Chapter one

Introduction and Literature Review

1.1.Introduction and background:-

Heamostasis is one of a number of protective processes that have involved in order maintaining a stable physiology. It interacts with other body defence such as inflammatory system and immune system. For example of these disseminated intravascular coagulation (DIC) can be initiated by Gram –negative septicemia.[Hoffbrand *et al*, 2016].

The heart is blood pump and net of vessels so the study is important. Several biomedical finding have established the effect of cardiovascular disease as arterial hypertension in the coagulation process. [Adaeze *et al* 2014].

Cardiovascular disease account for about one third of premature deaths in men and one quarter in women and arterial hyper tension is one of the most significant risk factors for cardiovascular disease .[Bernatova.I 2014]

Heart disease is major health problem and greatly affecting the economic and social status of such patients. [Chobanian.A.V *et al* 2003].

Cardiovascular disease develops 7to10 years later in women than in men and is still the main cause of the death in women. [Maas and Appelman, 2010].

Coronary artery disease is most common disease, is cause of death in male and female evaluation and treatment of the disease how ever can differ between male and female [Douglas and Ginsburg, 1996].

Ischemic heart disease is one of the common causes of mortality in the entire world;

also, according to the Framingham's studies, the risk of coronary

artery disease after age of 40 in men and women is respectively 45% and 32%..

Various parameters reflect the condition of platelets, including platelets count, platelet crit, and mean platelet indices (MPI) (mean platelet volume [MPV], platelet distribution width [PDW] and platelet large cell ratio [PLCR]).MPV reflects the average size of platelets. It is a marker that indicates subclinical platelet activation
and may be increased in some vascular conditions such as myocardial infarction (MI), coronary artery disease (CAD), cerebral ischemia, and PAD . Other platelet markers such as PDW, PLCR, and platelet crit (PCT), which reflect platelet morphology, are also important in vascular events such as atherosclerosis and thrombosis.

Platelet distribution width gives an indication of the distribution of platelet size.

Platelet Large Cell Ratio indicates the ratio of younger platelet group that has the largest volume, and PCT gives the total mass of platelets.

Nowadays Platelet counts (PC) and volumetric platelet indices are available routinely in most laboratories and reflect the level of mobility and production of platelets.

It seems that the excessive flexibility and size of the platelets and their local activation have correlation with extent of ischemic heart disease.

Through the world many studies were done in this topic due to the important of the heart in the body.
Other study platelets have been implicated in the pathogenesis of

coronary artery disease ,and number of studies have examined platelets function and coagulation parameters in such patients .

Other study is study the change in platelet count and indices in ischemic heart disease and assess their usefulness in predicting coronary events in 2009.

Other study is the relationship between platelet indices and clinical features of ischemic heart disease in 2013.

Also there is article about correlation between platelet indices and extent of coronary ischemic heart disease in 2016.

1.2 Literatuereviwe

1.2.1Haemostasis:

Haemostasis is one of a number of protective processes that have evolved in order to maintain a stable physiology. It has many features in common with (and to some extent interacts with) other defense mechanisms in the body, such as the immune system and the inflammatory response (Hoffbrand, et al., 2016).

1.2.2Haemostatic response:

1.2.2.1Vasoconstriction:

An immediate vasoconstriction of the injured vessel and reflex constriction of adjacent small arteries and arterioles is responsible for an initial slowing of blood flow to the area of injury. When there is a wide spread damage this vascular reaction prevents exsanguinations. The reduced blood flow allows contact activation of platelets and coagulation factors. The vaso-active amines and thromboxane A2 liberated from platelets, and the fibrin peptides liberated during fibrin formation, also have vasoconstrictive activity (Hoffbrand, *et al.,* 2016).

1.2.2.2 Platelet reactions and primary hemostatic plug formation:

Platelets are small cell fragments (average size 3–4 µm) that are important for hemostasis and coagulation. The normal platelet count is between 150,000 and 450,000/µL. Platelets are derived from megakaryocytes, which are very large cells with a large, multi-lobulated nucleus. The mean DNA content of megakaryocytes is at least eight times that of other somatic cells. One megakaryocyte can produceat least several thousands of platelets.

The formation and release of platelets is related to a preformed structure in the cytoplasm of megakaryocytes, the so-called3demarcation membrane system.

Megakaryocytes are derived from megakaryocyte progenitors, which in turn originate in the hematopoietic stem cell.

Megakaryocytes are mainly found in the bone marrow but can transit to many organs, including the lung, where part of the platelet release occurs.

The maturation of megakaryocytes and the production of platelets occur under the influence of thrombopoietin (TPO). TPO acts, together with certain other cytokines like IL-6 and IL-11, on early megakaryocyte progenitors as well as mature megakaryocytes. Under physiological conditions, the serum levels of TPO are lowat normal or elevated platelet counts and high in individuals with low platelet counts (Wintrobe, et al., 2009).

