

Review on Thrombocytopenia (Increasing Platelets Counts)

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Abstract—Thrombocytopenia is the result of falling the number of platelet from 150,000/ microliter. There are three main reasons of thrombocytopenia, a-Decreasing of making platelet b-Increasing of destruction platelet c-Changing of distribution platelet. Pseudo thrombocytopenia must be kept in mind too. Both hereditary and acquired reasons help thrombocytopenia have wide spreaded, but acquired causes are more common with increasing age. Thrombocytopenia separates three stages as numerical. Mild: 100,000 - 150,000/ microliter, Moderate: 50,000 - 100,000/ microliter. Severe: < 50,000/ microliter. However, thrombocytopenia is not usually detected clinically until the platelet count has fallen to levels below 100,000/ microliter. Severe thrombocytopenia, such as intracerebral and intra-abdominal bleeding may be life threatening. So, diagnosing the treatment immediately can save the life. Transfusion of platelet may not need in all thrombocytopenia's. Treatment of the underlying disease may be sufficient. The reason of thrombocytopenia can be temporary but also can be caused severe diseases. Causes of thrombocytopenia change development levels of countries, according to geographical distribution and application centers. In this review we emphasize common etiologies seen in adult patients with thrombocytopenia.

Index Terms—Thrombocytopenia; Etiology; Platelets; Treatment

DEFINATION

Platelets were diagnosed the first time in 1860 by Zimmerman. The role of clotting blood was suggested in 1878 by Zimmerman and Hayran. They are the shape of disc which are 2 - 4 μ m, 0.5 μ m wideness,

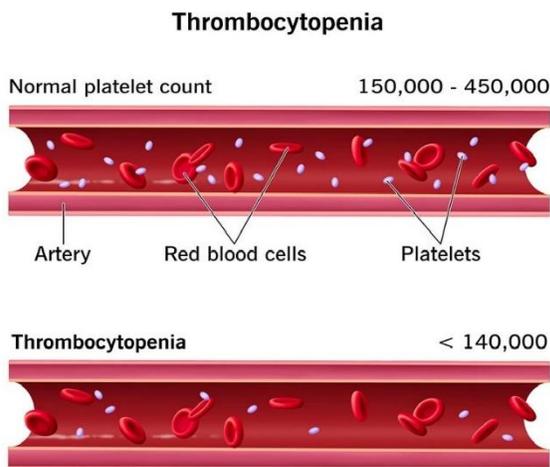
colorless, seedless. The average volume is 6 - 10 fL. There are about 150,000 - 450,000/micro-L platelet in peripheral circulation of blood. The average life of them is 8 - 10 days. Nears 2/3 platelet is in blood and 1/3 is in spleen

[1] Platelets can have a role both in primer and in seconder hemostasis. Platelets are essential for maintaining the integrity of the vascular endothelium and controlling hemorrhage from small-vessel injury through the formation of platelet plugs. More extensive injury and involvement of larger blood vessels requires, in addition to platelets, the participation of the coagulation system to provide a firm, stable, fibrin clot. The average of the platelet counts should be 150,000 - 450,000/microliter. If the numbers go down 150,000/microliter, it causes thrombocytopenia. 2.5 percent of the normal population have got thrombocytopenia. There can be bleeding after mild trauma at patients with thrombocytopenia. The deficiency of platelets or functional out of order causes some symptoms. In general, if the number of platelets is over 100,000/microliter, it can't be expected bleeding even have an operation. It may last longer than usual bleeding between 50,000 - 100,000/microliter in serious injuries. Even there can be bleeding between 20,000 - 50,000/microliter at mild trauma. There can be bleeding itself the lower numbers such as 20,000/microliter. There can be severe risks under 10,000/microliter.

