHOLESTROL,TG,HDL

Then data were analysed statistically according to chi -square for independence

Test using SPSS software version 26

Chapter 4

Result

The result show high ratio of patients who develop ischemia have risk factors

As DM , HT , smoking, high level of cholestrol and TG and low HDL

as shown in(fig.4.10) (table 11),(fig.4.11) (table 12),(fig.4.14) (table 15) ,(fig.4.18) (table 19),(fig.4.19)(table 20), (fig.4.20) (table 21)

respectively .

most of patients were male and between 50-60 years as shown in (fig.4.2) (table2),(fig.4.6)(table 7) respectively .

High ratio of patients with IHD come to hospital with chest pain and dysnea

Symptoms as shown in (fig.4.3) (table 3) (fig 4.4)(table 4)

And diagnosis can be reached by ECG and level of troponin as shown in

(fig. 4.15) (table 16), (fig.4.16) (table 17) respectively .

age / patient develop CAD

Crosstab

			patient develop CAD		
			Yes	No	Total
Age	40 - 49 years	Count	12	0	12
		% within age	100.0%	0.0%	100.0%
		% within patient develop CAD	14.0%	0.0%	12.0%
		% of Total	12.0%	0.0%	12.0%
	50 - 59 years	Count	27	3	30
		% within age	90.0%	10.0%	100.0%
		% within patient develop CAD	31.4%	21.4%	30.0%
		% of Total	27.0%	3.0%	30.0%
	60 - 69 years	Count	28	4	32
		% within age	87.5%	12.5%	100.0%
		% within patient develop CAD	32.6%	28.6%	32.0%
		% of Total	28.0%	4.0%	32.0%
	70 - 79 years	Count	11	5	16
		% within age	68.8%	31.3%	100.0%
		% within patient develop CAD	12.8%	35.7%	16.0%
		% of Total	11.0%	5.0%	16.0%
	80 - 89 years	Count	7	1	8
		% within age	87.5%	12.5%	100.0%
		% within patient develop CAD	8.1%	7.1%	8.0%
		% of Total	7.0%	1.0%	8.0%
	90 - 99 years	Count	1	0	1
		% within age	100.0%	0.0%	100.0%
		% within patient develop CAD	1.2%	0.0%	1.0%
		% of Total	1.0%	0.0%	1.0%
	20 - 29 years	Count	0	1	1
		% within age	0.0%	100.0%	100.0%
		% within patient develop CAD	0.0%	7.1%	1.0%
		% of Total	0.0%	1.0%	1.0%
Total		Count	86	14	100

	% within age	86.0%	14.0%	100.0%
	% within patient develop CAD	100.0%	100.0%	100.0%
	% of Total	86.0%	14.0%	100.0%

Table 1

age to patient develop CAD

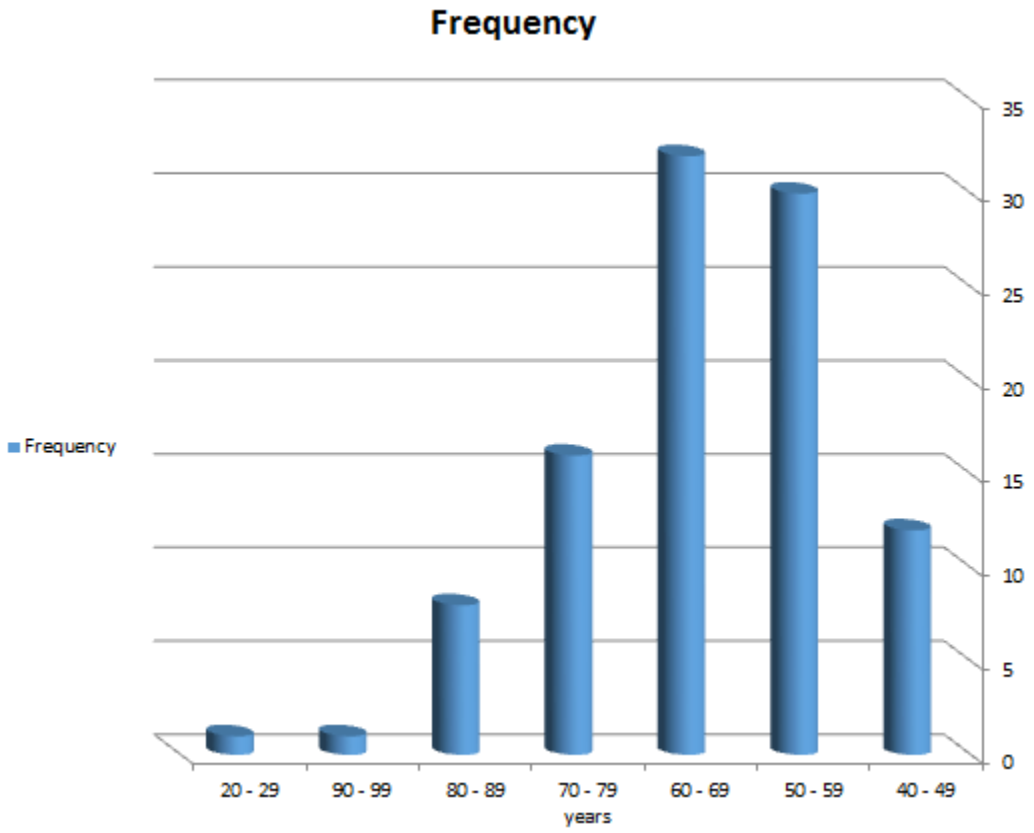

Figure 4.1

age to patient develop CAD

sex * patient develop CAD

Crosstab

			patient develop CAD		
			Yes	No	Total
Sex	Male	Count	59	9	68
		% within sex	86.8%	13.2%	100.0%
		% within patient develop CAD	68.6%	64.3%	68.0%
		% of Total	59.0%	9.0%	68.0%
	Female	Count	27	5	32
		% within sex	84.4%	15.6%	100.0%
		% within patient develop CAD	31.4%	35.7%	32.0%
		% of Total	27.0%	5.0%	32.0%
Total		Count	86	14	100
		% within sex	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

Table 2

type of sex to patient develop CAD

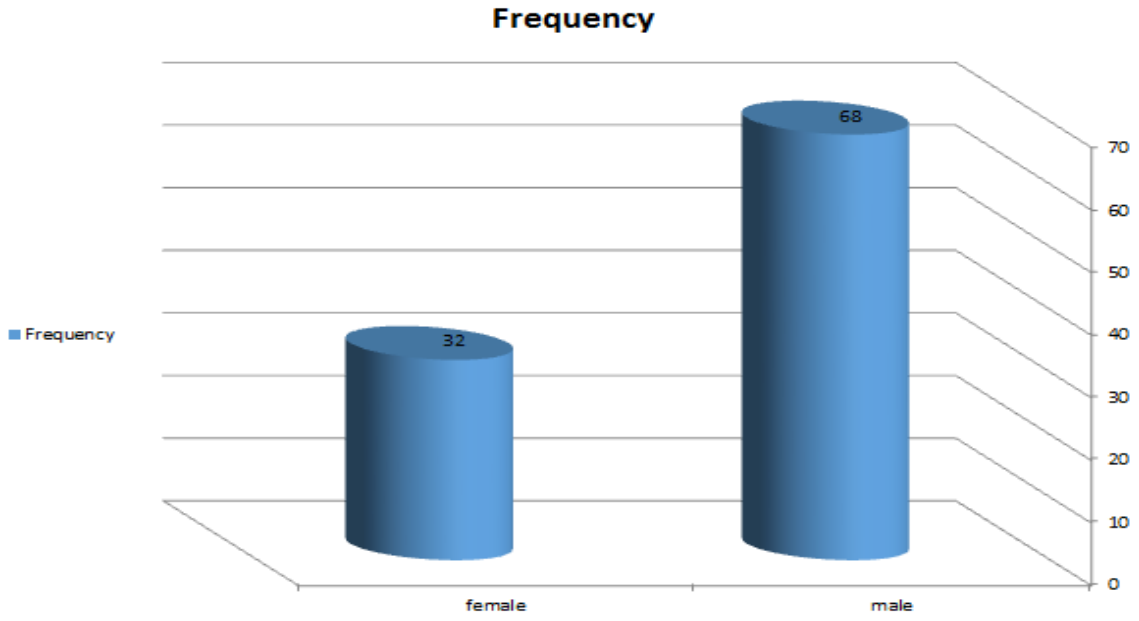

Figure 4.2

type of sex to patient develop CAD

chest pain * patient develop CAD

Table 3

chest pain in patient develop CAD

			patient develop CAD		
			Yes	No	Total
chest pain	Yes	Count	73	10	83
		% within chest pain	88.0%	12.0%	100.0%
		% within patient develop CAD	84.9%	71.4%	83.0%
		% of Total	73.0%	10.0%	83.0%
	No	Count	13	4	17
		% within chest pain	76.5%	23.5%	100.0%
		% within patient develop CAD	15.1%	28.6%	17.0%
		% of Total	13.0%	4.0%	17.0%
Total		Count	86	14	100
		% within chest pain	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

