Case Study 2
Anemia in Pregnancy – Case 21

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Professor Fischer – FNES 365
Written by Kimberly Tierney

Title: Case 21 – Anemia in Pregnancy

Instructions: The case study must be purchased from the publisher (see syllabus) (2.99$). Answer the questions below. Type your answers under the respective question. Save the document as yournameyourfirstnamecase2 and submit under assignments: Case study Anemia in Pregnancy on Blackboard.

Questions:

1. Evaluate the patient’s admitting history and physical. Are there any signs or symptoms that support the diagnosis of anemia?

Signs and symptoms which support the diagnosis of anemia consist of poor dietary intake, irregular use of prenatal vitamins, close proximity between pregnancies, vaginal spotting, fatigue, increased respiratory effort and rate, pale skin color and sclera of the eyes, and diminished bilateral pulses.

Pregnancy demands higher requirements of nutrient intake to meet the 15% increase in metabolism caused by fetal growth, such as folate and iron, to supply both the mother and fetus; Long term poor dietary intake, such as omission of iron containing sources of protein and nutrient dense vegetables, compounded with inconsistent use of prenatal vitamins and consumption of polyphenol contained within coffee results in reduced iron stores within the body. Iron deficiency may result from poor diet, excess blood loss and malabsorption of iron; however, the patient’s actions support poor diet as a possible cause of inadequate iron concentrations. Extended durations of reduced iron within the body is a cause of iron-deficiency anemia.

Close proximity of pregnancy indicates that the patient’s blood stores may not have had time to recuperate from the blood loss associated with the previous birth, leading to a decreased status of iron and red blood cells at the time of conception. The decreased iron and red blood cell status will be further agitated by the increased demands of the current pregnancy whereby the maternal blood supply increases resulting in greater demands of iron, as an additional 700 to 800 mg, to accompany the 20% to 30% red blood cell volume. Vaginal spotting increases blood and iron losses, as well as indicates a risk of iron-deficiency anemia.

Fatigue supports the diagnosis of anemia whereby a decreased concentration of oxygen carrying hemoglobin. Oxygen is required by tissues in the production of energy, therefore a decreased availability of oxygen will result in a feeling of fatigue and exhaustion. Fatigue is the most common symptom associated with anemia. However, iron is also an active component of the protein hemoglobin, therefore a decreased presence of hemoglobin will also indicate a lower presence of iron associated with hypochromic iron-deficient anemia.

A dyspneic respiratory state of increased rate and effort supports a diagnosis of anemia due to a decreased concentration of hemoglobin as a component of red blood cells to carry the oxygen from the lungs to the tissues, thereby creating a higher demand upon oxygen to the body, fetus, and lungs. This symptom is further compounded by the increased requirements of oxygen associated with pregnancy to meet the added demands of the fetus and a decreased ability to excrete carbon dioxide as a waste product in exchange of oxygen. The decreased threshold for carbon dioxide and decreased concentration of oxygen, accompanied by the constant pressure placed against the diaphragm from the growing fetus coexisting with decreased hemoglobin will aggregate to difficulty breathing.

Pale skin pigment, as well as pale sclera pigmentation, indicates inadequate hemoglobin concentrations upon erythrocytes, possibly resulting from prior blood loss in previous birth as well as nutritional deficits. Hemoglobin is an iron-rich pigmented protein which imparts the red coloration or pigment to erythrocytes. Low numbers of red blood cells and decreased concentrations of hemoglobin upon red blood cells symptoms of anemia; thereby, if there is less pigmented hemoglobin present upon fewer numbers of red blood cells circulating, the epidermis and sclera will appear pale. Diminished bilateral pulses indicate a weakened ability to circulate oxygen-depleted blood associated with increased cardiac effort relating to lower blood concentrations.
Diminished bilateral pulses may also be attributed to the decrease in diastolic blood pressure caused by peripheral vasodilation experienced within the first and second trimesters of pregnancy. Also associated with the second trimester of pregnancy is pain in the abdomen, however this sign may indicate injury acquired during the patient’s fall or anemia affecting the liver and spleen.


2. What laboratory values or other tests support this diagnosis? List all abnormal values and explain the likely cause for each abnormal value.

The laboratory values which support the diagnosis of hypochromic, microcytic anemia are the abnormal hematology values including red blood cell (RBC) concentration, hemoglobin (Hgb) concentration, hematocrit (Hct), mean cell volume (MCV), reticulocyte count (Retic), mean cell hemoglobin (MCH), mean cell hemoglobin content (MCHC), percent red blood cell distribution, total iron binding capacity (TIBC), serum ferritin, zinc protoporphyrin (ZPP), and folate concentration.

Anemia is defined as a decreased number or size of red blood cells (RBCs) within the body. A RBC value of $3.8 \times 10^6$/mm$^3$ is below the normal range of $4.2 \times 10^6$/mm$^3$ to $5.4 \times 10^6$/mm$^3$ for females indicating a lower concentration in the quantity of red blood cells supporting a diagnosis of anemia. The decreased RBC value may indicate nutritional deficits, hemorrhage, hemolysis, renal disease, and marrow failure; however, it is not indicative of iron deficiency. A blood smear utilizing RBCs would further confirm the pale color or pigment supporting hypochromic anemia, and small cell size supporting microcytic anemia. Therefore, the cause of a low RBC value is likely poor dietary intake, possibly marrow failure, and is compounded by blood loss as hemorrhage via previous birth and vaginal spotting. An iron deficient diet and lower iron status at time of conception may also cause a low production of RBCs because pregnancy requires an additional 500 mg of iron to perform hematopoiesis. Erythropoiesis of the pregnant mother also requires an increased dietary folate intake of 600 mcg/day from 400 ng/dL of non-pregnant women. However, in the nutrient deficient diet of the patient, folate values were compromised to 2 ng/dL, a value well below the normal limits of 5 to 25 ng/dL.

Hemoglobin (Hgb) is the oxygen-carrying, iron-containing protein of red blood cells utilized in the exchange of oxygen and carbon dioxide gases on mature erythrocytes within the peripheral blood; hemoglobin is the pigmented portion imparting the red pigment color to red blood cells. A Hgb concentration from laboratory values is a direct measure of iron deficiency, as compared with Hct, whereby it measures the quantity of total Hgb per RBCs. A Hgb value of 9.1 g/dL is below the normal range of 12 to 15 g/dL for females, as well as below the level designated for pregnant females as greater than 11 g/dL. A decreased concentration of Hgb lowers the red colored pigment imparted on RBCs producing a pale or hypochromic appearance. In a deficient iron state, hemoglobin synthesis will be limited due to the absence of iron available to bind with heme-protein. A deficiency in circulating hemoglobin is a characterization associated with iron-deficient microcytic anemia. A decreased value of Hgb may indicate nutritional deficits, hemorrhage or blood loss, hemolysis or destruction of RBCs, marrow failure; all of which may be causes of anemia. Hgb is not an indicator of iron, cobalamin, or folate deficiency. A Hgb value may also be decreased in the presence of renal disease.

A hematocrit (Hct) is the measure of packed cell volume or percent of RBCs within the total blood volume. Hct is a less direct measure of iron status because its measure is less specific than Hgb which quantifies per RBC. Generally the Hct value is three times the Hgb value, as is the case with this patient with a Hgb value of 9.1 g/dL and a Hct value of 33%. The patient’s Hct value of 33% is below the normal limits for a female of 35 to 47 percent, however, it is equivalent to the 33% value stated for a pregnant female due to an increased total blood supply during pregnancy. A decreased value of RBCs within total blood is an indicator of anemia which is defined as a deficiency in size or number of RBCs. The decreased Hct value may indicate nutritional deficits, hemorrhage or blood loss, hemolysis or destruction of RBCs, marrow failure; all of which may be causes of anemia, but is not an indicator of iron, cobalamin, or folate deficits. A Hct value may also be decreased in the presence of renal disease.
Mean cell volume (MCV) is a measure of the average size of RBCs used to classify the type of anemia based on cell size as either microcytic, normocytic, or macrocytic. The normal limits of MCV range from 82 to 92 μm³, interpreted as normocytic, normochromic RBCs. A value below 80 μm³, as the patient’s value of 72 μm³, may be interpreted as hypochromic, microcytic RBCs indicating anemia due to iron deficits. Microcytic anemia is determined by smaller than normal size of mature RBCs or erythrocytes. Although MCV is not a sensitive test for nutrient deficiency, the iron deficits causing a decreased MCV value may be a result of poor dietary intake, excess blood loss, malabsorption of iron, or the increased iron demands presented with pregnancy. Iron deficits, accompanied by microcytic RBCs may indicate chronic renal failure and chronic disease.