I. INTRODUCTION

Thrombocytopenia is a condition, where the platelet count drops below the normal level. The normal range of platelets, for every microliter of blood, is 150 000 to 450 000. Hence if the number of platelets, per microliter of blood, drops below 150 000, the individual is affected with Thrombocytopenia. When the platelet counts falls below 50 000 or less, there is a risk of mild bleeding. When the platelet counts falls below 20 000, enhanced bleeding occurs since platelets are necessary for blood clotting. Thrombocytopenia can last for either a few days or persist for years. Thrombocytopenia in the brain may be fatal, if there is bleeding. However, the general prognosis for thrombocytopenia is good, where the condition can be treated with medications or with transfusions of blood or platelets. Diagnosis of thrombocytopenia can be made with a simple blood test called the complete blood count

Figure No. 1 Thrombocytopenia



Platelets play an important role in the blood clot. Whenever there is a cut or injury in the body platelets aggregates together and form a clot to stop the bleeding and if there are not enough platelets in the blood, then the body cannot form clots and bleeding will not stop. There are so many reasons why blood does not contain enough platelets. Low platelet counts have symptoms like:

- Bruising: red, purple, or brown (purpura)
- Petechiae: rash with small red or purple dots on skin
- Nosebleed

- Bleeding gums
- Bleeding from wounds for a prolonged period of time or doesn't stop on its own
- Heavy menstrual bleeding
- In more serious cases, internal bleeding also occurs in urine, stool, rectum and sometime bleeding occur in the brain also.

There are so many cases of thrombocytopenia, it is generally occurring in pregnant women, about 7 - 10 % pregnant women are suffering from thrombocytopenia and 1- 4 % thrombocytopenic neonates are born every year. Thrombocytopenia is complicating disease and its treatment also depend upon the cause and severity of the disease, mild thrombocytopenia dose not required any treatment, moderate and severe thrombocytopenia where a serious bleeding occur either on the surface of the skin or inside the body which required a treatment like:

- Blood or platelet transfusions
- Changing medications that are causing low platelet count
- Steroids: used to suppress the immune system
- Immune globulin
- Corticosteroids: block platelet antibodies
- Immune-suppressant drugs
- Splenectomy, or the surgical removal of the spleen

Since there have been a remarkable increase in the number of cases with thrombocytopenia, this article will provide an insight view about the treatment and causes options available for thrombocytopenia. Platelets, characterized by their lack of a nucleus and small size, exhibit a multitude of functions while having a limited lifespan. The process of producing platelets, known as thrombopoiesis, predominantly occurs in the bone marrow. Hematopoietic stem cells are differentiated into polyploid megakaryocytes, which are big cells with a diameter of 50–100 μm . Proplatelets, which are lengthy, branching cytoplasmic protrusions, are released by these megakaryocytes. A complicated mechanism is involved in the change from megakaryocytes to platelets. Despite extensive research on the basic mechanics of platelet synthesis, ongoing studies aim to identify the precise molecular regulators and cellular processes involved in platelet generation and

release. In the human circulatory system, platelets persist for approximately 7–10 days, and subsequently, platelet clearance predominantly occurs within the spleen and liver. The liver produces thrombopoietin, which regulates the production and maturation of megakaryocytes in the bone marrow by stimulating the megakaryocytes' thrombopoietin receptor. Proplatelet production is sparked by this activation, especially when the bloodstream's platelet count is low. The daily production and elimination of 1011 platelets by the human body maintains a normal steady-state platelet count. The generation of platelets needs to be tightly controlled to prevent spontaneous bleeding, artery blockage, and organ damage. Numerous complex systems regulate the elimination and formation of platelets in both normal and pathological circumstances. Several approaches are used to mediate platelet clearance. One such approach works by using the aging (senescence) signals brought on by glycan degradation and apoptotic events. Additionally, immunological responses, more notably antibody reactions, can destroy platelets. Platelet clearance is mediated by the presence of glycans on the surface of platelets. In recent studies, the absence of sialic acid on the surface of platelets appears to contribute significantly to the recognition and elimination of senescent platelets from circulation. The hepatic Ashwell-Morrell receptor (AMR) aids in the clearance of sialic acid that is lost from platelets during circulation. The transmembrane heterooligomeric glycoprotein complex, also known as the AMR, is in charge of identifying and seizing platelets for expulsion. Moreover, antibodies can induce platelet desialylation, which leads to the convergence of signals for platelet removal, combining immune-mediated platelet elimination. Furthermore, in both clinical and experimental viral infections, activated platelets and heightened platelet-leukocyte aggregates are detected. The dynamics and consequences of platelet–leukocyte interactions hinge on the specific leukocyte involved, as well as the nature of the pathogen and the prevailing pathological conditions. Moreover, platelets are capable of interacting with bacterial LPS and subsequently forming platelet leukocyte aggregates (PLAs). Increased PLAs are pivotal in the development of thrombotic conditions. Nonetheless, platelet-monocyte aggregates (PMAs) have been identified as a more responsive indicator of platelet activation