Following a break in the endothelial lining, there is an initial adherence of platelets to exposed connective tissue, potentiated by VWF. Collagen exposure and thrombin produced at the site of injury cause the adherent platelets to release their granule contents and also activate platelet prostaglandin synthesis leading to the formation of thromboxaneA 2.

 Released ADP causes platelets to swell and aggregate. Additional platelets from the circulating blood are drawn to the area of injury. This continuing platelet aggregation promotes the growth of the hemostatic plug which soon covers the exposed collective tissue.

The unstable primary hemostatic plug produced by these platelet reactions in the first minute or so following injury is usually sufficient to provide temporary control of bleeding. And smooth muscle cells in

the vessel wall adjacent to the area of damage, is important in limiting the extent of the initial platelet plug (Wintrobe, *et al.,* 2009).

1.2.2.3 Stabilization of the platelet plug by fibrin:

Definitive hemostasis is achieved when fibrin formed by blood coagulation is added to the platelet mass and by platelet-induced clot retraction compaction.

Following vascular injury, the formation of extrinsic Xase (VIIa, TF, PL andCa2+) initiates the coagulation cascade. Platelet aggregation and release reactions accelerate the coagulation process by providing membrane phospholipid.

Thrombin generated at the injury site converts soluble plasma fibrinogen into fibrin, potentiates platelet aggregation and secretion and also activates factor XI and XIII and cofactors V and VIII. The fibrin component of the hemostatic plug increases as the fused platelets autolysis and after a few hours the entire hemostatic plug is transformed into a solid mass of cross-linked fibrin. Nevertheless, becauseof incorporation of plasminogen and tPA , this plug begging to auto digest during the same time frame (Wintrobe,*et al.,* 2009).

1.2.3Activation:

The complex process of converting inactive platelets into a platelet plug, is essential.

There are three phases to platelet function :-

*Adhesion :-

Endothelial cells are attached to the sub-endothelial collagen by von Will brand factor (VWF) . VWF is also stored in the Weibel-Palade bodies of the endothelialcells and secreted constitutively into the blood. Platelets store vWF in their alpha granules .when the endothelial layer is disrupted, collagen and VWF anchor platelets to

the sub-endothelium. Platelet GP1b-IX-V receptor binds with VWF;and GPVI receptor and integrin alpha2 - beta1 bind with collagen.(Dubois, *et al.,*2006)*.*

*Morphology change:-

Mitochondria hyper polarization is a key event in initiating changes in morphology. Intra platelet calcium concentration increases, stimulating the inter play between microtubule/actin filament complex. The continuous changes in shape from the un activated to the fully activated platelet. This dramatic increase insurface area comes about with neither stretching nor adding phospholipids to the platelet membrane.(Matarrese, *et al.,*2009).

*Granule secretion:-

Platelets contain dense granules, lambda granules and alpha granules. Activated

platelets secrete the contents of these granules through their canalicular systems tothe exterior. Simplistically, bound and activated platelets degranulate to release platelet chemotactic agents to attract more platelets to the site of endothelial injury.(Yip, *et al.*,2005).

1.2.4 Platelet disorders:

*Thrombocytopenia according to the classification of Geddis,(2013):

-Immune thrombocytopenias (ITP) – formerly known as immune thrombocytopenic purpura and idiopathic thrombocytopenic purpura

-Splenomegaly

-Chemotherapy

-Thrombotic thrombocytopenic purpura

-Hemolytic-uremic syndrome

-Pregnancy associated

-Aplastic anemia

-Transfusion associated

-Pseudo-thrombocytopenia

-idiopathic thrombocytopenic purpura

*Thrombocytosis and thrombocythemia according to the classification of Laidlaw *et al*.,(2012*):*

-Chronic infection

-Chronic inflammation

-Malignancy

-Hyposplenism (post-splenectomy)

-Iron deficiency

-Acute blood loss

-Myeloproliferative neoplasms – platelets are both elevated and activated

-Essential thrombocytosis

-Polycythemia vera

-Congenital.

* Altered platelet function according to the classification of Laidlaw *et al*.,(2012*):*

#Congenital:

-Disorders of adhesion

-Bernard-Soulier syndrome

-Disorders of activation

-Disorders of granule amount or release

-ADP Receptor defect

-Decreased cyclooxygenase activity

-Storage pool defects, acquired or congenital

-Disorders of aggregation

-Glanzmann'sthrombasthenia

#Acquired

-Disorders of adhesion

-Paroxysmal nocturnal hemoglobin uria

-Cancer

-Malaria

1.2.5 Platelets indices:

1.2.5.1 Mean platelet volume (MPV):

Is a measure of the average size of platelets, which are cells that help your blood clot with normal rang (7.4 _ 10.4 fl). A high MPV is usually a sign that there are more young platelets circulating in your bloodstream. If you have had a procedure such as major surgery, your body is using up platelets to repair the cuts to the blood vessels.