A reticulocyte (Retic) count is a measurement of the rate at which bone marrow synthesizes immature RBCs, also called reticulocytes. Reticulocytes generally account for about 1% of total blood volume. If bone marrow production is compromised, the production and maturation of reticulocytes will be inhibited, therefore this test is sensitive to determine the cause of anemia as either a disturbance within bone marrow, excess blood loss, or hemolysis. Reticulocyte counts are also a means of determining if the anemia is caused by nutritional or non-nutritional aspects, meaning that if values of platelet, reticulocyte, and leukocyte counts are affected then the cause is likely non-nutritional based but rather a disturbance in bone marrow. Bone marrow is also the site of hemoglobin synthesis requiring iron-saturated transferrin. Hemorrhage may be ruled out utilizing reticulocyte count because blood loss stimulates elevated RBC production to compensate for losses and is indicated by an elevated value in reticulocyte count. The patient’s value is 0.2%, below the normal limits stated as 0.8 to 2.8 percents. A decreased reticulocyte count supports a diagnoses of deficiency in red blood cell number, or anemia. However, the patient has also recently experienced blood loss with the previous and very recent birth and current vaginal spotting thereby limiting the amount of time blood cell levels have to recuperate to within normal limits.

Mean cell hemoglobin (MCH), or mean corpuscular hemoglobin, determines the amount of hemoglobin per RBC. In iron deficient microcytic anemia the MCH value decreases due to smaller cell size and thereby decreased hemoglobin content per RBC and less circulating hemoglobin. MCH values may be interpreted within the same parameters of implications as MCV whereby it is not a sensitive test for nutrient deficiency, the iron deficits causing a decreased MCV value may be a result of poor dietary intake, excess blood loss, malabsorption of iron, or the increased iron demands presented with pregnancy. Iron deficits, accompanied by microcytic RBCs may indicate chronic renal failure and chronic disease. The patient’s MCH value displays a decreased hemoglobin amount per RBC as 23 pg, below the normal limits ranging from 26 to 32 pg. A decreased hemoglobin content per RBC will cause a pale appearance to cells supporting the hypochromic anemia diagnosis.

Mean cell hemoglobin content (MCHC) is a measure of the hemoglobin concentration per RBC, or hemoglobin content in relation to cell size. Due to the decrease experienced in pregnancy correlating to increased blood volume, a slight decrease is anticipated from a range of 7.4 to 9.9 mmol/L in non-pregnant females to a value greater than 6.8 mmol/L in pregnant females. However, the patient’s MCHC value is stated as 28 g/dL which is below the normal range of 31.5 to 36 g/dL in combination with decreased RBC numbers and size thereby supporting the diagnoses hypochromic microcytic anemia caused by iron deficiency. Although MCHC is not a sensitive test for nutrient deficits, a deficiency in iron will cause the MCHC value to decrease.

RBC distribution measures the variation in RBC size and volume, when an increase in size and volume variation occurs, as in microcytic anemia, the RBC distribution value increases. The patient’s RBC distribution value of 22% is elevated above the normal limits stated as 11.6 to 16.5% supporting the diagnosis of microcytic anemia.

Total iron-binding capacity (TIBC) is the measure of how transferrin saturates or takes in iron. TIBC measures all binding capacities of protein to iron by serum transferrin receptors. Plasma transferrin concentrations increase by releasing iron from storage, therefore in iron deficiency, TIBC increases in attempts to bind as much iron as possible to an increase availability of serum transferrin receptors but also decrease iron stores. TIBC not only increases in people experiencing iron deficiency, but also women whom are pregnant. However, TIBC decreases when iron stores are deficient to compensate plasma transferrin, as is the case in poor dietary intake, as well as decreasing in relation to protein energy malnutrition, liver disease, and hemolytic anemia. The normal limits of TIBC are stated as 240 to 450 μg/dL. The patient’s value of 465 μg/dL is above normal limits indicating a negative iron balance as depleted stores to compensate for low transferrin plasma levels. A value greater than 410 μg/dL is classified as a stage IV anemia with clinical damage producing hypochromic
microcytic red blood cells because both serum and storage levels of iron are low; this evidence supports the diagnosis of hypochromic microcytic iron-deficient anemia, and further rules out anemia due to hemorrhage.

Serum ferritin is the storage protein form of iron within the liver, spleen and bone marrow. Ferritin storage levels are maintained in equilibrium with circulating plasma iron, or transferrin. When transferrin levels are low, the storage ferritin releases iron to compensate, thus it is the most sensitive indicator of iron balance. Negative iron balance is classified by a value of less than 10 μg/dL in stage IV iron deficient anemia with clinical damage. The patient’s value of 10 μg/dL is exceptionally below the normal limits stated as 20 to 120 μg/dL for females, and may be classified as stage III iron deficiency anemia affecting erythropoiesis. RBC synthesis disruption within the bone marrow supports the diagnosis of hypochromic microcytic iron-deficient anemia.

Zinc protoporphyrin (ZPP) is utilized to assess iron deficiency because protoporphyrin is the component containing iron which proteins in hemoglobin synthesis providing the red pigment. The patient’s increased value of 84 μmol/mol, above the normal range of 30 to 80 μmol/mol, can be interpreted as hemoglobin molecules containing higher protoporphyrin per molecule of hemoglobin supporting a diagnosis of hypochromic, or pale cells, in relation to iron deficiency.

Transferrin saturation is a measure which encompasses all criteria assessed in iron-deficiency anemia, including ferritin, iron, and transferrin, because hemoglobin status alone may be affected by chronic disease, value variation between individuals, and inability to determine iron-deficiency anemia from other forms of anemia. A definitive measure of iron-deficiency is found in assessment of transferrin saturation. A transferrin saturation below 16% determines that there is poor binding of iron to transferrin then distributed to tissues thereby inhibiting erythropoiesis. The patient’s transferrin saturation may be determined by dividing the serum iron ferritin by the TIBC, then multiplying the result by 100 to obtain percentage. The patient’s transferrin saturation is determined to be 2.1% by dividing 10 μL/dL ferritin by 465 μL/dL TIBC, then multiplied by 100. The patient’s transferrin saturation percent is less than 15% which classifies her again in stage IV negative iron balance and clinical damage, also supporting the diagnosis of iron-deficient hypochromic, microcytic anemia.


3. Mrs. Morris’s physician ordered additional lab work when her admitting CBC revealed a low hemoglobin. Why is this a concern? Are there normal changes in hemoglobin associated with pregnancy? If so, what are they? What other hematological values, if any, normally change in pregnancy?

A low hemoglobin value is a concern for anemia possibly caused by inadequate dietary intake of iron, or deficient iron stores. Poor hemoglobin synthesis and circulation, which require iron, will diminish intrauterine oxygen supplied to the fetus thereby compromising the mother’s, as well as fetus’, respiration and energy status.

Normal changes in hemoglobin levels are anticipated with pregnancy and adjusted from non-pregnant females levels of 7.4 to 9.9 μmol/L to a value of greater than 6.8 μmol/L in pregnant females, therefore hemoglobin levels are assessed within the range of 12 to 16 g/dL for non-pregnant women and greater than 11 g/dL for pregnant females. The decrease in the level of hemoglobin is associated with the expansion of maternal total blood volume by 50% at full term, in which 25% of cardiac output is provides uteroplacental blood flow. Therefore, an increased intake of iron to synthesize higher volumes of hemoglobin is required to compensate for a larger volume of maternal blood. If hemoglobin production is disrupted and unable to fulfill greater requirements to compensate for greater blood volume, the hemoglobin levels will decrease.

