Pediatric Nursing

Hematology 'part 2'

Lecture 11
DIC

• Disseminated intravascular coagulation, is a life threatening pathological process in which clotting factors are abnormally activated.

• Resulting in wide spread of clot formation in the small vessels throughout the body.

• It is complication of other serious disease such as infections, sepsis, hypoxia, shock, trauma, burn, liver disease, cancer.
Coagulation

Stage one

Intrinsic Factors
- From plasma
- Release factors: VIII, IX, XII

Extrinsic Factors
- From tissue
- Release factors: VII, X, III

+ platelets aggregation

↑ V.C
↓ B.F
Stage two

Presence *Complete thromboplastine time (III)* and converted to...

Stage three

Prothrombin (II) that converted to thrombin with help of Ca++ & K +

Stage four

Thrombin converted to fibrinogen that will converted into fibrin

Formation of the plug
Pathophysiology

1. Widespread formation of tiny blood clots within microcirculation of all body organs.

2. The fibrinolytic pathway is activated, promoting the dissolution of the clots that have been formed.

3. The amounts of thrombin that inter the circulation greatly exceed that of clotting inhibitors to regulate it.

4. The deposit of thrombin, decrease blood flow to organs leading to ischemia, infarction & necrosis.
Pathophysiology

5. The excessive amount of thrombin also activate platelets aggregation; causing thrombocytopenia.

6. Plasma begin to break down fibrin before a stable clot is formed.

5. Clotting factors are depleted, the ability to form clot is lost, and hemorrhage occurs.
Manifestations

- All symptoms of diffuse bleeding:
  1. Hematurea.
  2. Petechiae or purpura.
  3. Oozing at injection site.
  5. Major vessels thrombosis.
  6. Cyanosis, pallor, cool extremities.
  7. Weakness, malaise.
Treatment

• Clinical therapy is supportive.
• Identification of the underlying cause.
• Replacement of the depleted coagulation factors.
• Anticoagulation therapy (heparin).
Nursing managements

• Assessing bleeding, prevent injuries.
• Observe petechiae, ecchymoses & oozing every 1 - 2 hours.

• Assess extremities for capillary refill, warmth & pulse.

• Assess stool for presence of blood.

• Maintain skin integrity, gentle positioning.
Leukemia

Is a cancer of the blood or bone marrow and is characterized by an abnormal proliferation (production by multiplication) of blood cells, usually white blood cells (leukocytes).
Classifications

• Leukemia is divided according to the affected cells or into acute and chronic.

Acute leukemia

Is characterized by the rapid increase of immature blood cells. This crowding makes the bone marrow unable to produce healthy blood cells.

Immediate treatment is required in acute leukemias due to the rapid progression and accumulation of the malignant cells, which then spill over into the bloodstream and spread to other organs of the body.
Chronic leukemia

Is distinguished by the excessive build up of relatively mature, but still abnormal, blood cells.

Typically taking months or years to progress, the cells are produced at a much higher rate than normal cells, resulting in many abnormal white blood cells in the blood.

Chronic leukemia are monitored for some time before treatment to ensure maximum effectiveness of therapy.
• In **lymphoblastic** : **lymphocytic leukemias**, the cancerous change took place in a type of marrow cell that normally goes on to form lymphocytes.

• In **myeloid** : **myelogenous leukemias**, the cancerous change took place in a type of marrow cell that normally goes on to form red cells, some types of white cells, and platelets.
<table>
<thead>
<tr>
<th>Cell type</th>
<th>Acute</th>
<th>Chronic</th>
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<tbody>
<tr>
<td>Lymphocytic leukemia</td>
<td>Acute lymphocytic leukemia</td>
<td>Chronic lymphocytic leukemia</td>
</tr>
<tr>
<td>Myelogenous leukemia</td>
<td>Acute myelogenous leukemia</td>
<td>Chronic myelogenous leukemia</td>
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</table>
1- **Minor infection** as common cold that has failed to completely disappear.

2- Fever failed to disappear although use of antibiotic

3- Pale, restlessness.
4- Irritable, anorexia

5- Weight loss
6 - Bone + joint pain
7 - Sudden bleeding from teeth, mouth, nose.
Clinical manifestation

8 - Bruising without cause
9 - Recurrent infection
10 - Abdominal pain

11 - Decrease apatite
12 - Lymphoidadenopathy
13 - Dyspnea

14 - Anemia, fatigue, bleeding, decrease platelets
15 - Hepato - spleenomegally.
There is no single known cause for all of the different types of leukemia.

Researchers have strong suspicions about four possible causes:

- natural or artificial radiation
- certain kinds of chemicals
- some viruses
- genetic predispositions
Diagnostic evaluation

- History & physical examination
- WBC differentiation
- Bone marrow aspiration (biopsy)
- CBC, U/S
- Lymph node biopsy.
- Lumber puncture
Nursing role during chemotherapy

1- Teaching and support for family about side effect of medication and complication

2- Prepare family and child before start treatment.
Sickle Cell Anemia is an autosomal recessive condition in which the type of hemoglobin S (Hgbs) in a person’s RBCs differs either qualitatively or quantitatively from the hemoglobin that is usually contained in normal red cells (HgbA).

where red blood cells form “sickle” shapes instead of round ones.
SICKLE CELL TRAIT

A A

Sickle Cell Trait

A S S S

Sickle Cell Trait

Sickle Cell Disease

Usual
• **Defective hemoglobin caused by mutation at one amino acid:** _Valine replaces glutamic acid_

- Forming long hemoglobin fibers - cause sickle shape inside RBC

- Elongated cells block the capillaries, leads to microscopic obstruction and ischemia occur.

