



**Sudan University of Science and Technology**



**Collage of Graduate Studies**

**Measurement Of CBC And Prothrombin Time Among Patients With  
Gastrointestinal Bleeding In Khartoum State.**

**قياس التعداد الكامل للدم ومدة التخثر لدى مرضى النزف المعوي**

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A Thesis Submitted for Partial Fulfillment of the Requirements for M.Sc. Degree in  
Medical Laboratory Sciences (Haematology and Immunology)

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# بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قال تعالى:

﴿يَرْفَعُ اللَّهُ الَّذِينَ آمَنُوا مِنْكُمْ وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ وَاللَّهُ بِمَا تَعْمَلُونَ خَبِيرٌ﴾

صدق الله العظيم

سورة المجادلة

(الآية 11)

# *Dedication*

To my first lady my heaven thanks for giving me a light in my life

... My Mother

To the man who believed in me, supporting, encourage me to reaches my dreams

... My Father

To those who have stood beside me... My Sisters, My Brothers

# Acknowledgement

First thanks to my God

It gives me great pleasure to conduct this study, and I would like to thank everyone who has made it possible.

It is most appropriate that I begin by expressing my undying gratitude to my supervisor Dr. Selma E. Abdalla for her invaluable guidance with her super talent, professional expertise and immense patience, showing great care and attention to details and without her guidance this study would have been impossible.

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## ABSTRACT

The study descriptive (Cross sectional) aimed to measurement the mean level of complete blood count parameter and prothrombin time (PT) In patients with gastrointestinal bleeding (GIB).The study conducted through the period from June to Sep 2018, in Mohammed Salih Idres Center for gastrointestinal bleeding in Ibn Sina Hospital in Khartoum state. The study population compromise two groups of adults in different age for both sex (66 males and 24 females) in age range from(18-70 years old). 90 subject were recruited for this study, 60 patients with acute gastrointestinal bleeding with first time bleeding and 30 healthy volunteers as control group. The data was collected using laboratory investigation to obtain CBC result and prothrombin time test. Also data collected using questionnaire. Five of ml venous bloods were collected from all participants 2.5ml was added to trisodium citrate anticoagulant and 2.5ml was added in EDTA anticoagulant containers. The data analyzed by SPSS version 16.The data show that about 66% of GIB patients were males and 24% were female. The distribution of GIB patients according to age show that the mean age were 44 years .The clinical manifestation of patients distributed as 41% with hematemesis, 34.4% with melena, 18.9% with bilharzia and 13.3% with bloody stool. The present study revealed significantly ( $P.V = \leq 0.000$ ) lower Hb, RBC, PCV, MCV, MCH, PLT values in GIB patients compared with normal control (Table 3.1). The WBC and PT values were significantly ( $P.V = \leq 0.001$ ) and ( $P.V = \leq 0.000$ ) higher in GIB patients compared to the normal control respectively, while no significant change in MCHC among GIB patients. The study showed that the gastrointestinal bleeding patients may be at risk of anemia, hypoxia, thrombocytopenia and hypercoagulable state. Also the study proved the GIB have the quite similar effect in patients regardless of gender and age although the males were more frequently affected by disease.

## المستخلص

هذه دراسة وصفية مقطعية تهدف إلى قياس مستويات متوسط تعداد الدم الكامل ومدة التخثر في المرضى الذي يعانون من النزف المعوي. أجريت الدراسة من يونيو إلى سبتمبر 2018، في مركز محمد ادريس صالح للنزف المعوي في مستشفى ابن سينا في ولاية الخرطوم. قسم مجتمع الدراسة إلى مجموعتين من مختلف الفئات العمرية والجنس (66 رجل و24 امرأة) من فئة عمرية تتراوح بين 18-70 سنة. تم تعيين مجموعة من 90 شخص لهذه الدراسة 60 مريض يعانون من النزف المعوي الحميد مع التعرض للنزيف لأول مرة ، 30 متطوعين أصحاء كمجموعة ضابطة. تم جمع البيانات باستخدام الفحوصات المخبرية للحصول على نتائج تعداد الدم الكامل ومدة التخثر أيضا جمعت البيانات بواسطة الإستبيان. 5مل من الدم الوريدي تم جمعها من جميع المشاركين، 2,5مل تم وضعها في حاويات EDTA المضادة للتخثر ، و5مل تم وضعها في حاويات trisodium citrate المضادة للتخثر. وتم تحليلها بواسطة الحزمة الإحصائية للعلوم الإجتماعية الاصدار 16. أظهرت البيانات أن حوالي 66% من مرضى النزف المعوي كانوا من الذكور بينما 24% كانوا من الإناث. توزيع مرضى النزف المعوي أظهر أن متوسط الأعمار كان 44 سنة. المظاهر السريرية للمرضى قسمت إلى قىء دم بنسبة 41% و براز أسود بنسبة 34.4% و بلهارسيا بنسبة 18.9% والبراز المدمي بنسبة 13.3%. أظهرت هذه الدراسة إن متوسط خضاب الدم وعدد كريات الدم الحمراء وحجم خلايا الدم المضغوطة ومتوسط حجم الخلية ومتوسط تركيز الخلايا والصفائح الدموية في مرضى النزف المعوي انخفضت إنخفاضا ذو دلالة معنوية مقارنة بمجموعة الأصحاء. ارتفعت قيم عدد كريات الدم البيضاء ومدة التخثر ارتفاع ذو دلالة معنوية مقارنة مع مجموعة الأصحاء ، في حين لا يوجد تغيير في متوسط خضاب الدم في الخلايا بين مرضى النزف المعوي. أظهرت الدراسة إحصائية إصابة مرضى النزف المعوي بفقير الدم ونقص الأوكسجين ونقص الصفائح الدموية وحالة فرط تخثر الدم. كما أوضحت الدراسة أن النزف المعوي لديه تأثير مماثلا لكل المرضى بغض النظر عن السن والجنس والذكور كانوا أكثر تأثرا بالمرض.

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## **LIST OF ABBREVIATION**

<b>CBC</b>	Complete blood count
<b>RBC</b>	Red blood cell
<b>WBC</b>	White blood cell
<b>PCV</b>	Packed cell volume
<b>HB</b>	Haemoglobin
<b>MCV</b>	Mean corpuscular volume
<b>MCH</b>	Mean corpuscular haemoglobin
<b>MCHC</b>	Mean corpuscular haemoglobin concentration
<b>PLT</b>	Platelets
<b>PT</b>	Prothrombin time
<b>GIB</b>	Gastrointestinal bleeding
<b>UGIB</b>	upper gastrointestinal bleeding
<b>LGIB</b>	Lower gastrointestinal bleeding
<b>OGIB</b>	Occult gastrointestinal bleeding

# CHAPTER ONE

## INTRODUCTION AND LITERATURE REVIEW.1.

### 1.1 INTRODUCTION:

The main purpose of the gastrointestinal tract is the transport of food and the absorption of nutrients. Many pathologic conditions of the gastrointestinal tract impair either or both of these function.(Salminen *et al.*, 1998, Hall, 2015).