Other hematological values which normally change during pregnancy in association with the 50% expansion of total maternal blood volume are decreases in not only hemoglobin, but also decreases in serum proteins affecting total iron binding capacity, RBC count and hematocrit, total iron binding capacity, and the water-soluble vitamin folate required for RBC synthesis. RBC or erythrocyte mass increases by 30 to 35%, however packed cell volume or hematocrit decreases from 35 to 37% in non-pregnant to 33% in pregnant women. Maternal plasma volume increases by 45 to 55%.
Generally, hematological values relating to iron decrease due to the increased demands of iron levels associated with pregnancy and compounded by insufficient pre-pregnancy iron stores. Ferritin levels should be assessed within the first trimester of pregnancy and monitored throughout trimesters to avoid anemia. Levels should be maintained above 20 μg/L of serum ferritin, 32% in hematocrit, and 10.9 g/dL of hemoglobin.


4. There are several classifications of anemia. Define each of the following: megaloblastic anemia, pernicious anemia, normocytic anemia, microcytic anemia, sickle cell anemia, and hemolytic anemia.

Anemia is classified by the deficiency affecting red blood cell size, number, and/or amount of hemoglobin. The cause of most anemias is nutrient deficits of nutrients required for normal erythropoiesis, such as cobalamin, folate (folic acid), and iron. Both megaloblastic and pernicious anemias are macrocytic, meaning enlarged size of red blood cells. Megaloblastic anemia is a result of folate deficiency in which the blood contains red blood cells of abnormally large size and enlarged nucleus. Pernicious anemia is an autoimmune disease in which vitamin B12, cobalamin, is unable to be absorbed due to an absence of intrinsic factor secretion and achlorhydria; it is a chronic, macrocytic anemia with B12 deficiency. Normocytic anemia is a normal sized red blood cell with normal hemoglobin content. Microcytic anemia is a smaller than normal sized blood cell, as well as less circulating hemoglobin; Microcytic anemia occurs in iron-deficiency and thalassemia as a hemolytic disease caused by faulty globin synthesis. Normocytic and microcytic anemias may be caused by excess blood loss, as chronic or acute hemorrhage. Sickle cell anemia is an inherited disorder which has an abnormality of globin genes in hemoglobin caused by a recessive chromosome trait. The mutated hemoglobin causes a crescent- (or sickle-) shaped red blood cells leading to increased hemolysis. In hemolytic anemia, red blood cell size may be normal but the anemia is the result of hemolysis, or red blood cell destruction, caused by drugs, infections, antibodies, and toxins.


5. What is the role of iron in the body? Are there additional functions of iron during fetal development?

Iron is an integral participant in oxidation-reduction reactions within the body in which ferrous iron (Fe2+) is changed to ferric iron (Fe3+), then back to ferrous. The conversion of iron back and forth between isotopes within redox reactions created free radicals as a byproduct which may be damaging to DNA and cell membranes, therefore iron doesn’t often travel in free form, but rather bound by transport, functional and storage proteins.

Functional iron proteins include hemoglobin, myoglobin, and iron-containing enzymes. Iron transport proteins include transferrin and ferroportin, and iron storage proteins include ferritin and hemosiderin.

Iron is the component of hemoglobin circulating within the blood which pick up oxygen from the lungs for transport via mature red blood cells, or erythrocytes, and exchange the oxygen for carbon dioxide to then excrete as waste via lung expiration. When iron is inadequate upon hemoglobin, the oxygen capacity declines
and a hormonal signal is sent to the bone marrow to increase erythropoiesis, also increasing the demands for iron necessary in the synthesis of hemoglobin.

Within myocytes, iron is the oxygen-carrying component of the functional protein myoglobin which transports oxygen from erythrocytes to cardiac and skeletal myocytes. Therefore, the feeling of fatigue results from a deficit in oxygen supply which muscled metabolized for energy due to a deficit in iron-containing proteins.

Iron containing enzymes function in many metabolic cycles, including energy production via the citric acid cycle which necessitates the iron-containing enzyme aconitase in the conversion of citrate to isocitrate, and the electron transport chain, which utilizes the iron-containing enzymes cytochrome C, cytochrome oxidase, iron-sulfur enzymes succinate-ubiquinone oxidoreductase and NADH-ubiquinone oxidoreductase. The enzymes cytochrome C, aconitase, cytochrome oxidase, iron-sulfur enzymes succinate-ubiquinone oxidoreductase and NADH-ubiquinone oxidoreductase are sensitive to iron deficiency. The metabolism of alcohol and drugs within the liver occurs with the iron-containing enzyme P-450, and the detoxification of hydrogen peroxide, an organic compound is facilitated by the iron-containing enzyme catalase.

Iron functions in the development of cognitive brain function as a cofactor of neurotransmitter synthesis, and in the prevention of infection by aiding the immune system in synthesis of lymphocytes, thereby a deficit in iron compromises the immune system and increases the likelihood of infection.

As a function of fetal development, iron functions in the development of the cognitive brain, this correlates with long-term iron deficiency and decreased oxygen-carrying hemoglobin resulting in behavioral and developmental deficits, such as motor function. The maternal iron status affects the birth weight, pregnancy term duration, as well as bone formation, cell membrane integrity, and DNA synthesis. 


6. Several stages of iron deficiency actually precede iron-deficiency anemia. Discuss these stages—including the symptoms—and identify the laboratory values that would be affected during each stage.

The stages leading to iron-deficiency anemia begin with marginal deficiency or early iron depletion, then moderate depletion of iron stores or moderate deficiency, followed by severe depletion or deficiency of iron stores, and lastly iron deficiency anemia. The initial stage of early or marginal iron deficiency includes poor or decreased iron intake, increased iron losses, often associated with menses in women, decreased iron stores affecting the lab value of serum ferritin, increased transferrin receptors identifiable in the lab values of TIBC, transferrin saturation in ratio of serum ferritin to TIB, but no visible symptoms. In the moderate iron deficiency stage, iron stores are depleted as evident in the lab value of serum ferritin, transferrin saturation, TIBC as storage is released to concentrate serum transferrin, energy levels are negatively affected and observable in poor muscle function, immune function is compromised, and transport iron decreases as identifiable in lab values as serum transferrin and TIBC. In severe iron deficiency, hemoglobin synthesis and erythropoiesis are negatively affect as identifiable in lab values as affecting RBC count, Hct, Hgb, MCV, Retic, MCH, RBC distribution, and ZPP, as well as previous stage lab indicators; the transporting of oxygen is compromised and clinically evident in increased respiratory effort, rates, and dyspneic state, fatigue is observable as well as pale complexion due to decreased hemoglobin circulation and epithelial tissue defect, immunity is compromised allowing infection visible in white blood cell lab values, and late clinical findings include cardiac failure and inflammation of the gastrointestinal tract.

7. What potential risk factor(s) for the development of iron-deficiency anemia can you identify from Mrs. Morris’s history?

The potential risk factors for the development of iron-deficiency anemia identifiable in Mrs. Morris’s history include an inadequate intake of dietary iron in both heme and non-heme forms compounded with the increased demands for iron to compensate for expansion of blood volume associated with pregnancy, a near absence of ascorbic acid (vitamin C) to increase dietary absorption of iron, inclusion of polyphenol containing tea and coffee which decrease the absorption of iron, poor dietary sources of protein, excluding the steak protein and heme-iron source, poor sources of dietary folate, smoking cigarettes which decrease gastrointestinal absorption and produce free radicals, inconsistency in prenatal vitamin usage, and close proximity of pregnancies which increase the demands of iron over a short period of time as well as increased losses of iron due to blood loss associated with the cesarean surgery of the previous pregnancy, limit time to recuperate iron stores, and lead to a low pregravid iron status.

