

***Final Term Assignment***

***Technology***

*Medical Lab Technology(MLT)*

***Subject***

*WBCs and Platelets Disorder*

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**Q1. Write a note on Hodgkin lymphoma?**

**[Answer]**

**Hodgkin Lymphoma:**

Hodgkin lymphoma is a B-cell lymphoma characterized by the Reed-Sternberg cell, a tumor cell. These are large, irregular lymphocytes with multiple nuclei found on affected lymph node histology.

**Further types:**

Classical Hodgkin lymphoma (95 per cent)

Hodgkin lymphoma prevalent in the nodular lymphocyte (5 per cent of cases).

**Classical Hodgkin lymphoma is further categorized histologically into:**

* Classical Hodgkin lymphoma with nodular sclerosis
* A classical Hodgkin lymphoma rich in lymphocytes
* Classic Hodgkin Lymphoma Mixed cellularity
* Classical Hodgkin lymphoma weakened by the lymphocytes.

**Causes:**

A change in the DNA of a B-cell lymphocyte triggers Hodgkin lymphoma. This mutation causes huge numbers of irregular and over-sized B cells to accumulate in the lymphatic system, spreading to other organs over time. What causes DNA mutation isn't obvious.

A family history of a particular Hodgkin lymphoma sub - type is most closely associated with that subtype risk, supporting the likelihood of genetic predisposition.

In patients with chronic infections or autoimmune disorders the risk of lymphoma increases slightly.

Those with immune suppression, such as those who have undergone long chemotherapy courses for certain illnesses, or who are infected with viruses such as the human immunodeficiency virus (HIV) or the Epstein-Barr virus (EBV), may have an elevated risk of developing lymphoma. See also Lymph proliferative Conditions associated with Epstein – Barr virus.

**Clinical features:**

Hodgkin lymphoma typically starts inside a single lymph node, and then spreads through the lymph networks to neighboring lymph nodes before spreading to distant sites and organs[8,9]. This typically occurs with painless swelling of superficial lymph nodes (in the arm, axilla, or groin) or as an asymptomatic mass seen on an X-ray of the chest.

Many patients with non-specific, or 'B', symptoms present to their physicians. They may have one or more signs, such as:

* Temperature
* Drenching sweats by night
* Unexpected weight loss
* Maybe lethargy.

**Symptoms and Signs:**

Patients can sometimes present with symptoms and signs indicating the involvement of organs in the disease such as:

* Jaundice and itching due to hepatitis
* Tetany (muscular spasms) because of the high calcium levels
* Fatigue due to anemia
* Edema by involvement of the kidneys.

**Possible diagnosis:**

A thorough screening is the initial step in the diagnosis of Hodgkin lymphoma, which is confirmed in the pathological examination of a lymph node biopsy by the presence of Reed-Sternberg cells.

In test for symptoms of anemia and inflammation a full blood count and inflammatory markers are used.

Evaluation of the lymph node can be an excisional biopsy (where a full lymph node is removed), an incisional biopsy, i.e. (where part of a lymph node is removed), or a core biopsy (where part of a lymph node is extracted with a broad needle).

In patients with direct cutaneous infiltration with Hodgkin lymphoma, a skin biopsy of an infiltrated nodule that diagnose Hodgkin’s disease. Histology usually shows Reed-Sternberg cells in the process of invasion of small lymphocytes, histiocytes, eosinophils and plasma cells.

**Q2.What is Hemostasis , also explain steps and clotting factors?**

**[Answer]**

**Hemostasis:**

Hemostasis is the mechanism through which the body prevents bleeding from cutting or damage. Hemostasis is the normal cycle through which blood flow decreases and a clot develops to avoid blood loss after damage, with blood meaning hemo and stasis meaning stopping. Blood transitions from a liquid substance to a gelatinous state during hemostasis.

**Stages:**

**Vasoconstriction:**

Vasoconstriction is a reflex which narrows the blood vessels to increase blood pressure.

**Platelets plug formation:**

The plug formation of platelets involves the activation, aggregation and adherence of platelets in a plug which acts as a blood flow barrier.

**Coagulation:**

Coagulation requires a complex cascade in which fibrinogen cleaveth a fibrin network.

During formation of a clot, fibrin serves as a "molecular glue," holding the platelet plug together.

**Clotting Factors:**

The factors of coagulation are numbered in the order of its discovery. There are only 12 factors but 13 numerals. Factor VI was later found to be a part of a different factor.