- Causing swelling, inflammation, pain, and cell death.
• Sickeling may be triggered by any factor that increases the body’s need for oxygen or alert the transport of it, such as:

• Fever, emotional & physical stress.
• Increasing blood velocity (low intake).
• Hypoxia.
• Cold.
• Trauma.
• Infections.
Common forms of Sickle Cell Anemia:

1 - sickle cell anemia (HgbSS):
 homozygous form of the disease (Hgbss), in which valine, an amino acid, is substituted for glutamic acid at the sixth position of the B-Chain.

2 - sickle cell - c disease:
 heterozygous variant of SCD including both HgbS and hemoglobin C (Hgbsc) in which lysine is substituted for glutamic acid at position 6 of B-chains.

3 - sickle cell trait (HgbSA):
 Heterozygous condition (child has one normal and another affected one), child is carried.

4 - sickle thalassemia disease:
 combination of sickle cell trait and B-thalassemia trait.
Clinical manifestation

- Manifestations are classified according to the crisis that could occur

1- vaso-occlusive crisis.
2- spleenic sequestration.
3- aplastic crisis.
4- acute chest syndrome.
Clinical manifestations are related to the shorten life span of the blood cells (hemolytic anemia) and tissue destruction resulting from vaso-occlusion crisis.

Brain: CVA, headache, convulsion
Eyes: retinopathy, retinal detachment.
Bones: osteoporosis, osteomyilisis.
Heart: cardiomegally, exercise intolerance.
Lungs: infection, cough, fever, tachypnea.
Liver: hepatomegaly, cirrhosis.
Spleen: spleenomegally.
Kidneys: enuresis, hematurea, renal failure.
Skin: decreased peripheral circulation.
• The most common reason for hospitalization is due to acute painful episodes.

• Pain results from avascular necrosis of bone marrow, it experienced in back, abdomen, chest & joints.

• Generalized pain lasts for several days or weeks.
• The pain can be acute (sudden), chronic (long lasting), or a mixture of the two.
• **spleenic sequestration:**
  Profound anemia, hypovolemia, shock. Because blood pool in the spleen.

• **Aplastic crisis:**
  Anemia, pallor, fatigue. Because of decreased production of RBC & increase destruction of it.

• **Acute chest syndrome:**
  Fever, cough, chest & back pain, dyspnea, hypoxemia.
Diagnostic tests

• Hemoglobin electrophoresis.
• History & physical examination.
• Serum analysis that reviles anemia
• Hb (6-10 g/dl).
• Increased RBCs levels.
Clinical therapy

- Control the pain:
  1. By parenteral analgesia, such as morphine.
  2. Increase hydration.
  3. Oxygenation.

- Treat infections:
  1. By antibiotic such as penicillin.
  2. Monitor infections by cultures (blood, throat).
  3. Give immunization as scheduled.
• **Transfusion of RBCs:**
  1. To improve tissue oxygenation and temporary suspension of production of RBCs containing HbS.
  2. **Monitor for iron overload (hemosidrosis) with frequent transfusion.**
  3. **Give deferoxamine with vitamin C to enhance excretion of iron.**

• **Other therapy:**
  1. **Hydroxyurea:** is given that decreases the production of abnormal blood cells and leads to lesser pain
  2. **Hematopoietic stem cell transplantation.**
Nursing intervention

- Monitor child during blood transfusion.
- Monitor iron overload.
- Promote hydration.
- Pain management: Give analgesia as order.
- Emotional support.
- Infection control
- Educational support.
- Ensure adequate nutrition
Anemias

- Anemia is reduction in the number or RBCs, the quantity of hemoglobin, & the volume of packed red cells to below-normal levels.

- **It has several types:**
  1. **Normaocytic anemia**: decreased in production of RBC due to decrease stimulation. RBC is normal in color & size but low in number → anemia.
  2. **Acute blood loss anemia**: anemia that result from acute blood loss.
  3. **Anemia of acute infection**: infection leads to increase destruction of RBC → anemia occur.
4. **Anemia of renal diseases**: problems in kidneys leads to decreased erythropoietin secretion, thus, decreasing RBCs production → anemia (normaocytic)

5. **Anemia of neoplastic disorders**: invasion of bone marrow leads to decrease production of RBCs, thus, normaocytic anemia.

6. **Aplastic anemia**: (congenital or acquired), depression of hematopoietic activity in bone marrow will affect the RBC production with WBCs & platelets affected → anemia occur.

7. **Hypoplastic anemia**: same as previous condition, but the effect is on RBC production only.
8. Folic acid deficiency anemia: low level of folic acid will lead to arrest in of maturity process of RBC leading to abnormally large RBCs, \( \rightarrow \) anemia.

9. Iron deficiency anemia
Iron deficiency anemia

• As the body require iron to produce hemoglobin, insufficient quantities of iron limit hemoglobin production. And affecting RBC production resulting in anemia.

• It could occur secondary to blood loss, malabsorption or poor nutrition intake.

• Adolescent are at risk to develop this kind of anemia as well as the infants of 6 months of age.
Clinical manifestations

• Pallor
• Fatigue.
• Irritability.
• Nail bed deformities in prolonged anemia.
• Growth retardation.
• Developmental delay.
• Pica: consumption of non-food item.
Managements

• Correction of the iron by oral supplements.
• Consumption of food rich in iron (meats, fish, dried fruit, vegetables, whole cereal).
• Consumption of food rich in vitamin C, to enhance iron absorption. Such as (orange, strawberries, potatoes).
• Ferrous sulfate to correct the deficiency.
• Blood transfusion if Hb is less than 4 mg/dl.