8. What is the relationship between the health of the fetus and maternal iron status? Is there a risk for the infant if anemia continues?

Deficient maternal iron status may have detrimental effects on the cognitive brain development of a fetus as iron is required in the synthesis of the myelin sheath. Deficient maternal iron status may also result in low birth weights, preterm delivery, and in severe cases increasing chances of mortality. Increased risk of fetal anemia is still under study; however, infant mortality is more likely if the infant is born with a low iron storage status. The greatest occurrence of maternal-intrauterine iron transfer as carried by transferrin via placental entry and bound by ferritin, occurs late in pregnancy term around week 30 of pregnancy when the greater maternal blood volume expansion also occurs. If serum ferritin levels are in deficit, limited iron will transfer to the fetus and the infant will be born with increased chances of becoming anemic due to low iron status.


10. What are best dietary sources of iron? Describe the differences between heme and nonheme iron.

The best dietary sources of iron originate from meat sources for many reasons; the meat-fish-protein factor associated with amino acid content, the chelation of iron in accompaniment of metals for greater bioavailability from a more absorbable heme-iron source, and the source of heme-iron in animal flesh as containing hemoglobin and myoglobin has a greater bioavailability which is better for absorption and generally contains greater iron concentrations than non-heme plant-based sources. The richest sources of iron among US dietary consumption originate in animal flesh as heme-iron to include meats, as beef, pork and poultry, and seafood. The sources with greatest content include beef spleen, liver from both pork and beef, boiled lentils, kidney beans, beef, and spinach; however, the sources with the highest bioavailability and therefore absorption are highest in meat, then fish and poultry, and lowest bioavailability from liver, eggs, milk and cereals.

The difference between the dietary forms of iron as heme or nonheme iron is that heme iron has a higher bioavailability than non-heme iron because vegetables, grains, and some fruits contain
other components which bind to the non-heme iron decreasing its absorption capacity. Components which decrease the bioavailability and absorption of non-heme iron include polyphenols, like tannin in tea, and coffee, as well as oxalic acid in green leafy vegetable like spinach, and phytate in legumes and whole grains. However, consuming heme iron containing the MFP factor in conjunction with non-heme iron, as in eating vegetables with a meat source, will increase the absorption of non-heme iron.


11. Explain the digestion and absorption of dietary iron.

Absorption of bioavailable iron varies by individual in relation to amount of iron in storage. If an individual has adequate storage, 14-18% of dietary iron will be absorbed. If an individual has low storage amounts, requirements of greater absorption (35-40%) will be met. If iron storage is high or saturated, absorption will be 5%. Iron has higher bioavailability in the form of heme iron from animal-based source versus non-heme iron from plant-based source, and better absorption as ferrous iron (Fe^2+) versus ferric iron (Fe^3+) respectively. Since the excretion of iron via bile of fecal voids, 90% of the daily iron is recovered by the liver and spleen from RBCs about to expire and thereby iron is recycled within the body.

Iron digestion begins in the stomach with the separation of iron from its source in the presence chyme in an acidic environment with a gastric pH of 7 to which the ferric and ferrous forms of iron may enter the intestinal tract for absorption of mucosal iron facilitated by a carrier mediated mechanism across the brush border of the small intestine into enterocytes. If the form of iron is non-heme iron, it must first be reduced from ferric iron (Fe^3+) to ferrous iron (Fe^2+) for transport across the brush border microvilli into enterocytes simultaneously with heme; however, if the form is heme iron, then it is readily transported without conversion. Of the absorbed iron within the intestinal cell, some will be stored with ferritin and possibly lost to excretion during enterocyte sloughing, or it will again be transported across a second membrane, by ferroportin, called the basolateral membrane in ferrous form into the interstitial fluid. This second transport occurs in a coupled reaction with oxidation of the ferrous form back to the ferric iron form. The ferric iron within the serum then binds with transferrin for distribution to the iron storage locations of the liver, muscle, bone marrow, as well as the remainder to tissues dependent upon iron status.


**Calculations of patient’s BMI:**

Pre-pregnancy weight = 135 lbs
Height = 5 ft., 5 in. = 65 in.

\[
\text{BMI} = \frac{\text{weight (lb)}}{\text{height (in}^2)} \times 703
\]

\[
\text{BMI} = \frac{135 \text{ lb}}{(35 \text{ in}^2)} \times 703
\]
BMI = 22.53 % = normal healthy weight between 18.5 and 24.9%

Calculations of patient’s UBW:

ABW as actual pregnancy weight at 23rd week = 142 lbs

Usual body weight gain for pregnancy in 23rd week for a healthy pregravid BMI = 11 to 16 lbs

Therefore UBW = pre-pregnancy weight + 11 to 16 lbs

UBW = 135 lbs + 11 lbs = 146 lbs
UBW = 135 lbs + 16 lbs = 151 lbs

UBW range = 146 to 151 lbs

% UBW = (ABW/UBW) x 100
% UBW = (142 lb / 146 lb) x 100
% UBW = 97.2 % = 2.8 % below optimal pregnancy weight gain

% UBW = (ABW/UBW) x 100
% UBW = (142 lb / 151 lb) x 100
% UBW = 94.0 % = 6.0 % below optimal pregnancy weight gain

Therefore, the patient’s actual pregnancy weight of 142 lbs at the 23rd week of pregnancy is 2.8% to 6.0% below the UBW of weight gain she should achieve for optimal maternal and fetal health.


13. Check Mrs. Morris’s prepregnancy weight. Plot her weight gain on the maternal weight gain curve. Is her weight gain adequate? How does her weight gain compare to the current recommendations? Was the weight gain from her previous pregnancies WNL?

The patient, Mrs. Morris’s pre-pregnancy weight of 135 pounds calculated to a normal, healthy weight for her height interpreted as her BMI of 22.53% falling within the healthy BMI range of 18.5 to 24.9%. Her current pregnancy weight of 142 pounds as of the 23rd week of pregnancy is calculated to determine a weight gain of 7 pounds. The maternal weight gain curve shows that for a healthy pregravid body weight, a gain of 11 to 16 pounds is recommended for optimal pregnancy health for both the mother and fetus. The patient’s weight gain of 7 pounds at the 23rd week of pregnancy is 2.8% to 6.0% below the recommended UBW range of weight gain she should achieve for optimal maternal and fetal health. A total gestational weight gain of 25 to 35 pounds at the end of pregnancy term can still be achieved if dietary intake is increased and monitored to achieve a favorable maternal-fetal outcome.

As per the maternal weight gain curve, the weight gain from her previous pregnancies as a woman with healthy pregravid weight, as 15 pounds within 38 week pregnancy term and 20 pounds within 37 week pregnancy term, was suboptimal for the maternal-fetal health. Her weight gain should have been within the range of 23 to 33 pounds for pregnancies carried to terms of 37 or 38 weeks.
14. Determine Mrs. Morris’s energy and protein requirements. Explain the rationale for the method you used to calculate these requirements.

The rationale for the method used occurs respectively in the following steps. The estimated energy requirements are calculated for a healthy non-pregnant female and adjusted to account for a sedentary to mildly active physical activity level due to the patient’s responsibilities as a stay-at-home mother of two young children, as well as fatigue. The daily energy requirement is then adjusted to account for additional energy requirements associated with increased metabolic demands of pregnancy and fetus during the second trimester at 23 weeks to reflect the requirements to patient should be currently meeting with dietary intake. The protein requirements are calculated to fit the current demands at the current pregnancy weight.