In response, your bone marrow releases more of the young, larger platelets, and your MPV rises, a low MPV along with a low platelet count can point towards disorders affecting the bone marrow that slow down or decrease the production of platelets, such as a condition called a plastic anemia. In addition, alow MPV can be seen with high, low or normal platelet counts in sepsis (a life threatening reaction to infection in the body), splenomegaly (enlarged spleen),chronic kidney failure, or treatment with drugs that suppress blood production(Chandrashekar, (2013).

1.2.5.2 Platelet distribution width(PDW):

A measure of the variation in the size of platelets found in the circulating blood, with normal range (10.0 -14.0/fl).

Platelets recently released from bone marrow tend to be larger and to contain more RNA than older, smaller platelets, which discard

their endoplasmic reticulum as they mature. Medical dictionary, The volume is determined by a machine and a Complete Blood Profile, known as a CBC. This reading determine if a patient's body is producing larger than average platelets, indicative of platelet destruction or bone marrow diseases (Chandrashekar, 2013).

1.2.5.3 Platelet-crit (PCT):

Is a measure of total platelet mass. Values vary depending on mean platelet volume resulting in overlap between normal platelets, thrombocytopenia and thrombocytosis .with normal range(0.10_0.28%) (Chandrashekar.,2013).

1.2.5.4 Platelet large cell ratio(P-LCR):

Means Platelet large cell ratio with normal range (13.0_43.0%) and it's calculatedin automated blood analyzers .Increased percentage of large platelets (P-LCR) is observed in patients with Hyper-lipidaemia and suggest possible risk of thrombosis. an increase in P-LCR + MPV + PDW has been observed in autoimmune thrombocytopenic purpura. (Chandrashekar , 2013).

1.2.6 Laboratory evaluations:-

1.2.6 .1 Platelet count test:

Principle blood is diluted 1 in 20 in a filtered solution of ammonium oxalate reagent which lyses the red cells, platelets are counted microscopically using an improved neighbor ruled counting chamber and the number of platelet per liter of blood calculated .may be requested to investigate abnormal skin and mucosal bleeding .

also performed when patients are being treated with cytotoxic drugs or other drugs which may cause thrombocytopenia (Hoffbrand, *et al* .,2016).

1.2.6.2 Bleeding time:

Principle bleeding time is defined as the time taken for a standardized skin wound to stop bleeding. It is measures the ability of platelets to arrest bleeding and therefore, measures platelet number and function (Hoffbrand, *et al.*,2016).

1.2.6.3 Platelet aggregation :-

A known platelet aggregating factor such as collagen, ADP or thrombin is added to a suspension of the platelets under test and the degree of aggregation measured by decrease in turbidity of the suspension.(Hoffbrand, *et al.,* 2016).-

1-2.7. Normal cardiovascular system:-

Cardiovascular system may be considered as being composed of a pump (heart)and plumbing (vessels) that take nutrient to tissue and remove metabolites from them .Metabolites travel by blood , lymphatic return tissue fluid to blood via the thoracic ducts.

The major diseases implicated in the cardiovascular system for this death areatherosclerosis, thrombosis, embolism and infraction. [vardaxis, 2000]**.**

Figure 1.1; Normal heart anatomy [vardaxis , 2000]
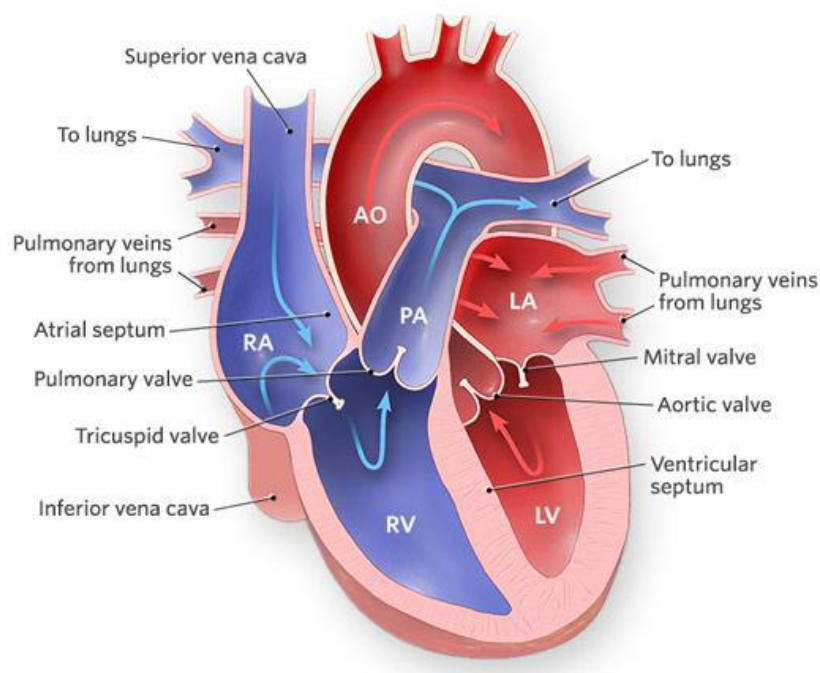
1-2.7.1. Heart failure :-