compared to platelet-neutrophil aggregates (PNAs) and the expression of platelet surface P-selectin (CD62P). The functional role of E-cadherin expression in platelets is probably associated with its capacity to facilitate interactions between platelets and extracellular factors. These factors, such as fibrinogen, play a role in influencing the formation and stabilization of platelet clots. Moreover, while circulating in a quiescent state, platelets express various receptors, including glycoprotein (GP) Ib-IX-V, GPVI, and $\alpha 2\beta 1$. These receptors have the capability to bind to collagen and von Willebrand factor (VWF) present on damaged endothelial surfaces, facilitating platelet adhesion and activation at injury sites. The formation of a stable clot is contingent upon thrombin generation and platelet-secreted factors like adenosine diphosphate (ADP) and thromboxane A2 (TxA2). These factors play crucial roles in promoting platelet spreading, aggregation, plug formation, and clot retraction. Despite this knowledge, numerous questions remain regarding the precise mechanisms that govern platelet numbers.

II. PLATELETS

Platelets play a crucial role in the regulation of bleeding (haemostasis) when a blood vessel wall sustains an injury, leading to the disruption of the endothelial cell layer and exposing the underlying extracellular matrix. In their resting state, circulating platelets exhibit a discoid shape and remain uninvolved in any interactions with the intact vessel wall. Furthermore, they serve vital functions beyond thrombosis and wound healing, as they actively participate in inflammation, immunity, and cancer biology. While platelets are mainly understood to be important participants in thrombosis, growing evidence suggests that they also play a substantial role in immunological responses and inflammatory processes in both healthy and pathological states. The importance of platelets' immunological and inflammatory capabilities, which go beyond their conventional involvement in clot formation, is now well understood. Risk of bleeding is increased after platelet numbers drop below 50, or mostly below 20. It is remarkable to normal platelet numbers are much high, indicating that much more platelets circulate than is needed for hemostasis. Moreover, the interaction between platelets and endothelial cells has

been the subject of substantial investigation due to the serious health effects associated with atherosclerosis. In a state of homeostasis, the endothelium plays an active role in preventing coagulation. This is particularly noteworthy, as microvascular thrombosis is a clinical characteristic observed in numerous viral infections. Endothelial cells synthesize and present heparan sulfate proteoglycans in the glycocalyx. These proteoglycans have the capability to bind to anti-thrombin III, facilitating the inhibition of thrombin molecules produced during the coagulation cascade. Furthermore, endothelial cells play a crucial role in preserving blood fluidity by deactivating thrombin, which, in turn, prevents platelet activation. Additionally, these cells generate and store von WVF in Weibel–Palade bodies. VWF, a sizable multimeric glycoprotein, is instrumental in various coagulation processes, including enhancing the interaction between platelets and collagen within the basement membrane.

Thrombocytopenia is a disorder associated with increased destruction, consumption, or decreased production of platelets. Platelets are also called thrombocyte. They are non-nucleated cytoplasmic that play a vital role in haemostasis and also involved in inflammation and immunity. Normal counts of platelets are 150,000 – 450,000 cells/ml in which 70 % circulates in the blood and remaining 30 % arrest by the spleen. The life cycle of platelets is about 7-10 days and then degraded in the liver or spleen. The laboratory test used to assess platelet function are bleeding time (*in-vivo* test) and platelet function tests. A normal bleeding time indicates sufficient platelet numbers or function and normal microvasculature.

First Max Schultze (German anatomist) published the accurate and convincing description of platelets in his newly founded journal *Archive für microscopes Anatomies* in 1865. He described “spherules” much smaller than red blood cells that occasionally clump and may participate in the collections of fibrous material. In 1882, Giulio Bizz zero (Italian pathologist) showed platelet being the first component of blood, which adhere to damaged blood vessel walls *in-vivo*, and that *in-vitro*.

Platelet’s structure and function

Platelets are tiny pieces of red blood cells about 20% of the diameter of red blood cells. Platelets originate from the cytoplasm of bone marrow, the same as red blood cell and white blood cells are produced. Platelets are produced from a very large bone marrow cells called megakaryocytes, about 1000 to 3000 platelets are produced by the megakaryocytes, which are released into the blood stream. It contains translational machinery and megakaryocyte derived messenger RNA (mRNA) which is required for the synthesis of protein but it does not contain genomic DNA. Platelets are in discoid shape, an average size of 2 to 4 µm in diameter and they are present in the blood about 200,000/µL in humans. Internal structure of Platelet can be divided into four zones: Peripheral zone, Sol-gel zone, Organelle zone and Membrane zone, which is important to stop bleeding. Peripheral zone contains glycoproteins, which is required for adhesion, activation, and aggregation. Sol-gel zone contain microtubules and microfilaments which maintain the platelet discoid shape. Organelle zone contains alpha granules which contain clotting mediators, required for creating a firm plug to seal blood vessel breaks. The Membrane zone also contains the enzymatic systems for prostaglandin synthesis (thromboxane A2 synthesis). Whenever there is an injury in the blood vessel platelets change shape and become round and extend long filaments which make contact with the broken blood vessel wall and then form a plug to seal the broken blood vessel.

Thrombocytopenia signs and symptoms may include:

- Easy or excessive bruising (purpura)
- Superficial bleeding into the skin that appears as a rash of pinpoint-sized reddish-purple spots (petechiae), usually on the lower legs
- Prolonged bleeding from cuts
- Bleeding from your gums or nose
- Heavy menstrual periods
- Blood in urine or stool
- Prolonged bleeding from cuts
- Spontaneous or excessive bleeding
- Blood in urine or stools
- Fatigue
- Enlarged spleen

III. CAUSES

Thrombocytopenia means have fewer than 150,000 platelets per microliter of circulating blood. Because each platelet lives only about 10 days, body normally renews the platelet supply continually by producing new platelets in bone marrow. Thrombocytopenia rarely is inherited; or it can be caused by a number of medications or conditions. Whatever the cause, circulating platelets are reduced by one or more of the following processes: trapping of platelets in the spleen, decreased platelet production or increased destruction of platelets.

Trapped platelets

The spleen is a small organ about the size of the fist situated just below the rib cage on the left side of the abdomen. Normally, spleen works to fight infection and filter unwanted material from the blood. An enlarged spleen which can be caused by a number of disorders can harbour too many platelets, which decreases the number of platelets in circulation

Decreased production of platelets

Platelets are produced in the bone marrow. Factors that can decrease platelet production include:

- Leukaemia and other cancers
- Some types of anaemia
- Viral infections, such as hepatitis C or HIV
- Chemotherapy drugs and radiation therapy
- Heavy alcohol consumption

Increased breakdown of platelets

Some conditions can cause the body to use up or destroy platelets faster than they're produced, leading to a shortage of platelets in the bloodstream. Examples of such conditions include:

- Pregnancy. Thrombocytopenia caused by pregnancy is usually mild and improves soon after childbirth.
- Immune thrombocytopenia. Autoimmune diseases, such as lupus and rheumatoid arthritis, cause this type. The body's immune system mistakenly attacks and destroys platelets. If the exact cause of this condition isn't known, it's called idiopathic thrombocytopenic purpura. This type more often affects children.

- Bacteria in the blood. Severe bacterial infections involving the blood (bacteremia) can destroy platelets.
- Thrombotic thrombocytopenic purpura. This is a rare condition that occurs when small blood clots suddenly form throughout the body, using up large numbers of platelets.
- Hemolytic uremic syndrome. This rare disorder causes a sharp drop in platelets, destruction of red blood cells and impairs kidney function.
- Medications. Certain medications can reduce the number of platelets in the blood. Sometimes a drug confuses the immune system and causes it to destroy platelets. Examples include heparin, quinine, sulfa-containing antibiotics and anticonvulsants.