Step 1: Estimated Energy Requirements (EER) utilizing Mifflin-St. Jeor Equations for healthy adult females:

Mifflin-St. Jeor Equation: kcal/day = 10 (wt) + 6.25 (ht) – 5 (age) – 161

Weight = actual body weight in kilograms
Height = centimeters
Age = years

Patient’s values:
Weight = 135 lbs / 2.2 kg = 61.36 kg pre-pregnancy
Height = 65 inches = 165.1 centimeters
Age = 31 years

Kcal/day = 10 (61.36 kg) + 6.25 (165.1 cm) – 5 (31) – 161

\[ \text{Kcal/day} = 1329.48 \text{ (EER of Mifflin-St. Jeor)} \]

Step 2: Factoring in a physical activity level (PAL) in the sedentary category, to account for the patient’s fatigue as well as energy requirements in responsibilities a stay-at-home mother of two young children, at a value of 1.4:

\[ \text{Patient’s EER} = \text{(EER of Mifflin-St. Jeor Equation) x (1.4 PAL value)} \]

\[ \text{Patient’s EER} = 1329.48 \text{ kcal/day x 1.4 PAL} = 1861.27 \text{ kcal/day} \]

Step 3: The estimated energy expenditure as a prediction of the PAL utilizing the Mifflin-St. Jeor equation EER for pregnant women is determined utilizing the following equation for adult women in the second trimester to account for additional energy required to support increased metabolic demands of pregnancy and fetal growth:

Second trimester = Adult EER + 160 kcal (8 kcal/wk x 20 wk) + 180 kcal

However, to account for the patients calories of 8 kcal/wk in the 23 week of pregnancy equaling 184 kcal, the equation is adjusted as follows:

Second trimester = Adult EER + 184 kcal (from 8 kcal/wk x 23 wk) + 180 kcal

Second trimester = 1861.27 kcal/day + 184 kcal/day + 180 kcal

\[ \text{Second trimester} = 2225.27 \text{ kcal/day requirement} \]
Step 4: Estimating Protein Requirements

The requirements of protein within the second half of pregnancy, as the patient is currently, increases from the 0.8 g/kg/day RDA of protein to 1.1 g/kg/day of pre-pregnancy weight, or an approximate 71 g/day.

At the actual current pregnancy weight of 142 pounds in the 23rd week of term:

\[
\text{Actual weight} = 142 \text{ lbs} = 64.5 \text{ kg}
\]

**Estimated protein requirements** = (1.1 g/day) \times (64.5 kg) = **70.95 g/day for the patient**


15. Using her 24-hour recall, compare her dietary intake to the energy and protein requirements that you calculated in Question 14.

Energy requirements: 2225.27 kcal/day

Protein requirements: 70.95 g/day

Patient’s 24-hour recall:

<table>
<thead>
<tr>
<th>Meal</th>
<th>Food Item/Beverage</th>
<th>Quantity</th>
<th>Kcal</th>
<th>Protein (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM</td>
<td>Frosted Flakes</td>
<td>2 c</td>
<td>301</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Whole Milk</td>
<td>½ c</td>
<td>74</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Black Coffee</td>
<td>1 c</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Lunch</td>
<td>Hot Dog on Bun</td>
<td>1 each</td>
<td>250</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Macaroni and Cheese</td>
<td>½ c</td>
<td>193</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Iced Tea (instant, sweetened)</td>
<td>1 c</td>
<td>47</td>
<td>0</td>
</tr>
<tr>
<td>Dinner</td>
<td>Salisbury Steak</td>
<td>3 oz</td>
<td>202</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>Green Beans</td>
<td>1 c</td>
<td>38</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Mashed Potatoes with Gravy</td>
<td>1 c</td>
<td>176</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Roll with Butter</td>
<td>1 each</td>
<td>100</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Iced Tea (instant, sweetened)</td>
<td>1 c</td>
<td>47</td>
<td>0</td>
</tr>
</tbody>
</table>

**Total Energy: 1432 kcal**  **Total Protein: 59 g**

According to the patient’s 24-hour recall, the total energy intake was 1432 kcal and the total protein intake was 59 g. The recommended requirements pertaining to the patient are an energy intake of 2225.27 kcal/day and protein intake of 70.95 g/day. Mrs. Morris’s dietary content did not achieve the adequate status of either recommendation. She achieved only 64% of her energy requirements and 83% of her protein requirements; these values may be interpreted as placing both the maternal and fetal health at risk.

A deficit in energy intake in macronutrient content will decrease the absorption of vitamins and minerals, as well as affect the fetus more so than the pregnant mother. A consequence of energy restriction in calorie intake affects pregnant women faster than non-pregnant women in relation to producing blood ketone-bodies at a greater rate and possibly leading to ketonuria.
16. Again using her 24-hour recall, assess the patient’s daily iron intake. How does it compare to the recommendations for this patient (which you provided in question #9)?

Estimate Average Requirement (EAR) of Iron: 22 mg/d

<table>
<thead>
<tr>
<th>Meal</th>
<th>Food Item/Beverage</th>
<th>Quantity</th>
<th>Iron (mg)</th>
<th>Iron Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM</td>
<td>Frosted Flakes</td>
<td>2 c</td>
<td>19</td>
<td>Non-heme</td>
</tr>
<tr>
<td></td>
<td>Whole Milk</td>
<td>½ c</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Black Coffee</td>
<td>1 c</td>
<td>0</td>
<td>Polyphenol decreases absorption</td>
</tr>
<tr>
<td>Lunch</td>
<td>Hot Dog on Bun</td>
<td>1 each</td>
<td>2</td>
<td>Heme</td>
</tr>
<tr>
<td></td>
<td>Macaroni and Cheese</td>
<td>½ c</td>
<td>1</td>
<td>Non-heme</td>
</tr>
<tr>
<td></td>
<td>Iced Tea (instant, sweetened)</td>
<td>1 c</td>
<td>0</td>
<td>Polyphenol may decrease absorption</td>
</tr>
<tr>
<td>Dinner</td>
<td>Salisbury Steak</td>
<td>3 oz</td>
<td>2</td>
<td>Heme</td>
</tr>
<tr>
<td></td>
<td>Green Beans</td>
<td>1 c</td>
<td>1</td>
<td>Non-heme</td>
</tr>
<tr>
<td></td>
<td>Mashed Potatoes with Gravy</td>
<td>1 c</td>
<td>1</td>
<td>Non-heme</td>
</tr>
<tr>
<td></td>
<td>Roll with Butter</td>
<td>1 each</td>
<td>1</td>
<td>Non-heme</td>
</tr>
<tr>
<td></td>
<td>Iced Tea (instant, sweetened)</td>
<td>1 c</td>
<td>0</td>
<td>Polyphenol may decrease absorption</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Total Iron: 27 mg</td>
</tr>
</tbody>
</table>

Although the demands for iron increase during pregnancy to compensate for a 50% expansion in maternal blood volume, as well as increasing the risk for iron-deficiency anemia, the body also compensates by increasing iron absorption during pregnancy by up to 3 times the normal absorption rate of iron. The average 2000 kcal/day diet consumed by adult females is averaged to provide 12 mg/day of iron. The estimated average requirement for an adult pregnant female of 31 years of age is 22 mg/day of iron which the patient has achieved and surpassed.

However, throughout the full term of the pregnancy, maternal iron intake must include an additional 700 to 800 mg of iron; the patient may have achieved recommended iron intake the day of the 24-hour recall, but may not be maintaining iron intake throughout full term. Also compounding an increased dietary requirement is the status of iron stores pregravid in which the patient recently birthed a child resulting in a depletion of iron stores via compensation for blood loss. Her iron stores and total iron status may not have had adequate time to recuperate from a lowered or deficient state prior to conception.


17. Identify the pertinent nutrition problems and the corresponding nutrition diagnoses.

Pertinent nutrition problems are emphasized further by increased demands required for pregnancy as iron deficiency manifesting with hypochromic microcytic anemia, diminished appetite and caloric energy and protein intake deficits leading to decreased energy intake, malnutrition, deficits in minerals and vitamins.

