"What to do with this child’s abnormal CBC?"

Nicola Jones, APRN, FNP-C, CPHON, AOCNP
Victoria Zelko, APRN, CPNP-AC, FNP-C, CPHON

Objectives

- To state the main components of a CBC which includes WBC, Hgb, and Platelet count. Describe the function of each component and state at least one age related implication.
- To recognize abnormal values in CBC results and state next appropriate action.
- To state 4 common disease processes related to abnormal CBC results and when referral to specialist would be indicated.

Back to Basics

White Blood Cells (WBC)

- White blood cells are comprised of neutrophils, monocytes, lymphocytes, eosinophils, basophils.
- Neutrophils are the primary defense in bacterial infections. Neutrophils are able to phagocytize and kill bacteria.
- Monocytes are the large phagocytic cells involved in the early inflammatory stage.
- Lymphocytes are involved in the development of antibodies and delayed hypersensitivity. Two types, B cells and T cells.
- Eosinophils have parasiticidal properties and can selectively destroy parasites.
- Basophils are seen in increased amounts during periods of inflammation and are responsible for histamine release.

Red Blood Cells (RBC)

- The major function of RBCs is to transport hemoglobin.
- Hemoglobin is a true indicator of the physiological potential of blood to transport oxygen to tissue.
- Hematocrit indicates the volume percentage of circulating RBCs and is approximately 1 times the concentration of Hgb.
- RBC indices are comprised of MCV (mean corpuscular volume), MCH (mean corpuscular hemoglobin), MCHC (mean corpuscular hemoglobin concentration) which are based on ratios of RBC volume, RBC count, and Hgb concentration.
- Hemoglobin is broken down into hemosiderin (iron) and bilirubin. Most of the bilirubin is reused by the bone marrow for the production of new RBCs or stored in the liver and other tissues for future use.
- Reticulocyte count is the direct measurement of the production of RBCs by the bone marrow and indicates the activity of the bone marrow.

Platelets (PLT)

- Platelets are also known as thrombocytes and are a component of the blood whose function (along with other clotting factors) is to react to bleeding from blood vessel injury.
- Platelets adhere to the endothelium to form a plug to stop bleeding.
- The first platelets which arrive at an injury site release substances that attract other platelets to the site. They release serotonin at the injury site which causes vasoconstriction.
Age related implications

It is important to recognize age related implications when interpreting CBC results and in making the decision to refer your patient to a hematology or oncology specialist.

*A very useful reference is the Harriet Lane Handbook, Hematology section*

<table>
<thead>
<tr>
<th>Age</th>
<th>WBC</th>
<th>Hgb</th>
<th>Hct</th>
<th>Plt</th>
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<td>9-30</td>
<td>16.5</td>
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<td>290</td>
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<td>6-17</td>
<td>12.0</td>
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<td>4.5-11 (males/females)</td>
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<td>150-350</td>
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<td>12-18 year old</td>
<td>14.0 females</td>
<td>41% females</td>
<td>150-350 (males/females)</td>
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Examples of age related differences

Common Disease Processes Related to Abnormal CBC results

- Leukemia
- Iron deficiency anemia
- ITP
- Sickle Cell Trait
- Sickle Cell Disease
- Thalassemia
- Autoimmune hemolytic anemia
- Spherocytosis
- Chronic Anemia
- Infection/Leukocytosis
- G6PD deficiency
- Factor VIII deficiency

Iron deficiency anemia

- Iron deficiency is the most common nutritional deficiency worldwide.
- Anemia is the most important indicator of iron deficiency (ID); however, ID may develop in the absence of anemia and the tissues may be affected by this condition.
- History may include excessive intake of cow’s milk, increased fatigue, irritability, difficulty sleeping, behavioral disorder, and learning difficulty. In teenage girls, heavy menses can be a contributing factor.
- Blood loss should always be considered in the work up.
- Physical exam may include pallor, lethargy, tachycardia, neurocognitive deficits.
- CBC findings may reveal decreased RBC, Hgb/Hct, MCV, MCH, MCHC and increased RDW and occasionally thrombocytosis.
- Additional lab work may also reveal decreased ferritin, serum iron, elevated TIBC, low transferrin saturation.
- Referral to Hematology should be considered if patient has not responded or is intolerant to oral iron therapy.

Leukemia

- Every year ~4,900 new cases of ALL occur in the United States.
- History could include illness, fever, bleeding, CNS symptoms (headache, vision changes, facial palsy), bone pain.
- Physical exam may include splenomegaly, hepatomegaly, testicular swelling, lymphadenopathy.
- **Pancytopenia with or without blasts, high WBC with low Hgb/Plts would warrant immediate referral to Hem/Onc service or ER. It is also very important to stress that the provider should not scare the parents, especially for pancytopenia without blasts as the more common cause is infection.**

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Clinical pearl

- Thalassemia trait can also present with microcytosis (decreased MCV) and anemia. If not iron deficient, a Mentzer Index is useful to do.
- The Mentzer Index is calculated from the results of a complete blood count: MCV/RBC. Greater than 13 indicates iron deficiency, less than 13 suggests thalassemia trait.
**Chronic Anemia**

- Primary chronic anemias are true chronic anemias, in which anemia is part of the basic process (sickle cell disease or hereditary spherocytosis.)
- Secondary chronic anemias are chronic anemias which may provide a diagnostic clue to an underlying pathology. They are the consequence of a nonhematologic problem such as chronic blood loss, chronic renal failure, osteomyelitis, or inflammatory bowel disease.
- Patients with chronic anemia are usually asymptomatic and often can tolerate surprisingly low Hgb levels (~5-6 g/dL).
- All patients with primary chronic anemia should be routinely followed by a hematologist. If concern for secondary chronic anemia, the patient may benefit from referral to help reveal underlying pathology and direct appropriate therapy.

**ITP**

- Idiopathic thrombocytopenic purpura is primarily a disease of increased platelet destruction, with most patients having antibodies to specific platelet membrane glycoproteins.
- An average estimate of the incidence in children is 50 cases per 1,000,000 per year and new cases of chronic refractory ITP comprise approximately 10 cases per 1,000,000 per year. Peak prevalence occurs in children aged 2-6 years.
- Acute ITP often follows acute infection and has spontaneous resolution within 2 months.
- History provided by family may include recent viral illness, abrupt onset of symptoms such as purpura, epistaxis, gingival bleeding, and bruising.
- Physical exams may include non-palpable petechiae (mostly in dependent regions), hemorrhagic bullae on mucous membranes, purpura, gingival bleeding, signs of GI bleeding, menorrhagia, retinal hemorrhages, evidence of intracraniad hemorrhage (possible neuro symptoms), non-palpable spleen.
- CBC will reveal isolated thrombocytopenia (decreased platelet count, the WBC and Hgb are typically normal unless hemorrhage has occurred.) Coag studies are usually normal and bleeding times are not helpful. Referral to Hematologist is necessary, spontaneous bleeding can occur with plt count <20K; if plt count <20K referral to ER is indicated.

**Sickle Cell Anemia and Trait**

- Sickle cell disease is an autosomal recessive inheritance pattern most often found in people of African descent but can also be found among ethnic groups from the Mediterranean, Caribbean, and India.
- In the United States, SCD occurs in about 1 of every 400 black infants.
- SCD describes a group of complex, chronic disorders that are characterized by hemolytic, nonhemolytic acute complications which can become life threatening, and possible development of chronic organ damage.
- Children who have SCD do not form the normal Hgb A molecule but rather synthesize hemoglobin S which carries the amino acid valine instead of glutamic acid. This process damages the RBC by giving it a “sickled” appearance and causes a chronic hemolytic anemia with associated anemia and vaso-occlusive problems.
- Children with sickle cell trait who are heterozygous for the gene essentially have a benign course. Their RBCs contain only 30-40% of Hgb S and sickling generally does not occur. Their newborn screen will be FAS.