requirement of the metabolic tissues of the body or being able to do so at increased It is clinical condition characterized by the in ability of heart pump blood with filling pressures .It may be divided in to systolic or diastolic failure depending on whether there is abnormality in the cardiac contractility.[GargandGupta ,2013].

1.2.7.2 .Left ventricle failure:-

Mainly caused by ischemic heart disease, hypertension, aortic or mitral valc11ular disease and myocardial disease. The feature includes hyper atrophy and fibrosis in the myocardium .Arterial involvement result in the development of arterial fibrillation which is responsible for thrombus or embolic stroke. .[Garg and Gupta,2013].

1-2.7.3.Sudden cardiac death:-

It is defined as the death of an individual within 1 hour of onset of symptom commonly due to ventricular fibrillation.

In case of coronary vessel occlusion leading to ischemia, there is physiological compensatory vasodilatation resulting in augmentation of coronary blood follow.[Garg and Gupta ,2013].

1-2.7.4.Stable angina:-

Occur when the myocardial oxygen demand is more than the supply .Is take place when the coronary artery occluded more than 75% .Characterized by the pain one xertion is relieved on taking rest or taking vasodilators.[Garg and Gupta ,2013].

1-2.7.5.Prinzmetal or variant angina:-

It is an episodic angina due to coronary artery spasm resulting in pain at rest it is characterized by ST segment elevation on the ECG.[Garg and Gupta ,2013]

1-2.7.6.Unstable or crescendo angina:-

It is induced by a the rosclerotic plaque disruption with superimposed partial thrombosis or vasospasm or both of them .The pain occur with increasing frequency and for a longer duration and characteristically precipited by progressively less exertion.[Garg and Gupta ,2013].

1-2.7.7.Myocardial infractions (MI) :-

Table 1-1 type of myocardial infarctions..[Garg andGupta,2013].

Subendocardial MI	Transmural MI
*Ischemic necrosis limited to 1|3rd of ventricular wall	*Ischemic necrosis involves full thickness of ventricular wall
*Caused by complete coronary artery occlusion	*Caused by sever coronary atherosclerosis with acute plaque rupture and occlusive thrombosis

1-2.7.8.Infective endocarditis (IE):-

It is colonization invasion of heart valve and mural end cardium by microbiologic agent leading to formation of bulky, friable vegetation composed of thrombotic debris and organism with destruction of under lying cardiac tissue..[Gupta ,2013].

1-2.7.8.1. Morphology:-

The friable, bulky destructive vegetations containing fibrin. When the vegetation rode into myocardium, they can form an abscess, the systemic embolistion can result in septic interact.[Gupta ,2013].

1-2.7.9.Rheumatic Heart Disease (RHD):-

Rheumatic fever is an acute immunologically mediated multi system inflammatory disease that occur few weeks after an attack of group A,B-hemolytic streptococcal pharyngitis not disease mainly in children between 5-15 years .Only 3% of patients with group A streptococcal pharyngitis develop acute rheumatic fever [Gupta,2013].

1-2.7.10.Congenital heart disease:-

About 1 in 200 babies as born have a congenital heart defect .About 5 % of cardiac defects are attributed to the chromosomal abnormalities ,Or may be to other disease .Defect are variables in severity ,ranging

from the trivial and subclinical to the rapidly fatal .There are many types of congenital heart disease ,but only the most common are :

1-2.7.10-1.Left to right shunt:-

Arterial septal defect is a connection between right and left arterial due to a hole in the inter arterial septum. About 90% congenital heart disease most of this the occurrence in adult. A complication associated with untreated cases of this diseases the occurrence of paradoxical emboli.[Vardaxis,2000].

1-2.7.10.2.PlumonaryStenotic lesions:-

This involve narrowing of the pulmonary artery or the pulmonary valve which reduce the amount of blood .So the affected babies appear blue cyanotic this is probably due to emoblis of megakrocyte from the bone marrow. [Vardaxis ,2000].

1-2.7.10-3.Coarctation of the aorta:-

Is the condition in which there is narrowing or blockage of the aorta, is about 5% of congenital heart disease .

There are 2 type:

1-2.7.10.3.1.preductal type (infant):-

Sever narrowing of segment of the aorta, a patient duct allow blood to enter the systemic circulation from the pulmonary circulation.