Diagnostics as:

**Energy Balance**
- NI – 1.4: Inadequate energy intakes
- NI – 1.6: Predicted suboptimal energy intake
- NI – 2.1: Inadequate oral intake; Fluid intake and prenatal vitamin supplementation
- NI – 2.9: Limited food acceptance
- NI – 3.1: Inadequate fluid intake
- NI – 4.1: Inadequate bioactive substance intake; Prenatal vitamin inconsistency
- NI – 4.2: Excessive bioactive substance intake; 2 cigarettes per day
- NI – 5.1: Increased nutrient needs required in pregnancy
- NI – 5.2: Malnutrition
- NI – 5.3: Inadequate protein-energy intake
- NI – 5.6.1: Inadequate fat intake
- NI – 5.7.1: Inadequate protein intake
- NI – 5.8.5: Inadequate fiber intake

**Vitamins**
- NI–5.9.1: Inadequate vitamin intake of A, C, D, E, K, Thiamin, Riboflavin, Niacin, Folate, B6, B12; inconsistent prenatal vitamin consumption

**Mineral**
- NI–5.10.1: Inadequate mineral intake of calcium, iron, magnesium, potassium, phosphorous, zinc

**Multinutrient**
- NI-5.11.1: Predicted suboptimal nutrient intake

**Functional**
- NC-1.4: Altered GI function; absence of fecal voids, possible constipation, possible malabsorption

**Biochemical**
- NC – 2.1: Impaired nutrient utilization
- NC – 2.2: Altered nutrition related laboratory values relating to iron

**Weight**
- NC-3.1: Underweight; at current weight in pregnancy

**Knowledge and Beliefs**
- NB - 1.1: Food- and nutrition-related knowledge deficit
- NB – 1.3: Not ready for diet/lifestyle change
- NB - 1.4: Self monitoring deficit
- NB - 1.5: Disordered eating pattern
- NB – 1.6: Limited adherence to nutrition-related recommendations; prenatal vitamin inconsistency
- NB – 1.7: Undesirable food choices
Physical Activity and Function
- NB - 2.1: Physical inactivity
- NB - 2.3: Inability or lack of desire to manage self-care
- NB – 2.4: Impaired ability to prepare foods/meals
- NB - 2.5: Poor nutrition quality of life

Food Safety and Access
- NB – 3.2: Limited access to food or water; financial restrictions
- NB – 3.3: Limited access to nutrition-related supplies; financial restrictions


18. Write a PES statement for each nutrition problem.

Iron deficiency manifesting with hypochromic microcytic anemia related to altered nutrition related laboratory values pertaining to iron status, inadequate mineral intakes of iron, inadequate energy intakes, limited food acceptance, predicted suboptimal nutrient intake, disordered eating pattern, undesirable food choices, and poor nutrition quality of life as evidence by fatigue, pale skin pigment and ocular sclera, poor dietary intake, irregular use of prenatal vitamins, close proximity between pregnancy as well as recent cesarean surgery, vaginal spotting, increased respiratory effort and rate as dyspneic state, and diminished bilateral pulses (NI – 1.4, 2.9, 5.10.1, 5.11.1, NC – 2.2, NB – 1.5, 1.6, 1.7, 2.5).

Inadequate energy intake related to predicted suboptimal and inadequate energy intakes, limited food acceptance, increased nutrient needs required in pregnancy, inadequate protein-energy intake, underweight at current ABW in pregnancy term, predicted suboptimal nutrient intake, self monitoring deficit, disordered eating pattern, limited adherence to nutrition-related recommendations as inconsistent prenatal vitamin use, undesirable food choices, impaired ability to prepare foods, and poor nutrition quality of life as evidenced by inadequate energy intake as per usual intake recall and 24-hour recall, picky eating, poor dietary sources, inadequate pregnancy weight, irregular consumption of prenatal vitamins needed for metabolism, and fatigue. (NI – 1.4, 1.6, 2.9, 5.1, 5.3, 5.11.1, NC – 3.1, NB – 1.4, 1.5, 1.6, 1.7, 2.4, 2.5)


19. Mrs. Morris was discharged on 40 mg of ferrous sulfate three times daily. Are there potential side effects from this medication? Are there any drug–nutrient interactions? What instructions might you give her to maximize the benefit of her iron supplementation?

The ferrous form of a supplement is already in the more bioavailable and absorbable form of iron as ferrous in comparison to ferric, and chelated compared to bound iron, as well as in oral form unless malabsorption disorders exist. However, maximizing absorption can be achieved if taken in conjunction with dietary sources of ascorbic acid (vitamin C) and meat sources containing amino acids with the MFP factor. Conversely, the iron will be best absorbed without the addition of food, when the stomach is empty, but unbound ferrous iron may lead to gastric upset with negative side effects including nausea, bloating, diarrhea, constipation, a darkening color of stool, and increased stomach acid as heartburn. Gastric discomfort can be reduced by taking the oral medication with a meal. Despite chelated ferrous oral supplementation’s high absorption capacity, components
which normally reduce the absorption of iron should be avoided or limited while continuing supplementation, such as food items containing phytate, oxalic acid and polyphenols, as well as phosphate and calcium.

Interactions between iron supplementation with zinc and calcium may occur, and may be more likely if zinc and calcium are also in supplementation form due to competition between the same absorption pathways in ionic form. There is concern that zinc and calcium may inhibit the absorption of iron during competition.


21. List factors that you would monitor to assess her pregnancy, nutritional, and iron status.

Factors monitored to assess the patient’s pregnancy status will be achieved in the monitored factors of nutritional and iron status and restoration of overall maternal-fetal health. Factors monitored to assess the patient’s nutritional and iron status are total energy intake, beverage intake as oral fluids, food intake as amount of food, types of food, meal pattern, diet quality index, food variety, bioactive substance intake relating to nicotine use, macronutrient intake as total protein intake and total carbohydrate intake, micronutrient intake as prenatal multivitamin consistency specific to folate, B6, B12, and vitamins A, C, D, and K, and mineral intake as calcium, iron, and zinc, adherence to diet orders as general, healthful diet and diet/nutrition education, correction in misuse of prenatal vitamin, increased level of knowledge pertaining to iron-deficiency hypochromic, microcytic anemia, adjustment in behavior as self-management at agreed upon specifics, patient fatigue during feeding process resulting in inadequate intake, physical activity as duration, intensity, and strength, nutrition-related patient-centered measure as nutrition quality of life responses, anthropometric measurements as weight, weight change, pregnancy growth pattern, pregnancy compartment estimates, biochemical data and medical tests as gastrointestinal profile to include gastric emptying time, small bowel transit time, as metabolic rate profile to include resting metabolic rate, all inclusive nutritional anemia profile and vitamin profile, nutrition-focused physical findings as overall appearance in weight change, skin pigment, digestive system stool production and color, and most closely monitored factors to assess nutritional status are comparative standards as energy needs and intake, macronutrient needs and intakes, fluid needs and intakes, micronutrient needs and intake, as well as adequate adjustments in desired pregnancy growth pattern. (FH - 1.1.1.1, 1.2.1.1, 1.2.2.1, 1.2.2.2, 1.2.2.3, 1.2.2.4, 1.2.2.5, 1.4.2.5, 1.5.2.1, 1.5.3.1, 1.6.1.12, 1.6.2.1, 1.6.2.3, 1.6.2.8, 2.1.1.1, 2.1.2.2, 3.1.3, 4.1.1, 4.1.2, 5.1.5, 5.4.7, 7.3.4, 7.3.5, 7.3.7, 8.1.1, AD – 1.1.2, 1.1.4, 1.1.6, 1.1.7, BD – 1.4.20, 1.4.21, 1.8.1, 1.10, 1.13, PD – 1.1.1, 1.1.5, 1.1.8, CS – 1.1.1, 2.1.1, 2.2.1, 2.3.1, 2.4.1, 3.1.1, 4.1, 4.2, 5.1.3)

## A - Assessment

### S - Subjective

**Chief Complaint:** Experienced vaginal spotting and some abdominal pain following fall on ice; Presented to ER in 23rd week of gestation; R/O premature labor secondary to fall