Gastrointestinal bleeding is a common problem medical practitioners encounter in the emergency department and in the primary care setting (kim,*et al*,2014).It is a symptom of a problem rather than a disease itself. It usually happens due to conditions that can be cured or controlled, such as hemorrhoids. It differ from internal bleeding where blood leaks from the blood vessels in another way cant not seen outside the body(Saljoughian,2009).It known as any bleeding in the GI tract from the mouth, oesophagus, stomach, small intestines, large intestines, to the anus,it differentiated into upper and lower GI bleeding, with the point of division being proximal or distal to the ligament of treitz respectively (El-tawil,2011).The clinical evaluation of gastrointestinal bleeding depends on the hemodynamic status of the patient and the suspected source of the bleeding(Ghosh,2002). Upper gastrointestinal bleeding constituted with oesphgal varices about 90,3% in sudanese patients.(Salih , *et al*,2009).

Lower gastrointestinal bleeding constituted 5.37% of total cases of gastrointestinal bleeding in Sudanese parients. (Arabi,*et al*,2018).The most common risk factor for gastrointestinal bleeding include: helibactor pylori infection, non steroidal anti-inflammatory edication, aspirin, selective serotonin reuptake inhibtor and other anti platelet and anticoagulant medication.(Tielleman,2015).

Upper endoscopy or colonoscopy are generally considered the best methods to identify the source of bleeding (Ghosh,2002).

## **1.2 Literature Review:**

### **1.2.1 Gastrointestinal tract:**

Gastrointestinal tract also known as the digestive tract, alimentary tract, or gut.(Guyton and Hall, 2006)

#### **1.2.1.1. Function of digestive tract:**

The gastrointestinal tract has several function includes the digestion which mean the break down complex food into simpler one, the absorption involve transport.

the product that digested to the blood stream and excretion .(Khurana and khurana,2018)

#### **1.2.1.2. Layers of digestive tract:**

The wall of the GI tract is formed by four layers: Mucus layer, Submucus layer, Muscular layer and Serous or fibrous layer. (Sembulingam and Sembulingam, 2012).

- **Mucous layer:**

Is the innermost layer of the wall of GI tract,it is also called gastrointestinal mucosa. (Sembulingam and Sembulingam, 2012)

It consist of : a mucosal layerconsists of a layer of epithelial cells, a lamina propria, and a muscularis mucosae. The epithelial cells are specialized to carry out absorptive and secretory functions ,the lamina propria consists primarily of connective tissue, but it also includes blood and lymph vessels and the muscularis mucosae consists of smooth muscle cells contraction of the muscularis mucosae changes the shape and surface area of the epithelial cell layer.(Khurana, 2014)

Submucus layer:

is also present in all parts of GI tract, except the mouth and pharynx

It contains loose collagen fibers, elastic fibers, reticular fibers and few cells of connective tissue. Blood vessels, lymphatic vessels and nerve plexus are present in this layer. (Sembulingam and Sembulingam, 2012).

- **Muscular Layer:**

Muscular layer in lips, cheeks and wall of pharynx contains skeletal muscle fibers the esophagus has both skeletal and smooth muscle fibers. Wall of the stomach and intestine is formed by smooth muscle fibers. (Sembulingam and Sembulingam, 2012) .

- **Serous or fibrous layer:**

Outermost layer of the wall of GI tract is either serous or fibrous in nature. The serous layer is also called serosa or serous membrane and it is formed by connective tissue and mesoepithelial cells. It covers stomach, small intestine and large intestine, the fibrous layer is otherwise called fibrosa and it is formed by connective tissue, it covers pharynx and esophagus. (Hall, 2015)

### **1.2.1.3 Organs:**

GIT consist of two parts: upper GI tract which include (Mouth, pharynx, esophagus, and stomach) and lower GI tract (small intestine, large intestine and anus). In addition to associated gland such as (salivary gland, pancreas and liver). (Herrington, 2014).

#### **1.2.1.3.1. Mouth:**

Mouth is loosely used term to denote the external opening and for the cavity it leads to. The cavity containing anterior two-thirds of tongue and teeth is the mouth cavity or oral cavity. (Khurana, 2014).

#### **1.2.1.3.2. Tongue:**

The tongue is a mass of striated muscle covered by mucosa, which manipulates ingested material during mastication and swallowing. The muscle fibers are oriented in all directions, allowing a high level of mobility. Connective tissue between the small fascicles of muscle is penetrated by the lamina propria which makes the mucous membrane strongly adherent to the muscular core.

The lower surface of the tongue is smooth, with typical lining mucosa. The dorsal surface is irregular, having hundreds of small protruding papillae of various types on its anterior two-thirds and the massed lingual tonsils on the posterior third, or root of the tongue. The papillary and tonsillar areas of the lingual surface are separated by a V-shaped groove called the sulcus terminalis. (Junqueira and Mescher, 2013).

#### **1.2.1.3.3. Salivary gland:**

The salivary glands are tubuloalveolar glands and may contain mucous cells, serous cells, or both. The parotid gland is purely serous. The submandibular gland is mixed type but is predominantly serous, whereas

the sublingual gland though also a mixed gland is predominantly mucous type.

The secretory acini of the major salivary glands are drained by ducts lined by: low cuboidal epithelium in the intercalated portion, tall columnar epithelium in the intralobular ducts and simpler epithelium in the secretory ducts.

The product of major salivary glands is saliva which performs various functions such as lubrication for swallowing and speech, and has enzyme amylase and antibacterial properties. (Harsh, 2010)

#### **1.2.1.3.4. Teeth:**

The teeth consist of three specialized mineralized tissues with underlying soft-tissue pulp. Dentine is a thick layer of tubular, calcified, collagenous tissue that surrounds the pulp. On the crown of the tooth, the dentine is covered by enamel, an acellular tissue consisting largely of calcium apatite crystals in a delicate organic matrix. Cementum overlies the root dentine. At the apex of each root is one or more foramina through which vessels and nerves enter the pulp.

The teeth are attached to the jaws by the periodontium, a specialized supportive complex comprising cementum, the periodontal ligament, alveolar bone, and gingiva.

Adult complement of 32 teeth by the late teens. (Herrington, 2014).

#### **1.2.1.3.5. Pharynx:**

The pharynx is a musculofascial tube, incomplete anteriorly, which extends from the base of the skull to the oesophagus and which acts as a common entrance to the respiratory and alimentary tracts. From above downwards, it is made up of three portions the nasopharynx: lying behind the nasal fossae and above the soft palate; the oropharynx : lying behind the anterior pillars of the fauces; and the laryngopharynx: lying behind the larynx. (Donner *et al.*, 1985).

#### **1.2.1.3.6. Esophagus:**

Averaging some 25 cm in length, the oesophagus is a muscular tube with a well-defined origin at the cricoid cartilage. Its function is a simple one, namely the conduction of food from the pharynx to the stomach. This simplicity is reflected in its structure. The mucosal lining is of stratified squamous epithelium, whereas the underlying submucosa includes numbers of mucinous glands, which lubricate the lining. (Herrington, 2014).



#### **1.2.1.3.7. Stomach:**

Stomach is a hollow organ situated just below the diaphragm on the left side in the abdominal cavity. Volume of empty stomach is 50 mL. Under normal conditions, it can expand to accommodate 1 L to 1.5 L of solids and liquids, however, it is capable of expanding and still further up to 4L. (Sembulingam and Sembulingam, 2012)

#### **1.2.1.3.8. Pancreas:**

The pancreas is a mixed exocrine-endocrine gland that produces both digestive enzymes and hormones. It is an elongated retroperitoneal organ, with a large head near the duodenum and more narrow body and tail regions that extend to the left, it has a thin capsule of connective tissue, from which septa extend to cover the larger vessels, and ducts to separate the parenchyma into lobules. The secretory acini are surrounded by a basal lamina that is supported only by a delicate sheath of reticular fibers with a rich capillary network, endocrine function of the pancreas involves primarily smaller cells similar to enteroendocrine cells located in variously sized clusters called the pancreatic islets (islets of Langerhans). (Mescher, 2013).

#### **1.2.1.3.9. Liver:**

The liver is the largest organ in the body weighing 1400-1600 gm in the males and 1200-1400 gm in the females. There are 2 main anatomical lobe right and left the right being about six times the size of the left lobe. The right lobe has quadrate lobe on its inferior surface and a caudate lobe on the posterior surface. The right and left lobes are separated anteriorly by a fold of peritoneum called the falciform ligament, inferiorly by the fissure for the ligamentum teres, and posteriorly by the fissure for the ligamentum venosum (Harsh, 2010)

#### **1.2.1.3.10 Gall bladder:**

The gallbladder is a pear-shaped organ, usually 10 cm in length and 3 to 5 cm in diameter that is attached to the inferior surface of the liver. Two thirds of the gall bladder is covered by peritoneum. The fundus of the gallbladder projects beyond the liver; the body (or corpus) is in contact with the second portion of the duodenum and the colon and the infundibulum (Hartmann pouch), located; at the free edge of the lesser omentum, bulges forward toward the cystic duct. The neck is the part of the gallbladder between the body and the cystic duct. (Floch *et al.*, 2010)

#### **1.2.1.3.11. Small intestine:**

The small intestine extends from the pylorus to the ileocecal valve and may measure 3.5 to 6.5 m in length. The small intestine is divided into three parts, duodenum: Primarily retroperitoneal, ends at the ligament of treitz supplied by the pancreaticoduodenal branch of the hepatic artery, jejunum: Contains plicae circularis giving the mucosa a folded appearance; supplied by the superior mesenteric artery and ileum contains peyer patches (submucosal lymphoid aggregates); supplied by the superior mesenteric artery (Alpers *et al.*, 2011).

#### **1.2.1.3.12. Large intestine:**

The large intestine consists of the colon and rectum and measures 0.9 to 1.25 m in length. The colon is subdivided into the cecum, ascending colon, transverse colon, descending colon, and sigmoid colon. The superior mesenteric artery supplies the proximal half of the large intestine, and the inferior mesenteric artery supplies the distal half. The regular out pouchings of the colon are termed haustra (Davenport, 1966)

#### **1.2.4 . Gastrointestinal bleeding :**

Gastrointestinal bleeding (GIB) is a bleeding that starts in the gastrointestinal tract, which extends from the mouth to the large bowel. (Bresci,2009)

##### **1.2.4.1. Types of git bleeding:**

##### **1.2.4.2. Upper git bleeding:**

Upper gastrointestinal bleeding (UGIB) is defined as bleeding that results from lesions located above the ligament of Treitz.(Masoodi and Saberifiroozi, 2012).

The colour of vomited blood depends on the concentration of hydrochloric acid in the stomach in addition to the duration of its contact with the blood. When vomiting takes place shortly after the onset of bleeding it will appear dark red, and when it stays longer it will appear brown or black. The coffee ground appearance is due to the action of hydrochloric acid on hemoglobin which will change to hematin giving it this characteristic appearance(Isselbacher *et al.*, 1996).The UGIB characterize by:

- **Hematemesis:**

Which is vomiting of red-colored blood usually from an arterial source or varix. (Marignani,2005).

- **Melena:**

Passage of black tarry stools are common when there is bleeding from any part of upper GIT ◦ The black color of melenic stool is caused by iron sulfide, formed by the action of byproducts from colonic bacteria on heme iron.(Davenport, 1966).

#### **1.2.4.2.1 Causes of upper gastrointestinal bleeding:**

- **Peptic ulcer disease:**

Peptic ulcer is a broad term which includes ulcers of digestive tract in the stomach or the duodenum. The causative agent is infection caused by the bacteria *H. pylori* or reaction to certain medicines like non-steroidal anti-inflammatory drugs (NSAIDs)<sup>1</sup>. Symptoms of peptic ulcers include abdominal discomfort and pain. Other symptoms include weight loss, poor appetite, bloating, nausea, and vomiting. Some may also experience blood in stool and vomit, and black stools that indicate gastrointestinal bleeding. (Amandeep *et al* ,2012).

- **Mallory-Weiss Tears:**

is a common cause of non-variceal upper gastrointestinal bleeding. Its characterized by linear, non-perforating mucosal laceration at the lower part of the esophagus and/or upper part of the stomach, with an incidence of 5% to 15%. Although most patients with bleeding MWT require no intervention other than hemodynamic support. (Koo *et al.*, 2015).

- **Other causes:**

Esophageal varices ,Miscellaneous, cancer of stomach, duodenal erosion and esophagitis. (Guelrud, 2017).

#### **1.2.4.2.2. Management of upper git bleeding:**

Intravenous access , oxygen, sedation, blood volume replacement, endoscopy, pharmacological measure, proton pump inhibitor ,octreotide and surgery. .(Hall, 2015)

#### **1.2.4.3 Lower GIT bleeding :**

Lower GI bleeding is defined as bleeding that originates from a site distal to the ligament of treitz. (Key, 2017).

Lower GI bleeding has been further subdivided into mid GI bleeding coming from the small bowel between the ampulla of Vater to the terminal ileum, and lower GI bleeding coming from the colon ( Raju, 2007).It

classically presents with hematochezia passing of red blood from rectum (Cappell and Friedel, 2008)

Hematochezia might also result from vigorous upper GI bleeding with rapid transit of blood through the intestines. (Marignani,2005)

#### **1.2.4.3.1 Causes of LGIB:**

- **Diverticulosis:**

Diverticular bleeding is a common cause of lower gastrointestinal hemorrhage. Its develop at sites of weaknesses in the colonic wall that occur where the vasa recta penetrate the circular muscle layer. As a diverticulum herniates, the vasa recta drape over the dome of the diverticulum and become susceptible to trauma and disruption. Inflammatory changes are not seen histologically, and diverticulitis does not usually coexist with diverticular bleeding. Although diverticula typically occur throughout the colon, diverticular bleeding tends to occur in the thinner-walled ascending (right) colon, Patients typically present with massive and painless rectal hemorrhage.(Kaltenbach *et al.*, 2012)

- **Angiodysplasia:**

Angiodysplasia (angioectasia or vascular ectasia) are abnormally dilated, tortuous, thin-walled vessels, involving capillaries, veins, and arteries. They are visualized within the mucosal and submucosal layers of the gut. They are lined by endothelium with little or no smooth muscle, and lack inflammatory or fibrotic changes, They are the most common cause of small bowel bleeds, particularly in patients older than 40 years of age.(Gunjan,2014)

Other causes may be associated with LGIB such as:

( Ischemic colitis ,colonic ulcer, hemorrhoid and neoplasia).(Ghassemi and Jensen,2013)

#### **1.2.4.3.2. Management of LGIB:**

Rectal examination anoscopy , sigmoidoscopy, excluded an upper tract source, technetium-99 RBCS scan , colonoscopy and angiography.  
(Lhewa and Strate, 2012)

#### **1.2.4.4. Occult GIT bleeding:**

Defines as the initial Presentation of a positive fecal occult blood test (FOBT) result and/or iron-deficiency anemia when there is no evidence of visible blood loss to the patient or clinician.( Raju, 2007)

##### **1.2.4.4.1. Causes of OGIB:**

inflammatory, vascular, and infectious more common causes include colorectal cancer (especially right-sided colon), severe esophagitis, gastric or duodenal ulcers including from the use of aspirin and other NSAIDs, inflammatory bowel disease, gastric cancer, celiac disease, vascular ectasias (any site), diverticula, and portal hypertensive gastropathy.(Kim *et al* ,2014)

#### **1.2.5 . Complete blood count :**

##### **1.2.5.1 . Blood :**

Blood is a specialized connective tissue in which cells are suspended in fluid extracellular material called plasma, Propelled mainly by rhythmic contractions of the heart, about 5 L of blood in an average adult move unidirectional within the closed circulatory system so-called formed circulating elements in the plasma are erythrocytes(red blood cells), leukocytes (white blood cells), and platelets.(Junqueira and Mescher, 2013).

##### **1.2.5.2 . Plasma :**

Plasma is an aqueous solution, pH 7.4, containing substances of low or high molecular weight that make up 7% of its volume. As summarized in, the dissolved components are mostly plasma proteins, but they also include nutrients respiratory gases, nitrogenous waste products, hormones, and inorganic ions, collectively called Electrolytes.(Junqueira and Mescher, 2013).

### **1.2.5.3. Red blood cell:**

RBCs are a nucleate, biconcave, discoid cells filled with a reddish protein, hemoglobin (HGB), which transports oxygen and carbon dioxide. RBCs appear pink to red and measure 6 to 8 mm in diameter with a zone of pallor that occupies one third of their center reflecting their biconcavity. (Keohane.*et al.*,2016).

#### 1.2.5.4 . Heamoglobin :

Haemoglobin is a conjugated protein of molecular weight 64 000, consisting of two pairs of polypeptide chains to each of which a haem is attached. Human haemoglobin exists in a number of types, which differ slightly in the structure of their globin moiety. (Firkin, *et al.*,2011).

#### 1.2.5.5. HEMATOCRIT

Blood consists of a fluid portion (plasma) and a solid portion that includes RBCs, WBCs, and platelets. More than 99 percent of the total blood cell mass is composed of RBCs. The Hct or packed RBC volume measures the proportion of RBCs in a volume of whole blood and is expressed as a percentage.(Cavanaugh, 2003).

#### 1.2.5.6. MCV:

The mean red cell volume (MCV) provides information on red cell size. It is measured in femtolitres(fl) and is determined from the PCV and electronically obtained RBC count. It can be calculated as follow:

$$MCV = \frac{PCV}{RBC \times 10^{-12} \text{ L/L}}$$

A femtolitre (fl) is 10<sup>-15</sup> litre.

Interpretation of MCV values:

There is some variation in reference ranges for MCV depending on the method used by manufacturers of blood cell analyzers to obtain the MCV value and how an instrument has been calibrated. ( Cheesbrough, 2006).

#### 1.2.5.7. MCH:

The MCH gives the amount of haemoglobin in picograms (pg) in an average red cell It is calculated from the haemoglobin and electronically obtained RBC count:  $MCH = \frac{Hb \text{ g/L}}{RBCs \times 10^{-12} /L}$

A picogram (pg) is 10<sup>-12</sup> of a gram( Cheesbrough , 2006).

#### 1.2.5.8. MCHC:

It gives the concentration of haemoglobin in g/l in 1 litre of packed red cells. It is calculated from the haemoglobin (Hb) and PCV  $MCHC = \frac{Hb \text{ g/L}}{HCT \text{ L/L}}$

If using g/dl divide the g/l figure by 10. (Cheesbrough, 2006).

#### **1.2.5.9. Platelets:**

Platelets are small fragments of megakaryocyte cytoplasm with an average volume of 7–8 fl.40,41 When seen in Romanowsky-stained blood smears, most platelets have a diameter of 2–3  $\mu\text{m}$ . They have an irregular outline, stain light blue and contain a number of small azurophilic granules that are usually concentrated at the center.(Porwit *et al.*, 2011).

#### **1.2.5.10. White blood cell:**

White blood cells are produced from pluripotent stem cells located within the bone marrow. Development of white blood cells along different lineages is governed by external stimuli including cytokines, matrix proteins, and other cellular products within the marrow environment. The combination of specific cytokines and growth factors influence the maturation of white blood cell progeny along specific lineages. (Hoffbrand *et al.*, 2016).

#### **1.2.5.11. Neutrophil:**

Neutrophil granulocytes have a mean volume of 500 fl and, in dried fixed smears, a diameter of 9–15  $\mu\text{m}$ . Their cytoplasm is slightly acidophilic and contains many very fine granules that stain with neutral dyes; the granules stain a faint purple color with Romanowsky stains The nucleus usually contains two to five nuclear segments (Porwit *et al.*, 2011).

#### **1.2.5.12 . Eosinophil :**

Are little larger than neutrophils 12-17 $\mu\text{m}$  in diameter they usually have two nuclear lobes or segments and the cytoplasm is packed with distinctive spherical gold/orange granules.(Bain *et al.*, 2016).

#### **1.2.5.13 . Basophil :**

Are the rarest <1% of the circulating leucocytes, their nuclear segment tend to fold up on each other, resulting in compact irregular dense nucleus resembling a closed lotus flower, the distinctive larger visibly sized dark blue or purple granules of the cytoplasm. (Bain *et al.*, 2016).



#### **1.2.5.14 . Monocytes :**

Are the largest of circulating leucocyte 15-18  $\mu\text{m}$  in diameter. They have bluish-grey cytoplasm that contains variable numbers of fine reddish granules, the nucleus is large and curved, often in the shape of a horseshoe but may be folded or curled. (Bain *et al.*, 2016)

#### **1.2.6 . Hemostasis :**

Coagulation (also known as clotting) is the process by which blood changes from a liquid to a gel, forming a blood clot. It potentially results in hemostasis, the cessation of blood loss from a damaged vessel, followed by repair. The mechanism of coagulation involves activation, adhesion, and aggregation of platelets along with deposition and maturation of fibrin. Disorders of coagulation are disease states which can result in bleeding (hemorrhage or bruising) or obstructive clotting (thrombosis). (David *et al.* 2009).

Coagulation begins almost instantly after an injury to the blood vessel has damaged the endothelium lining the vessel. Leaking of blood through the endothelium initiates two processes: changes in platelets, and the exposure of subendothelial tissue factor to plasma Factor VII, which ultimately leads to fibrin formation. Platelets immediately form a plug at the site of injury; this is called primary hemostasis. Secondary hemostasis occurs simultaneously: Additional coagulation factors or clotting factors beyond Factor VII respond in a complex cascade to form fibrin strands, which strengthen the platelet plug. (Furie, 2005)

The coagulation cascade of secondary hemostasis has two initial pathways which lead to fibrin formation. These are the contact activation pathway (also known as the intrinsic pathway), and the tissue factor pathway (also known as the extrinsic pathway) which both lead to the same fundamental reactions that produce fibrin. It was previously thought that the two pathways of coagulation cascade were of equal importance, but it is now known that the primary pathway for the initiation of blood coagulation is the tissue factor (extrinsic) pathway. The pathways are a series of reactions, in which a zymogen (inactive enzyme precursor) of a serine protease and its glycoprotein co-factor are activated to become active components that then catalyze the next reaction in the cascade, ultimately resulting in cross-linked fibrin. Coagulation factors are generally indicated by Roman numerals, with a lowercase letter appended to indicate an active form. (Pallister *et al.*, 2010)

The coagulation factors are generally serine proteases (enzymes), which act by cleaving downstream proteins. There are some exceptions. For

example, FVIII and FV are glycoproteins, and Factor XIII is a transglutaminase. The coagulation factors circulate as inactive zymogens. The coagulation cascade is therefore classically divided into three pathways. The *tissue factor* and *contact activation* pathways both activate the final common pathway of factor X, thrombin and fibrin (Hoffbrand,2002)

#### **1.2.6.1 Tissue factor pathway (extrinsic):**

The main role of the tissue factor pathway is to generate a "thrombin burst", a process by which thrombin, the most important constituent of the coagulation cascade in terms of its feedback activation roles, is released very rapidly. FVIIa circulates in a higher amount than any other activated coagulation factor. The process includes the following steps:

Following damage to the blood vessel, FVII leaves the circulation and comes into contact with tissue factor (TF) expressed on tissue-factorbearing cells (stromal fibroblasts and leukocytes), forming an activated complex (TF-FVIIa). TF-FVIIa activates FIX and FX.

FVII is itself activated by thrombin, FXIa, FXII and FXa, the activation of FX (to form FXa) by TF-FVIIa is almost immediately inhibited by tissue factor pathway inhibitor (TFPI).

FXa and its co-factor FVa form the prothrombinase complex, which activates prothrombin to thrombin. then activates other components of the coagulation cascade, including FV and FVIII (which forms a complex with FIX), and activates and releases FVIII from being bound to vWF. FVIIIa is the co-factor of FIXa, and together they form the tenase complex, which activates FX; and so the cycle continues. (Tenase is a contraction of "ten" and the suffix "-ase" used for enzymes.) (Pallister *et al.*, 2010).

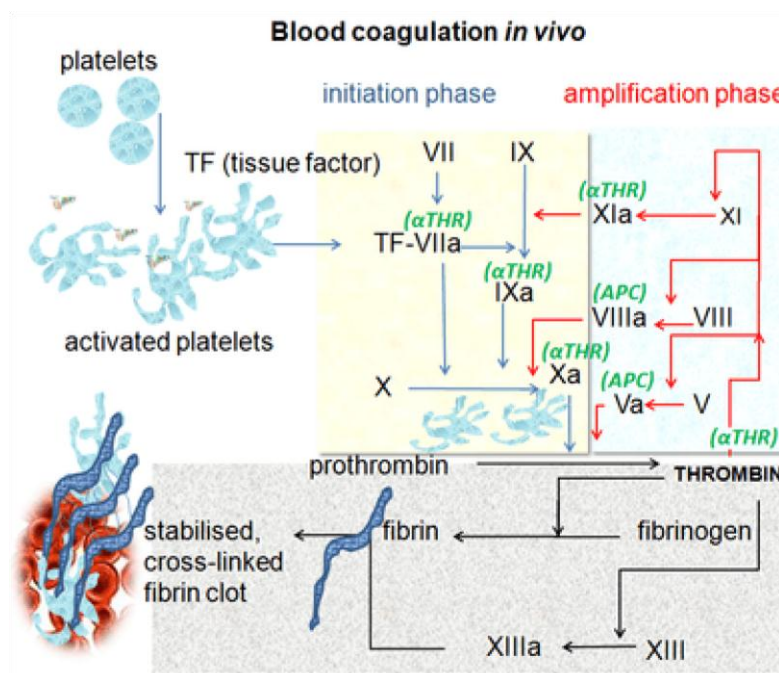
#### **1.2.6.2. Contact activation pathway (intrinsic):**

The contact activation pathway begins with formation of the primary complex on collagen by high-molecular-weight kininogen (HMWK), prekallikrein, and FXII (Hageman factor). Prekallikrein is converted to kallikrein and FXII becomes FXIIa. FXIIa converts FXI into FXIa. Factor XIa activates FIX, which with its co-factor FVIIIa form the tenase complex, which activates FX to FXa. The minor role that the contact activation pathway has in initiating clot formation can be illustrated by the fact that patients with severe deficiencies of FXII, HMWK, and

prekallikrein do not have a bleeding disorder. Instead, contact activation system seems to be more involved in inflammation (Pallister *et al.*, 2010).

### 1.2.6.3. Final common pathway:

The division of coagulation in two pathways is mainly artificial, it originates from laboratory tests in which clotting times were measured after the clotting was initiated by glass (intrinsic pathway) or by thromboplastin (a mix of tissue factor and phospholipids). In fact thrombin is present from the very beginning, already when platelets are making the plug. *Thrombin* has a large array of functions, not only the conversion of fibrinogen to fibrin, the building block of a hemostatic plug. In addition, it is the most important platelet activator and on top of that it activates Factors VIII and V and their inhibitor protein C (in the presence of thrombomodulin), and it activates Factor XIII, which forms covalent bonds that crosslink the fibrin polymers that form from activated monomers, following activation by the contact factor or tissue factor pathways, the coagulation cascade is maintained in a prothrombotic state by the continued activation of FVIII and FIX to form the tenase complex, until it is down-regulated by the anticoagulant pathways. (Pallister *et al.*, 2010)



**Blood coagulation pathways in vivo showing the central role played by thrombin (Furie *et al.*, 2005)**

### 1.2.7. Previous studies:

A study of Low hemoglobin levels are associated with upper gastrointestinal bleeding submitted by Tomizawa *et al.*,(2016).

Patients were selected from all of those who had endoscopy at the National Hospital , organization Shimoshizu Hospital, Yotsukaido, Chiba, Japan from October 2014, to September 2015. Endoscopy was performed in 1,023 patients, 431 men (mean age  $68.1 \pm 12.9$  years), and 592 women ( $66.4 \pm 12.3$  years ).

Screening blood tests included white blood cell count (WBC), hemoglobin (Hb), platelet (Plt), C-reactive protein (CRP), total protein, albumin (Alb), alkaline phosphatase, aspartate aminotransferase, alanine aminotransferase (ALT),  $\gamma$ -glutamyl transpeptidase, lactate dehydrogenase, uric acid, blood urea nitrogen (BUN), creatinine (Cre), triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, blood glucose, and HbA1c.

To identify blood test variables associated with upper GI bleeding, oneway analysis of variance was performed. WBC ( $P=0.02$ ), Plt ( $P=0.005$ ), and BUN ( $P=0.04$ ) were elevated and Hb ( $P.V < 0.001$ ) and Alb ( $P=0.005$ ) were reduced in patients with upper GI bleeding. (Tomizawa *et al*,2016).

Another study of Assessment of some coagulation parameters in GIB Patients in a Sudanese Center for GI by( El-khair, 2011).

This study included 100 (50 were selected randomly from patients with GIT bleeding who were attending to Mohammed Saleh Idres center for GI bleeding at Ibn Seena Hospital, Khartoum Sudan the other 50 were healthy volunteers , matched in age and sex with patients as control group All participants were investigated for dependent study variables (prothrombin time (PT), activated partial thromboplastin time (APTT)and platelets count) and independent variables (cause, history of GI bleeding, age and gender) using automated coagulometer. The results obtained showed that PT and APTT were significantly prolonged in patients than in control ( $P=0.00$ ), while platelets count was significantly decreased in patients than in control ( $P=0.00$ ). (Elkhair,2011).

Also another study done in the same hospital by munsoor,2012 from January to march 2010 to assessment of complete blood count in gastrointestinal bleeding patients referring gastrointestinal bleeding center. The patients were adult Sudanese with GIB (44 males and 21 females) at age range from 16-80, (50 patients and 15 controls) The results of this study showed that, the mean of patientes hemoglobin (Hb)

was 8.01 gm/dl, the mean of packed cell volume (PCV) was 24.91%, platelets count was 157.90/mm<sup>3</sup>, mean corpuscular hemoglobin (MCH) was 2.39 pg, mean corpuscular hemoglobin concentration (MCHC) was 31.37g/dl and total of white blood cell (WBC) count was 10,100 cells/mm<sup>3</sup>. These values were statistically different ( $p < 0.05$ ) when compared to that of control. This study concluded that, patients with GI bleeding were at risk of anemia, hypoxia and other complication.

### **1.3. Rationale:**

Gastrointestinal bleeding is a common clinical problem frequently requiring hospitalization. Its type of bleeding originated in GI. So the complete blood count is necessary to assess the level of blood loss and prothrombin time to assess the bleeding risk, help in prognosis of coagulation disturbance.

### **1.4. Objectives:**

#### **1.4.1 General objective:**

Assessment of complete blood count and some coagulation parameter (PT) in patients with gastrointestinal bleeding attending to Mohammed Salih bleeding center.

#### **1.4.2. Specific objective:**

To measure HB, PCV, RBC, WBCS, PLT, MCV, MCH and MCHC in patients and control group

To measure PT in control group and patients.

Correlation between result and demographic data age and gender.

## CHAPTER TWO

### 2. MATERIAL AND METHOD

#### 2.1 Study design and area :

This is descriptive (cross sectional) study was conducted through the period from June to September 2018 .The study was carried out at Khartoum state in Mohammed salih idres gastrointestinal bleeding center in ibn sina hospital .

#### 2.2 Study population :

The study population comprises two groups of adult in different age for both sex was included 60 patients with acute GIB bleeding and 30 normal as control group.

##### 2.2.1 . Inclusion criteria :

All Sudanese patients with GIB and first time bleeding.

##### 2.2.2 . Exclusion criteria :

Any patients who takes aspirin or other medication, smoker and pregnancy, that interfere or affect the test result.

#### 2.3 Data collection tools :

Data was collected using structured questionnaire type including age and sex. Laboratory investigation was performed to obtain CBC and prothrombin result.(Appendix)

#### 2.4 Sample collection :

Five ml of venous blood sample was collected 2.5 ml was added slowly to 0.25 ml of 0.38% trisodium citrate for prothrombin test.

The rest 2.5ml was added to EDTA container for CBC.

#### 2.5 Methodology :

Complete blood count (HB, PCV,RBC, MCV, MCH, MCHC,TWBC and PLT) was done using hematological analyzer sysmex kx21n. (Cooperation japan)The coagulation test(PT) was performed using sysmex CA 50.(Cooperation japan)

### **2.5.1 Principle of kx21n :**

The Sysmex KX-21 is an automatic multi-parameter blood cell counter for in vitro diagnostic use in clinical laboratories. (Sysmex cooperation, Kobe japan).

The KX-21 processes approximately 60 samples an hour and displays on the LCD screen the particle distribution curves of WBC, RBC, and platelets, along with data of 18 parameters, as the analysis results.

The KX-21 employs three detector blocks and two kinds of reagents for blood analysis. The WBC count is measured by the WBC detector block using the DC detection method .

The RBC count and platelets are taken by the RBC detector block, also using the DC detection Method.

The HGB detector block measures the hemoglobin concentration using the noncyanide hemoglobin method.

### **2.5.2. Analysis mode:**

This instrument works in two analysis modes: whole blood mode and pre diluted mode.

#### **Whole blood mode:**

This is the mode of analyzing collected blood sample in the whole blood status. The tube cap is opened and the sample is aspirated through the sample probe one after another.

#### **Pre-diluted mode:**

This mode is used in analyzing a minute amount of child's blood, for instance, collected from the earlobe or fingertip. In this mode, blood sample diluted into 1:26 before analysis is used. The sample aspiration procedure is the same as in the whole blood mode.

### **2.5.3. Prothrombin time:**

Tissue factor (in the form of thromboplastin) and calcium are added to plasma that has been anticoagulated with citrate during collection. Tissue factor reacts with factor VIIa to activate the “extrinsic” pathway and thus form a clot. Use of the Prothrombin Time Test.

The PT is sensitive to deficiencies of factors VII, X, V, and II, and fibrinogen. The PT is particularly useful in monitoring anticoagulation in patients on vitamin K antagonist therapy such as warfarin and should be reported as International Normalized Ratio (INR) in such patients. (Nigel, *et al.*, 2017).

#### **2.5.4. Principle of sysmex CA-50:**

The CA-50 is an automated blood coagulation analyzer that can quickly analyze samples with a high degree of accuracy. The CA-50 can analyze samples using a Coagulation Method; and the analyzed data can be displayed on its LCD screen and printed by the built-in printer.

#### **2.5.5. Procedure:**

##### **Coagulation Method :**

##### **1.Coagulation Reaction Detection Method (Scattered Light Detection Method) :**

Irradiates red light (660 nm) onto a mixture of blood plasma and reagent and detects the change in turbidity (when the fibrin clots are formed) as the change in scattered light. And measures the coagulation time.

##### **2. Coagulation Point Detection Method (Percentage Detection Method):**

Calculates the coagulation time as the time required to achieve the amount of scattered light that is set for the coagulation detection point, using the amount of scattered light that is present just after the start of detection as 0%, and the amount of scattered light that is present at the completion 100%. of coagulation.

##### **Required Sample :**

- Prothrombin Time (PT): 50 uL
- Activated Partial Thromboplastin Time (APTT): 50 uL
- Fibrinogen (Fbg): 10 uL
- Thrombotest (TTO): 20 uL
- Normotest (NT): 10 uL
- Thrombin Time (TT): 100 uL Extrinsic Factor TT:
- Deficiency Assay (II, V, VII, X): 5 uL
- Intrinsic Factor Deficiency Assay (VIII, IX, XI, XII): 5 uL

##### **Detection Time:**

Detects within the maximum detection time, and measures the result.



Typical maximum detection time: 100 seconds for PT and Fbg; 190 seconds for others.

Maximum detection time: 600 seconds for each parameter

**Display:**

Displays characters on a liquid crystal display (LCD).

Printing Permits graphic printing through a built-in printer. (Printer paper specification: F1-1, width: 58 mm)

External Input/Output:

Equipped with an RS-232C serial port (bit serial voltage signal).

Detector: 4 wells (Scattered light detector)

Incubator: 5 wells

Reaction tube holder: 8 wells Temperature Control:

Detector: 37C,0.5C

Incubator: 37C,1C

Applies when room temperature is within 15C - 35C.

Time to Reach Temperature Setting:

Reaches preset temperature within 30 minutes after power is turned ON (when the room temperature is within the specified range)

**2.6 Ethical approval :**

Ethical clearance from the ethical committee of the sudan university of sciences and technology, college of graduate studies. was taken from the patients participated in the study. Permission was obtained from ibn sina hospital.

**2.7 Data analysis:**

The data was extracted from the questionnaire and the lab reports into a major spread sheet and then fed on the statistical software SPSS version 16.0.Descriptive statistics and probability testing was done by using independent T-test. The result obtained were presented in tables and figures. Level of significances was set at <0.05. The mean was calculated  $\pm$ SD.

## CHAPTER THREE

### 3. RESULT

#### 3.1. Demographic data:

The study investigated the effect of GIB on CBC and prothrombin time

Fig 3.1 Indicate that about 66% of GIB patients are male and 24% are female in Khartoum state.

Fig 3.2 The distribution of GIB patients according to their age, recorded the mean age were 44 years.

Fig 3.3 Reveled the most common symptoms of GIB is hemostasis about 41%,Melena 34.4%, bilharzia 18.9% and bloody stool 13.3%.

#### 3.2. Laboratory data:

The result show the effect of GIB on CBC in the (table 3.1)

The Haemoglobin concentration value in GIB Patients was 7.95g/dl compared to 14.527g/dl in control group. The data indicated significant ( $p \leq 0.000$ ) lower value in case compared with normal group.

The total erythrocyte count value in case patients was 3.076 c/cm compared to 5.097 c/cm in control group. The case patients had significantly ( $p \leq 0.000$ ) lower value compared with normal control.

The total leukocyte count value in GIB patients was 8.563 c/cm compared to 5.743 c/cm in normal group (control) .The WBC was significantly ( $p \leq 0.001$ ) higher value in case compared with normal control.

The PCV value in case patients was 25.637% compared to 46.000% in control group. The case patients had significantly ( $p \leq 0.000$ ) lower value compared with normal control.

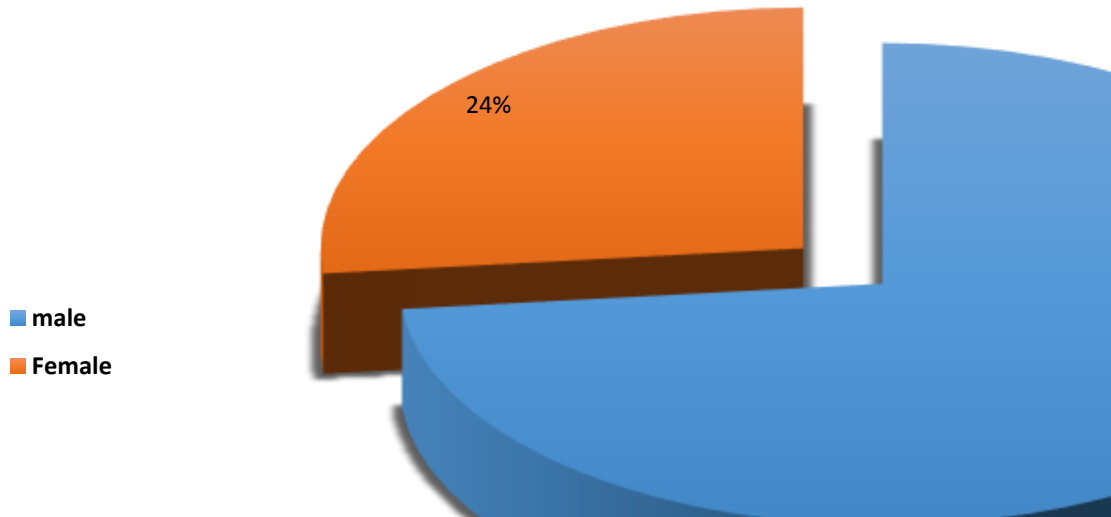
The MCV in GIB patients was 82.307fl compared to 90.197fl in normal group (control). The MCV was significantly ( $p \leq 0.000$ ) lower value in case compared with normal control.

The MCH value in case patients was 25.890 pg compared to 28.283pg in control group. The case patients had significantly ( $p \leq 0.000$ ) lower value compared with normal control.

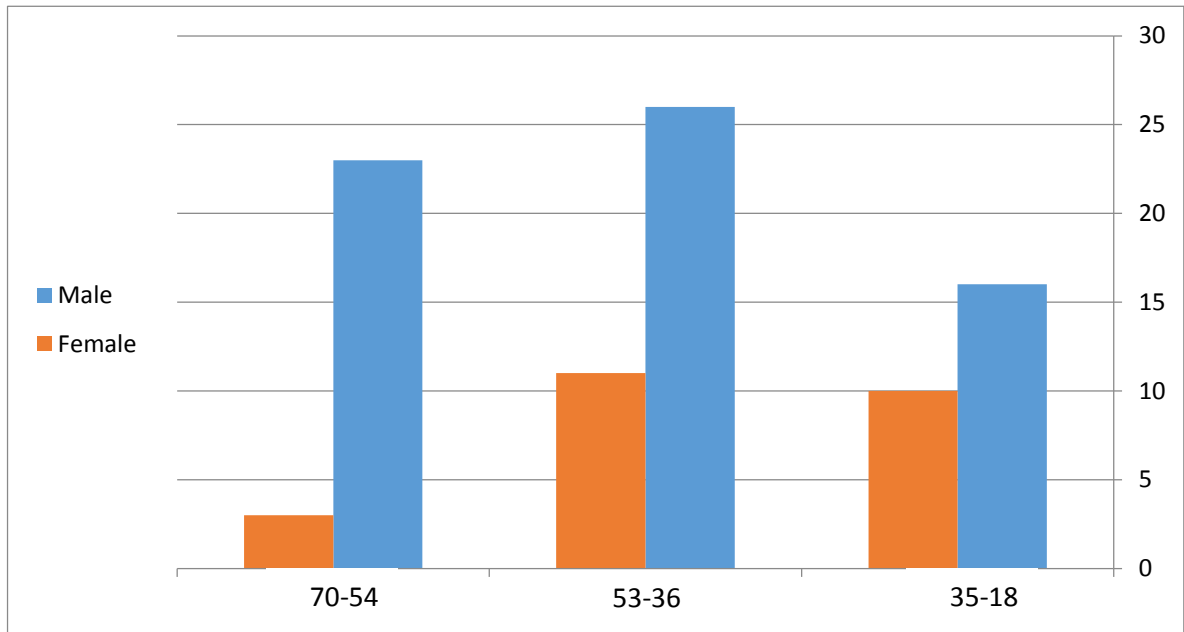
The PLts value in GIB patients was 188.367 c/cm compared to 261.367c/cm in normal group (control) The platelet was significantly ( $p \leq 0.000$ ) lower value in GIB patients compared with normal control.

Also show there was slight difference in MCHC between GIB patients and normal group .