**Sickle Cell Disease**

- Most infants with sickle cell disease are identified by routine neonatal screening. To confirm newborns screened by High performance, the patient should not have PRBC transfusions within 3 months and do not repeat Hgb electrophoresis on FAS until 3-6 months of age.
- Symptoms generally begin to emerge in the second 6 months of life as the amount of Hgb S increases and Hgb F declines.
- Physical exam concerning for vaso-occlusive crisis may be exhibited by the following: pallor, jaundice, painful swelling of hands/feet (common in infancy), fever, painful involvement of larger bones (older patients), gingival, abdominal pain, chest pain, enlarged spleen (sequestration of blood) (Note: after 5 years old, splenomegaly tends to disappear due to auto-infarction.
- CBC evaluation may reveal Hct of 20-29% with sickled cells, nucleated RBCs and Howell-Jolly bodies on peripheral smear, Hgb 6-10, MCV >90, elevated retic 5-15%, normal to increased WBC and Plt count. High electrophoresis after infancy reveals a predominance of Hgb S and no Hgb A.
- If you have concern that patient is experiencing vaso-occlusive crisis urgent referred to hematologist is indicated and patient should be directed to nearest ER.
Thalassemia

The thalassemias are a group of hereditary, hypochromic anemias associated with the absence or decreased synthesis of the normal Hgb polypeptide chains, usually the alpha and beta chains.

Anemia associated with alpha thalassemia can be absent or can range from mild to severe depending on the number or alpha genes affected. For example, one gene deletion is of no clinical significance; two gene deletion or trait may be associated with asymptomatic mild anemia; three gene deletion (Hgb H disease) can present with moderate to severe anemia requiring regular transfusions; four gene deletion, hydrops fetalis, is not compatible with life.

Beta thalassemias cover a broad clinical spectrum of disorders which are classified according to patterns of inheritance and severity of anemia. Populations affected are usually those of Mediterranean and Southeast Asian descent. Louisville has community of Southeast Asians

In contrast homozygous beta thalassemia major is associated with severe anemia which results from decreased or absent production of Hgb A and hemolysis caused by precipitation of excess alpha chains in the RBC which results in premature erythrocyte destruction (hemolysis). Incidence is approximately 1 per 80,000 persons/year.

RBC normally survive in peripheral circulation for 100-120 days. In AIHA, anemia develops when hemolysis occurs at a rate faster than RBC production.

Though AIHA has many causes (autoimmune conditions such as SLE, infections such as hepatitis, EBV, mycoplasma pneumonia, and effect from medications such as penicillin), more than 50% are idiopathic.

Physical exam may include malaise, pallor (look at conjunctiva, oral mucosa), jaundice, palpitations, tachycardia, dyspnea, dizziness, dark or tea colored urine, hypoxia, splenomegaly, hepatomegaly.

Lab evaluation: CBC will reveal anemia which can be mild or severe. Increased retic count. Positive direct Coombs test. Increased unconjugated bilirubin, increased LDH, decreased haptoglobin, and hemoglobinuria.

If AIHA is suspected, referral to hematologist is essential. Decision to send patient to ER vs outpatient depends on Hgb, retic, and clinical status. Generally, we do not recommend sending to ER if Hgb is 9 or higher and clinically stable.

Autoimmune Hemolytic Anemia

AIHA is a group of disorders characterized by a malfunction of the immune system where antibodies are produced against antigen (proteins on the surface of the RBC) which results in premature erythrocyte destruction (hemolysis). Incidence is approximately 1 per 80,000 persons/year.

Spherocytosis

Hereditary spherocytosis (HS) is a heterogeneous disorder in which abnormalities of RBC structural proteins lead to loss of erythrocyte membrane surface area, resulting in spherical shape, hyperdense, poorly deformable RBC with shortened lifespan.

Though HS is rare, it occurs worldwide and affects individuals from all racial and ethnic groups. Incidence is about 1 in 2,000 births.

With neonates, they often are not anemic in the first week of life and jaundice will be most common presenting symptom. Older children and adults will often exhibit a triad of symptoms including anemia, splenomegaly, and jaundice.

If you suspect HS in a jaundiced neonate, obtain CBC for interpretation of the RBC indices. Typically a neonate will have elevated MCHC. If the MCHC is >36.5-37 g/dL, HS is likely.

If you suspect HS, referral is necessary to hematologist for management.

Other diagnostic lab work, peripheral blood smear revealing spherocytes, elevated retic count with or without anemia, elevated indirect bilirubin, and positive osmotic fragility test. Note, PCP should not do osmotic fragility testing before six months of age as results are not accurate, would defer this testing to hematology office.
Leucocytosis/Infection

- "Can you differentiate bacterial from viral pediatric infections based on the CBC?" was an article published by the Journal of Family Practice in 2007. The evidence-based answer was "NO." The CBC alone does not have adequate sensitivity or specificity to differentiate these infections. However, when used with other clinical parameters, the CBC can help detect serious bacterial infections in patients with fever.

In a retrospective study of 5,353 infants ages 3-89 days presenting to an ER for evaluation of fever revealed that 3 of 4 infants with bacterial meningitis would have been missed if the WBC alone were used to predict which infants needed lumbar puncture.

G6PD deficiency

- Glucose-6-phosphate dehydrogenase (G6PD) deficiency is an inherited disorder caused by a genetic defect in the RBC enzyme G6PD. This disorder is the most common enzymatic disorder of RBCs affecting 400 million people worldwide.

- There are a few diagnostic tools you can utilize such as Heinz body prep, and G6PD enzyme level.

- The severity of hemolytic anemia varies among individuals with G6PD, making the diagnosis challenging. Numerous variants have been described and targeted therapy is often used in situations associated with chronic hemolytic anemia.

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- The majority of individuals are asymptomatic and do not have hemolysis in the steady state. Episodes of acute hemolysis can be triggered by medications (e.g., Primaquine, Dapsone, Rasburicase), certain foods (e.g., fava beans), acute illness, and especially infections.

- Important tip: G6PD patients should have their pharmacy chart labeled with G6PD diagnosis!!!

- Physical examination during an acute hemolytic state would reveal jaundice, pallor, dark urine, and may also include back pain or abdominal pain.

- CBC will reveal an abrupt fall in Hgb by 3-4 g/dL and peripheral blood smear reveals microspherocytes, eccentrocytes, and blister cells.

- Referral to hematologist would be recommended if there is a concern for G6PD deficiency or if family history is concerning. If concern for acute hemolysis, patient should be referred to ER for evaluation.

Factor VIII deficiency

- Hemophilia A is known as factor VIII deficiency or classic hemophilia. Occurs in 1 in 10,000 births. It is estimated that approximately 400,000 people have hemophilia and those with the disease, 80-85% have hemophilia A.

- Hemophilia is a sex linked recessive disorder. The abnormal gene responsible for hemophilia is carried on the X chromosome. Males only have one X chromosome and thus, if affected, express the trait.

- Clinical presentation includes bleeding from circumcision, multiple raised bruises without trauma, prolonged mouth bleeding, prolonged bleeding from heel or finger stick. Many children also present when reaching developmental milestones such as crawling/pulling to stand and become easily injured with bruising, swollen/red joints, or mouth bleeding.

- CBC will appear normal. However additional work up should include PT/PTT. PT measures deficiencies on the intrinsic pathway of coagulation, therefore, PT will be normal and PTT will be prolonged. If the PTT is prolonged, the diagnosis is confirmed by low or absent Factor VIII. Referral is always indicated if hemophilia is a concern.
Case study I

- **HPI:** 2-year old previously healthy female who presented with history of bruising for approximately 6 months and decreased platelet count. At her WCC, CBC demonstrated platelet count of 55,000. Repeat CBC obtained 1 week later was significant for platelet count of 28,000 and she was referred to Hematology/Oncology clinic for further evaluation. Upon evaluation in the Hematology/Oncology clinic, CBC demonstrated platelet count of 18,000 with normal WBC’s and Hgb. Parents report that bruising began mostly on shins, but has recently included her arms and flank.

