بِسْمِ ٱللَّهِ ٱلرَّحْمَٰنِ ٱلرَّحِيمِ

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Evaluation of PT and APTT among Pregnant Women in Atbara City at 2018.

Assessment (PT), (APTT)among pregnant women form first to third trimester in Atbara locality at 2018.

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الآيـــة

قال تعالى :

(إِن كُلُّ مَن فِي السَّمَاوَاتِ وَالأَرْضِ إِلاَّ آتِي الرَّحْمَنِ عَبْدًا (93) لَقَدْ أَحْصَاهُمْ وَعَدَّهُمْ عَدًّا

(94) وَكُلُّهُمْ آتِيهِ يَوْمَ الْقِيَامَةِ فَرْدًا (95) إِنَّ الَّذِينَ آمَنُوا وَعَمِلُوا الصَّالِحَاتِ سَيَجْعَلُ لَهُمُ

الرَّحْمَنُ وُدًّا (96))

صدق الله العظيم

سورة مريم الآية (93-96)

الإهـــداء

إلي الوالدين اللذان هما مصدر إلهامنا في الحياة

إلي كل الإخوة والأخوات

إلي كل الأساتذة الأجلاء الذين لم يبخلوا

علينا بكل ما آتاهم الله من علم

إلى كل الأصدقاء

شكر وعرفان

الشكر أولا وأخيرا لله سبحانه وتعالى

ونصلي ونسلم على سيدنا محمد صل الله عليه وسلم .

كل الشكر والامتنان إلي من له الفضل الاكبر

في التوجيه والمساعدة في إتمام هذا العمل على أكمل وجه

الأستاذ: محمد هاشم

والشكر ممتد إلي الجامعة وكل الأساتذة.

Abstract

This is cross sectional study aimed to measure APTT and PT in pregnant women with control group the study inclusion Any well healthy pregnant women and exclusion any complicated pregnant women. in Atbara city at the period between March and July 2018. The study included 50 pregnant women and 50 non pregnant as control . 2.5 ml of citrated blood was collected under ideal conditions and analyzed PT and APTT manually. The study proved after analyzing the results statistically by Statistical Package for Social Sciences SPSS that found increased significant in PTT and PT when compare to control. we show the result of APTT was significant increased Found the mean of APTT was 61.18(2.745) sec when compare to control group found mean of control group was 39.82(2.745) sec and the result of PT was slightly increase we found the mean of PT cases was 19.64(0.916) sec when compare to control found the mean of PT 18.90(0.584) sec. This study aim to assessment the coagulation profile among pregnant women form first to third trimester to observed any change in coagulation profile PT , APTT .

ملخص البحث

هذه دراسة تهدف لقياس الثرمبوبلاثتين والبروثرومبين للنساء الحوامل مقارنا بنساء غير حوامل، متضمنة الحوامل الاصحاء مع تجاهل الحوامل الغير أصحاء في مدينه عطبرة في الفترة ما بين مارس إلي يوليو 2018. هذه الدراسة تضمنت 50 امرأة حامل و50 امرأة غير حامل كعينة ضابطة .تم جمع 2.5 مل من الدم في مضاد تجلط سترات الصوديوم الثلاثي وتم فصل العينة تحت ظروف مثالية وتم تحليلها يدوياً. أثبتت الدراسة بعد تحليل النتائج إحصائيا بواسطة برنامج الحزم الإحصائية للعلوم الاجتماعية أنه يوجد زيادة ذو دلالة احصائية في البروثرومبين والثرومبوبلاثتين مقارنة بالعينة الضابطة. حيث وجدنا البروثرومبين (0.916)19.64 ثانية مقارنة مع العينة الضابطة (0.584)18.90 ثانية والثرومبوبلاثتين (2.745)61.18 ثانية مقارنة مع العينة الضابطة (39.82)2.745 ثانية.

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Abbreviation	Term
APTT	Activated Partial Thromboplastin Time
FDPs	Fibrin Degradation Products
HMWK	High Molecular-Weight Kininogen
INR	international normalized ratio
KCCT	kaolin cephalin clotting time
LMP	last menstrual period
PPP	platelet poor plasma
PT	Prothrombin Time
PTTK	partial thromboplastin time with kaolin
SPSS	statistical package for social sciences
TFPI	Tissue Factor Pathway Inhibitor
TT	Thrombin Time
VWF	Von Willebnllld Factor'

Chapter One

Introduction
Justification
Objectives

1.1. INTRODUCTION:

1.1.1. Pregnancy:

Pregnancy ,also known as gestation , is the time during which one or more offspring develops inside a women .A multiple pregnancy involves more than one offspring, Such as with twins. Pregnancy can occur by sexual intercourse or assisted reproductive technology. Child birth typically occurs around 40 weeks from last menstrual period (LMP).Pregnancy is typically divided into three trimesters, The first trimester is from week one through 12 and includes conception. Conception is when the sperm fertilizes the egg. The second trimester is from week 13 until 28weeks .Around the middle of the second trimester movement of the fetus .The third trimester is from 29 week through 40 weeks.[1]Pregnancy is associated with profound anatomical ,physiological, biochemical and endocrine changes that affect multiple organ and system .This changes are essential to help the women adapt to the pregnancy state and to aid fetal growth and survival .the hematological system must adapt in a number of ways such as provision of vitamin and mineral hematopoiesis (iron, vitamin B12,folic acid).which can exacerbate maternal anemia and preparation for bleeding at delivery , which enhanced hemostatic function.[2]

1.1.2. Hemostasis:-

The maintenance of circulatory hemostasis is achieved through the process of balancing bleeding (hemorrhage) and clotting (thrombosis). Hemostasis, the arresting of bleeding, depends on several components. The four major components are the vascular system, platelets (thrombocytes),blood coagulation factors, and fibrinolysis and ultimate tissue repair.[3]

1.1.2.1. The Mechanism of Coagulation:-

Many chemical reactions occur in hemostasis, from the initial stimulus that triggered bleeding to the final formation of stable clot. To understand the process more easily, portions of the normal coagulation sequence are artificially segregated into smaller sections such as the extrinsic and intrinsic pathways.

These pathways are not actual physiological pathways of hemostasis but allow for the grouping of factor defect sand the focusing of laboratory assays. The initiation of the coagulation process may occur via one of two pathways: the extrinsic pathway and the intrinsic pathway. Regardless of the initiating pathway, the two pathways converge into a final common pathway. The outcome of this process is the conversion of circulating insoluble coagulation factors into a gelatinous fibrin clot with entrapped blood cells, a blood clot. As repair of damaged tissue takes place, the clot is lysed and the particulate matter is removed by the mononuclear phagocytic system.[3]

1.1.2.2. Physiological Coagulation (In Vivo):

The original theory of coagulation used a cascade or waterfall theory. This description depicted the generation of thrombin by the soluble coagulation factors and the initiation of coagulation. This theory identified two starting points for the generation of thrombin: the initiation of the Intrinsic pathway with factor XII and surface contact, and the extrinsic pathway with factor VIIa and tissue factor. These two pathways meet at the common pathway, where they both generate factor Xa from X, leading to a common pathway of thrombin from prothrombin and the conversion of fibrinogen to fibrin. This process holds true under laboratory conditions The discovery of a naturally occurring inhibitor of hemostasis, tissue factor pathway inhibitor (TFPI), is able to block the activity of the tissue factor VIIa complex, soon after it becomes active.[4]

1.1.2.3. Laboratory Model of Coagulation:

Laboratory testing looks at the in vitro effect of the coagulation process which is measured by the prothrombin time (PT), activated partial thromboplastin time (aPTT), thrombin time (TT), fibrin degradation products (FDPs), and D-dimer. This section will focus on PT and a PTT. While the coagulation cascade does not reflect what goes on in vivo, it provides a model in which the laboratory relates to for testing. However, the coagulation cascade reflects the mechanisms that the laboratory uses for results. The screening tests provide a tremendous

amount of information to the physician. They can be performed both quickly and accurately.[4]

1.1.2.4. The coagulation cascade

Blood coagulation involves a biological amplification system in which relatively few initiation substances sequentially activate by proteolysis a cascade of circulating precursor proteins (the coagulation factor enzymes) which culminates in the generation of thrombin; this, in turn, converts soluble plasma fibrinogen into fibrin .Fibrin enmeshes the platelet aggregates at the sites of vascular injury and converts the unstable primary platelet plugs to firm, definitive and stable hemostatic plugs .The operation of this enzyme cascade requires local concentration of circulating coagulation factors at the site of injury. Surface-mediated reactions occur on exposed collagen, platelet phospholipid and tissue factor. With the exception of fibrinogen, which is the fibrin clot subunit, the coagulation factors are either enzyme precursors or cofactors. All the enzymes, except factor XIII, are serine proteases (i.e. their ability to hydrolyse peptide bonds depends upon the amino acid serine at their active centre. **(4)**

1.1.2.5.Classification of Coagulation Factors:

Coagulation factors may be categorized into substrates, cofactors, and enzymes. Substrates are the substance upon which enzymes act. Fibrinogen is the main substrate. Cofactors accelerate the activities of the enzymes that are involved in the cascade. Cofactors include tissue factor, factor V, factor VIII, and Fitzgerald factor. All of the enzymes are serine proteases except factor XIII which is a transaminase.[4]

There are three groups in which coagulation factors can be classified:

1. The fibrinogen group consists of factors I, V, VIII, and XIII. They are consumed during coagulation. Factors V and VIII are labile and will increase during pregnancy and inflammation.

2. The prothrombin group: Factors II, VII, IX, and X all are dependent on vitamin K during their synthesis. This group is stable and remains preserved in stored plasma.

3. The *contact* group: Factor XI, factor XII, prekallikrein, and high-molecular-weight kininogen (HMWK) are involved in the intrinsic pathway, moderately stable, and not consumed during coagulation.[2]

- **Factor I, Fibrinogen:**

Substrate for thrombin and precursor of fibrin, it is a large globulin protein. Its function is to be converted into an insoluble protein and then back to soluble components. When exposed to thrombin, two peptides split from the fibrinogen molecule, leaving a fibrin monomer to form a polymerized clot.[4]

- **Factor II, Prothrombin:**

Precursor to thrombin, in the presence of Ca2, it is converted to thrombin (IIa), which in turn stimulates platelet aggregation and activates cofactors protein C and factor XIII. This is a vitamin K–dependent factor.[4]

- **Factor III, Thromboplastin:**

Tissue factor activates factor VII when blood is exposed to tissue fluids.

- **Factor IV, Ionized Calcium:**

This active form of calcium is needed for the activation of thromboplastin and for conversion of prothrombin to thrombin.

- **Factor V, Proaccelerin or Labile Factor:**

This is consumed during clotting and accelerates the transformation of prothrombin to thrombin. A vitamin K dependent factor, 20% of factor V is found on platelets.

- **Factor VI, Nonexistent:**
- **Factor VII, Proconvertin or Stable Factor:**

This is activated by tissue thromboplastin, which in turn activates factor X. It is a vitamin K–dependent factor.

- **Factor VIII, Ant hemophilic:**

This cofactor is used for the cleavage of factor X-Xa by IXa. Factor VIII is described as VIII/vWF: VIII:C active portion, measured by clotting, VIII:Ag is the antigenic portion, (vWF):Ag measures antigen that binds to endothelium for platelet function; it is deficient in hemophilia A.

- **Factor IX, Plasma Thromboplastin Component:**

A component of the thromboplastin generating system, it influences amount as opposed to rate. It is deficient in hemophilia B, also known as Christmas disease. It is sex linked and vitamin K–dependent

- **Factor X, Stuart-Prowers:**

Final common pathway merges to form conversion of prothrombin to thrombin, activity also related to factors VII and IX. It is vitamin K–dependent and can be independently activated by Russell's viper venom.

- **Factor XI, Plasma Thromboplastin Antecedent:**

Essential to intrinsic thromboplastin generating of the cascade, it has increased frequency in the Jewish population. Bleeding tendencies vary, but there is the risk of postoperative hemorrhage.

- **Factor XII, Hageman factor:**

This surface contact factor is activated by collagen. Patients do not bleed but have a tendency to thrombosis.

- **Factor XIII, Fibrin Stabilizing Factor:**

In the presence of calcium, this transaminase stabilizes polymerized fibrin monomers in the initial clot. This is the only factor that is not found in circulating plasma.

- **High-Molecular-Weight Kininogen:**

This surface contact factor is activated by kallikrein.

- **Prekallikrein, Fletcher Factor:**

This is a surface contact activator, in which 75% is bound to HMWK.

1.1.2.6. Coagulation Pathways:-

Initiation of clotting begins with either the extrinsic or the intrinsic pathway. Factor X activation is the point of convergence. Factor X can be activated by either of the two pathways and subsequently catalyzes the conversion of prothrombin to thrombin.[3].

1.1.2.6.1. The Extrinsic Coagulation Pathway:-

The extrinsic pathway is initiated by the entry of tissue thromboplastin into the circulating blood. Tissue thromboplastin is derived from phospholipoproteins and organelle membranes from disrupted tissue cells. The semembrane lipoproteins, termed **tissue factors**, are normally extrinsic to the circulation. Platelet phospholipids are not necessary for activation of the extrinsic pathway because tissue factor supplies its own phospholipids. Factor VII binds to these phospholipids in the tissue cell membranes and is activated to factor VIIa, a potent enzyme capable of activating factor X to Xa in the presence of ionized calcium. The activity of the tissue factor–factor VII complex seems to be largely dependent on the concentration of tissue thromboplastin. The proteolytic cleavage of factor VIIa by factor Xa results in inactivation of factor VIIa. Factor VII participates *only* in the extrinsic pathway. Membranes that enter the circulation also provide a surface for the attachment and activation of factors II and V. The final step is the conversion of fibrinogen to fibrin by thrombin.[3]

1.1.2.6.2. The Intrinsic Coagulation Pathway:-

The intrinsic pathway involves the contact activation factors prekallikre in, HMWK, factor XII, and factor XI. These factors interact on a surface to activate factor IXto IXa. Factor IXa reacts with factor VIII, PF3, and calcium to activate factor X to Xa. In the presence of factor V, factor Xa activates prothrombin (factor II) to thrombin, which in turn converts fibrinogen to fibrin. Strong negatively charged solids that can participate in the activation of factor XII include glass and kaolin in vitro as well as elastin, collagen, platelet surfaces, kallikrein, plasmin, and high–molecular-weight kininogen in vivo. Collagenex

posed by blood vessel injury greatly influences the rate of reaction. Factor XIIa interacts in a feedback loop to convert prekallikrein to additional kallikrein. This reaction is facilitated by the action of HMWK. In the absence of prekallikrein, factor XIIa is generated more slowly. Ionized calcium plays an important role in the activation of certain coagulation factors in the intrinsic pathway. Calcium is not required for the activation of factor XII, prekallikrein, or factor XI but is necessary for the activation of factor IX by factor XIa .[3]

1.2.6.3.Final Common Pathway:-

Once factor X is activated to Xa, the extrinsic and intrinsic pathways enter a common pathway. Factor II, prothrombin, is activated to thrombin (factor IIa), which normally circulates in the blood as an inactive factor. Following the activation of factor Xa, it remains platelet bound and activates factor V. The complex of factors Xa and Va on the platelet surface is formed near platelet-bound factor II molecules. In turn, the platelet-bound Xa/ Va complex cleaves factor II into thrombin, factor IIa. The stage is accelerated by factor V and ionized calcium. [3]

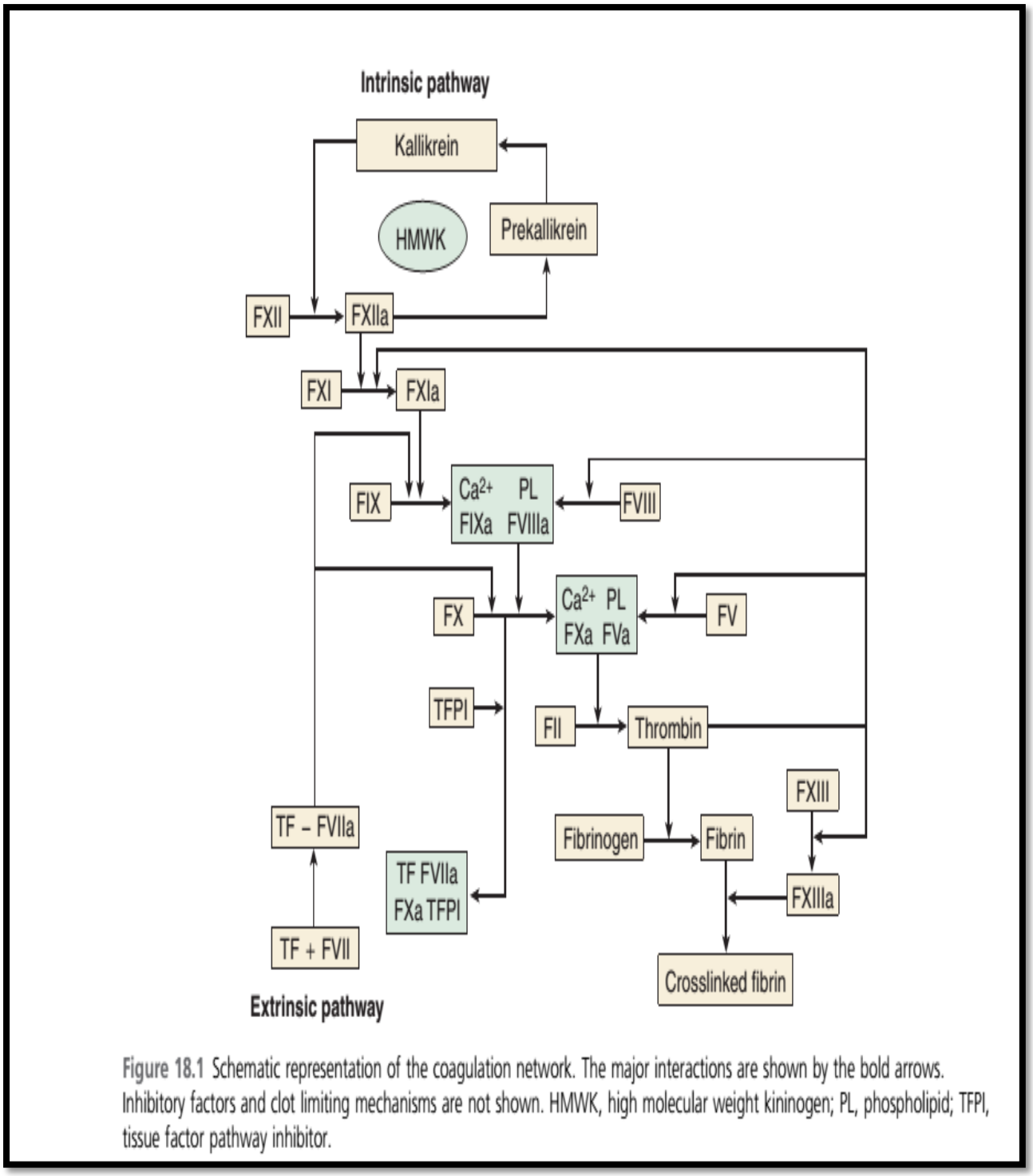


Figure 18.1 Schematic representation of the coagulation network. The major interactions are shown by the bold arrows. Inhibitory factors and clot limiting mechanisms are not shown. HMWK, high molecular weight kininogen; PL, phospholipid; TFPI, tissue factor pathway inhibitor.

Figure (1.1) coagulation cascade .(5)

1.1.3. Coagulation test:-

1.1.3.1. Prothrombin time:

The PT test measure the clotting time of recalcafied plasma in the presence of an optimal concentration of tissue extract (thromboplastin) and indicate the over all of efficiency of the extrinsic clotting system although originally though to measure prothrombin ,the test is now known to depend on reaction with factor v, Vii and x and on the fibrinogen concentration of the plasma.[4]

- Reagent thromboplastin:

Thr0mboplasten were originally tissue extract obtained from different species and different organs contained tissue factor and phospholipid. Because of the potential hazard of viral and other infection from handling human brain it showed no longer be used as a source of thromboplastin .The majority of animal thromboplastin know is used are extract of rabbit brain or long. The introduction of recombinant thromboplastin has resulted in a move away from rabbit brain thromboplastin. There are manufactured using recombinant human tissue factor produce in E. coli and synthetic phospholipids. Which do not contain any other clotting .Normal Range :Defend on thromboplastin used .A mathematical (correction)of the PT ratio for differences in the sensitivity of the thromboplastin reagent .

INR is PT ratio one would have obtained if the reference Thromboplastin had been use .Allows for comparison of result between labs and standardizes reporting of prothrombin time.[4]

1.1.3.2.Activated Partial Thromboplastin Time:-

Specific variations of the APTT test are known as the partial thromboplastin time with kaolin (PTTK) and the kaolin cephalin clotting time (KCCT), reflecting the methods used to perform the test. Reagents PPP From the patient and a control, Kaolin. 5 g/l (laboratory grade) in barbitone buffered saline, pH

7.4.Add a few glass beads to aid suspension. The suspension is stable at room temperature. Other insoluble surface active substances such as silica, celite or ellagic acid can also be used. Phospholipid. Many reagents are available; these contain different phospholipids. When choosing a reagent for the APTT, it is important to establish that the activator phospholipid combination is sensitive to deficiencies of factors VIII, IX and XI at concentrations of`1 0.35–0.4 iu/ml. too insensitive for routine use the system should be responsive to un fractionated heparin over the therapeutic range of approximately 0.3–0.7 iu/ml. In addition, some laboratories will wish the system to be sensitive to the presence of lupus-like anticoagulants.CaCl2. 0.025 mol/l.[4].

1.1.4. Physiological change to coagulation during pregnancy :

Pregnancy is associated with changes in hemostasis , including an increase in the majority of clotting factors, a decrease in the quantity of natural anticoagulants and a reduction in fibrinolytic activity .these changes result in a state of hypercoagulability , are likely due to hormonal changes and increase the risk of thromboembolism . the increase in clotting activity is greatest at the time of delivery with placental expulsion ,releasing thromboplastic substances. These substances stimulate clot formation to stop maternal blood loss.

1.1.5. Justification:-

-To prevent blood lost during pregnancy and after delivery, There was limited or none published data of the PT & APTT, there for this study was conducted to assessment of bleeding profile among pregnant women

1.1.6. Objective:-

1.1.6.1. General Objective:

Evaluation of PT and APTT among pregnant women in Atbara city at 2018.

1.1.6.2. Specific Objectives:

- To estimate of prothrombin time in pregnant women and compare with INR

-To estimate of activated partial thromboplastin time for pregnant women .

-To correlate between prothrombin time and pregnancy stage.

-To correlate between activated partial thromboplastin time and pregnancy stage.

Chapter Two

Materials and Methodology

2. Materials and Methodology

2.1. Study design:.

Cross sectional study with control group.

2.2. Study area :

In Atbara city.

2.3. Study duration:

From Marsh to July at 2018

2.4. Study population:

(100)blood sample p(50) blood samples as Study group of pregnant and (50) as control group.

2.5. Sample size:

(50) citrated venous blood samples was collected from pregnant women.

2.6. Inclusion criteria:

Any well healthy pregnant women are include in the study.

2.7. Exclusion criteria:

Pregnant whom have previous history of thrombosis, bleeding ,maternal sepsis , hypertensive disease ,obstructive labor and pregnancy with abortive outcome.

2.8. Data collection tools:

The primary data will be collect by using questionnaire.

2.9. Sampling technique:

Manual method.

2.10. Blood Sampling:

venous blood was collected using sterile disposable plastic syringe, the vein puncture area is clean with 70% ethanol, and the blood is add to the anticoagulant at ratio of 2.5ml of blood to 0.25 ml of citrate (3.2%) buffer sodium citrate and gently mix The sample is centrifuge at 25000SP for 15min to obtain platelet poor plasma (PPP).

2.11. Principle of APTT:

The APTT was performed by manual testing. it measure over all activity of in intrinsic pathway. Undiluted, platelet poor plasma was incubated at 37 ° c with a particulate factor XII activator (ellagic acid) A reagent containing phospholipids (partial thromboplastin) was added, followed by $CaCl_2$ .the time required for clot formation after the addition of $CaCl_2$. Prekallikrein is required as a cofactor for the auto activation of factor XII by factor XIIa. XI is activated and requires a cofactor of HMWK. XIa activates IX to IXa, which in the presence of VIIIa converts X to Xa. Also present are platelet phospholipids PF3.Calcium is required for the activation of X to proceed rapidly. The reaction then enters the common pathway where both systems involve factors I, II, V, and X. This results in a fibrin monomer polymerizing into a fibrin clot. Factor XIII, or fibrin stabilizing factor, follows activation by thrombin. This will convert initial weak hydrogen bonds, cross-linking fibrin polymers to a more stable covalent bond.

2.12. Reagents and materials:

- Ellagic Acid (Activator), $cacl_2$.
- Cotton, automatic piped, water path, alcohol, stop watch

2.13. Assay procedure:

0.1ml of citrated plasma was added in test tube then 0.1 ml of the thromboplastin also wad added and mixed and incubated in 37 ° c for 5 mints. After that add 0.1 ml of the pre warm $cacl_2$ was added and the stop watch was started. The test was remained in water path with gentle mix for 20sec, the tube was tilted back and forth until clot was formed, and this point was the time for APTT.

2.14. Normal value:

50_57 seconds (depend on APTT reagent).

2.15. Principle of PT:

Tissue thromboplastin in the presence of calcium activates the extrinsic pathway of human blood coagulation mechanism. When reagent is added to normal

anticoagulant plasma ,the clotting mechanism is initiated ,forming a solid gel clot within a specified period of time .the time required for clot formation would be prolonged if there is a deficiency of factor/factor activity in the extrinsic pathway of the coagulation mechanism.

2.16. Reagents and materials:

- Cotton, automatic piped, water path, alcohol, stop watch
- Tissue thromboplastin, calcium

2.17. Assay procedure:

1-Aspirate from the reagent vial enough reagent for immediate testing requirements in a thoroughly clean and dry test tube .

2-bring the reagent to room temperature before prewarming at 37 for testing purpose.

3-recap the reagent vial and replace immediately to 2-8c.

4-toa12x75mm tube add 0,1 ml of (PPP) and place the tube in a water bath for 3to 5 minutes at 37c.

5-to the tube forcibly add ,2ml of reagent and simultaneously start a stop watch .shake the tube gently to mix contents.

6-gently tilt the tube back and forth and stop the stopwatch as soon as the first fibrin strand is visible and the gel/clot formation begins. record the time in seconds.

7-repeat steps 4-6 for a duplicate test on the same samples .

8-find the average of the duplicate test values .this is the prothrombin time (PT).

2.18. Normal value:

15_25 seconds (depend on PT reagent).

2.19. Ethical considerations:

Procedure of blood sampling is explain to the women and their participants. All women and their participants are inform about the research objectives and procedures during the interview period. A verbal consent is obtain from all participants.

2.20. Data analysis:

The data are compare by using statistical analysis perform with statistical package for social sciences (SPSS), to obtain means and standard deviation of activated partial thromboplastin time.

2.21. Data presentation:

By graph(Bie , Bar chart), frequency table and scatter plot.

Chapter Three

Results

3. Result

This is a descriptive cross sectional study that conducted in Atbara during period between March to July 2018. This study measured PT&APTT among pregnant women included 50 pregnant women and 50 none pregnant as a control , 2.5ml of citrated blood was collected under ideal condition then analyzed manually. The study provide after analyzing the results statistically by Statistical Package for Social Sciences SPSS that found increased significant in PTT and PT when compare to control.

Table (3-1) distribution of study group according to age

Age group	Frequency	Percent
< 20 years	7	14%
20 - 29 years	25	50%
30 - 39 years	18	36%
Total	**50**	**100%**

This table mean: The frequency of age of pregnant women in(< 20) was (14)% , frequency in(20-29) was 50% and in (30-39) was 36% .

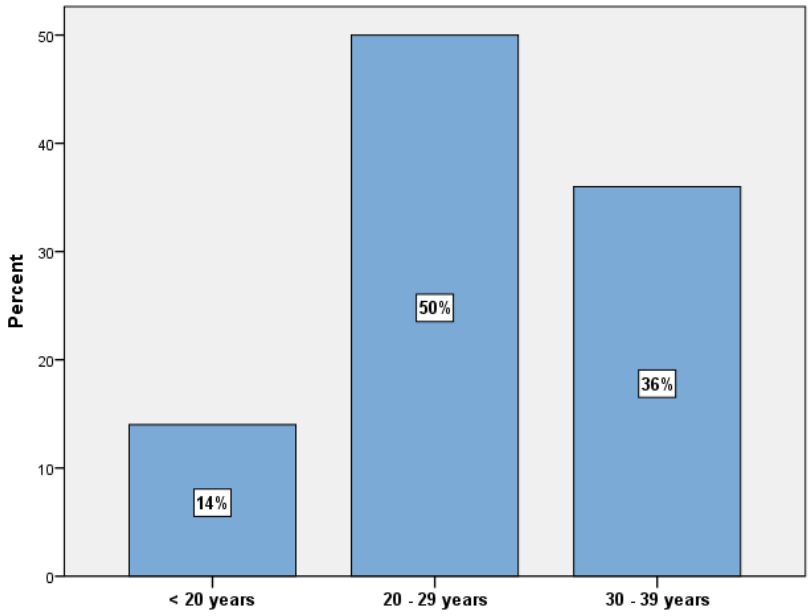


Figure (3-1) distribution of study group according to age

Table (3-2) distribution of study group according to normal or pregnant women

Normal or pregnant	Frequency	Percent
Pregnant women	50	100%
Normal women	0	0%
Total	**50**	**100%**

This table mean: The frequency according to normal women was 0% and according pregnant women was 100% .

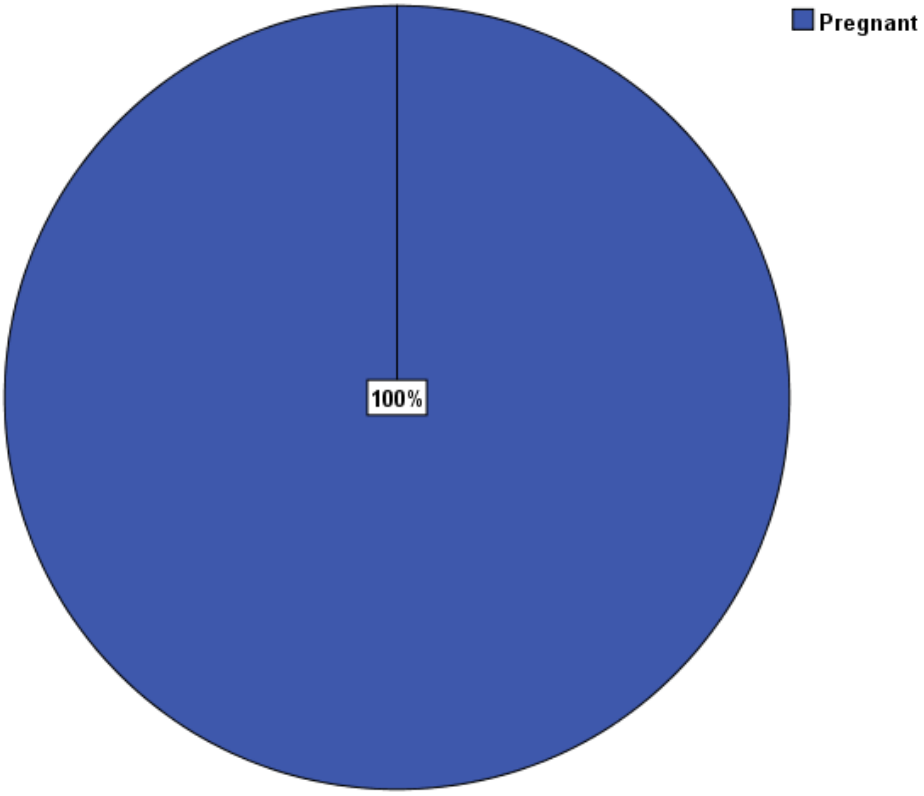


Figure (3-2) distribution of study group according to normal or pregnant women

Table (3-3) distribution of study group according to trimester

Trimester	Frequency	Percent
First	2	4%
Second	21	42%
Third	27	54%
Total	**50**	**100%**

This table mean: The frequency of pregnancy in first trimester was 4% in second trimester was 42% and frequency of pregnancy in third trimester was 54% .

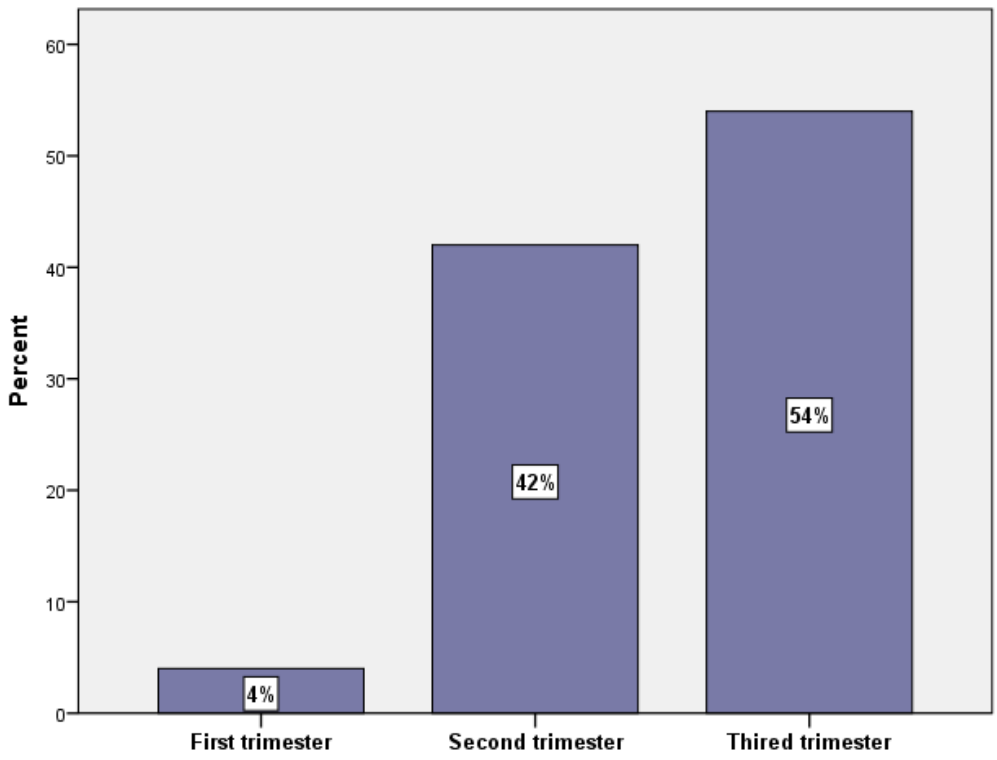


Figure (3-3) distribution of study group according to trimester

Table (3-4) distribution of study group according to number of pregnancy

Number of pregnancy	Frequency	Percent
1 – 2	22	44%
3 – 4	24	28%
5 – 6	9	18%
More than 6	2	4%
No previous pregnancy	3	6%
Total	**50**	**100%**

This table mean: the frequency of number of pregnancy (1-2) was 44% and frequency (3-4) was28% and frequency in time more than 6 was 4% and frequency according to No pregnancy was 6% .

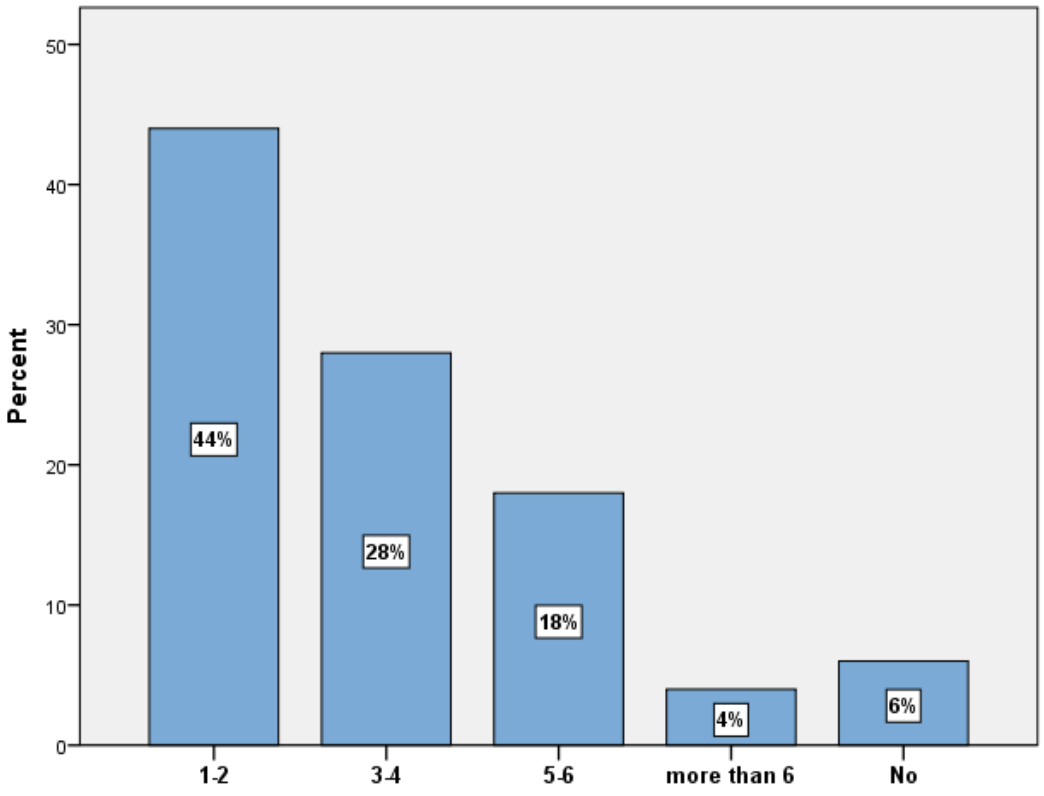


Figure (3-4) distribution of study group according to number of pregnancy

Table (3-5) distribution of study group according to normal or complicated pregnancy

Normal or pregnant	Frequency	Percent
Normal pregnancy	50	100%
Complicated pregnancy	0	0%
Total	**50**	**100%**

This table mean: The frequency according to normal women was 100% and according complicated pregnant women was 0% .

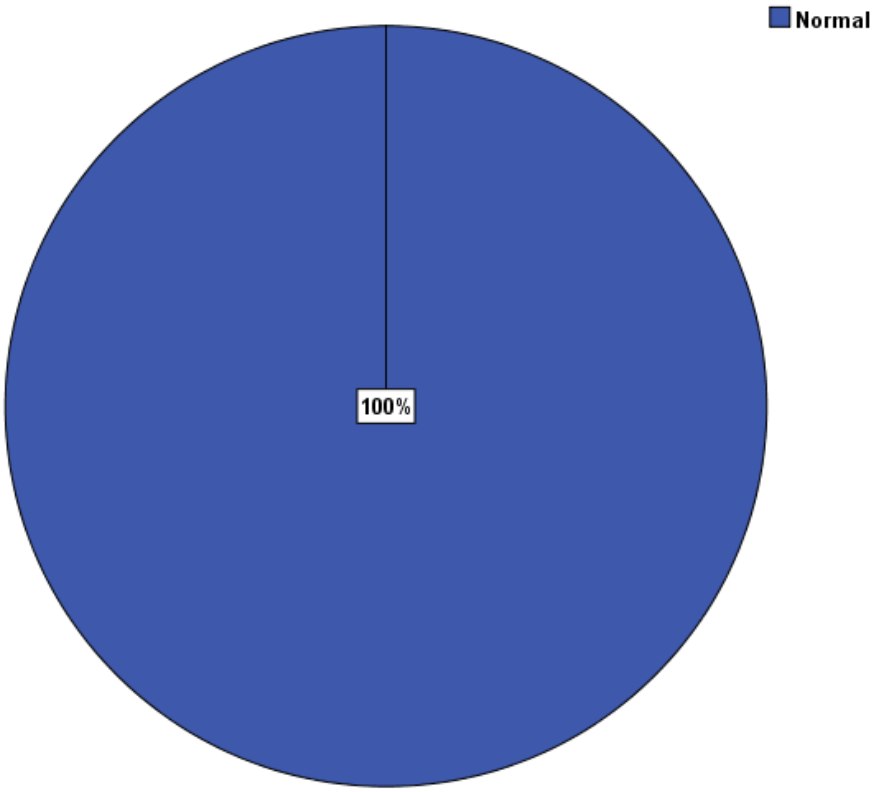


Figure (3-5) distribution of study group according to normal or complicated pregnancy

Table (3-6) distribution of study group according to period between pregnancy

Period between pregnancy	Frequency	Percent
1 year	1	2%
1.5 years	5	10%
2 years	13	26%
3 years	8	16%
More than 3 years	8	16%
No previous pregnancy	15	30%
Total	**50**	**100%**

This table mean: The frequency according to the period between pregnancy (1 year) was 2% , in (1.5 year) was 10% in (2 year) 26% , in (3 year) was16% , in period more than 3 year 16% , according to No pregnancy was 30% .

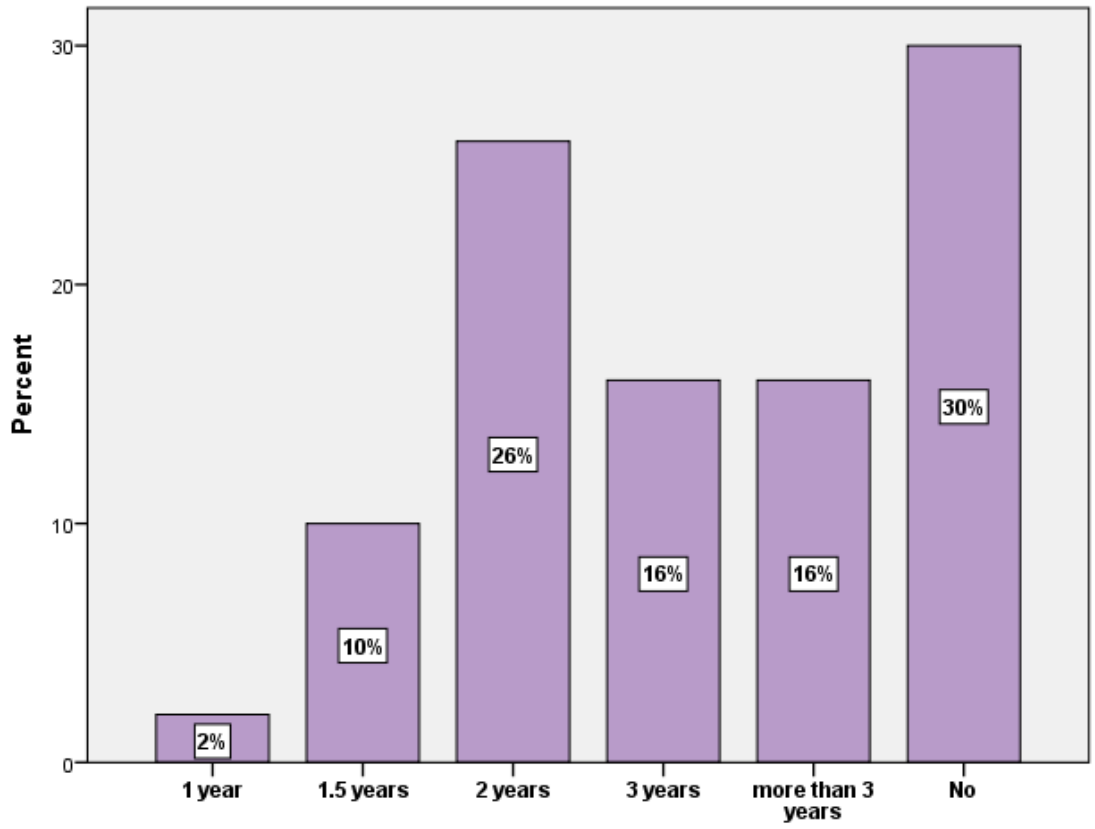


Figure (3-6) distribution of study group according to period between pregnancy

Table (3-7) distribution of study group according to disease

Disease	Frequency	Percent
No disease	50	100%
Other diseases	0	0%
Total	**50**	**100%**

This table mean: frequency to disease was 100% and according to other disease 0% .

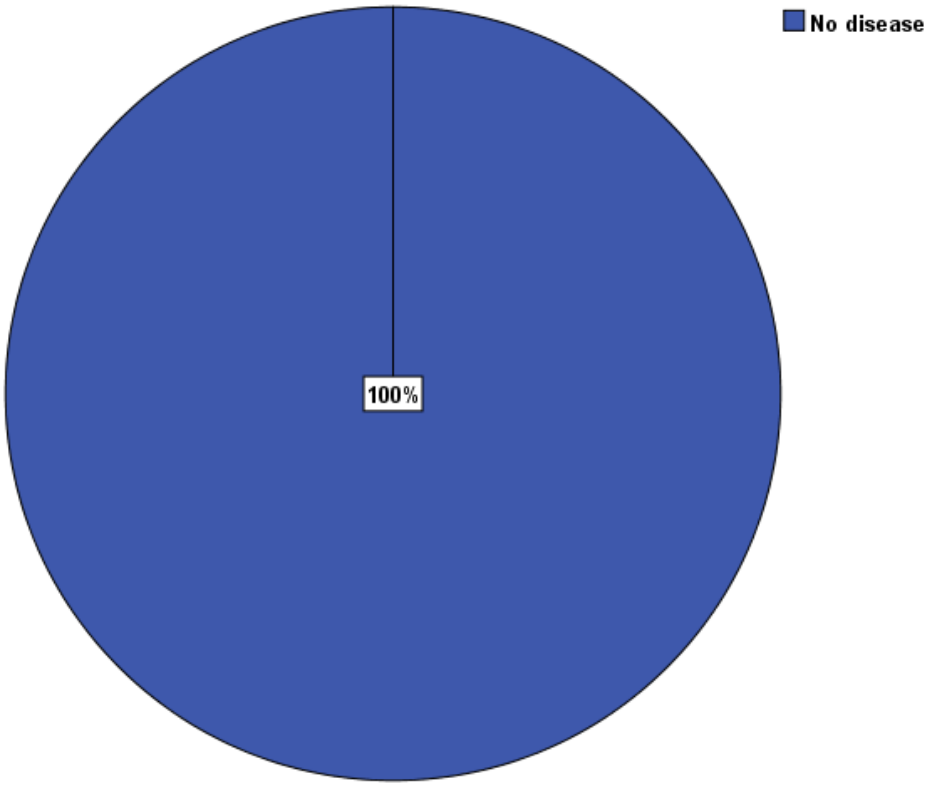


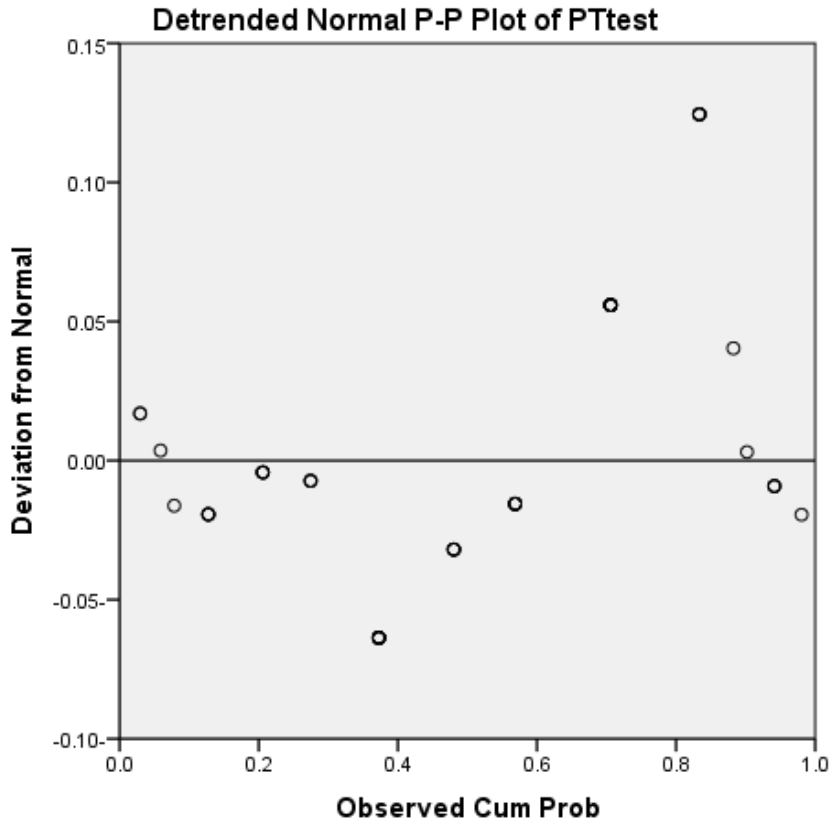
Figure (3-7) distribution of study group according to disease

Table (3-8) Comparison of mean (SD) and mean difference of PT and APTT between case (n= 50) and normal control (n=50)

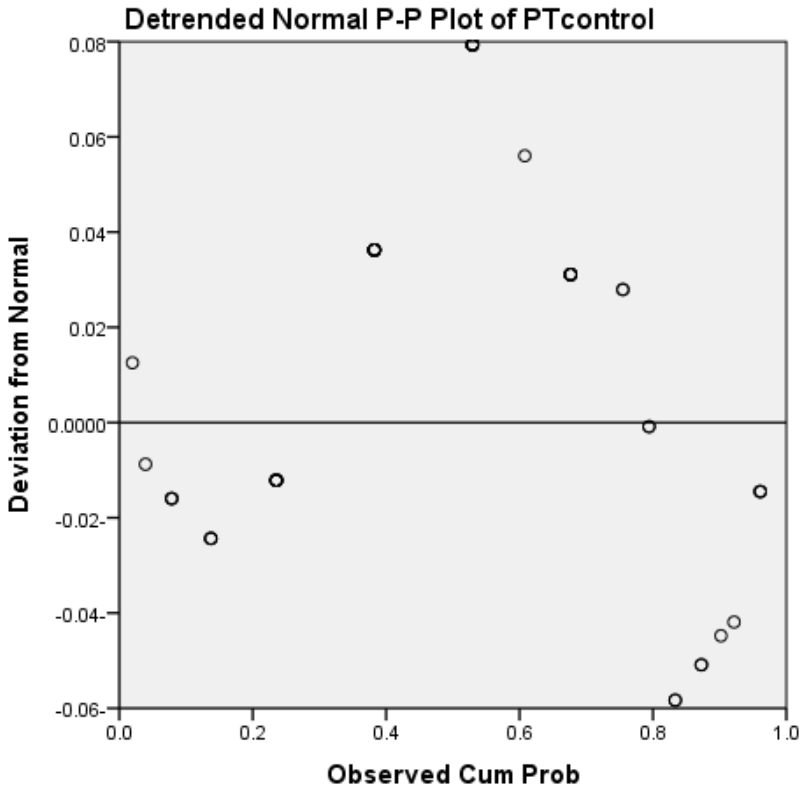
Group	Mean (SD)	MD	*p*-value
PT case	19.64(0.916)	6.474	0.035
PT control	18.90(0.584)	4.127	0.027
APTT case	61.18(2.745)	19.411	0.017
APTT control	39.82)2.745	17.293	0.004

a Independent t. test was applied. *P*-value set as 0.05 is significant

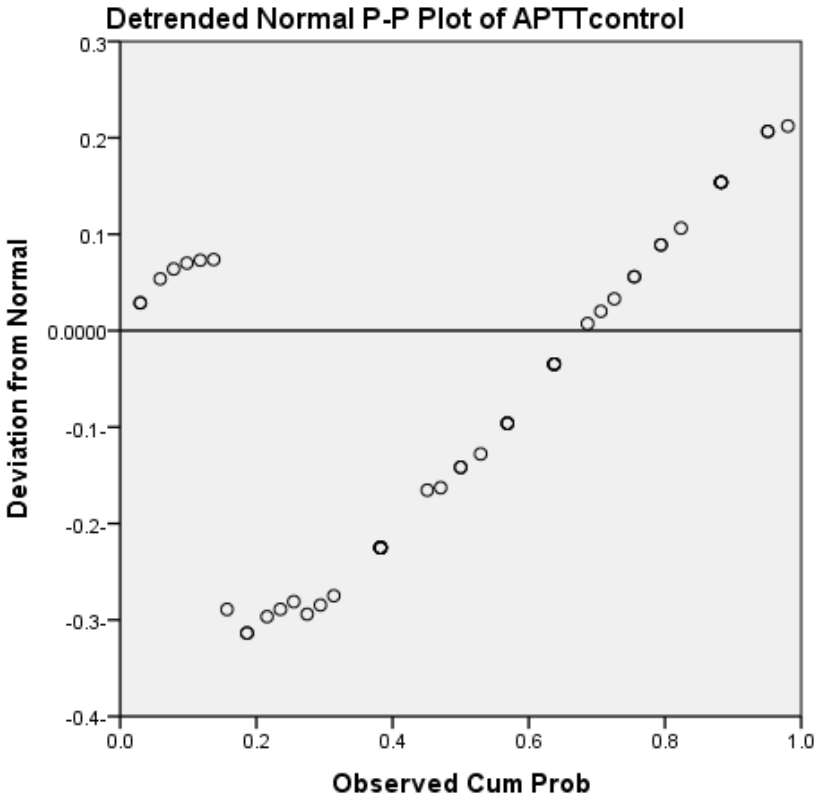
This table mean : there was slightly increase in PT among pregnant women ,and there was significant increase in APTT among pregnant women .



Transforms: natural logarithm



Transforms: natural logarithm



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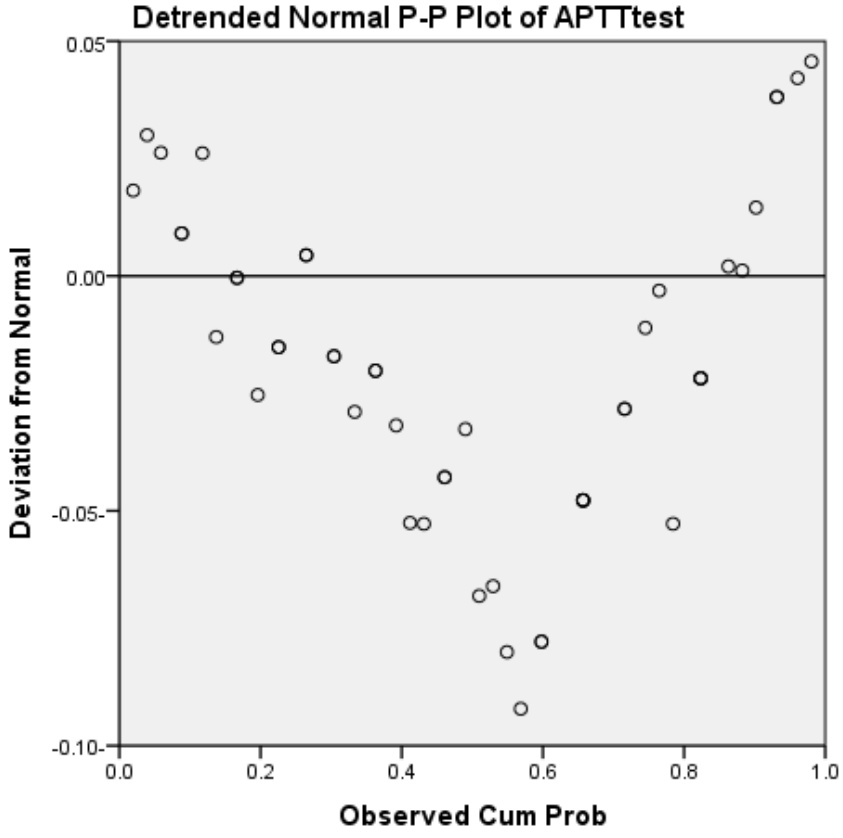

Transforms: natural logarithm

Chapter Four

Discussion

Conclusion

Recommendations

4.1. Discussion

Pregnancy ,also known as gestation , is the time during which one or more offspring develops inside a women .The maintenance of circulatory hemostasis is achieved through the process of balancing bleeding (hemorrhage) and clotting (thrombosis).This study conducted to determine the partial thromboplastin time and prothrombin time on pregnant women.

Found the mean of APTT was(61.18)(2.745), mean of normal control was (39.82)(2.745).the result was significant increased when compare to control and the men of PT was(19.64)(0.916) sec it slightly increase sec.A study done by Mona Aowad in Shandi city in2017 reported the mean of APTT was (40.2750), mean of normal control was (31.8250).which agree with our study[10].

Another study done by Ibeh N, etal Niger J med 2000 reported The means of the APTT were significantly lower in the first ,second and third trimesters with controls (35.59±4.95 second ,32.22±5.79seconds and 29.60±3.66seconds; P=0.01) which dis agreement with our study[6].

A study done by ALkhansa Osman Mohamed ,Khalda Mirghani hamza ,Assad Mohamed ,Ahmed Babiker Department of Hematology and immunohematology Collage of medical laboratory science, Sudan university of Sudan and Technology .Khartoum Sudan.-Department of medical laboratory science ALghad international college for applied medical science .ALmadina ALmunawera. Saudi Arabia received September 30-2015 . Accepted November 3-2015 .reported - Show that the PT and the PTT remain unchanged among pregnant women which dis agreement with our study [9].

Another study done by Hellgren M .semin thromb hemost 2003. Reported Show changes representative of hypercoagulability during pregnancy which agree with our study [7].

Another study done by stCerhecaf, etal.Eurs obstet Gynecol Reprod boil.1997. reported Variation during pregnancy in PT and other parameters which agree with our study [8].

4.2.Conclusion

This study concluded that there was significant increase in APTT among pregnant women and slightly increase in PT among pregnant women.

4.3.Recommendations

1. Another study with large sample size should be done .
2. Further studies were needed to give accurate results. Like factor VIII assay.
3. APTT should be used as clinical routine investigation for treatment and follow up the pregnant women.
4. Pregnant women should make continuous measurement of PT to avoid bleeding disorder and to guarantee safe life for him and baby until delivery.
5. Full care for pregnant women and routine follow up till delivery.

Chapter Five

References

Appendix

5.1 References:-

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2. http://www.reserchgate.net/pubication/283578996.
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6. A study done by Ibeh N, etal Niger J med 2000 reported The means of the APTT were significantly lower in the first ,second and third trimesters with controls (35.59±4.95 second ,32.22±5.79seconds and 29.60±3.66seconds ;P=0.01).
7. study done by Hellgren M .semin thromb hemost 2003. Reported Show changes representative of hypercoagulability during pregnancy.
8. study done by stCerhecaf, etal.Eurs obstet Gynecol Reprod boil.1997. reported Variation during pregnancy in PT and other parameters.
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10. study done by Mona Aowad in Shandi city in2017 reported the mean of APTT was (40.2750), mean of normal control was (31.8250). the result was significant increased when compare to control.

5.2. Appendix

University of Alshakh Abdal Albadri

Faculty of medical laboratory senesce

Questionnaire about the effect of pregnancy ON PT and APTT

Age:……...…… ….years.

Pregnant or non-pregnant:……

Trimester:……..……

Period between pregnancy...…….

number of pregnancy:…… …….

Complicated or normal:……

Disease:…….

Result:-

PT:……..sec.

APTT:…….sec.

INFORMED CONSENT FOR COLLECTION OF BLOOD SAMPLES FOR REASEARCH

This sample is being collected solely for purpose of research .The research pertains reference of change in conglutination profile PT and APTT among pregnant women on Atbara people. The procedure for sample collection involves only the withdrawal of 2-3 ml of blood. It is such a harmless process .The results of the study may not be of immediate benefit to the patient. Complete confidentiality will be maintained in the handling and processing of samples.

The above statement has been read out or explained to me, and having understood the same, I put my signature or thumb impression. I hereby consent to collection of the blood sample of myself.

Phone number:

Address: signature

Date:

استمارة موافقه للأشخاص المشاركين في الدراسة

هذه الدراسة لغرض البحث العلمي فقط وهى بغرض دراسة التغيرات في عوامل التجلط الثرومبين و البروثرومبين بين النساء الحوامل في مدينه عطبرة. وتتطلب اخذ 2-3 مل من الدم ولا يوجد ازى او خطورة تنتج منها عليك و حال موافقتك على المشاركة في هذه الدراسة سيبقى اسمك قيد الكتمان كما لن يكون هنالك أي تعويض مالي .لقد اطلعت على استمارة الموافقة وادركت مضمونها واطلعني الباحث عن فوائد بحثه واهميته العلمية والعملية وبناء عليه فاني حرا مختارا وبمحض إرادتي اوافق على المشاركة في هذه الدراسة كما اوضح بان مشاركتي فيها طوعا منى ، وان باستطاعتي رفض المشاركة كما ان بإمكاني ان لا اجيب على أي سؤال لا ارغب في اجابته ، كما تم إعلامي بان مشاركتي بالبحث لن تحملني أي نفقات او مسائله من شانها الضرر بمهنتي او شخصي . وان المعلومات الناتجة عن مشاركتي سوف تعامل بسريه تامه ولن يطلع عليها أي شخص غير معنى بالبحث وان هذه المعلومات ونتائجها هي لأغراض علميه فقط ولن تكون هنالك اشاره الى شخص او عائلتي في أي منشور عن هذه الدراسة ولأجل هذا فاني اوافق بمشاركتي في هذه الدراسة.

رقم التلفون:

التوقيع: العنوان:

التاريخ :