<table>
<thead>
<tr>
<th>UBW: pregravid: 135#</th>
<th>Nutritional supplement:</th>
</tr>
</thead>
<tbody>
<tr>
<td>recommended UBW for 23 wk gestation: 146-151#</td>
<td><strong>Vitamins</strong> / herbs: prenatal; inconsistent use</td>
</tr>
<tr>
<td>Weight change: <strong>gain</strong> / loss: 7# in 23 weeks</td>
<td>Food preparation: prepared by patient; prepares for 2 young children &amp; husband</td>
</tr>
<tr>
<td>Appetite: current: good appetite, picky eater</td>
<td>Factors affecting food intake: fatigue, picky eater, previous trimester +/- N/V from morning sickness, financial restrictions</td>
</tr>
<tr>
<td>previous trimester: decreased/poor appetite,</td>
<td>Social / cultural / religious / financial – financial restriction, previously on WIC, responsible for 2 children &amp; husband, current occupation: stay-at-home mother</td>
</tr>
<tr>
<td>Chewing / swallowing problem / sore mouth – no difficulty</td>
<td>Other: nicotine use: 1-2 cigarettes/day</td>
</tr>
<tr>
<td>Nausea / vomiting / diarrhea / <strong>constipation</strong> – possible constipation, absence of fecal voids, previous trimester: +/- N/V</td>
<td></td>
</tr>
<tr>
<td>Food intolerance / allergies: none, picky eater</td>
<td></td>
</tr>
<tr>
<td>Diet prior to admit: no restrictions/ normal diet; not fulfilling increased requirements of pregnancy, poor dietary sources, deficit in calorie intake per day</td>
<td></td>
</tr>
</tbody>
</table>

### O - Objective

**Current Diet Order:** NPO upon admit, no restrictions prior to admit

- Upon D/C from hospital: increase dietary sources of iron & folate, dec interacting components; polyphenols, oxalic acid, phytate, minimize interaction of iron with zinc & calcium

**Medical Diagnosis:** Hypochromic, microcytic anemia

- R/O: premature labor secondary to fall

**Past Medical History:**

- Sx: s/p appendectomy at 12 yo, cesarean section 18 months previously

**Nutrition Focused Physical Signs & Symptoms:** normal RR: 19 bpm, inc RE: dyspneic, diminished bilateral pulse: 88 bpm, Skin: pale, Eyes: pale sclera, Urinary output exceeding fluid input: 67.3 mL/kg, poor dietary sources & calorie intake, inconsistent use of prenatal vitamin supplementation, close proximity between pregnancy & recent cesarean section Sx, vaginal spotting, fatigue, abdominal pain

### Nutritionally Relevant Laboratory Data:

**Hematology:**

- RBCs: dec RBC count: 3.8 x 10^6/mm³, dec Hct: 33%, dec Retic: 0.2%, inc RBC distribution: 22%, dec Folate: 2 ng/dL, dec MCV: 72 µm³,
- Hgb: dec Hgb: 9.1 g/dL, dec MCH: 23 pg, dec MCHC: 28 g/dL
- Iron: inc TIBC: 465 µg/dL, dec Ferritin: 10 µg/dL, inc ZPP: 84 µmol/mol, dec transferrin saturation: 2.1%

**Chemistry:** low Lipase: 5 U/L, low Amylase: 26 U/L

**Drug Nutrient Interaction:** Prior to admit & upon D/C from hospital: prenatal vitamin, possible interactions with nicotine, dietary sources of zinc and calcium

- In hospital: IVF: LR @ 100 mL/hr, no interactions
- Upon D/C from hospital: began 40 mg ferrous sulfate PO, possible interactions if consumed with dietary zinc & calcium source, & nicotine use

**Estimated Energy Need:**

| kcal / day | 2225.27 |
| Base on: | 34.5 kcal/kg/d |

**Estimated Protein Need:**

| g/day | 70.95 |
| Base on: | 1.1 g/kg/d |

**Estimated Fluid Need:**

| ml / day | 2275.5 |
| Base on: | 35 mL/kg/d |
### Nutrition Diagnosis (D)

#### A - Assessment (A)

State no more than 2 priority Nutrition Diagnosis statements in PES Format. Use Nutrition Diagnosis Terminology sheet ND Term (Problem) related to (Etiology) as evidenced by (Signs and Symptoms):

1. Iron deficiency manifesting with hypochromic microcytic anemia related to altered nutrition related laboratory values pertaining to iron status, inadequate mineral intakes of iron, inadequate energy intakes, limited food acceptance, predicted suboptimal nutrient intake, disordered eating pattern, undesirable food choices, and poor nutrition quality of life as evidence by fatigue, pale skin pigment and ocular sclera, poor dietary intake, irregular use of prenatal vitamins, close proximity between pregnancy as well as recent cesarean surgery, vaginal spotting, increased respiratory effort and rate as dyspneic state, and diminished bilateral pulses (NI – 1.4, 2.9, 5.10.1, 5.11.1, NC – 2.2, NB – 1.5, 1.6, 1.7, 2.5).

2. Inadequate energy intake related to predicted suboptimal and inadequate energy intakes, limited food acceptance, increased nutrient needs required in pregnancy, inadequate protein-energy intake, underweight at current ABW in pregnancy term, predicted suboptimal nutrient intake, self monitoring deficit, disordered eating pattern, limited adherence to nutrition-related recommendations as inconsistent prenatal vitamin use, undesirable food choices, impaired ability to prepare foods, and poor nutrition quality of life as evidenced by inadequate energy intake as per usual intake recall and 24-hour recall, picky eating, poor dietary sources, inadequate pregnancy weight, irregular consumption of prenatal vitamins needed for metabolism, and fatigue. (NI – 1.4, 1.6, 2.9, 5.1, 5.3, 5.11.1, NC – 3.1, NB – 1.4, 1.5, 1.6, 1.7, 2.4, 2.5)

### Nutrition Intervention (I)

#### P - Plan

List Nutrition Interventions. Use Nutrition Intervention Terminology sheet. (The intervention(s) must address the problems (diagnoses),

- **ND – 1.1**: General healthful diet
- **ND – 1.2**: Modify distribution, type or amount of food & nutrients within meals
- **ND – 1.3**: Specific food groups; iron containing sources
- **ND – 3.2.9**: Folate vitamin supplement
- **ND – 3.2.12**: Prenatal vitamin supplementation; consistent use
- **ND – 3.2.3**: Mineral iron supplement
- **ND – 3.3.5**: Manage bioactive substance; reduce/cease nicotine use
- **E – 1.1**: Purpose of the nutrition education
- **E – 1.2**: Priority modification
- **E – 1.4**: Nutrition relationship to health/disease
- **E – 1.5**: Recommended modifications
- **E – 2.1**: Result interpretation
- **C – 2.1**: Motivational interviewing
- **C – 2.2**: Goal setting
- **C – 2.3**: Self-monitoring
- **C – 2.4**: Problem solving
- **C – 2.9**: Relapse prevention

**Goal(s):**

- To increase gestational weight gain to adequate parameters via increased daily caloric intake from nutrient dense, energy rich sources, restore iron status from deficient to adequate of optimal levels, restore overall metabolic equilibrium for maternal-fetal health, and improving nutritional status by increasing nutrient intake and education.

### Plan for Monitoring and Evaluation (M E)

List indicators for monitoring and evaluation. Use Nutrition Assessment and Monitoring & Evaluation sheets. (Upon follow-up, the plan for monitoring would indicate if interventions are addressing the problems).