- **PMH:** Treated for Strep throat 3 weeks prior to admission; h/o mild eczema
- **PSH:** No previous surgeries
- **Medications:** None
- **Allergies:** NKDA
- **Immunizations:** UTD including the flu vaccine
- **Family history:** Maternal uncle with diabetes; paternal grandmother with h/o stroke
- **Social history:** Lives at home with parents and two older brothers. Attends an in-home daycare.

- **Physical exam:**
  - Oxygen supplementation: Room air
  - General: well appearing, NAD, happy and playful
  - Head: NCAT
  - Eyes: PERLA, EOMI, conjunctiva pink, sclera nonicteric
  - ENT: oropharynx, oral mucosa, gums, hard and soft palate, tongue, tonsils, posterior pharynx are clear and without lesion. Nasal mucosa, septum and turbinates grossly normal. External ear canals clear.
  - Neck: no LAD, no thyromegaly or nodules, supple, no rigidity
  - Chest: CTAB, no increased WOB, no rhonchi/rales/wheeze, chest wall without deformity, no retractions, no nasal flaring
  - Heart: RRR, no murmur
  - Abdomen: NTND, no HSM or masses, BS present in all 4 quadrants. Soft, bruising on left flank
  - Extremities: full ROM, no deformity, symmetric in bulk, tone and movement
  - Skin: eczematous rash on arms and legs, scattered bruises on legs, bruising of BL AC fossas
  - Neuro: cranial nerves II to XII grossly intact. Motor and sensory without gross deficit.

- **Laboratory studies:** WBC 15.5 (5-14.5); Hgb 11.4 (11.5-13.5); platelets 18,000 (140K-440K). Differential: neutrophils 36%, lymphocytes 54%, monocytes 3%, eosinophils 2%, atypical 1%; ANC 5,590. PT 11.7 (10.3-13.3), INR 1, PTT 32.5 (25.3-35); uric acid 3.3 (2.5-8.5); CMP unremarkable; PO4 6.2 (3.8-6.7); Mg 1.9 (1.6-2.3); Coombs test negative

- **Imaging:** None indicated

What do you think?
Diagnosis: Peripheral blood smear revealed benign lymphocytosis and severe thrombocytopenia; negative for blasts/malignancy. Consistent with ITP.

Treatment: IVIG 1 gm/kg

(Note: While this child was treated with IVIG, an alternative option is use of steroids. WinRho is no longer indicated for treatment.)

Case Study 2

- HPI: 2-year old Hispanic female referred for evaluation due to abnormal newborn screen. Patient's newborn screen showed FAS. Hemoglobin electrophoresis completed at the age of 6 months and revealed Hgb AS (A 53.7%, S 40.2%, F 2%).
- PMH: No significant illnesses reported
- PSH: No prior surgeries
- Medications: None
- Allergies: NKDA
- Immunizations: Up to date
- Family history: Father and paternal grandfather with history of sickle cell trait
- Social history: Lives with parents and an older brother.

Case Study 2

- Physical exam:
  - General appearance: Alert and happy
  - Skin and Hair: Skin turgor and tone are normal, no petechiae and no lesions
  - Head and Face: Normocephalic, atraumatic, no sinus tenderness
  - ENT: External ear canals are clear, TM's translucent with normal light reflex; oral mucosa, hard and soft palate, tongue, mouth, posterior pharynx clear without lesions.
  - Lungs: normal respiratory effort, clear to auscultation bilaterally, no rhonchi, no rales, no wheezes and no accessory muscle use
  - Cardiac: PMI is not displaced, no thrills, no murmurs, normal S1 and S2
  - Abdomen: Soft, nontender, bowel sounds active in all 4 quadrants, no hepatosplenomegaly
  - Genitourinary: deferred
  - Musculoskeletal: FROM, no clubbing, no cyanosis, no edema
  - Neurological: Oriented to time, place, and person and CN II-XII grossly intact. Motor and sensory without gross deficit. Muscle strength and tone within normal limits, gait within normal limits for age.

Laboratory studies: Need to include copy of newborn screen; Hemoglobin electrophoresis: Hgb A1 53.7% (>92%), Hgb S 40.2% (0%), Hgb F 2% (<8%), Hgb A2 4.1% (<2.7%); Reticulocyte count 1.5% (0.5%-2.9%); CBC-WBC 7.4 (4.5-15.5), Hgb 13 (10.5-14.5), platelets 339K (150K-350K), ANC 3300.

- Imaging: None

What do you think?

Sickle Cell Trait

Treatment:
- Educate parents that sickle cell trait rarely causes medical problems. There are possible risks involved in areas of high and low altitude-instruct to seek medical attention medical attention if chest pain occurs in these environments. Precautions should be taken if patient chooses to participate in high-intensity sports as persons with sickle cell trait should seek immediate medical attention if chest pain or muscle weakness occurs during intense sports participation. NCAA websites are a good reference for the athlete with sickle cell trait. Important to emphasize that normal childhood play and sports are not a risk factor for persons with sickle cell trait. Advise to avoid dehydration.
- Recommendation for genetic counseling when age appropriate.
Case Study 3

- HPI: 12-year old Caucasian female referred Hematology/Oncology clinic after being diagnosed with iron deficiency anemia on routine testing 3 months earlier. Hgb was 11.1 g/dL and iron studies consistent with iron deficiency - iron saturation 3%, ferritin 3.8. Patient has not started menses. No bleeding symptoms. She eats a balanced diet; drinks 8 oz milk/day. Has been on iron supplementation with no improvement.
- PME: Previously healthy
- PSH: No prior surgeries
- Medications: None
- Allergies: NKDA
- Immunizations: Up to date
- Family history: No pertinent family history
- Social history: Attends middle school, in 7th grade. Reports making good grades.

Physical exam:
- General appearance: Alert and well nourished.
- Skin and Hair: Skin turgor and tone are normal, no petechiae and no ecchymoses
- Head and Face: Normocephalic, atraumatic, no sinus tenderness
- ENT: External ear canals are clear, TMs translucent with normal light reflex; oral mucosa, hard and soft palate, tongue, uvula, posterior oropharynx, no lesions.
- Eyes: conjunctiva are pink, sclerae nonicteric, bilateral PERRLA and ocular movement is equal
- Neck: supple, no jugular venous distension and thyroid normal without masses
- Lymphatic: no cervical, supraclavicular, or infraclavicular nodes palpable
- Lungs: normal respiratory effort, clear to auscultation bilaterally, no rhonchi, no rales, no wheezes and there is no accessory muscle use
- Cardiovascular: PMI is not displaced, no thrills, no murmurs, normal S1 and S2
- Abdomen: Soft, nontender, bowel sounds active in all 4 quadrants, no hepatosplenomegaly
- Genitourinary: deferred
- Musculoskeletal: FROM, no clubbing, no cyanosis, no edema
- Neurological: Oriented to time, place, and person and CN II-XII grossly intact. Motor and sensory without gross deficit. Muscle strength and tone within normal limits, gait within normal limits for age

Laboratory studies: CBC-WBC 9.5 (4.1-10.9), Hgb 10.2 (12-18), Hct 33.7 (37-51), platelets 444K (140K-440K), ANC 6000, MCV 70.6 (80-97), MCH 21.6 (26-32), MCHC 30.3 (31-36), RDW 16.3 (11.5-14.5). Stool negative for occult blood.

Imaging: None

What do you think?

- Iron Deficiency Anemia
- Treatment:
  - Start NovaFerum 150 mg PO daily
  - Start Senna 8.6 mg PO twice daily
  - Patient advised to take iron with orange/apple juice to aid with absorption.
  - Review dietary iron sources such as red meat, green leafy vegetables, legumes, pumpkin seeds, quinoa

Clinical Pearls
Case Study 4

HPI: "Mom states pt was at urgent care tonight and referred here for anemia and elevated WBC. Pt pale in color with dark circles under eyes."

This is a previously healthy 3 year old female presenting from ICC with anemia, thrombocytopenia, and elevated WBC. She presented to ICC the night before due to fever up to 103F, limping for the last 24 hours. Parents had also 2 episodes of M+R fever 34 for 24 hours the last 3 months. Patient was evaluated at the time, but extensive workup was negative and referred to be seen at ICC. On admission, patient had intermittent fever, limp, and refused to move her left leg. Mother reported that patient's urologist's office is "very busy," and the patient developed multiple bruises on her legs and arms.

PMH: Previously healthy. No prior hospital admissions.

PSH: None

Medications: None

Allergies: NKDA

Immunizations: UTD

Family history: Maternal grandmother with Factor V Leiden deficiency

Social history: Lives at home with parents and 2 brothers. Attends preschool 3 days a week.

Physical exam:
- Pain Assessment: denies pain
- General Appearance: Pale but alert and active, well-nourished, no acute distress.
- Skin: Normal turgor and tone; no petechiae, ecchymoses or lesions. No erythema, rash, sclerodermatous changes. Peripheral IV in right AC site clear, nontender.
- Head: Normocephalic; atraumatic; no sinus tenderness.
- Eyes: Conjunctivae pink; sclera nonicteric. EOM full; Pupils equal, round, reactive to light.
- ENT: Oropharynx, oral mucosa, gums, hard and soft palate, tongue, tonsils, posterior pharynx are clear and without lesions. Nasal mucosa, septum and turbinates grossly normal. External ear canals clear.
- Neck: Supple. No masses. Trachea midline. Thyroid normal, no JVD.
- Lymph: No preauricular, cervical, supraclavicular, axillary, inguinal, femoral, posterior popliteal or other nodes palpable.
- Lungs/chest: Normal respiratory effort with symmetric chest expansion. Clear to auscultation with normal breath sounds. No rhonchi, rales or wheezes.
- Heart/CV: PMI not displaced; tachycardic with regular rhythm; normal S1 and S2; no murmur or gallop or rub. Peripheral pulses 2+ and equal. Cap refill 2-3 seconds.
- Abdomen: Soft, nontender, non-distended; bowel sounds are normal. No liver, spleen, kidneys or masses palpable.
- Extremities: Full range of motion; no clubbing, cyanosis or edema. No signs of infection, inflammation or ischemia. No calf tenderness.
- Neurologic: Cranial nerves II to XII grossly intact. Motor and sensory without gross deficit.

Laboratory studies:
- WBC 66.5 (5-14.5), Hgb 6.2 (11.5-13.5), platelets 48,000 (140K-440K). Differential: neutrophils 0%; lymphocytes 10%, blasts 90%; ANC 0.
- ESR 76% (0-20); CRP 9.9 (<1); CMP unremarkable with the exception of AST of 63 (15-46); Mg 2.4 (1.6-2.3); PO4 6.3 (2.5-4.5); uric acid 7.2 (2.5-8.5); LDH 1403 (313-618); UA unremarkable; PT 18.7 (9.9-12.7), PTT/INR unremarkable; fibrinogen 610 (200-400).

Imaging: Chest radiograph WNL.

What do you think?
- Acute Lymphoblastic Leukemia
- Diagnosis: Peripheral flow: "IMMUNOPHENOTYPING REVEALS UPTO 94% B LYMPHOBLASTS."
- Bone marrow aspirate/biopsy-consistent with acute B-cell lymphoblastic leukemia
- CSF-negative for malignancy
- Treatment: CVL was placed surgically and treatment was initiated per COG AALL1131

Clinical pearls:
- Ferritin is an acute phase reactant, so it may be falsely elevated in a sick child
- Ferrous sulfate is the standard of treatment for IDA.
- However, NovoFerrum can be offered as an alternative as it tastes much better. It costs approximately $30.
- Often false TCP results are due to a poor sample (difficult stick leading to clumping) and specimen not processed timely. If patient is asymptomatic, should repeat CBC at a local hospital before referring.
Clinical pearls

- IDA should be treated for 3 months to replace iron stores/correct ferritin.
- Dosing is 5 mg/kg/day for pediatric patients over 1 year of age.
- Treatment should be initiated if patient has documented low Fe studies while waiting for referral.
- Recommend increasing dietary iron sources.
- Decrease milk to 16 oz/day for toddlers during iron replacement.
- Teenagers are noncompliant, so parents should supervise iron administration.

References


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