1. Factor I Fibrinogen
2. Factor II Prothrombin
3. Factor III Thromboplastine (tissue factor)
4. Factor IV calcium ionized (Ca++),
5. Factor V the labile or proaccelerin factor
6. Factor VI undetermined
7. Factor VII Secure or Proconvert element
8. Factor VIII antimicrobial factor
9. Factor IX part of thromboplastine plasma, factor for Christmas
10. Factor X Stuart-Prower
11. Factor XI history plasma thromboplastine
12. Factor XII The factor Hageman
13. Factor XIII Factor to Stabilize Fibrin

Vitamin K must be used in the liver to develop Factors II, VII, IX and X. Dietary vitamin K comes from plant and animal sources and is readily available. Normal intestinal flora produces it too.

**Q3.Explain Hemophilia its types, symptoms, and lab diagnosis ?**

[Answer]

**Hemophilia:**

Hemophilia is a bleeding condition, which delays the cycle of blood clotting. People with this condition experience continuous bleeding or oozing after injury, surgery or the pulling of a tooth. For serious hemophilia cases, recurrent bleeding occurs after mild injuries or sometimes without damage (spontaneous bleeding).

**Types of hemophilia:**

**Hemophilia A:**

Hemophilia A is a genetic disease caused by missing or deficient protein Factor VIII. It is inherited but is caused by a random genetic mutation in around one third of reported cases.

All racial groups are affected similarly by the blood condition. More than half of the people with hemophilia A have a serious type of disease.

Hemophilia A is transmitted by the X chromosome. This is inherited in a recessive manner and is X-linked. As such, for the disorder to be present in women, two hemophilia-carrying X chromosomes must be inherited but for men only in one X chromosome.

**Hemophilia B:**

Hemophilia B is a genetic disorder caused by absent or defective protein coagulation by factor IX. It is also inherited and in one-third of cases, much like hemophilia A, it can be caused by a random genetic mutation. This form of hemophilia also has an equal effect on all ethnic groups, but it is about four times as severe as hemophilia A.

Hemophilia B is often transmitted in the X chromosome in an X-linked recessive pattern, indicating that it is essential to inherit two hemophilia-carrying X chromosomes for the disorder to be active in women, but only one X chromosome in men.

**Symptoms:**

Hemophilia symptoms and signs vary, depending on your clotting factor level. If your level of the coagulation factor is reduced significantly, you may bleed only after surgery or trauma. If you have a serious deficiency you can experience spontaneous bleeding.

**Spontaneous bleeding marks and symptoms include:**

* Excessive and unexplained bleeding from wounds or burns, or during surgery or dental work
* Several bruises, broad or small,
* Unusual bleeding following vaccinations
* Pain, tightness or swelling in the joints
* Blood in your stool or urine
* Nosebleeds which have no known cause
* Unprecedented irritability in children

**Diagnosis:**

Diagnosis includes tests for the screening and tests for the coagulation factor. Screening tests are blood tests which show whether the blood is properly coagulating. To diagnose a bleeding disorder, tests of the coagulation factor, also called factor assays, are needed. A blood test indicates the form and extent of hemophilia.

**Screening Test:**

Screening tests are blood tests which show whether the blood is properly coagulating. Screening Control Types:

* Total Blood Count (CBC)
* Activated Partial Thromboplastine Time Test (APTT)
* Prothrombin time test (PT)
* Fibrinogen
* Clotting factor

To diagnose a bleeding disorder, tests of the coagulation factor, also called factor assays, are needed. A blood test indicates the form and extent of hemophilia.

**Q4. Describe Von Wille Brand disease?**

**[Answer]**

**Von Willebrand disease:**

The condition is named after Erik von Willebrand, a Finnish physician who first described it in the 1920's.

Von Willebrand’s disease (VWD) is a genetic disorder caused by Willebrand’s missing or defective factor (VWF), a protein that coagulates. VWF binds factor VIII, an important protein for clotting, and platelets in the walls of the blood vessels, helping to form a platelet plug during the coagulation process.

VWD is the most familiar bleeding disorder that affects up to 1 per cent of the population in the United States. It is borne on chromosome 12 and occurs in men and women alike.

**Types:**

There are three major forms of VWD dependent on VWF's qualitative (type 2 and platelets type) or quantitative (type 1 and 3) faults. A fourth form, acquired from VWD, is not inherited.

**Type 1:**

In 60 per cent -80 per cent of patients, type 1 VWD is reported. People with type 1 VWD suffer from a quantitative VWF deficiency. VWF levels range from 20 percent -50 percent of normal in the blood. In general the symptoms are mild.

**Type 2:**

Type 2 VWD is found in patients with 15 percent -30 percent. Those with Type 2 VWD provide their VWF with a qualitative shortcoming. Type 2 is split into four subtypes: Type 2A, Type 2B, Type 2 M and Type 2N, depending on multimer presence and behavior, VWF molecular chains. Symptoms are moderate to mild.

**Type 3:**

Type 3 VWDs are found in 5% -10% of patients. People with type 3 VWD suffer from a quantitative VWF deficiency. Symptoms are usually serious, and often involve episodes of sporadic bleeding within their joints and muscles.

**Acquired VWD:**

A form of VWD in adults results from an autoimmune disease disorder, such as lupus, or from heart disease or certain forms of cancer. This can also happen after other drugs have been taken.

**Causes:**

Willebrand disease is caused by a genetic mutation. The type of von Willebrand disease you have depends on whether a mutated gene was passed on to you by one or both of your parents. For example, you can only grow type 3 Von Willebrand if both of your parents have inherited a mutated gene.

If only one copy of the mutated gene is inherited, you will develop a type 1 or 2 von Willebrand disease.

**Symptoms:**

Individuals with VWD suffer regular nosebleeds, quick swelling and heavy bleeding during and after surgical operations, such as dental extractions and surgery. Women also experience extreme menstrual bleeding (heavy menstrual cycles that last longer than average), and post-birth hemorrhage.

**Blood tests may analyze:**

**Levels of VWF:**

* In order to determine the type of von Willebrand disease, the structure of the vWF and its multimers or protein complexes and how its molecules break down
* Cofactor operation of ristocetin to demonstrate how well vWF functions and how it coagulates the blood adequately
* Factor VIII Coagulation activity, determining factor VIII levels
* Platelet function
* Bleeding time, to see how long a small wound takes to avoid bleeding

**Q5. Explain Hemolytic uremic syndrome and its types?**

**[Answer]**

**Hemolytic uremic syndrome:**

Hemolytic uremic syndrome (HUS) is a disease characterized by red blood cell loss, low platelet count and renal failure.

**Types:**

Hemolytic uremic syndrome has two types;

**Typical:**

Typical HUS fits a frequently induced diarrheal infection. E.Coli.

**Atypical:**

Atypical HUS is not associated with digestive tract infection, and has a less beneficial effect.

**Symptoms:**

HUS symptoms can vary. Possible signs include:

* Bloody diarrhoea
* Abdominal pain
* Blank skin
* Irritability
* Fatigue
* Temperature
* Bleeding or unexplained bruises
* Reduced urination
* Tummy swelling
* Blood in Urine
* Confusion
* Vomiting
* Gull face
* Gull arms
* Seizures (rare)

**Causes:**

HUS occurs where blood cells are destroyed by an immune reaction. It contributes to low rates of red blood cells, low levels of platelets and kidney injury

HUS in Children:

For children the most common underlying cause of HUS is Escherichia Coli (E. coli) infection. There are several different types of E.coli, and most are not causing trouble. Finally E.coli bacteria are commonly present in healthy human and animal intestines. But some particular strains of E.Coli, transmitted by infected food, are responsible for infections which may lead to HUS. Water bodies that are polluted with feces can hold too. E.coli. Certain bacteria that can induce HUS, such as Shigella dysenteriae and Salmonella typhi.

**HUS for Adults:**

An infection also causes HUS in adults with E.Coli. There are also other, less common, non-bacterial causes of HUS in adults, including:

* Pregnancy
* HIV / AIDS infection
* Quinine (Applied to muscle cramps)
* Chemotherapy and treatment for immunosuppression
* Pills used for birth control
* Anti-platelet drug
* Cancer
* Systemic Lupus and glomerulonephritis.

**Diagnosis at laboratory:**

Laboratory research is far more specific, and includes:

* Full blood count showing hemolysis in the form of erythrocyte fragmentation, seen within one day of infection.
* Microangiopathic hemolytic type anemia, with a mean concentration of 6g / dL in hemoglobin.
* Thrombocytopenia is seen in more than 90 percent of HUS patients due to platelet sequestration in specific end-organ vascular beds, with a count below 50 000 / mm3.
* Unmodified clotting time.
* Leukocytosis exists.
* The count of reticulocytes may increase.
* The CRP is high.
* Coomb’s test is negative.
* Hematuria presence on urinalysis. It may also be that proteinuria is present.
* Acute renal impairment shown by renal function tests indicating azotemia, as a result of micro thrombi obstruction of renal blood flow in the renal parenchyma.
* Stool cultures should be taken for STEC but after the first six days of infection, the patient may not be shedding the microbes. This will validate the etiological diagnosis, which is essential to public health. Stool checking for toxin Shiga is faster and more accurate.