IV. PATHOPHYSIOLOGY OF THROMBOCYTOPENIA

Thrombocytopenia, characterized by a reduced platelet count, involves complex mechanisms that disrupt the balance between platelet production, distribution, and destruction. The pathophysiology encompasses several key processes:

1. Decreased Platelet Production

The bone marrow's ability to produce platelets can be compromised due to:

- Bone Marrow Failure: Conditions like aplastic anemia and paroxysmal nocturnal haemoglobinuria (PNH) lead to insufficient platelet production.
- Bone Marrow Suppression: Exposure to certain drugs (e.g., valproic acid, daptomycin, chemotherapy agents), irradiation, chronic alcohol abuse, inherited thrombocytopenia, and viral infections can suppress bone marrow activity.

2. Increased Platelet Destruction

- Immune Thrombocytopenia (ITP): An autoimmune disorder where antibodies target platelet membrane antigens, leading to their destruction primarily in the spleen.
- Disseminated Intravascular Coagulation (DIC): A condition causing widespread clotting that

consumes platelets, leading to thrombocytopenia.

- Thrombotic Thrombocytopenic Purpura (TTP): A disorder characterized by small blood clots forming throughout the body, consuming large numbers of platelets.
- Hemolytic Uremic Syndrome (HUS): Often triggered by infections, leading to platelet destruction.

3. Platelet Sequestration

An enlarged spleen (splenomegaly) can sequester excessive platelets, reducing their number in circulation.

4. Dilutional Thrombocytopenia

Massive transfusions or fluid resuscitation can dilute circulating platelets, leading to a relative decrease in platelet concentration.

V. CLASSIFICATION OF THROMBOCYTOPENIA

Thrombocytopenia, defined as a platelet count below 150,000 per microliter of blood, can be classified based on its underlying mechanisms:

1. Decreased Platelet Production:

- Bone Marrow Disorders: Conditions such as aplastic anemia, myelodysplastic syndromes, and leukemia can impair platelet production.
- Nutritional Deficiencies: Deficiencies in vitamin B12 or folic acid may lead to reduced platelet synthesis.
- Infections: Certain viral infections can suppress bone marrow function, leading to decreased platelet production.
- Medications and Toxins: Exposure to chemotherapy agents, radiation, or alcohol can negatively affect platelet formation.

2. Increased Platelet Destruction or Consumption:

- Immune-Mediated Destruction: Autoimmune conditions like immune thrombocytopenic purpura (ITP) result in the body's immune system attacking its own platelets.
- Non-Immune Consumption: Conditions such as disseminated intravascular coagulation (DIC)

and thrombotic thrombocytopenic purpura (TTP) involve increased platelet consumption.

- Drug-Induced Thrombocytopenia: Certain medications can trigger platelet destruction through immune mechanisms.

3. Increased Platelet Sequestration:

- Splenomegaly: An enlarged spleen can sequester more platelets than normal, reducing their number in circulation. Conditions like liver disease leading to portal hypertension can cause splenomegaly.

4. Dilutional Thrombocytopenia:

- Massive Transfusions: Large-volume blood transfusions or fluid resuscitation can dilute circulating platelets, leading to a relative decrease in platelet concentration.

VI. ETIOLOGIS OF THROMBOCYTOPENIA

Common Etiologies

Artificial thrombocytopenia

Sometimes the platelet counts can be less even though it may not be thrombocytopenia. The persons who have got thrombocytopenia but also they don't have petechia and ecchymosis must suspect about artificial thrombocytopenia. This situation occurs due to giant thrombocyte and thrombocyte satellitism, the most important common is pseudothrombocytopenia. Formation of thrombocyte in pseudothrombocytopenia occurs anticoagulin which depends on platelet agglutinant. The most farming occurs in EDTA when it is used. It occurs in anticoagulant when they come together such as citrate, acid citrate, dextrose, oxalate and heparin. In most of the patients forming activity of platelets increases under 37 oC, 22 oC or 4 oC is maximum level. Forming the platelets occurs in several minutes and it increases 60 – 90 minutes. They are called platelet cold agglutinate. Platelets in vitro condition which blood with anticoagulant on the epitope (usually GPIIb/IIIa complexity knowing antibody can form IgG). EDTA separates calcium from GPIIb/IIIa complexity and it causes new epitopes on GPIIb. Antibodies sometimes both platelet GPIIb/IIIa with epitope and also entering the reaction with neutrophil and leukocyte FcγIII receptor and it helps the platelets to stick to the round of leucocytes as a rosette. It is called platelet satellitism. The giant

platelets are counted in blood measuring machine as it supposed lymphocyte; in fact they cause the low platelet counts [1].

Immune thrombocytopenia

Immune thrombocytopenia is an autoimmune illness which is developed against platelets auto antibodies and owing to destruction of premature platelets. The number of platelet in ITP is under the level 150,000/microL. The lifetime of platelet cut down. There are antiplatelet antibodies in plasma. Although most of ITP are autoimmune especially viral infection starts this. Antibody of antiplatelet IgG, stick to the membran of palelelets are kept through the macrophage Fc receptors in spleen, causes levaning from circulation. So, platelet counts in circulation decreases [5]. In scintigraphy works the lifetime of platelet decreases from 5 - 7 days to 1 - 4 hours [9].

Although ITP can be seen in every age especially, it can be more seen at children and young. The rate in women is one and half times more often than men. The acute ITP which is seen in childhood shows acute bleeding. Chronic ITP which is shown in teenagers starts insidiously. Bleeding is spontaneously, it increases after trauma and bleeding which are different parts of body can be seen at same time. The skin bleeding is like purpura and ecchymosis. Epistaxis, gum bleeding, vaginal and gastrointestinal bleeding can be seen. Hematuria, cerebral and retinal bleeding, menorrhagia can be seen in women. For the diagnosis of ITP medical history and with the completing physical examining in laboratory researching the reasons which are producing seconder thrombocytopenia should be eliminated [10, 11]. The most important laboratory finding is thrombocytopenia and platelet anisocytosis. There have been platelets which are big and small in the shape of atipic in widespreading of blood. If there is loosing blood at the beginning even normochromic normocytes finds anemia, after the long bleeding the deficiency of iron can be seen. The number of leukocyte is usually normal. If there is loosing blood, it can be seen leucocytosis. The Associate of American Hematology, do not suggest researching of bone marrow especially under the age of 60 and the one who responses well treatment [5]. With these International Consensus suggest myelodysplasia and over the age of

40. Bone marrow is normal. Megakaryocytes may be increased. Immature, megakaryocyte which is big and has got one nucleus can be seen [12]. Risk of bleeding is related platelet number's of chronic ITP which is seen in adults. The platelet counts are over 30,000/microL it is followed without treatment. But if a great operation will be treatment necessary.

Although the number of platelet is under 30,000/microL or over 30,000/microL, and also if there is mucous bleeding, it should be treated. In ITP of mature first step is drug corticosteroid and

1 - 2 mg/kg/day should be given. The patients who responses treatment well the number of platelet raises in first week and later it raises between two and four weeks. If there is bleeding which is threat the life, they should have done parenteral corticosteroid or intravenous immunoglobulin [5, 10]. The 20-30% percent of patients can not give response to the corticosteroid. Although they have taken corticosteroid for six

months and can not give response, the severe thrombocytopenia (lower than 10,000/microL) continue or the patients who take treatment for three mounts and then platelet counts lower than 30,000/microL are suitable to take out spleen. After increasing the number of platelets and in order to keep the platelet at this level and the patients who need 15 mg and over it prednisolone everyday, in spite of patients who take low dose steroid, have got side effects of it and condition contraindicate of steroid are needed to splenectomy for

the second step therapy. They can have complete remission 60-80% after splenectomy. The patient whose platelet counts increases after operation 24 - 48 hours is responded well to splenectomy. If there is not any response to splenectomy, you should find accessory of it. Patients who have not response to splenectomy, thrombopoetin agonist and rituximab are used in third step therapy. The other treatments are azathioprine

(3 - 4 mg/kg/day), cyclophosphamide (2 mg/kg/day) 6-mercaptopurine (100 mg/m²/day), vinkristin sulphate are used 1 - 2 mg three-four weeks [10, 12].

Decreased platelet production or function Alcohol use disorder* Bone marrow failure, suppression, or infiltration Congenital platelet disorders Drug-induced nonimmune thrombocytopenia Hepatic disease* Infections‡ Myelodysplastic syndrome Nutritional deficiencies (vitamin B12 and folate) Sepsis*	Increased peripheral consumption Alloimmune destruction Disseminated intravascular coagulation* Drug-induced immune thrombocytopenia Heparin-induced thrombocytopenia Immune thrombocytopenia Infections‡ Mechanical destruction Paroxysmal nocturnal haemoglobinuria Preeclampsia/HELLP syndrome Thrombotic microangiopathy (thrombocytopenic purpura, haemolytic uremic syndrome, drug-induced thrombotic microangiopathy) Vaccine-induced immune thrombotic thrombocytopenia	Sequestration/ other Alcohol use disorder* Dilutional thrombocytopenia Gestational thrombocytopenia Hepatic disease* Hypersplenism Pseudo thrombocytopenia
<p>HELLP -haemolysis, elevated liver enzymes, and low platelet count. More than one mechanism of action. In some cases, the abnormality is quantitative (decreased platelet production) or qualitative (platelet dysfunction). Examples include Alport syndrome, Bernard-Soulier syndrome, Fanconi anaemia, gray platelet syndrome, Glanzmann thrombasthenia, MYH9-related disorders, platelet-type or pseudo-von Willebrand disease, and Wiskott-Aldrich syndrome. Thrombocytopenia with infection is usually caused by bone marrow suppression. In some cases, the thrombocytopenia is also immune mediated. Infections include babesiosis, cytomegalovirus, dengue fever, ehrlichiosis, Epstein-Barr virus, <i>Helicobacter pylori</i>, hepatitis C virus, HIV, malaria, measles, mumps, parvovirus B19, rickettsia, rubella, SARS-CoV-2, varicella-zoster virus, and Zika virus.</p>		

Table no. 1 Etiologies of Thrombocytopenia

VII. DIAGNOSIS

Thrombocytopenia is not a disease but is a diagnosis. The detailed knowledge must be acquired from patients who have been suffering from the thrombocytopenia. Detailed examining and the test of laboratory should be done which are related to etiology. Some of the situation about thrombocytopenia must be checked. Recent new drugs or drugs that are only taken intermittently, recent infection, previously diagnosed hematologic disease, nonhematologic diseases known to decrease platelet counts (eg, eclampsia, sepsis, DIC, anaph. lactic shock, hypothermia, massive transfusions), positive family history of bleeding and/or thrombocytopenia, recent virus vaccination, poor nutritional status, pregnancy, recent organ transplantation from a donor sensitized to platelet alloantigen's, recent transfusion of a platelet-containing product in an all sensitized recipient. History pertaining to alcohol consumption and HIV risk factors should be obtained. When faced with an asymptomatic patient with a low platelet

count, the clinician should initially seek to exclude artefactual or "pseudo thrombocytopenia" as the etiology. Also, the story o can be eliminated in patient or the abnormality which causes thrombocytopenia can be recognized [5]. f the family must be questioned for the congenital thrombocytopenia reasons [4]. Thrombocytopenia is not only bleeding for patient, but it depends on heparin. Disseminate intravascular coagulation and as paroksismal nocturnal haemoglobinuria can be with thrombosis clinic. In physical examination the thrombocytopenia which depends on diagnosis petechia, purpura, nose bleeding, gum bleeding, haematuria, menorrhagia or as cerebral haemorrhage is understood also some symptoms which depend on thrombosis can be fixed. The peripheric smear must be done every patient who is thrombocytopenia. In normal magnify there can be seen 3 - 10 platelet in every part.

VIII. CONCLUSION

Thrombocytopenia is a second most common haematological disorder after anemia. It has various causes such as drugs, chemicals, clinical disorders, foods and additives. It can affect both male and female, but mostly likely in pregnant women. Understanding the auto pathology is important in the treatment of thrombocytopenia. Treatment of thrombocytopenia depends upon the severity and condition of the patients. Corticosteroids it is the first line of therapy for thrombocytopenia usually prednisone drug is used, which can help raise the platelet count by decreasing the activity of the immune system and in critical cases, platelet transfusion is the most suitable option.

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