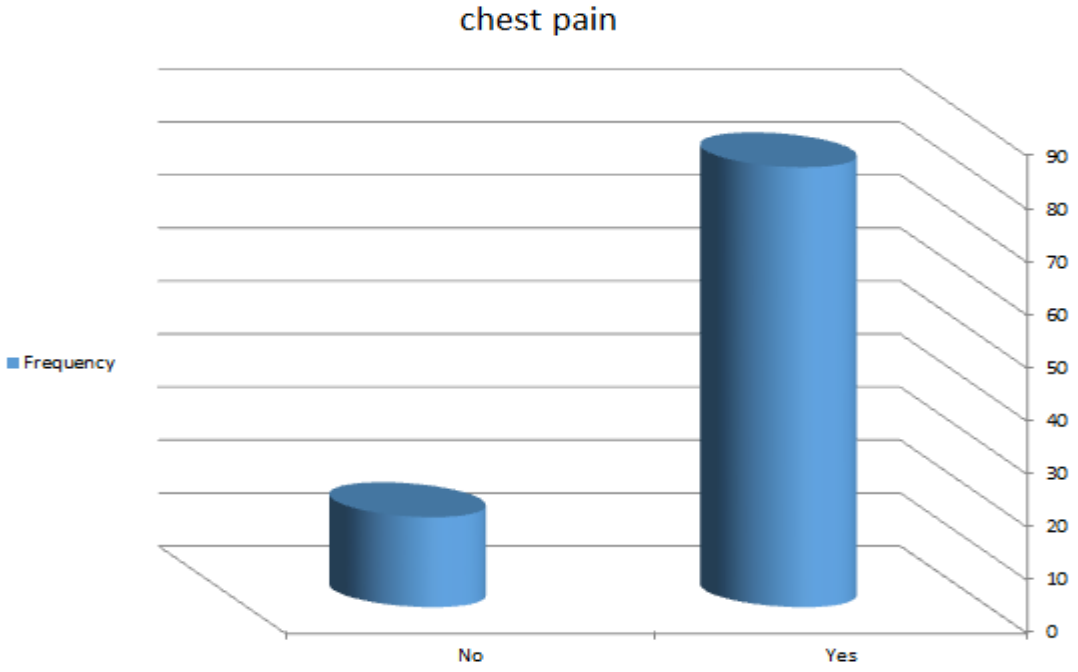

Figure 4.3

chest pain in patient develop CAD

dysnea * patient develop CAD

Crosstab

			patient develop CAD		
			Yes	No	Total
Dysnea	Yes	Count	52	6	58
		% within dysnea	89.7%	10.3%	100.0%
		% within patient develop CAD	60.5%	42.9%	58.0%
		% of Total	52.0%	6.0%	58.0%
	No	Count	34	8	42
		% within dysnea	81.0%	19.0%	100.0%
		% within patient develop CAD	39.5%	57.1%	42.0%
		% of Total	34.0%	8.0%	42.0%
Total		Count	86	14	100
		% within dysnea	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

Table 4

dysnea in patient develop CAD

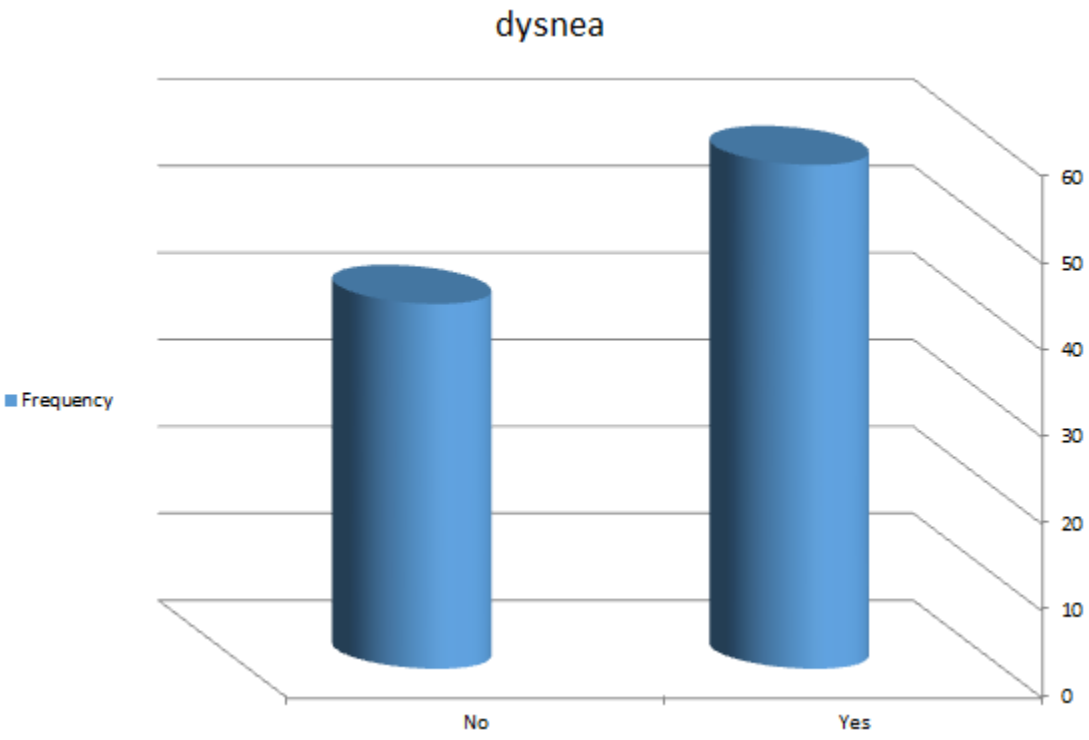

Figure 4.4

dysnea in patient develop CAD

palpitation * patient develop CAD

Table 5

palpitation in patient develop CAD

			patient develop CAD		
			Yes	No	Total
palpitation	Yes	Count	2	2	4
		% within palpitation others	50.0%	50.0%	100.0%
		% within patient develop CAD	2.3%	14.3%	4.0%
		% of Total	2.0%	2.0%	4.0%
	No	Count	84	12	96
		% within palpitation others	87.5%	12.5%	100.0%
		% within patient develop CAD	97.7%	85.7%	96.0%
		% of Total	84.0%	12.0%	96.0%
Total		Count	86	14	100
		% within palpitation others	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

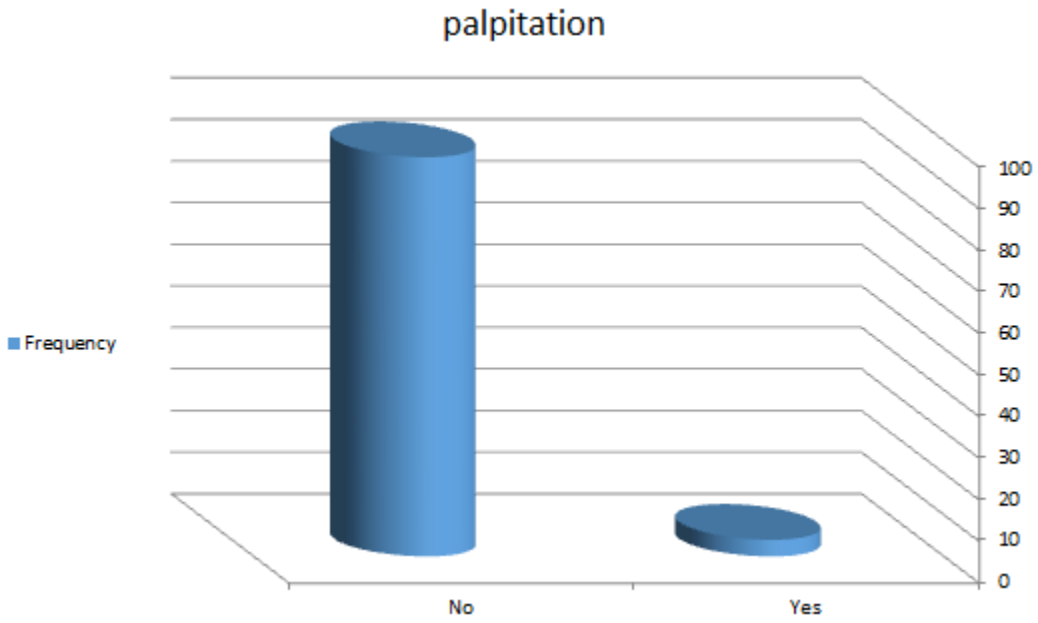

Figure 4.5

palpitation in patient develop CAD

others * patient develop CAD

Crosstab

			patient develop CAD		
			Yes	No	Total
others	Yes	Count	25	2	27
		% within others	92.6%	7.4%	100.0%
		% within patient develop CAD	29.1%	14.3%	27.0%
		% of Total	25.0%	2.0%	27.0%
	No	Count	61	12	73
		% within others	83.6%	16.4%	100.0%
		% within patient develop CAD	70.9%	85.7%	73.0%
		% of Total	61.0%	12.0%	73.0%
Total		Count	86	14	100
		% within others	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

Table 6

Others symptoms in patients develop CAD

duration * patient develop CAD

table 7

duration * patient develop CAD

			patient develop CAD		
			Yes	No	Total
duration	3 hours	Count	14	0	14
		% within duration	100.0%	0.0%	100.0%
		% within patient develop CAD	16.3%	0.0%	14.0%
		% of Total	14.0%	0.0%	14.0%
	2 hours	Count	7	0	7
		% within duration	100.0%	0.0%	100.0%
		% within patient develop CAD	8.1%	0.0%	7.0%
		% of Total	7.0%	0.0%	7.0%
	5 hours	Count	7	0	7
		% within duration	100.0%	0.0%	100.0%
		% within patient develop CAD	8.1%	0.0%	7.0%
		% of Total	7.0%	0.0%	7.0%
	6 hours	Count	1	0	1
		% within duration	100.0%	0.0%	100.0%
		% within patient develop CAD	1.2%	0.0%	1.0%
		% of Total	1.0%	0.0%	1.0%
	7 hours	Count	9	1	10
		% within duration	90.0%	10.0%	100.0%
		% within patient develop CAD	10.5%	7.1%	10.0%
		% of Total	9.0%	1.0%	10.0%
	1 hours	Count	9	3	12
		% within duration	75.0%	25.0%	100.0%
		% within patient develop CAD	10.5%	21.4%	12.0%
		% of Total	9.0%	3.0%	12.0%
	24 hours	Count	18	4	22
		% within duration	81.8%	18.2%	100.0%
		% within patient develop CAD	20.9%	28.6%	22.0%
		% of Total	18.0%	4.0%	22.0%
	minutes	Count	1	0	1
		% within duration	100.0%	0.0%	100.0%
		% within patient develop CAD	1.2%	0.0%	1.0%
		% of Total	1.0%	0.0%	1.0%
	half hour	Count	3	2	5

		% within duration	60.0%	40.0%	100.0%
		% within patient develop CAD	3.5%	14.3%	5.0%
		% of Total	3.0%	2.0%	5.0%
	15 min.	Count	6	3	9
		% within duration	66.7%	33.3%	100.0%
		% within patient develop CAD	7.0%	21.4%	9.0%
		% of Total	6.0%	3.0%	9.0%
	4 hours	Count	9	1	10
		% within duration	90.0%	10.0%	100.0%
		% within patient develop CAD	10.5%	7.1%	10.0%
		% of Total	9.0%	1.0%	10.0%
	8 hours	Count	2	0	2
		% within duration	100.0%	0.0%	100.0%
		% within patient develop CAD	2.3%	0.0%	2.0%
		% of Total	2.0%	0.0%	2.0%
Total		Count	86	14	100
		% within duration	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

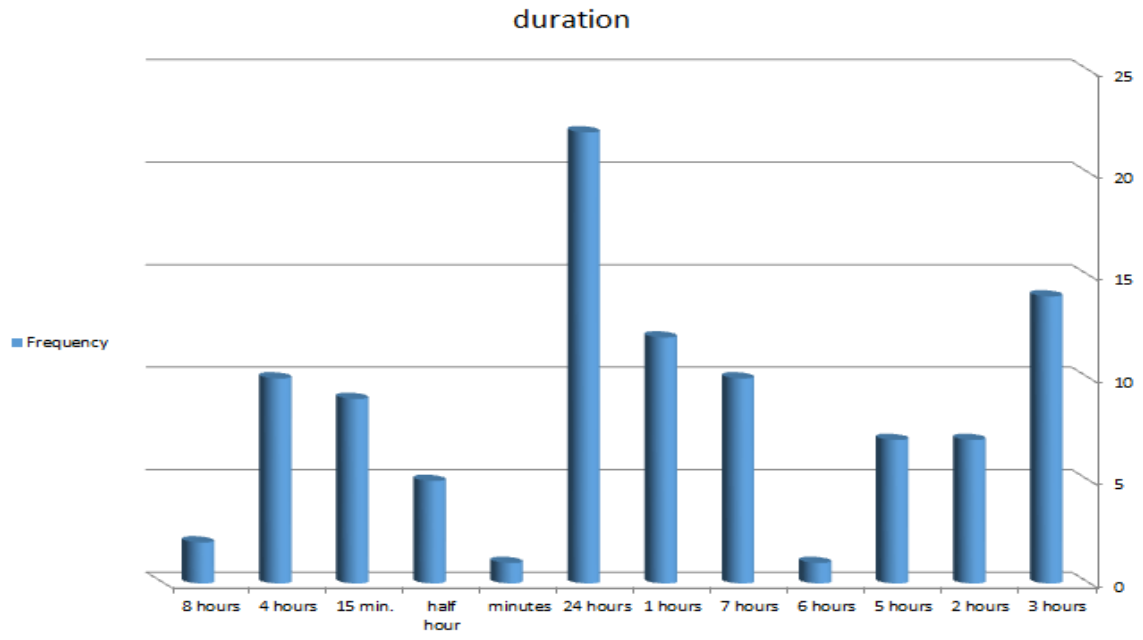

Figure 4.6

duration * patient develop CAD

HF * patient develop CAD

Table 8

HF in patient develop CAD

			patient develop CAD		
			Yes	No	Total
HF	Yes	Count	16	0	16
		% within HF	100.0%	0.0%	100.0%
		% within patient develop CAD	18.6%	0.0%	16.0%
		% of Total	16.0%	0.0%	16.0%
	No	Count	70	14	84
		% within HF	83.3%	16.7%	100.0%
		% within patient develop CAD	81.4%	100.0%	84.0%
		% of Total	70.0%	14.0%	84.0%
Total		Count	86	14	100
		% within HF	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

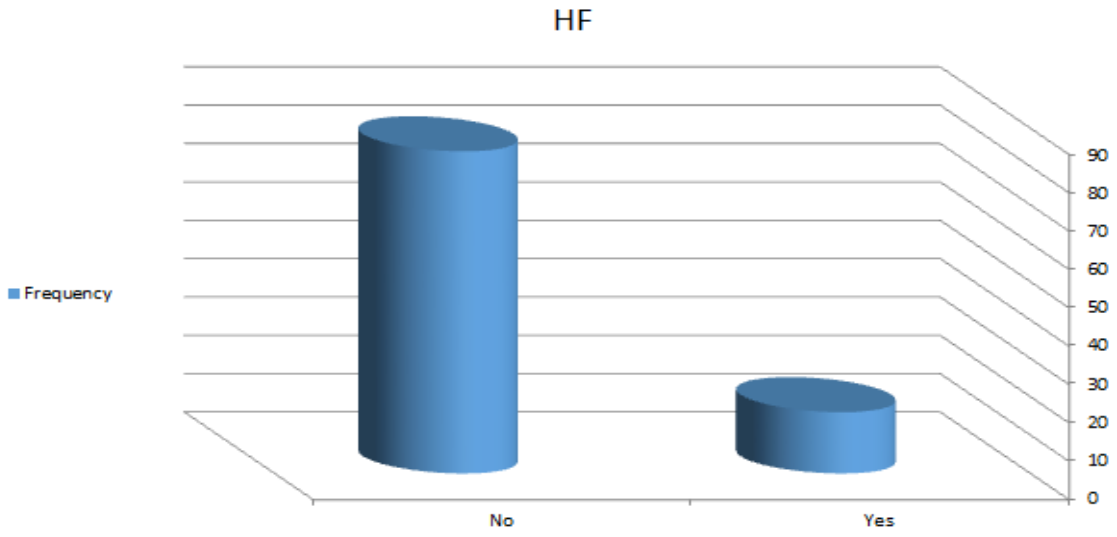

Figure 4.7

HF in patient develop CAD

arrhythmea * patient develop CAD

table 9

arrhythmea in patient develop CAD

			patient develop CAD		
			Yes	No	Total
Arrhythmea	Yes	Count	47	6	53
		% within arrhythmea	88.7%	11.3%	100.0%
		% within patient develop CAD	54.7%	42.9%	53.0%
		% of Total	47.0%	6.0%	53.0%
	No	Count	39	8	47
		% within arrhythmea	83.0%	17.0%	100.0%
		% within patient develop CAD	45.3%	57.1%	47.0%
		% of Total	39.0%	8.0%	47.0%
Total		Count	86	14	100
		% within arrhythmea	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

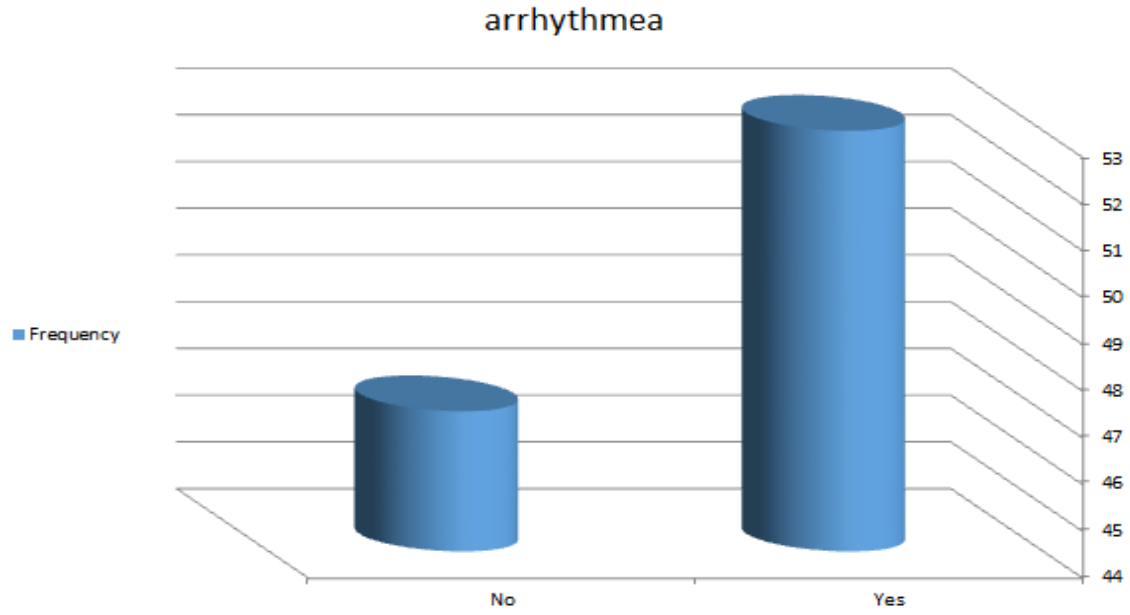

Figure 4.8

arrhythmea in patient develop CAD

stroke * patient develop CAD

Table 10

stroke in patient develop CAD

			patient develop CAD		
			Yes	No	Total
stroke	Yes	Count	6	0	6
		% within stroke	100.0%	0.0%	100.0%
		% within patient develop CAD	7.0%	0.0%	6.0%
		% of Total	6.0%	0.0%	6.0%
	No	Count	80	14	94
		% within stroke	85.1%	14.9%	100.0%
		% within patient develop CAD	93.0%	100.0%	94.0%
		% of Total	80.0%	14.0%	94.0%
Total		Count	86	14	100
		% within stroke	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

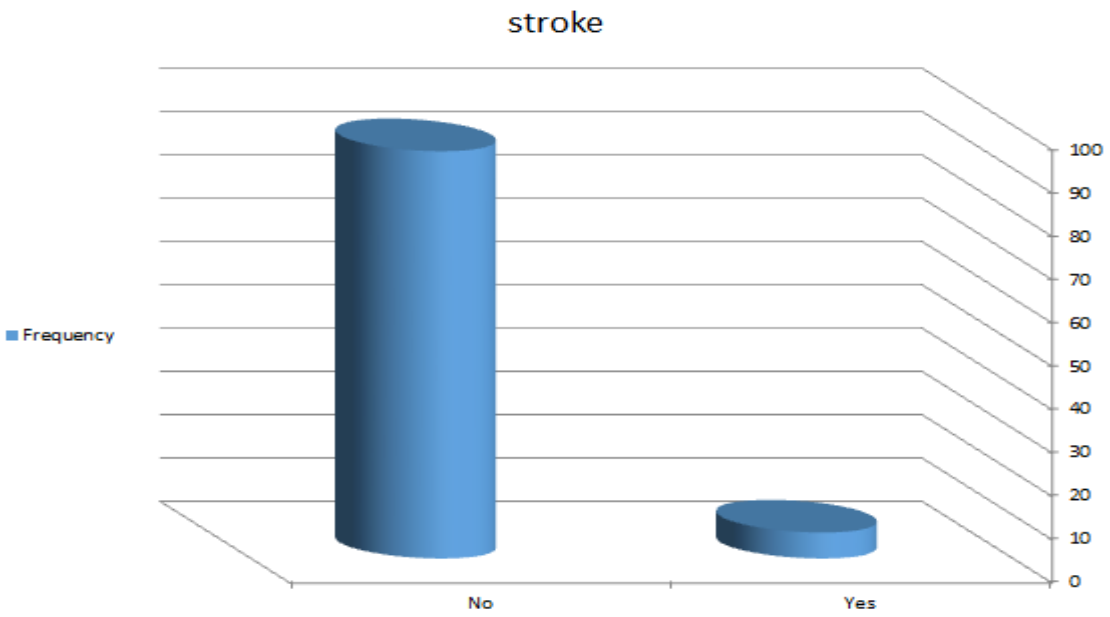

Figure 4.9

stroke in patient develop CAD

DM * patient develop CAD

Table 11

DM in patient develop CAD

			patient develop CAD		
			Yes	No	Total
DM	Yes	Count	50	9	59
		% within DM	84.7%	15.3%	100.0%
		% within patient develop CAD	58.1%	64.3%	59.0%
		% of Total	50.0%	9.0%	59.0%
	No	Count	36	5	41
		% within DM	87.8%	12.2%	100.0%
		% within patient develop CAD	41.9%	35.7%	41.0%
		% of Total	36.0%	5.0%	41.0%
Total		Count	86	14	100
		% within DM	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

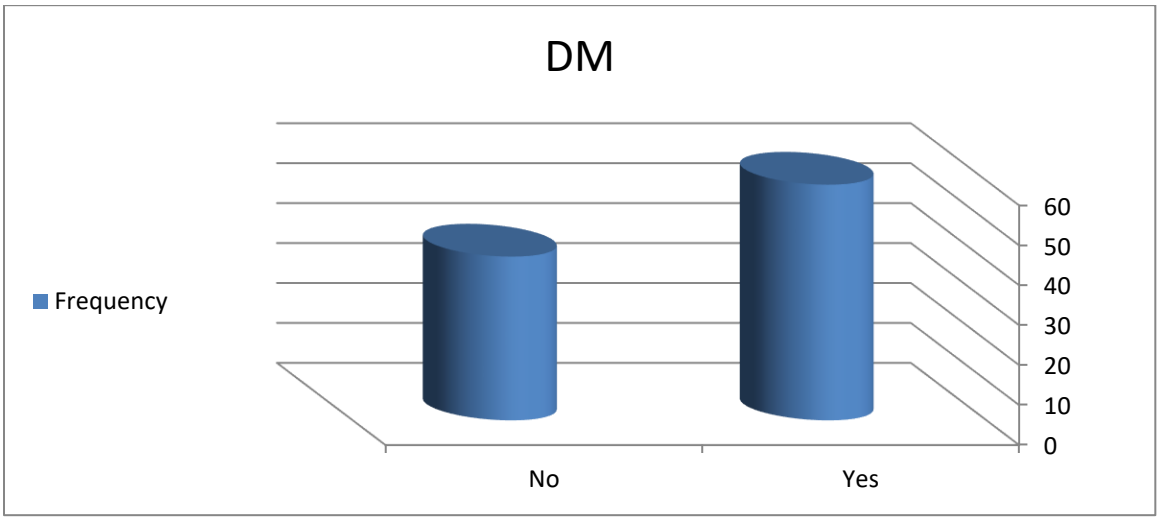

Figure 4.10

DM in patient develop CAD

HT * patient develop CAD

Table 12

HT in patient develop CAD

			patient develop CAD		
			Yes	No	Total
HT	Yes	Count	59	10	69
		% within HT	85.5%	14.5%	100.0%
		% within patient develop CAD	68.6%	71.4%	69.0%
		% of Total	59.0%	10.0%	69.0%
	No	Count	27	4	31
		% within HT	87.1%	12.9%	100.0%
		% within patient develop CAD	31.4%	28.6%	31.0%
		% of Total	27.0%	4.0%	31.0%
Total		Count	86	14	100
		% within HT	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

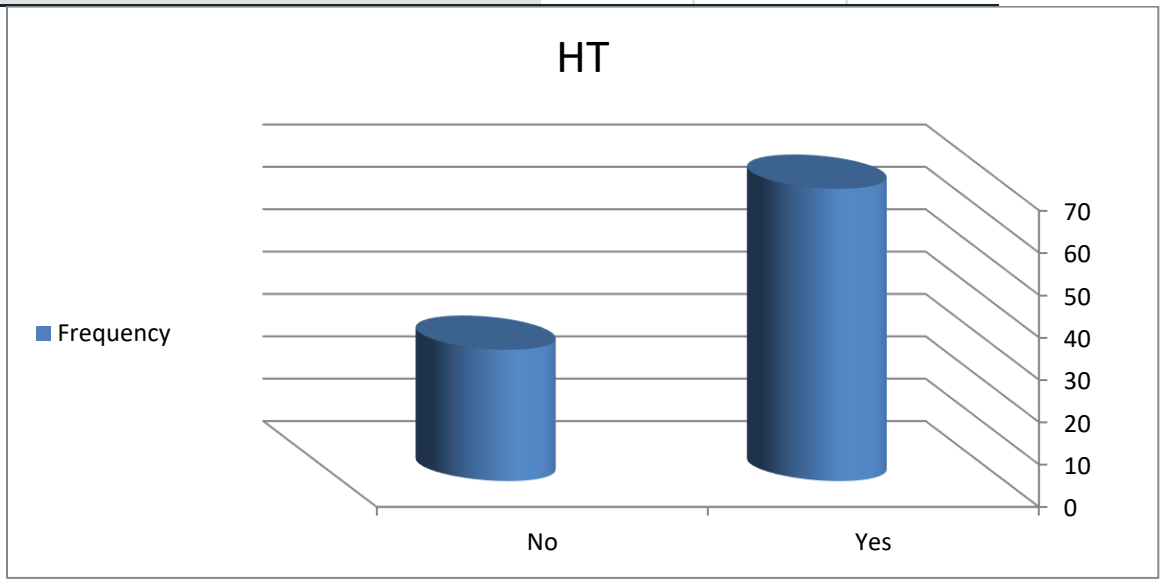

Figure 4.11

HT in patient develop CAD

IHD * patient develop CAD

Table 13

IHD * patient develop CAD

			patient develop CAD		
			Yes	No	Total
IHD	Yes	Count	35	5	40
		% within IHD	87.5%	12.5%	100.0%
		% within patient develop CAD	40.7%	35.7%	40.0%
		% of Total	35.0%	5.0%	40.0%
	No	Count	51	9	60
		% within IHD	85.0%	15.0%	100.0%
		% within patient develop CAD	59.3%	64.3%	60.0%
		% of Total	51.0%	9.0%	60.0%
Total		Count	86	14	100
		% within IHD	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

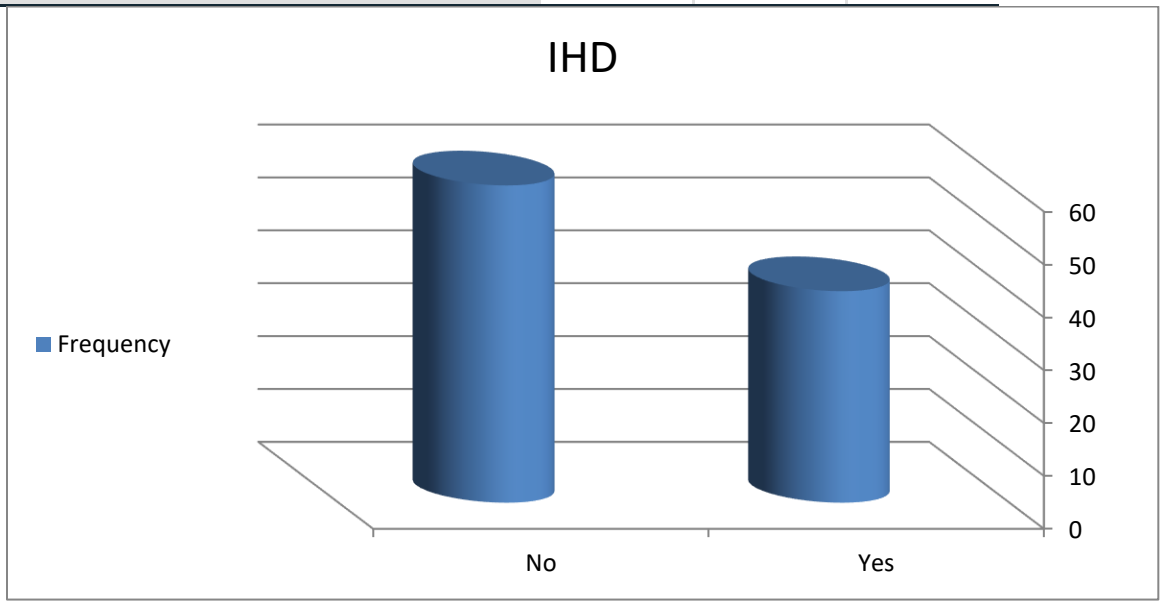

Figure 4.12

IHD * patient develop CAD

thyroid dys * patient develop CAD

Table 14

thyroid dys * patient develop CAD

			patient develop CAD		
			Yes	No	Total
thyroid dys	Yes	Count	1	0	1
		% within thyroid dys	100.0%	0.0%	100.0%
		% within patient develop CAD	1.2%	0.0%	1.0%
		% of Total	1.0%	0.0%	1.0%
	No	Count	85	14	99
		% within thyroid dys	85.9%	14.1%	100.0%
		% within patient develop CAD	98.8%	100.0%	99.0%
		% of Total	85.0%	14.0%	99.0%
Total		Count	86	14	100
		% within thyroid dys	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

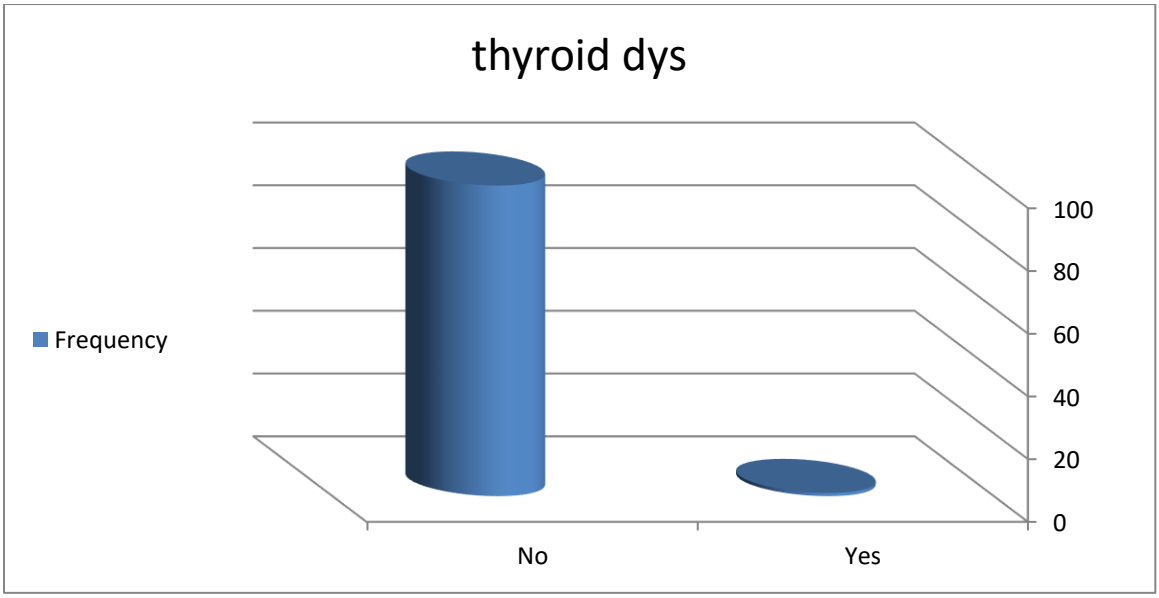

Figure 4.13

thyroid dys * patient develop CAD

smoking * patient develop CAD

Table 15

smoking * patient develop CAD

			patient develop CAD		
			Yes	No	Total
smoking	Yes	Count	43	3	46
		% within smoking	93.5%	6.5%	100.0%
		% within patient develop CAD	50.0%	21.4%	46.0%
		% of Total	43.0%	3.0%	46.0%
	No	Count	43	11	54
		% within smoking	79.6%	20.4%	100.0%
		% within patient develop CAD	50.0%	78.6%	54.0%
		% of Total	43.0%	11.0%	54.0%
Total		Count	86	14	100
		% within smoking	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

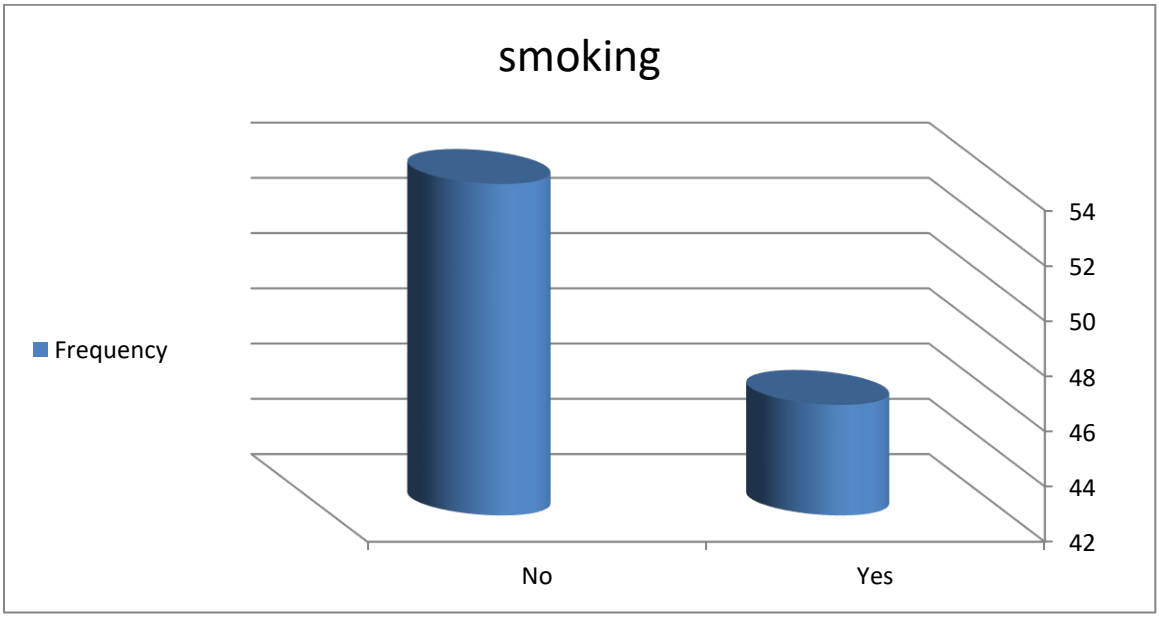

Figure 4.14

smoking * patient develop CAD

ECG abnormalty * patient develop CAD

Table 16

ECG abnormalty * patient develop CAD

			patient develop CAD		
			Yes	No	Total
ECG 2ty	Yes	Count	77	0	77
		% within ECG 2ty	100.0%	0.0%	100.0%
		% within patient develop CAD	89.5%	0.0%	77.0%
		% of Total	77.0%	0.0%	77.0%
	No	Count	9	14	23
		% within ECG 2ty	39.1%	60.9%	100.0%
		% within patient develop CAD	10.5%	100.0%	23.0%
		% of Total	9.0%	14.0%	23.0%
Total		Count	86	14	100
		% within ECG 2ty	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

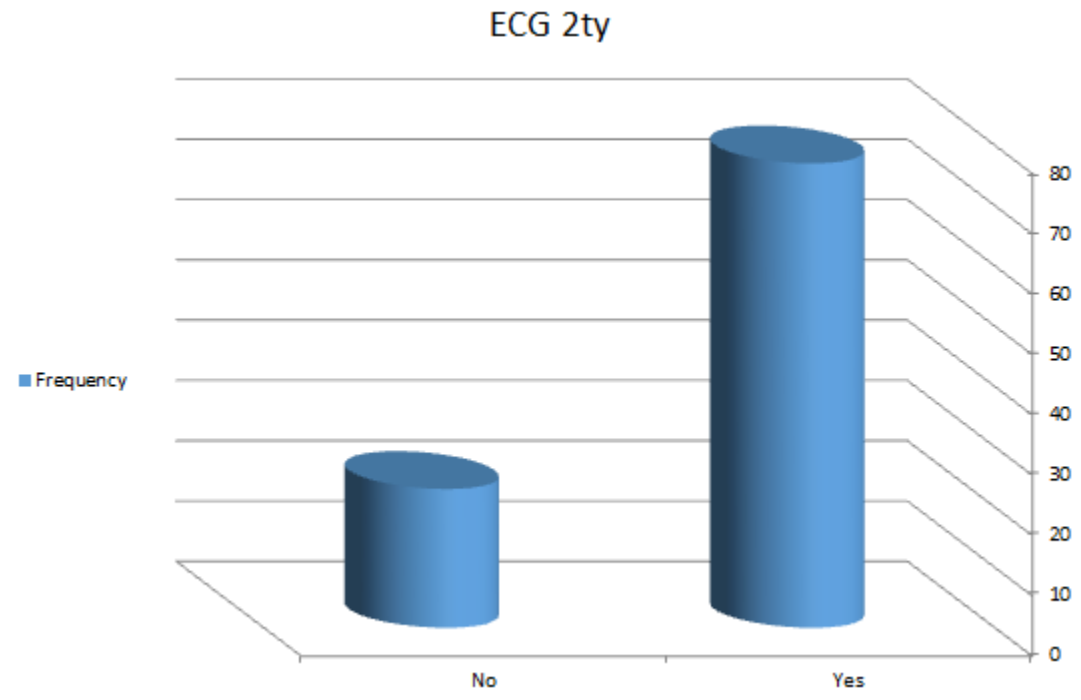

Figure 4.15

ECG abnormalty * patient develop CAD

TROPNINE * patient develop CAD

Table 17

TROPONIN * patient develop CAD

			patient develop CAD		
			Yes	No	Total
TROPNINE	Yes	Count	74	3	77
		% within TROPNINE	96.1%	3.9%	100.0%
		% within patient develop CAD	86.0%	21.4%	77.0%
		% of Total	74.0%	3.0%	77.0%
	No	Count	12	11	23
		% within TROPNINE	52.2%	47.8%	100.0%
		% within patient develop CAD	14.0%	78.6%	23.0%
		% of Total	12.0%	11.0%	23.0%
Total		Count	86	14	100
		% within TROPNINE	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

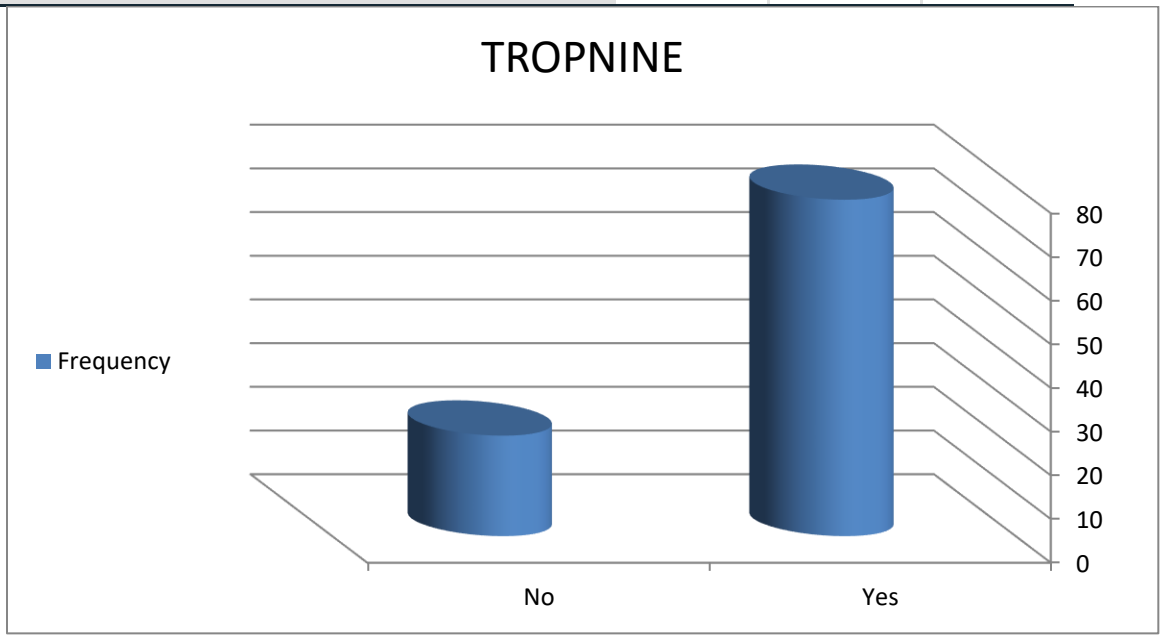

Figure 4.16

TROPONIN * patient develop CAD

glucose * patient develop CAD

Table 18

glucose * patient develop CAD

			patient develop CAD		
			Yes	No	Total
glucose	Normal	Count	35	5	40
		% within glucose	87.5%	12.5%	100.0%
		% within patient develop CAD	40.7%	35.7%	40.0%
		% of Total	35.0%	5.0%	40.0%
	Elevated	Count	51	9	60
		% within glucose	85.0%	15.0%	100.0%
		% within patient develop CAD	59.3%	64.3%	60.0%
		% of Total	51.0%	9.0%	60.0%
Total		Count	86	14	100
		% within glucose	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

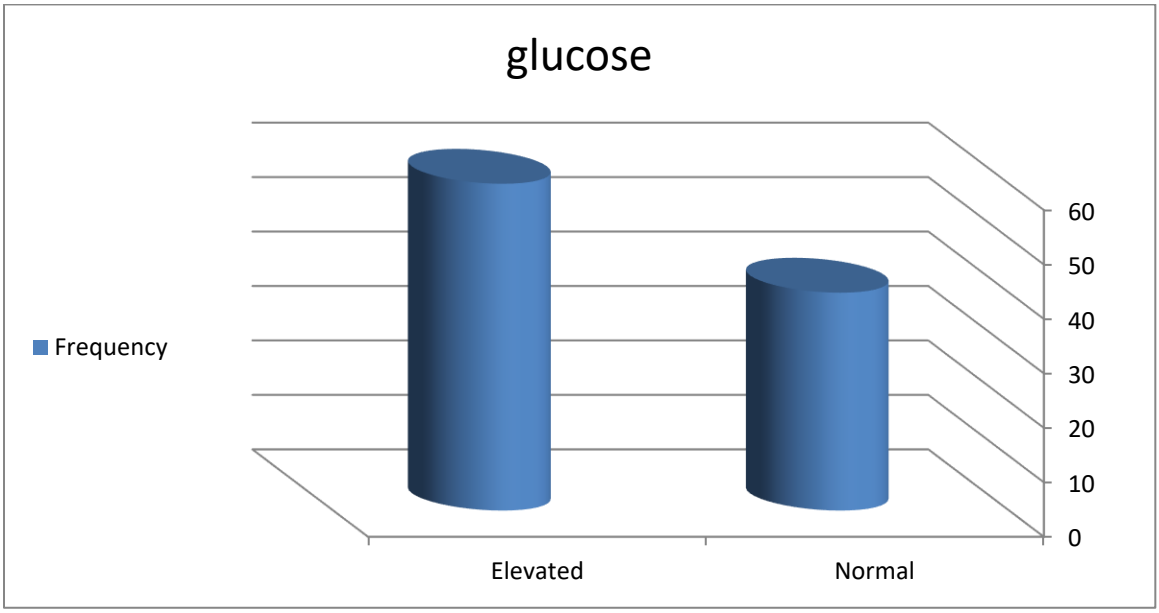

Figure 4.17

glucose * patient develop CAD

cholestrol * patient develop CAD

Table 19

cholestrol * patient develop CAD

			patient develop CAD		
			Yes	No	Total
cholestrol	Normal	Count	26	1	27
		% within cholestrol	96.3%	3.7%	100.0%
		% within patient develop CAD	30.2%	7.1%	27.0%
		% of Total	26.0%	1.0%	27.0%
	Elevated	Count	32	9	41
		% within cholestrol	78.0%	22.0%	100.0%
		% within patient develop CAD	37.2%	64.3%	41.0%
		% of Total	32.0%	9.0%	41.0%
	Unknown	Count	28	4	32
		% within cholestrol	87.5%	12.5%	100.0%
		% within patient develop CAD	32.6%	28.6%	32.0%
		% of Total	28.0%	4.0%	32.0%
Total		Count	86	14	100
		% within cholestrol	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

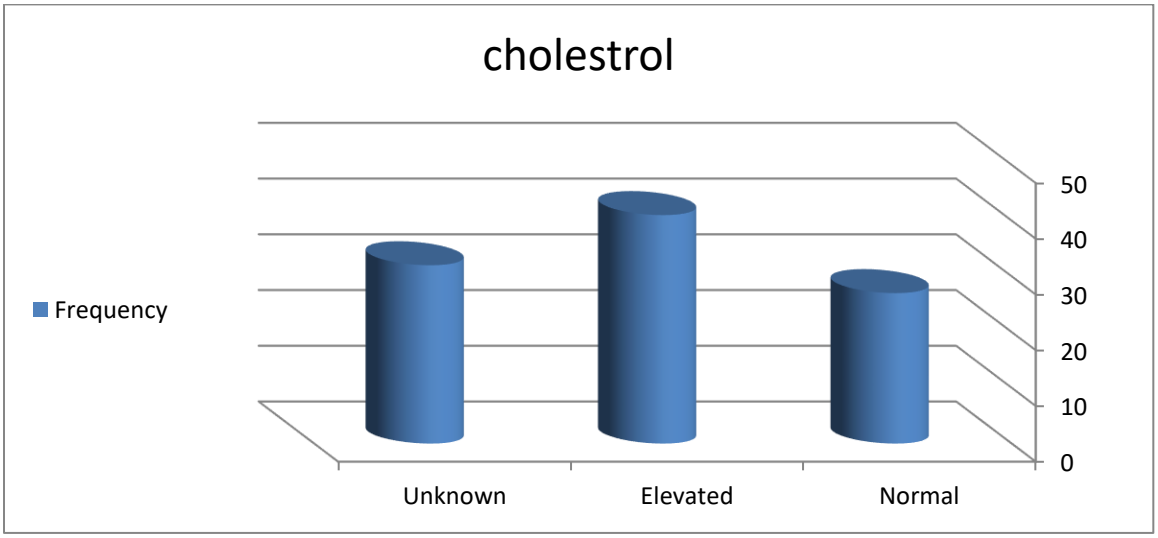

Figure 4.18

cholestrol * patient develop CAD

TG * patient develop CAD

Table 20

TG * patient develop CAD

			patient develop CAD		
			Yes	No	Total
TG	Normal	Count	29	1	30
		% within TG	96.7%	3.3%	100.0%
		% within patient develop CAD	33.7%	7.1%	30.0%
		% of Total	29.0%	1.0%	30.0%
	Elevated	Count	29	9	38
		% within TG	76.3%	23.7%	100.0%
		% within patient develop CAD	33.7%	64.3%	38.0%
		% of Total	29.0%	9.0%	38.0%
	Unknown	Count	28	4	32
		% within TG	87.5%	12.5%	100.0%
		% within patient develop CAD	32.6%	28.6%	32.0%
		% of Total	28.0%	4.0%	32.0%
Total		Count	86	14	100
		% within TG	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

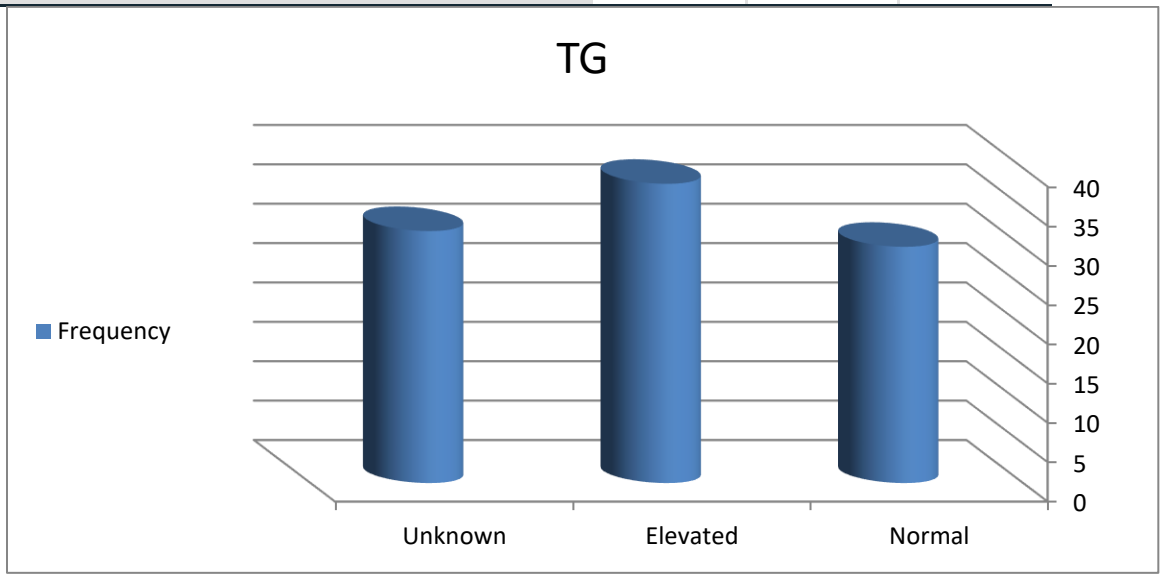

Figure 4.19

TG * patient develop CAD

HDL * patient develop CAD

Table 21

HDL * patient develop CAD

			patient develop CAD		
			Yes	No	Total
HDL	Normal	Count	14	2	16
		% within HDL	87.5%	12.5%	100.0%
		% within patient develop CAD	16.3%	14.3%	16.0%
		% of Total	14.0%	2.0%	16.0%
	Low	Count	44	8	52
		% within HDL	84.6%	15.4%	100.0%
		% within patient develop CAD	51.2%	57.1%	52.0%
		% of Total	44.0%	8.0%	52.0%
	Unknown	Count	28	4	32
		% within HDL	87.5%	12.5%	100.0%
		% within patient develop CAD	32.6%	28.6%	32.0%
		% of Total	28.0%	4.0%	32.0%
Total		Count	86	14	100
		% within HDL	86.0%	14.0%	100.0%
		% within patient develop CAD	100.0%	100.0%	100.0%
		% of Total	86.0%	14.0%	100.0%

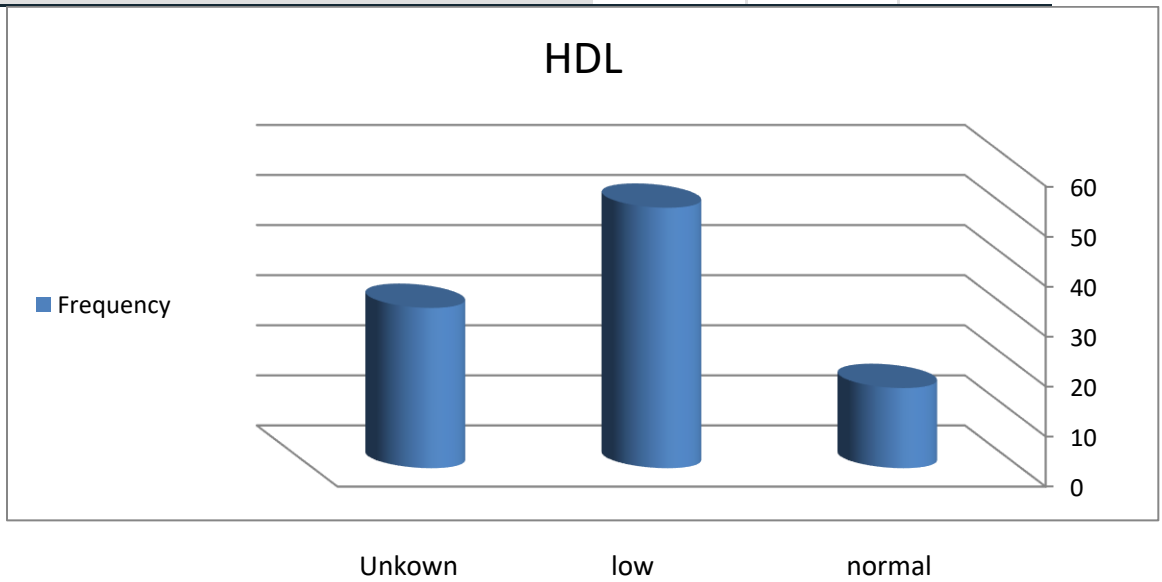

Figure 4.20

HDL * patient develop CAD

Chapter 4

5.1.Discussion

According to these data we can see the great effect of hypertension ,DM,smoking , high level of cholestrol and TG , low level of HDL on development

Ischemic heart disease (IHD)

As high ratio of patient who develop ischemia either stable angina or MI

Have these risk factors

As DM , HT and smoking destroy blood vessels

And cholestrol and TG help in atherosclerosis and plaque formation these lead to narrowing of blood vessel and ischemia of end tissue

And ischemia lead to life threating complication like arrhythmia , HF, stroke .

The diagnosis could be reached easly by ECG changes like ST elevation in MI

Or ST depression in stable angina .

the dignosis could be proven by troponin level measurement .

Most of patients was between 50 -60 years old because with age blood vessel loss its elasticity

And most of patient who develop IHD male gender

High ratio of patients with IHD come to hospital with chest pain and dysnea

Symptoms

5.2.Conclusion

This asymptomatic stage should be concentrated if want to eliminate a majority of coronary events.

These should include earlier identification and treatment of high blood pressure, eliminating tobacco, and lowering average LDL levels.

Earlier identification of atherosclerosis lead to lower event rates if the proper lifestyle and possibly drug therapies are initiated.

In the future, new strategies and risk profiles (possibly with genetic profiling) may help to better identify those at risk.

New treatments specifically targeting inflammation might also result in a reduction in events as long as the treatments do not interfere with a patient's natural immunity and erase any potential benefits.

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