1-2.7.10.3.2. post ductal type (adult):-

More common and involves a shorter segment of the aorta frequently in ring fibrosis occurs in wall of aorta.[Vardaxis,2000].

1.2.7.11 Ischemic heart disease:

Also called coronary heart disease which affects the blood vessels that supply the heart with blood and oxygen.

Fatty material called pluge builds up in the lining of the blood vessels this is called atherosclerosis.

The inside of the blood vessels become narrow and less blood can get through .

IF the arteries become too clogged the heart may not be able to work properly.

There is no single cause of coronary heart disease but there are risk factors that increase the chance for getting it.

*Risk factor:

_ High (bad) cholesterol.

_ Cigarette smoking (or being expose to other people smoke) .

_ Lake of exercise.

_ High blood pressure.

_ Obesity.

_ Diabetes.

_ Depression.

There are some factor we cannot change:

_family history.

_age.

_sex (men are greater risk for coronary heart disease)

1.3 Rationale

Recent studies indicate that patients who had heart disease may have ability to stimulate haemostatic disorders coagulation deficiency, platelets dysfunction and thrombocytopenia as results of heart disease. Patients show variable abnormalities in platelet count and platelet indices and some experience no change from the base line value.

1.4 Objective:

1.4.1 General objectives:

1. to Measurement of platelet count and platelet indices among Sudanese ischemic Heart disease Patients.

1.4.2 Specific objective:

1. to Measure Platelet count in ischemic heart disease patients compared to control.

2. to Measure platelet indices in ischemic heart disease patients compared to control .

3. to compare of platelets counting and platelet indices in study group according to age group and gender and duration of disease.

Chapter Two

Material and methods

Chapter Two

Material and Method

2. Material and method:
2-1 .Study design:
A case control study was conducted for evaluation of PLTS count and indices in patients with ischemic heart disease.
2-2 .Study area:
The study conducted in Khartoum state at Ashaab hospital teaching .
2-3-.Study population:
Patients with ischemic heart disease investigated for PLTS count and PLTS indices.
2-4.Sampling:
The frame included all ischemic heart disease patients.
2-5.Inclusion criteria:
All patients who had a confirmed diagnosed as ischemic heart disease patients.
2-6.Exclusion criteria:
All patients who were not diagnosis as ischemic heart disease patients.
2-7. Sample size:
 100 samples of patients were collected (40 from male and 30 from post monoposal female) matched by 30 samples as control(20 male and 10 female).
2-8. Tool of data collection:
The data collected by using of laboratory investigation to obtain Platelet count and platelet indices.
Also the interviews used to obtain age ,sex ,family history ,clinical features and using of questionnaire as instrument .
2-9. Data analysis:
The data after collection analyzed to obtain the mean, standard deviation and the probability (p_ value).between patients and control by using SPSS computer program.

2-10. Ethical consideration:

All information that obtained from patients kept as highly confidential and specimens or result not permitted.

The participants provided with information about the study and any risk that may arise especially when collection technique applied.

2-11. Time line:

The time of research began from May to September 2018.

2-12. Sampling:

2.5 ml collect in EDTA of platelets count and platelet indices.

2-13. Methodology:

2-13-1. Collection technique:

EDTA container.

Cotton.

Alcohol (70%).

Syringes.

Tourniquet.

2-13-2. Procedure:

1. Patients' comfortable sitting, tourniquet applied above elbow and superficial antecubital for arm vein identified.

2. The skin sterile with 70% ethanol and allowed to dry.

3. Syringe needle inserted correctly into the vein ,and 2.5ml of blood sample are collected.

2-13-3.Platelets count:

By sysmex 21Automated Hematology Analyzer.

2-13-3-1. Reagent and material:

1.Cell pack.

1. Stromatolyser.

2. Detergent.

3. Cell cleaner.

2-13-3-2 .Principle of sysmex 21Automated Hematological Analyzer:

Measurement of blood cells (red blood cells ,white blood cells, and platelets) and hemoglobin concentration obtained by aspiration of small volume of EDTA blood by sample probe and mixed isotonic diluents in nebulizer diluted mixture aspiration delivered to RBCs aperture bath for providing

information about RBCs and platelets based on cell sizes, particles of 2 to 20 femtoliter counted as platelets ,above 36 femtoliter counted as Red blood cells .some portion of aspirated mixture induced into white blood cells bath in hemolytic reagent (Stromatolyser) was added automatically to measure hemoglobin concentration in build colorimeter ,based on cynomethemglobin method (HiCN). Blood cells counted and size information generated in triplicate pulses according to electronic conductivity, and translated into digital number using in build calculator programmed and designed for that RBCs, WBCs counts .

hence three values were directly measured (RBC,WBC,Hb) and displayed on (LCD).other values of red cell indices, platelets counts, leukocyte differential and absolute count calculated from given information and automated constructed histogram, the result printed out according to the setting mode.[Bendette*et al* 1995].

Statistical analysis

Statistical analysis was performed by using SPSS computer program version 16 .

the value were expressed as mean ± Std .Devision by using Independent –T-test ,frequency and graph to get mean ages and distribution between gender.

Chapter three

Results

Chapter Three

Results

3 . Results:

100 venous blood sample were collected , 70 sample collected from
ischemic heart disease patients were recognized according to gender as:
40 samples from male (57.1%) and 30 samples from postmonoposal
female (42.9%) .

Also recognized according to the age as: 52 patients are more than 50
years (74.3%). And 18 patients are less than 50 years (25.7%) .

And 30 samples were collected from healthy persons as control with the
same age range of the patients (20 male 66.7% and 10 female 33.3%).

**Table (3.1): Comparison of PLTS count, PMV,PDW and PLCR
between study group and control group.**

Parameters	Mean ± STD		P.value
	Study group	**control group**	
PLTS thousands per cumm	247.9± 93.4	268.2 ± 66.01	.220
PMV fl	10.08 ± 1.3	9.4 ± 0.5	0.01
PDW fl	13.1 ± 2.6	11.4 ± 1.3	0.001
PLCR %	26.8 ± 7.8	20.7 ± 4.8	0.000

* Significant level at (P-value ≤ 0.05).

Table(3.2):Age group in study group:-

Group	Range	Frequency
Group 1	More than 50 years	52
Group2	Less than 50 years	18
Total		70

Table(3.3):Comparison of PLTs count, PMV, PDW and PLCR between(group1,group2) in study group:-

Parameters	Mean ± STD		p-value
	group1	group 2	
PLTS count thousand per cumm	285.1± 127.2	235 ± 75	0.1
PMV fl	10.2 ± 1.25	10 ± 1.33	0.5
PDW fl	12.8 ± 2.1	13.3 ± 2.1	0.4
PLCR %	28.1 ± 8	26.4 ± 7.8	0.4

* Significant level at (P-value ≤ 0.05).

Table(3.4):Comparison of PLTs count, PM V, PDW and PLCR between male group and
female group in study group .

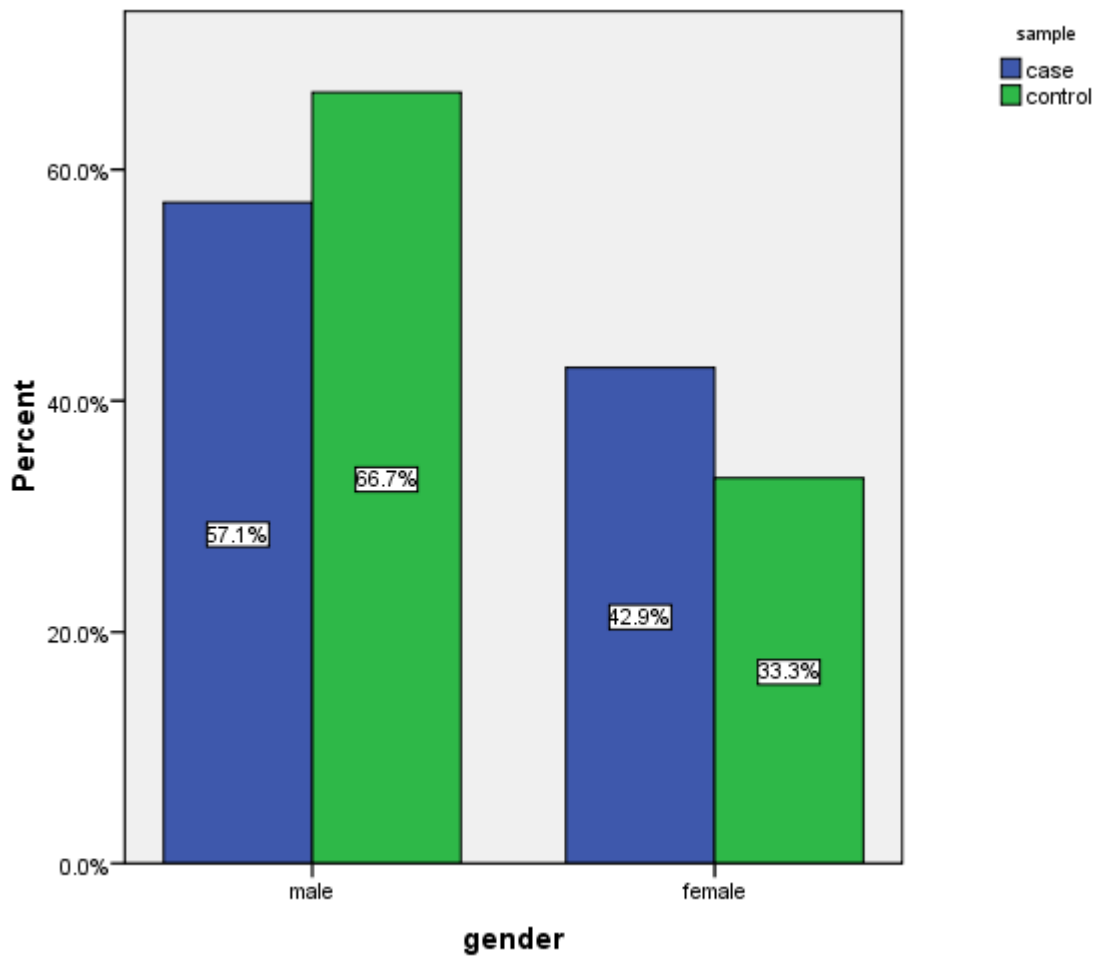| Parameters	Mean ± STD		P-value
	Male	Female	
PLTS count thousand per cumm	229.1± 74.8	272 ± 110	0.06
PMV fl	10.04 ± 1.3	10.1 ± 1.2	0.7
PDW fl	13 ± 2.3	13.4 ± 3.1	0.4
PLCR %	26.2 ±7.2	27.7 ± 8.6	0.4

* Significant level at (P-value ≤ 0.05).

Figure3- 1;The percentage of male and female in study group compared
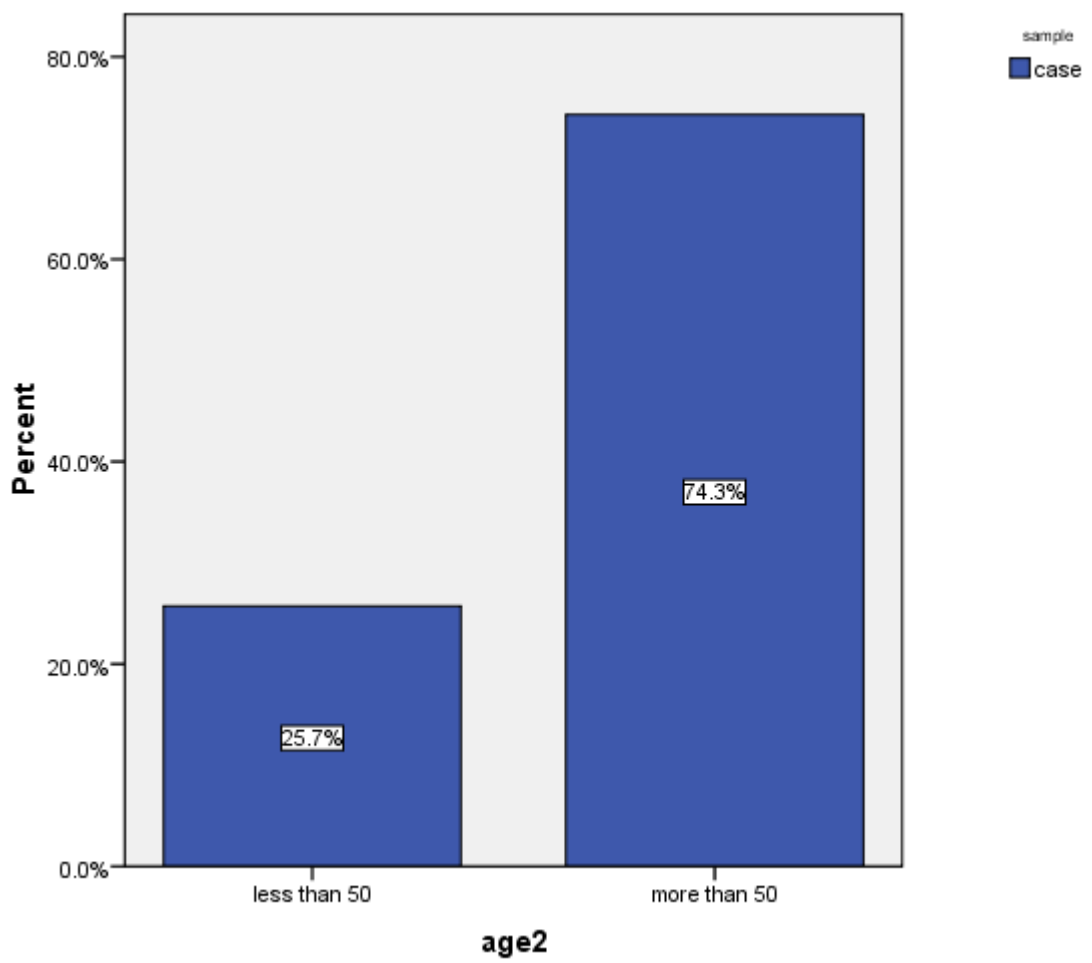to control.

Figure3-2 :The percentage of age group in study group

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Chapter four

Discussion, conclusion and Recommendation

Chapter four

Discussion, Conclusion and Recommendation

4-1.Discussion

Platelets are heterogeneous blood elements with diverse sizes and densities. Platelet activation is a hallmark of acute coronary syndrome. It has been shown that platelet size, when measured as mean platelet volume (MPV), is a marker of platelet function and is positively associated with indicators of platelet activity.

An increased MPV, an indicator of larger and more reactive platelets, has been associated with myocardial damage in ACS and has been found to be predictive of an unfavorable outcome among survivors of AMI (Boss et al 2006).

Platelet activation leads to the formation of free arachidonic acid, which can be transformed into prostaglandins, such as thromboxane A2, one of the most potent vasoconstriction and platelet-aggregating substances, or into leukotrienes, which can amplify the acute inflammatory response (Ross et al 1999).

Platelet function and size correlate because larger platelets, produced from activated megakaryocyte in the bone marrow, are likely to be more reactive than normal platelets because large platelets contain more secretary granules and mitochondria and are known to be more active than small platelets (Dalby et al 1998).

Consequently, larger and hyperactive platelets play a vital role in accelerating the formation and propagation of intracoronary thrombus, leading to the occurrence of acute thrombotic events(Smith et al 1990).

In the present study, the platelet indices; MPV, PDW, and P-LCR p.value(< 0.05) were assessed in a group of IHD patients and compared

to normal populations. A higher significant difference was detected in patients with IHD than in normal controls.

 Similar result to my study was reported by(Jasmin et al 2014),(Assiri et al 2012) ,(Salim et al 2013), and(Khode et al 2012),(Khandekar et al 2006),(Awad elkareem et al 2016), which suggested that all platelet volume indices are significantly raised in patients with IHD compared to normal control.

Platelets count in my study have no statistical difference between patients and normal controls (p. value: 0.22),which agree with(Awad elkarrem et al 2016) and disagree with other above studies those found significant higher in platelets count of patients compared with control and also disagree with(Mathur et al 2001) that found platelet counts to be significantly low in patients compared with control. .

No statistical difference was detected in my study for platelets count and platelets indices between patients and normal controls according to age group and gender , agree with the above previous studies. (p. value: >0.05),

4-2.Conclusion

The study showed that ;-
1-The mean of Platelets count in the patients (247.9±93.4) thousand cells per cumm were in control(268.2± 66.01) thousand per cumm,(p.value: .220). with no significance decrease or increase between case and control.
2-The mean of PMV in patients (10.08 ± 1.3)fl , were in control 9.4±0.5 fl (p.value:0.01) with higher significant differences in patients compared to control.
3-The mean of PDW in patients (13.1±2.6) fl , were in control 11.4±1.3 fl (.p.value 0.001) with higher significant differences in patients compared to control.
4- The mean of PLCR in patients (26.8 ±7.8) % ,were in control 20.7± 4.8% (p.value:0.000) with higher significant differences in patients compared to control.
5- No significant change in PLTS count ,PMV ,PDW and PLCR in study group according to age group.
6- No significant change in PLTS count , PMV ,PDW and PLCR in study group according to gender .

4-3 .Recommendations

1. Using more advanced technique for platelets function as the confirmatory test.

2. Regular checkup of hematological parameters for IHD patients should be performed to avoid the sudden crises which may occur.

3 -Coagulation screening should be done as routine test for all patients with heart disease and must be check monthly to patients to prevent heart attack that leads to sudden death.

4. More studies should be conducted with a large sample size:

a.To confirm the state of the PLT such as platelets aggregation, coagulation profile, Thrombin time, D.Dimer.

b. To assess Platelet count and indices as risk factors for cardiovascular complications in IHD patients.

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Appendix 1:-

Sudan University of science &technology

Collage of Graduate studies

Measurement of Platelet Count and indices among Ischemic Heart Disease Patients on Khartoum state

Questionnaire

- Name: ……………………………………… ID: …………………………
- Age:……………………………………………………………………………….
- Sex : Male (   )   Female(   )
- Do you have previous a blood or platelet transfusion? Yes……   No ………
- Do you have taken any Treatment of anticoagulant?   Yes…….   No……….
- Do you suffer from a chronic disease?   Yes……   No………

*Investigation :

1-CBC …………………………………………………………………………

*Platelet count……………………………………………………………..c\ cumm

*MPV……………………………………………………………………………fl

*PDW……………………………………………………………………………...fl

*P -LCR……………………………………………………………………………%

Signature……………………   **Date..**

Appendix 2

sysmex 21Automated Hematology Analyzer

sysmex 21Automated Hematology Analyzer