Factors monitored to assess the patient’s pregnancy status will be achieved in the monitored factors of nutritional and iron status and restoration of overall maternal-fetal health. Factors monitored to assess the patient’s nutritional and iron status are total energy intake, beverage intake as oral fluids, food intake as amount of food, types of food, meal pattern, diet quality index, food variety, bioactive substance intake relating to nicotine use, macronutrient intake as total protein intake and total carbohydrate intake, micronutrient intake as prenatal multivitamin consistency specific to folate, B6, B12, and vitamins A, C, D, and K.
mineral intake as calcium, iron, and zinc, adherence to diet orders as general, healthful diet and diet/nutrition education, correction in misuse of prenatal vitamin, increased level of knowledge pertaining to iron-deficiency hypochromic, microcytic anemia, adjustment in behavior as self-management at agreed upon specifics, patient fatigue during feeding process resulting in inadequate intake, physical activity as duration, intensity, and strength, nutrition-related patient-centered measure as nutrition quality of life responses, anthropometric measurements as weight, weight change, pregnancy growth pattern, pregnancy compartment estimates, biochemical data and medical tests as gastrointestinal profile to include gastric emptying time, small bowel transit time, as metabolic rate profile to include resting metabolic rate, all inclusive nutritional anemia profile and vitamin profile, nutrition-focused physical findings as overall appearance in weight change, skin pigment, digestive system stool production and color, and most closely monitored factors to assess nutritional status are comparative standards as energy needs and intake, macronutrient needs and intakes, fluid needs and intakes, micronutrient needs and intake, as well as adequate adjustments in desired pregnancy growth pattern. (FH -1.1.1.1, 1.2.1.1, 1.2.2.1, 1.2.2.2, 1.2.2.3, 1.2.2.4, 1.2.2.5, 1.4.2.5, 1.5.2.1, 1.5.3.1, 1.6.1.12, 1.6.2.1, 1.6.2.3, 1.6.2.8, 2.1.1.1, 2.1.2.2, 3.1.3, 4.1.1, 4.1.2, 5.1.5, 5.4.7, 7.3.4, 7.3.5, 7.3.7, 8.1.1, AD – 1.1.2, 1.1.4, 1.1.6, 1.1.7, BD – 1.4.20, 1.4.21, 1.8.1, 1.10, 1.13, PD – 1.1.1, 1.1.5, 1.1.8, CS – 1.1.1, 2.1.1, 2.2.1, 2.3.1, 2.4.1, 3.1.1, 4.1, 4.2, 5.1.3)

- Determine progress made by patient in food sources, nutrient and calorie intake outcomes and if goals are met by the following follow up labs and dietary corrections:
  - Hematology of nutritional anemia panel:
  - Total caloric intake: actual dietary calorie intake & supplements, weight gain per pregnancy gestation

- Evaluation: Anticipate nutrient adjustments at home with increase calorie and sources of dietary intake, along with regular use of prenatal and iron supplementation.

### Nutrients Report

Your plan is based on a default **2000 Calorie** allowance.

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>Target</th>
<th>Average Eaten</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Calories</td>
<td>2000 Calories</td>
<td>1432 Calories</td>
<td>Under</td>
</tr>
<tr>
<td>Protein (g)***</td>
<td>46 g</td>
<td>59 g</td>
<td>OK</td>
</tr>
<tr>
<td>Protein (% Calories)***</td>
<td>10 - 35% Calories</td>
<td>16% Calories</td>
<td>OK</td>
</tr>
<tr>
<td>Carbohydrate (g)***</td>
<td>130 g</td>
<td>207 g</td>
<td>OK</td>
</tr>
<tr>
<td>Carbohydrate (% Calories)***</td>
<td>45 - 65% Calories</td>
<td>58% Calories</td>
<td>OK</td>
</tr>
<tr>
<td>Dietary Fiber</td>
<td>25 g</td>
<td>11 g</td>
<td>Under</td>
</tr>
<tr>
<td>Total Fat</td>
<td>20 - 35% Calories</td>
<td>27% Calories</td>
<td>OK</td>
</tr>
<tr>
<td>Saturated Fat</td>
<td>&lt; 10% Calories</td>
<td>12% Calories</td>
<td>Over</td>
</tr>
<tr>
<td>Monounsaturated Fat</td>
<td>No Daily Target or Limit</td>
<td>10% Calories</td>
<td>No Daily Target or Limit</td>
</tr>
<tr>
<td>Polyunsaturated Fat</td>
<td>No Daily Target or Limit</td>
<td>3% Calories</td>
<td>No Daily Target or Limit</td>
</tr>
<tr>
<td>Linoleic Acid (g)***</td>
<td>12 g</td>
<td>4 g</td>
<td>Under</td>
</tr>
<tr>
<td></td>
<td>Target</td>
<td>Average Eaten</td>
<td>Status</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-----------------</td>
<td>---------------</td>
<td>--------</td>
</tr>
<tr>
<td><strong>Linoleic Acid (% Calories)</strong>*</td>
<td>5 - 10% Calories</td>
<td>2% Calories</td>
<td>Under</td>
</tr>
<tr>
<td>α-Linolenic Acid (g)**</td>
<td>1.1 g</td>
<td>0.7 g</td>
<td>Under</td>
</tr>
<tr>
<td>α-Linolenic Acid (% Calories)**</td>
<td>0.6 - 1.2% Calories</td>
<td>0.4% Calories</td>
<td>Under</td>
</tr>
<tr>
<td>Omega 3 - EPA</td>
<td>No Daily Target or Limit</td>
<td>1 mg</td>
<td>No Daily Target or Limit</td>
</tr>
<tr>
<td>Omega 3 - DHA</td>
<td>No Daily Target or Limit</td>
<td>0 mg</td>
<td>No Daily Target or Limit</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>&lt; 300 mg</td>
<td>126 mg</td>
<td>OK</td>
</tr>
<tr>
<td><strong>Minerals</strong></td>
<td>Target</td>
<td>Average Eaten</td>
<td>Status</td>
</tr>
<tr>
<td>Calcium</td>
<td>1000 mg</td>
<td>448 mg</td>
<td>Under</td>
</tr>
<tr>
<td>Potassium</td>
<td>4700 mg</td>
<td>1885 mg</td>
<td>Under</td>
</tr>
<tr>
<td>Sodium**</td>
<td>&lt; 2300 mg</td>
<td>2569 mg</td>
<td>Over</td>
</tr>
<tr>
<td>Copper</td>
<td>900 µg</td>
<td>823 µg</td>
<td>Under</td>
</tr>
<tr>
<td>Iron</td>
<td>18 mg</td>
<td>27 mg</td>
<td>OK</td>
</tr>
<tr>
<td>Magnesium</td>
<td>310 mg</td>
<td>148 mg</td>
<td>Under</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>700 mg</td>
<td>657 mg</td>
<td>Under</td>
</tr>
<tr>
<td>Selenium</td>
<td>55 µg</td>
<td>89 µg</td>
<td>OK</td>
</tr>
<tr>
<td>Zinc</td>
<td>8 mg</td>
<td>8 mg</td>
<td>OK</td>
</tr>
<tr>
<td><strong>Vitamins</strong></td>
<td>Target</td>
<td>Average Eaten</td>
<td>Status</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>700 µg RAE</td>
<td>656 µg RAE</td>
<td>Under</td>
</tr>
<tr>
<td>Vitamin B6</td>
<td>1.3 mg</td>
<td>3.5 mg</td>
<td>OK</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>2.4 µg</td>
<td>9.4 µg</td>
<td>OK</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>75 mg</td>
<td>34 mg</td>
<td>Under</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>15 µg</td>
<td>5 µg</td>
<td>Under</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>15 mg AT</td>
<td>1 mg AT</td>
<td>Under</td>
</tr>
<tr>
<td>Vitamin K</td>
<td>90 µg</td>
<td>30 µg</td>
<td>Under</td>
</tr>
<tr>
<td>Folate</td>
<td>400 µg DFE</td>
<td>797 µg DFE</td>
<td>OK</td>
</tr>
<tr>
<td>Thiamin</td>
<td>1.1 mg</td>
<td>2.6 mg</td>
<td>OK</td>
</tr>
<tr>
<td>Riboflavin</td>
<td>1.1 mg</td>
<td>2.8 mg</td>
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</tr>
<tr>
<td>Niacin</td>
<td>14 mg</td>
<td>36 mg</td>
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</tr>
<tr>
<td>Choline</td>
<td>425 mg</td>
<td>202 mg</td>
<td>Under</td>
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</tbody>
</table>

Sources:


