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Research Article

THROMBOCYTOPENIA ASSOCIATED WITH PREGNANCY IN HAIL REGION 2013

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ABSTRACT

Thrombocytopenia is second to anemia as the most common hematologic abnormality encountered during pregnancy. Pregnancy is associated with numerous metabolic, immunologic, and other homeostatic changes that require careful consideration when attempting to define the cause of thrombocytopenia in a particular individual Thrombocytopenia is classically defined as a platelet count of less than 150,000/μL. Counts from 100,000 to 150,000/μL are considered mildly depressed, 50,000 to 100,000/μL are moderately depressed, and less than 50,000/μL are severely depressed. Pregnancy complicated with thrombocytopenia is a challenge to the clinician. The myriad of disease processes, either pregnancy-induced disorders or preconception medical conditions, can cloud the correct diagnosis. It is important to remember the great majority of patients will have a benign condition, but minorities of patients who have a more serious disease are at risk for serious morbidity and mortality. This descriptive cross-sectional hospital based study that aims to determine the prevalence of thrombocytopenia among pregnant women in Hail, KSA. And also to determine the correlation between thrombocytopenia and Haemoglobin concentration, MPV, Age, Number of Pregnancies and Gestational age. Simple random 100 pregnant in different gestational period participated in the study. In our study 11% show thrombocytopenia, 64% of them in the third trimester, and statistically no significant correlation between low platelets and low haemoglobin, also thrombocytopenia do not correlate to age or number of pregnancies but platelets count proportionate reversely with MPV .

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INTRODUCTION

Thrombocytopenia is second to anemia as the most common hematologic abnormality encountered during pregnancy. The prevalence of a platelet count < 150 x 109/L in the third trimester of pregnancy is 6.6 to 11.6%. A platelet count of < 100 x 109/L, the definition for thrombocytopenia adopted by the International Working Group, is observed in only 1% of pregnant women. There are many potential causes of pregnancy associated thrombocytopenia; some of these are unique to pregnancy, whereas others may also occur in the nonpregnant setting. Pregnancy is associated with numerous metabolic, immunologic, and other homeostatic changes that require careful consideration when attempting to define the cause of thrombocytopenia in a particular individual. Moreover, because therapeutic interventions used to treat thrombocytopenic disorders in pregnant women may have toxicities unique to pregnancy, management approaches must be carefully considered (1). Platelets are one of the cellular components of the blood along with white and red blood cells. Platelets play an important role in clotting and bleeding. Platelets are made in the bone marrow similar to other cells in the blood (1). Platelets are the smallest of the three major types of blood cells. Platelets originate from megakaryocytic which large cells are found in the bone marrow. The fragments of these megakaryocytic are platelets that are released into the blood stream. The circulating platelets make up about two third of the platelets that are released from the bone marrow. The other one third is typically stored (sequestered) in the spleen (1).

Platelet Function

Platelets are not only the smallest blood cell, they are the lightest. Therefore they are pushed out from the center of

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flowing blood to the wall of the blood vessel. There they roll along the surface of the vessel wall, which is lined by cells called endothelium. The endothelium is a very special surface, like Teflon, that prevents anything from sticking to it(1). However when there is an injury or cut, and the endothelial layer is broken, the tough fibers that surround a blood vessel are exposed to the liquid flowing blood. It is the platelets that react first to injury. The tough fibers surrounding the vessel wall, like an envelope, attract platelets like a magnet, stimulate the shape change that, and platelets then clump onto these fibers, providing the initial seal to prevent bleeding, the leak of red blood cells and plasma through the vessel injury.(2)

Disorders of Platelets Number: Too Many Platelets

Rare conditions result in the bone marrow producing too many platelets, sometimes as many as one million or two million per microliter. In some of these patients, there are increased risks for blood clots, but many patients with these disorders have no problems

Disorders of Platelet Number: Too Few Platelets

Disorders with low platelet counts are called thrombocytopenias, a term derived from an old name for platelets, "thrombocytes". This name describes the platelets as the cells ("cytes" is a word for cell) that contribute to thrombosis, or blood clotting. The last part of the word, "-penia", refers to too few cells (5). Thrombocytopenia can be caused by failure of the bone marrow to produce normal numbers of platelets. Bone marrow failure has multiple causes. Thrombocytopenia can also be caused by increased destruction of platelets once they are produced and released into the circulating blood. The many different causes of thrombocytopenia are detailed below. These causes are not mutually exclusive and more than one may be responsible for an abnormal platelet count (.3)

Thrombocytopenia

Thrombocytopenia occurs commonly during pregnancy, and may result from diverse etiologies. Awareness of these many causes facilitates proper diagnosis and management of thrombocytopenia in the pregnant setting.(2) Some causes of thrombocytopenia are unique to pregnancy and may not be familiar to hematologists. In the review, we will discuss the differential diagnosis of thrombocytopenia in pregnancy.(2)

Disorders That Cause Thrombocytopenia in Pregnancy

Gestational Thrombocytopenia

Gestational thrombocytopenia, also known as incidental thrombocytopenia of pregnancy, is the most common cause of thrombocytopenia in pregnant women, accounting for approximately 75% of all cases. Normal pregnancy is associated with a physiologic fall in the platelet count that is characterized by a leftward shift in the platelet count distribution (3). The reason for this decline is unknown, although it has been speculated that these changes may reflect dilution, decreased platelet production, or increased platelet turnover during pregnancy.(4) Regardless, the fall in the platelet count during normal pregnancy results in some pregnant women developing platelet counts that fall into the thrombocytopenic range(.2) Generally, these individuals have mild thrombocytopenia that first becomes apparent in the mid-second to third trimester of pregnancy. Although there is no well-established minimum value for the platelet count in gestational thrombocytopenia, most experts consider this diagnosis to be less likely when the platelet count falls below 70,000/μL. However, reports exist of more severe thrombocytopenia in pregnant women that was not responsive to steroid therapy and resolved postpartum, and thus was consistent with gestational thrombocytopenia.(10) Because there is no diagnostic testing available for gestational thrombocytopenia, this disorder is a diagnosis of exclusion (5). Patients with a history of primary or secondary immune thrombocytopenia(ITP), thrombocytopenia of any etiology preceding pregnancy, or any reason for thrombocytopenia other than uncomplicated pregnancy itself are generally not considered to have gestational thrombocytopenia. However, in many cases, it may not be possible to distinguish gestational thrombocytopenia, particularly a more severe case, from ITP (.3) Gestational thrombocytopenia is not associated with adverse outcomes to either the mother or fetus (6). The incidence of fetal or neonatal thrombocytopenia in the offspring of such patients is no higher than that of nonthrombocytopenic women, and when it occurs often results from coincident neonatal alloimmune thrombocytopenia (7.8). The degree of maternal thrombocytopenia is generally not severe enough to increase the risk of bleeding with delivery, although some cases may compromise the ability to deliver epidural anesthesia. Because such cases may be difficult to distinguish from ITP, a short trial of ITP therapy (eg, corticosteroids or intravenous immunoglobulin [IVIg]) may be useful both diagnostically and therapeutically.(6) In the absence of a platelet increment, platelet transfusion may be used to raise the platelet count to a level deemed safe for epidural catheter placement if desired.(3) Gestational thrombocytopenia is self-limited and resolves within 1 to 2 months after delivery.

Miscellaneous Causes of Thrombocytopenia

In we will briefly discuss several miscellaneous causes of thrombocytopenia in pregnancy. These and other causes that are not discussed. Disseminated intravascular coagulation (DIC) may arise from a number of events in pregnant women. Some causes of fulminant DIC include placental abruption, amniotic fluid embolism, and uterine rupture; in each of these situations, a rapid release of tissue factor-rich material into the maternal circulation leads to profound activation of coagulation, with consumption of coagulation factors and severe hypofibrinogemia. DIC may also be present in association with retained fetal products, but in these cases is often compensated and more gradual in onset, and thrombocytopenia may be the presenting symptom(9)An increased D-dimer level may be useful diagnostically. Acute fatty liver of pregnancy is a rare disorder that usually presents in the third trimester of pregnancy with nausea, vomiting, malaise, right upper quadrant pain, and cholestatic liver dysfunction.(10). Most patients develop DIC due to acquired antithrombin deficiency, with thrombocytopenia and deficiencies of fibrinogen and other clotting factors. Due to the coagulopathy, bleeding is common, despite only mild thrombocytopenia. Some cases of acute fatty liver, as well as HELLP, may be associated with fetal mitochondrial fatty acid oxidation defects, most commonly due to deficiency of long-

chain 3-hydroxyacyl coenzyme A dehydrogenase. Treatment involves supportive care with blood product support for the underlying coagulopathy.(16) Type IIb von Willebrand disease is characterized by a mutant VWF molecule that binds to its primary platelet receptor, glycoprotein Ib, with increased affinity, thereby inducing platelet agglutination, accelerated platelet clearance, and thrombocytopenia. A number of underlying mutations, all in exon (17), have been described during pregnancy, levels of endogenous mutant VWF increase in response to the estrogen-rich hormonal milieu, and thrombocytopenia induced by the mutant VWF may become more apparent. In occasional patients, platelet counts have been reported to fall to as low as 20,000 to 30,000/μL, with improvement after delivery. In addition to primary ITP, ITP secondary to infection (HIV, hepatitis C, cytomegalovirus, H pylori, Epstein-Barr virus, etc), or autoimmune disorders-such as systemic lupus erythematosus or the antiphospholipid syndrome-should also be considered in the differential diagnosis. (13) Antiphospholipid antibodies have been associated not only with a secondary ITP, but also a microangiopathic thrombocytopenia as well.(14)Drug-induced thrombocytopenia may result from a variety of prescription drugs, as well as illicit medications, such as cocaine. Myelopthistic causes of thrombocytopenia, although unlikely in women of child-bearing age, include infiltrative marrow disorders (eg, metastatic neoplasms, fungal, or other types of infections), as well as primary bone marrow syndromes (including myelofibrosis, myelodysplasia, and related disorders). Inherited thrombocytopenia, such as the May-Hegglin anomaly, may first be detected during pregnancy, and may be detected through identification of abnormal platelet morphology on review and assessment of the peripheral blood film of family members.(14). During pregnancy induced hypertension vasodilators like prostaglandins, nitric oxide synthesis is decreased and vasoconstrictors such as angiotensin II, thromboxane A2 and endothelin –1 are increased, There were significant differences between normal pregnancy and severe PIH cases. Thrombocytopenia is an associated phenomenon of PIH. (18)

Treatment of thrombocytopenia

Treatment of thrombocytopenia may vary depending on the cause and the severity. Therapeutic interventions used to treat thrombocytopenic disorders in pregnant women may have toxicities unique to pregnancy, management approaches must be carefully considered.

MATERIALS AND METHODS

Study design

A descriptive cross-sectional hospital based study

Study duration

February - April; 2013

Study population

Pregnant women's in Hail Maternity and children hospital.

Study area

Hail maternity and children hospital.

Inclusion criteria

All pregnant women attended In Hail Maternity and children hospital during the study period was included.

Variables

Platelets count, MPV, Hb, PCV, RBC count,Age,Gestational age and number of pregnancies.

Sample size

A number of 100 pregnant women were included

Sample technique

A total of 100 Pregnant (randomly) in different gestational period, participated in the study, blood samples were taken from each pregnant in the morning (between 8 and 9 a.m.) A proximately 4 ml of blood from each of them were taken in EDTA tube

Steps1

Complete blood count (CBC)

Principle

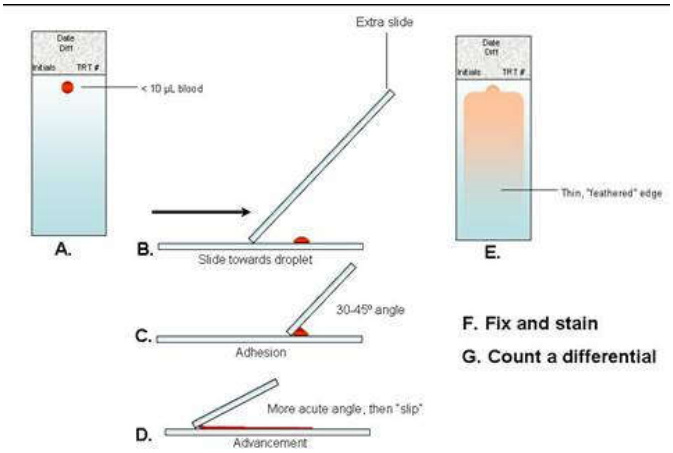
The complete blood count (CBC) is one of the most commonly ordered blood tests. The complete blood count is the calculation of the cellular (formed elements) of blood. These calculations are generally determined by special machines that analyze the different components of blood in less than a minute. A major portion of the complete blood count is the measure of the concentration of white blood cells, red blood cells, and platelets in the blood.

Steps2

Blood film

Procedure

To do a blood smear you need two slides. On one slide the blood sample is placed - this is the "sample" slide. The second slide is used to smear the drop of blood - this the "spreader" slides. Clean the sample slide by wiping it with alcohol. Handle slides by edges only. (Any grease on the slide will cause the dried blood to flake off during staining). Place a very small drop of blood near the end of the sample slide. Place the end of the spreader slide on the sample slide so that the short sided edge of the spreader is just below the drop of blood. The next two steps should be done quickly to avoid smears that are too thick. Holding the spreader at an angle of 45 ^{o} (relative to the sample slide), push the spreader so that the edge just barely touches the drop of blood. By capillary action, a thin line of blood will spread along the edge of the spreader. Quickly drag the spreader along the entire length of the sample slide in one fluid motion. Note that the blood is being dragged by the spreader. If the correct amount of blood was used, and the technique was performed correctly, the smear should end before the end of the sample slide. The smear should also end in a "feathered edge" - a region where the blood cells are well separated. Air dry the sample slide. Fix and stain the slide



Staining smears

1. First prepare the buffer. The stock buffer should be kept in the refrigerator, but if not possible, can be stored at room temperature for several weeks. Make working buffer which can be stored at room temperature for a few days. Buffer should be pH 7.0
2. A high-quality Giemsa should be used. Not all Giemsa stains are equal in quality. We place a layer of stain in the bottom of a glass coplin jar (about 4.5 mL), then add buffer to a level that will just cover the slides (except for frosted ends!) when they are in the jar. A little practice will tell the amount of buffer to add. Place the slides, back-to-back into the slots of the jar, and stain at room temperature for about 10 minutes.
3. Remove slides, rinse by dipping a few times into plain buffer, then stand on end to dry. Some workers prefer to run a thin stream of tap water over the slide to remove all the remaining stain; we have not found this necessary. Be sure to wash out the coplin jars after each use. If not properly washed, stain builds up inside the jar and reduces the quality of staining.
4. There is no need to cover-ship the slides. Immersion oil can be placed directly on the smear for observing under 1000x.

Preparing staining buffer

Stock buffers (two)

The alkaline stock is Sodium phosphate, dibasic anhydrous, N2HPO4, Sigma Chemical S-0879. Mix 9.5 gm with distilled water to make 1000 mL.

The acid stock is Potassium phosphate monobasic anhydrous, KH2PO4, SigmaP5379, mix 9.07 gm with distilled water to make 1000 Ml

Working buffer: Mix 39 mL of acid stock with 61 mL of the alkaline stock, and 900 mL of distilled water. Check pH, and adjust to ph 7 or 7.2 by adding the acid buffer stock to lower pH or alkaline to raise pH. Just a very few mL should be necessary to reach the required pH.

Estimation of Platelets from Blood Film

Examination of a stained blood film provides a rapid estimate of platelet numbers. Normally, there are 8to 20 platelets per 100× (oil) immersion field in a properly prepared smear (where the erythrocytes barely touch or just overlap). At least 10 different fields should be carefully examined for platelet estimation. The average number (e.g., 14) can be multiplied by a factor of 20,000 to arrive at an approximation of the quantitative platelet concentration. If an average number of 14 platelets is multiplied by 20,000, the approximate platelet concentration would be 280,000 or 280 × 10^9/L. Although the estimation of platelets from a blood smear does *not* replace an actual quantitative measurement, it should be done as a cross-check of the quantitative measurement. "pseudothrombocytopenia," a benign condition in which platelets agglutinate or adhere to leukocytes (satellitism) when blood is collected with EDTA as anticoagulant.

Ethical considerations

- Approval from the faculty of Medical laboratory Sciences were obtained. A letter of explanation was sent to medical directorate of Hail Maternity and children hospital about the study objectives and approval was also obtained
- Patients were informed by the objectives of the study and their verbal consent were taken
- Patient confidentiality is guaranteed.

Analysis

Data was entered, checked, and analyzed using SPSS, Excel computer software.

RESULTS AND DISCUSSION

Prevalence of Thrombocytopenia is 11% in all pregnancies Fig (1), MPV frequency distribution fig (2). Frequency and correlation of hemoglobin concentration to platelets count appear in Fig (3). Age and number of pregnancies do not affect thrombocytopenia in pregnancy Fig (4) and Fig (5) respectively. Thrombocytopenia is common in third trimester Fig (6).

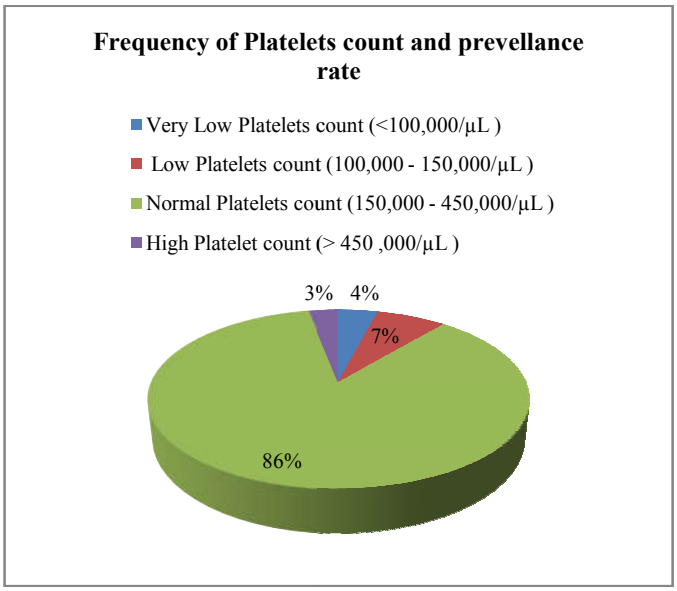


Fig 1

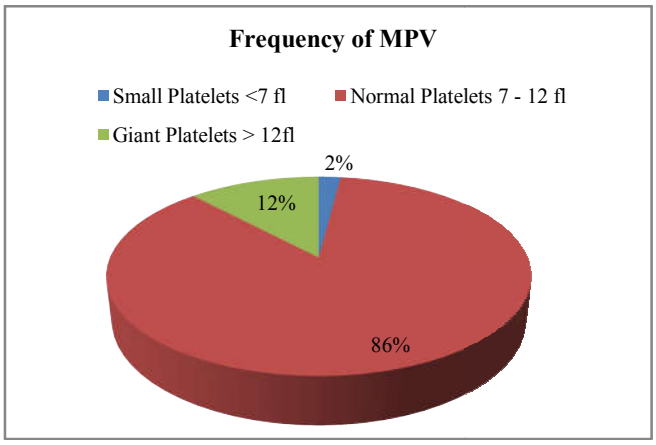


Fig 2

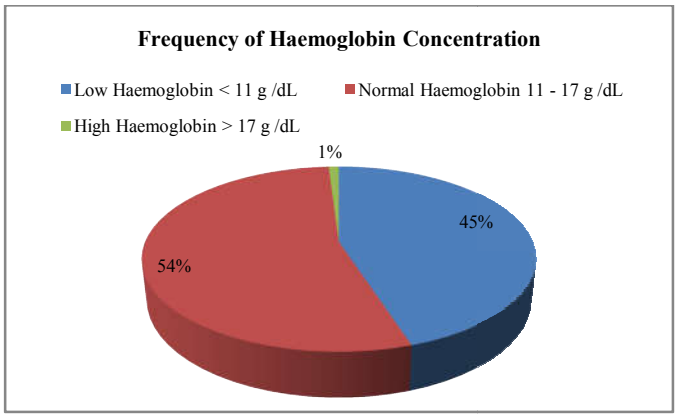


Fig 3

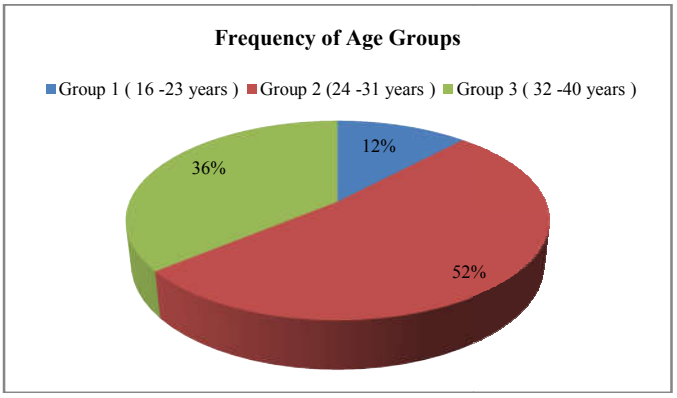


Fig 4

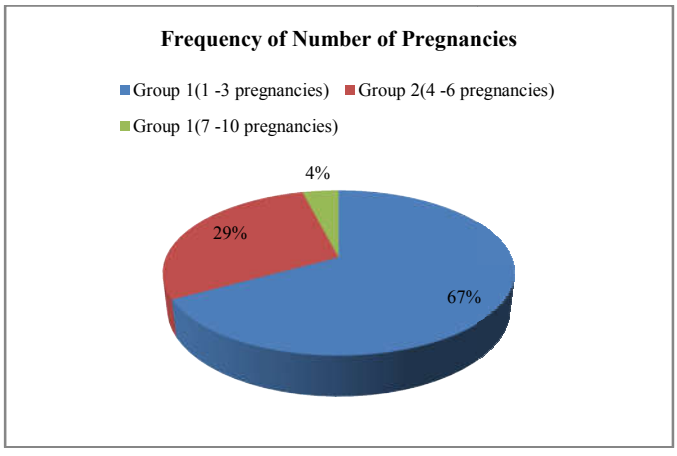


Fig 5

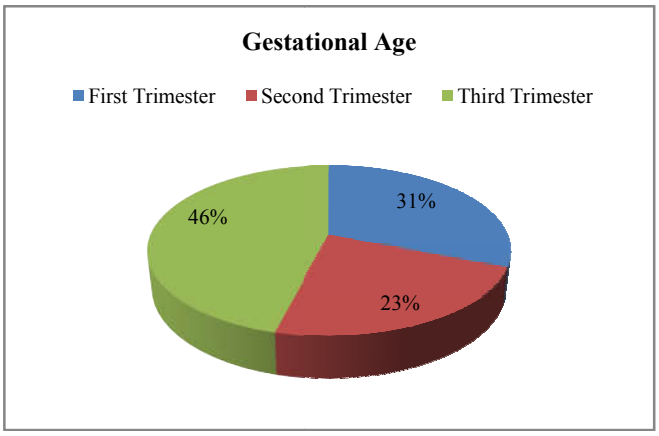


Fig 6

DISCUSSION

Thrombocytopenia is encountered in 6-11% of all pregnancies in previous studies. The modern recognition of the condition predominantly is due to automated CBCs, which routinely include platelet counts.

In our study 11% show thrombocytopenia, 64% of them in the third trimester, and statistically no significant correlation between low platelets and low haemoglobin, also thrombocytopenia do not correlate to age or number of pregnancies but platelets count proportionate reversely with MPV.

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