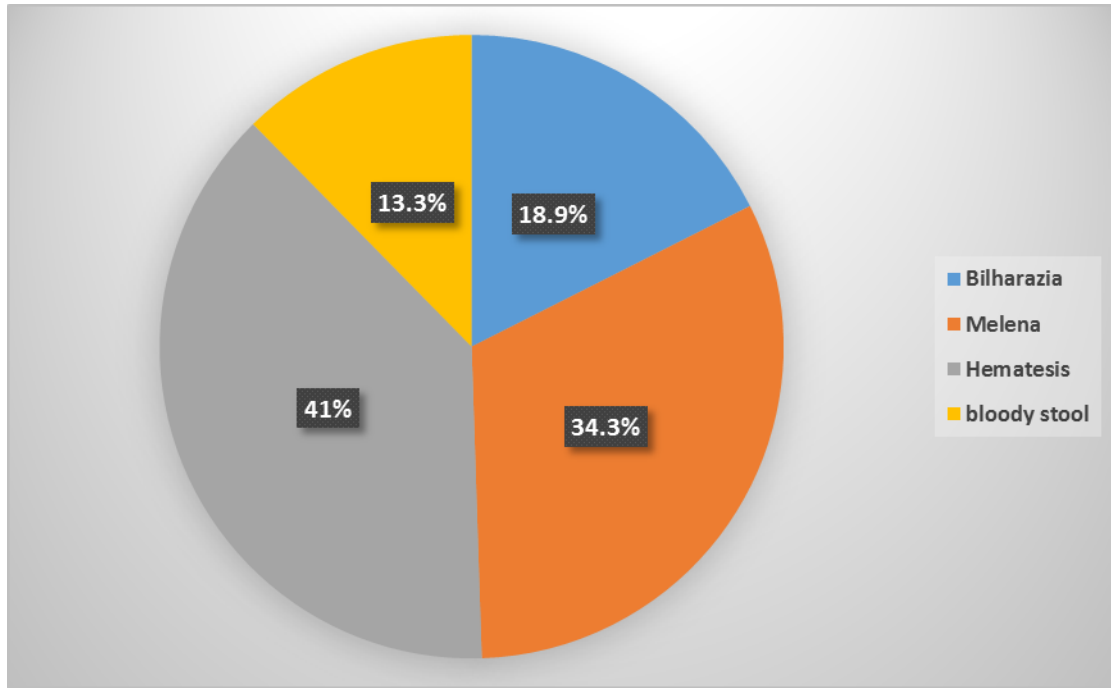
Table (3.2) shows the PT values in normal and GIB patients, the PT value in case group was 18.912 sec compared to 14.353 sec in normal group. The data indicate that the GIB patients group had significantly ( $p \leq 0.00$ ) higher value compared with normal control.



**Figure (3-1): Distrubtion of patients with GIB according to gender  
(n= 60)**



**Figure (3-2) Distribution of patients with GIB according to age group  
(n = 60)**



**Fig. (3-3) Distribution of GIB patients according to symptoms  
(n = 60)**

**Table 3-1 Mean of CBC in case and control in the study****(n=90)**

<b>Sample</b>	<b>N</b>	<b>Mean</b>	<b>P. value</b>
Hb g/dl	Case 60	7.95±2.84	0.000
	Control 30	14.527±.9836	
RBC c/cm	Case 60	3.076±1.1166	0.000
	Control 30	5.097±.5021	
WBC c/cm	Case 60	8.563±5.9723	0.001
	Control 30	5.743±1.5035	
PCV%	Case 60	25.637±9.1009	0.000
	Control 30	46.000±3.2516	
MCV fl	Case 60	82.307±9.2386	0.000
	Control 30	90.197±4.2447	
MCH pg	Case 60	25.890±4.3265	0.000
	Control 30	28.283±1.6725	
MCHC %	Case 60	31.088±3.0961	0.504
	Control 30	31.393±1.1855	
PLT c/cm	Case 60	188.367±101.8941	0.000
	Control 30	261.367±44.0818	

**Table3-2 Mean of thrombin time in case and control group  
(n=90)**

<b>Sample</b>	<b>N</b>	<b>Mean</b>	<b>P. value</b>
PT sec Case	60	18.912±3.9738	0.000
Control	30	14.353±1.0006	



## CHAPTER FOUR

### 4. Dissuaction-Conculsion and Recommandation

#### 4.1 Discussion:

The study investigated the effect of gastrointestinal bleeding on the blood constituents, estimating the mean level of various complete blood parameter (Hb, RBCS, Erythrocyte indices, WBC, Plt and some hemostatic parameter (PT) in adult Sudanese patients with gastrointestinal bleeding with age range from 17-80 years.

The present study indicate that the mean age of patients with GIB was 44 (Fig.3.2). The result agree with the finding of (Obara, 2006) and (fedial *et al*, 1993) in Sudan, these observation were supported by pervious work (Mansour and Elkhair, 2011), who reported the mean valued of GIB in Khartoum state were 33 years. this is also comparable to that reported in japan were the mean age of GIB patients 69 years. (Tomizawa.*et al*, 2016).

In this study the most common causes of gastrointestinal is hematemesis about 41%, melena 34%, bilharzia 18.9% and bloody stool 13.3%.

The present result revealed there was significantly lower PCV, Hb concentration and RBC values and was associated with decrease in MCV and MCH values, the drop in PCV lead to shifting of water from the interstitial fluid compartment to restore blood volume .(Zuckerman,2007)

Hb, MCV ,MCH and MCHC values are useful to diagnosis different types of anemia because reflect the size and hb concentration (Zuckerman,2007).

Most patients with upper gastrointestinal bleeding presents with PCV less than 30%.(Balderas.*et al*,2011).

Previous studies suggested strongly association between low Hb concentration and upper GIB, these finding may be associated with losing of blood leading to anemia. (Tomizawa.*et al*, 2014).

In the present there was significant increase in WBC in patients with gastrointestinal bleeding compared with control group this may be the hematopoietic system respond in bimodal manner, abrupt increase of white blood cell count in response to the stress of bleeding and frequently an outpouring platelet into the peripheral circulation (Sleisnger,1978). (Tomizawa.*et al*, 2014) reported that the WBC and C-reactive protein

level were elevated, because were useful indicator for inflammation, the present result agreement with (Tomizawa.*et al*, 2014) and (Mansour, 2012).

The present there was significant decrease in platelet count and prolongation of PT in patients with gastrointestinal bleeding compared with normal control group, this may be related to vitamin k deficiency from malnutrition , it disorder may give rise to vitamin k deficiency lead to impairment of in production of many coagulation factors (v,vii,ix,x) and sequent impaired the coagulation mechanism and hence git bleeding .(Aoki and Jchino,2011) .In contrast Tomizwa. *et al*,2016) reported that the platelet count were elevated in patients with upper gastrointestinal bleeding.

The result above help to management the disease through the laboratory investigation.

## **4.2 CONCLUSION:**

The study revealed that the HB, PCV, RBC, MCV, MCH, PLT had lower value in patients with gastrointestinal bleeding compared with normal control.

WBCS had higher value in patients with gastrointestinal bleeding compared with normal, there was statically significant (P.V =  $\leq 0.100$ ).

PT had higher value in patients with gastrointestinal bleeding compared with normal, there was statically significant (P.V =  $\leq 0.000$ ).

There was no significant differences among patients with gastrointestinal bleeding in MCHC.

Patients with gastrointestinal bleeding may be at risk for develop anemia, coagulation disturbance and other medical consequence.

### **4.3 RECOMMENDATIONS:**

- Provide a health center for gastrointestinal bleeding in various states of Sudan
- Follow up by laboratory examination.
- For prolongation of prothrombin time factor assay it necessary to determine which factor deficiency.

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## Appendix (A1)



Hematological analyzer (Sysmex kx21n)

## Appendix (A2)



Coagulometer (Sysmex CA-50)

**Appendix (A3)**  
**Sudan University For Sciences And Technology**  
**Faculty Of Graduate Study**  
**College Of Medical Laboratory Sciences**  
**Department Of Hematology and Immunology**  
**Research Questionnaire**

2/ Name

3/ Age

4/ Gender

Male

Female

5/ Social Status

Single

Married

6/ State

Region

7/ The Patient Complain Of:

i/ Pervious Git Bleeding	Yes ( )	No ( )
ii/ Blood Vomiting	Yes ( )	No ( )
iii/ Melena (Black Stool)	Yes ( )	No ( )
iv/ Bloody Stoll	Yes ( )	No ( )
v/ Bilharziasis	Yes ( )	No ( )

8/ CBC Result:

9/ Coagulation Profile:

i\PT.....