Back To Chiropractic CE Seminars

Labs for the DC:
Diabetes Type II, Hyperlipidemia, Iron Deficiency Anemia
6 Hours

- Presented by John B. Campise, D.C.-

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This course counts toward your California Board of Chiropractic Examiners CE. (also accepted in other states, check our website or with your Chiropractic State Board). The California Board requires that you complete all of your CE hours **BEFORE** the end of your Birthday month. We recommend that you send your chiropractic license renewal form and fee in early to avoid any issues.

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John B. Campise, Doctor of Chiropractic

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EDUCATION

-Doctor of Chiropractic, March 2001 – Life Chiropractic College West, Hayward, CA
-Undergrad 90 quarter hours, June 1997 – Santa Clara University, Santa Clara, CA

CHIROPRACTIC TECHNIQUE ADVANCED STUDY

-Neuro-Emotional Technique Certification, January 2006, Dr. Scott Walker, D.C., NET, Inc., Carlsbad, CA

-Applied Kinesiology Certification, May 1999, Tim Francis, D.C., ICAK USA, Sunnyvale, CA

-Carrick Institute Chiropractic Neurology Diplomate Course: 250 hours audited, May 1999

John B. Campise, Doctor of Chiropractic

CONFERENCE PRESENTATIONS

-Neuro-Emotional Technique “Success Seminars” 25th Anniversary. *24 hour clock acupuncture theory correlations to NET and homeopathic support of the chiropractic adjustment.*

CHIROPRACTIC PRACTICE

-Campise Chiropractic private practice, June 2001 - Present, Fresno, CA. General Practice with a focus on nutrition, wellness, and rehabilitation of traumatic brain injuries.

-Dr. Kotsonis, D.C., DACNB Chiropractic Office, Jan 2015 - Dec 2015, Clinton Township, MI. General practice with a focus on stroke rehabilitation. For 2 weeks every month Dr. Campise was trained by and filled in for Dr. Kotsonis while he recovered from lumbar spinal fusion surgery.

Legal Disclaimer

Please be advised that everything contained within this PowerPoint and/or within this lecture is not intended to be clinical advice, counseling or advice for any patient.
The examples contained herein are for the comprehension of nutrition/labs and is intended for nothing other than informative continuing education purposes.
All protocols herein should not be used until proper history, exam and special studies have been completed and assessed.

Laboratory Interpretation: For the Chiropractor ~ 6hrs

Overview

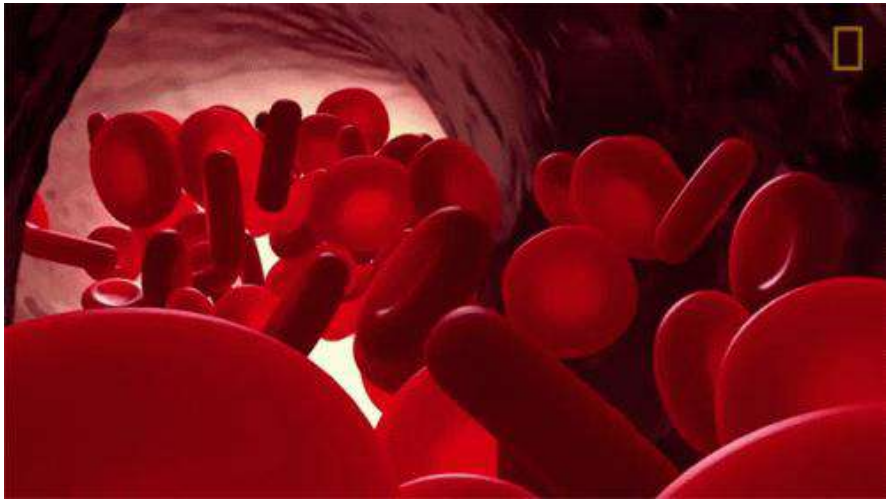
The 3 conditions with lab interpretation covered:

1) Red Blood Cell Anemia (iron deficiency type)
2) Diabetes, Type II
3) Hyperlipidemia

Each condition has the following sections:

- Clinical overview
- Causes of condition
- Signs and Symptoms
- Labs findings and interpretation
- Medical vs alternative treatment options

Anemia



Anemia

Red Cell Anemia

We will cover 1 type:
Iron deficiency

White Cell Anemia

AKA: Leukopenia

Platelet Anemia

AKA: Thrombocytopenia

Anemia

Clinical Overview

“Emia” is latin for blood and “An-” means “without or lacking.” So anemia simply means “lacking blood.” But it specifically means a lack of blood cells (either red or white) or a lack of the parts that make up blood cells or produced by blood cells (ie. hemoglobin, platelets, etc.).

Anemia

Clinical Overview

There are several types of anemia and generally several causes of each type. It is important to know which type and even more important to know which cause of which type in order to best help the patient.

Anemia

Clinical Overview

Red Blood Cell (RBC) anemia

Various types:

1) Nutrient Deficiency Anemia
2) Hemolytic Anemia
3) Megaloblastic Anemia
4) Microcytic Anemia
5) Pernicious Anemia
6) Aplastic Anemia

Anemia

Clinical Overview

Red Blood Cell (RBC) anemia

Various causes:

1) B2 deficiency
2) B6 deficiency
3) B9 (folate) deficiency
4) **B12 deficiency**
5) Copper deficiency
6) **Iron deficiency**
7) **Protein deficiency**
8) Autoimmune disease
9) Infection
10) Kidney issues
11) Liver issues
12) Spleen issues
13) **Internal bleeding**

Anemia

Clinical Overview

White Blood Cell (WBC) anemia: Leukopenia

Various causes:

1) AIDS
2) Bone marrow failure
3) Liver or Spleen disease
4) Cancer or Chemotherapy

Anemia

Clinical Overview

Platelet anemia (Thrombocytopenia)

Various causes:

1) Genetic disorders
2) B9 (Folate) deficiency
3) B12 deficiency
4) Vitamin K deficiency
5) Blood thinning medication
6) Autoimmune diseases
7) Infections
8) Enlarged Spleen
9) Blood clotting diseases (ie., TTP, DIC)
10) Cancers

Anemia

Iron Deficiency

Causes

1) **internal bleeding**
2) excessive menstrual bleeding
3) severe malnutrition
4) various intestinal diseases interfering with absorption of iron

MUST RULE OUT FIRST, especially if no signs or symptoms of #2-4.

Anemia

Iron Deficiency

Causes

1) **internal bleeding:**

Must first rule out:

-intestinal cancer
-kidney or bladder disease
-pancreas or liver cancer
-colon polyp or diverticulitis
-stomach ulcer bleeding

Anemia

Iron Deficiency

Causes

1) **internal bleeding:**

Ask Patient:

-Have they noticed any blood in stool or urine?

-What color is their stool?

-Black stool (oreo cookie) could be upper GI bleeding.

Anemia

Iron Deficiency

Causes

1) **internal bleeding:** If the patient says they have noticed blood in their stool or urine, then refer to a GI specialist or Urologist to find the cause of the bleeding.

Anemia

Iron Deficiency

Patients cannot see and will not notice microscopic amounts of blood in their stool or urine.

Causes

1) **internal bleeding:**

If the patient says they DO NOT have blood in their stool or urine, **THIS IS NOT GOOD ENOUGH**. Microscopic levels of blood loss over time can lead to anemia. A urine and stool test must be ordered to rule out.

Anemia

Iron Deficiency

Causes

2) Excessive menstrual bleeding

Refer to a Gynecologist to rule out uterine diseases.

Anemia

Iron Deficiency

Causes

2) Excessive menstrual bleeding

Also refer to a holistic practitioner who can help to correct their hormone imbalances by assessing thyroid, adrenal, diet, and stress issues.

Anemia

Iron Deficiency

Causes

3) Severe malnutrition

Patients at high risk:
-Vegans
-Elderly
-Cancer patients
-Anorexic or bulimic patients

Anemia

Iron Deficiency

Causes

4) various intestinal diseases interfering with absorption of iron

- Celiac disease (severe gluten intolerance)
- Crohn's disease
- Tropical Sprue (infection leading to chronic diarrhea)
- Inflammatory Bowel Disease

Anemia

Iron Deficiency

Signs and Symptoms

- Fatigue
- Slow wound healing
- Brittle nails
- Shortness of breath
- Lightheadedness
- Cold Hands and Feet
- Headaches
- Blue sclera
- Pale skin
- Weakness

Anemia

Iron Deficiency

Labs to order:

Complete Blood Count (CBC): includes RBC, hemoglobin, hematocrit, MCV, MCH, MCHC, RDW.

Iron Panel: includes ferritin, serum iron, transferrin, UIBC/TIBC (unsaturated/total iron binding capacities)

Anemia

Iron Deficiency

Fun Fact:

uL = microliter

1 mL = 1,000 microliters

Labs Findings and Interpretation:

RBC (Red Blood Cell count)
tends to be low.

RBC normal range:

men: 4.1-5.8 million cells per uL

women: 3.7-5.2 million cells per uL

Anemia

Iron Deficiency

Labs Findings and Interpretation:

Why does iron deficiency cause low **RBC**?

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2730642/

"Iron deficiency in the body limits the synthesis of heme, a prosthetic group of hemoglobin that in turn limits the synthesis of hemoglobin and *decreases the production of red blood cells* (RBCs) in the bone marrow resulting in anemia. Since cellular energy metabolism is dependent on oxygen, anemia has a wide range of clinical consequences. One of the consequences of severe iron deficiency is a *decrease in the life span of RBC*s in circulation that further exacerbates the anemic condition."

Anemia

Iron Deficiency

Fun Fact:

dL = deciliter

1 liter = 10 deciliters

Labs Findings and Interpretation:

Hemoglobin will tend to be low.

Normal range men: 13-17.7 g/dL

Normal range women: 11-15.9 g/dL

Anemia

Iron Deficiency

Labs Findings and Interpretation:

Why does iron deficiency cause low **Hemoglobin**?

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2730642/

"Iron deficiency in the body limits the synthesis of heme, a prosthetic group of hemoglobin that in turn **limits the synthesis of hemoglobin**..."

Anemia

Iron Deficiency

Labs Findings and Interpretation:

Hematocrit tends to be low.

Normal range men: 40 - 54%

Normal range women: 36 - 48%

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Hematocrit**?

https://www.ncbi.nlm.nih.gov/books/NBK542276/

“HCT measures the volume of packed red blood cells (RBC) relative to whole blood. Hence, it is also known and reported as a packed cell volume (PCV). It is a simple test to identify conditions like anemia or polycythemia and also to monitor response to the treatment. The term "hematocrit (HCT)" originated from English “hemato-“ and Greek “krites.” HCT measures the volume of packed red blood cells (RBC) relative to whole blood…

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Hematocrit**? (cont.)

https://www.ncbi.nlm.nih.gov/books/NBK542276/

…A glass tube and a centrifuge machine are sufficient to measure HCT. After centrifugation, the component of blood separates into three distinct parts. From below upwards, the layers are - a layer of red blood cells (RBC), a layer of white blood cells (WBC) and platelets, and a layer of plasma at the top. HCT calculation is by dividing the lengths of the packed RBC layer by the length of total cells and plasma."

Anemia

Iron Deficiency

Labs Findings and Interpretation:

Hematocrit:

- Another way of measuring RBC's

- Hematocrit will tend to be **LOW** with iron deficiency anemia.

Anemia

Iron Deficiency

Fun Fact:

fL = femtoliter

1 mL = 1 trillion femtoliters

Labs Findings and Interpretation:

MCV (mean corpuscular volume) tends to be normal.

Normal range (men): 80-100 fL

Normal range (women): 80-100 fL

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **MCV** (mean corpuscular volume)?

Defines the size of the red blood cell.

- A low MCV is a sign of microcytic anemia.
- A high MCV is a sign of macrocytic anemia.
- Iron deficiency anemia will usually present as normocytic.

Anemia

Iron Deficiency

Labs Findings and Interpretation:

MCV (mean corpuscular volume):

Defines the size of the red blood cell.

MCV is expected to be NORMAL with iron deficiency anemia.

Anemia

Iron Deficiency

Fun Fact:

pg = picogram

1 gram = 1 trillion picograms

Labs Findings and Interpretation:

MCH (mean corpuscular hemoglobin) tends to be LOW.

Normal range: 29 (+/- 2) pg

Anemia

Iron Deficiency

Labs Findings and Interpretation:

MCH (mean corpuscular hemoglobin):

Quantifies the average amount of hemoglobin per red blood cell.

MCH is expected to be **LOW** with iron deficiency anemia.

MCH generally follows the RBC trend, high or low.

Anemia

Iron Deficiency

Fun Fact:

dL = deciliter

1 liter = 10 deciliters

Labs Findings and Interpretation:

MCHC (mean corpuscular hemoglobin concentration) tends to be LOW.

Normal range: 34 (+/- 2) g/dL

Anemia

Iron Deficiency

Labs Findings and Interpretation:

MCHC (mean corpuscular hemoglobin concentration):

MCHC correlates the hemoglobin content with the volume of the cell.

MCHC is expected to be **LOW** with iron deficiency anemia.

Anemia

Iron Deficiency

Labs Findings and Interpretation:

RDW (RBC Distribution Width) tends to be HIGH.

Normal range: 13 (+/- 1.5) %

Anemia
Iron Deficiency

Fun Fact:

ug = microgram

1 g = 1 million micrograms

Labs Findings and Interpretation:

Serum Iron will tend to be low, but often normal.

Normal range men: 38-169 ug/dL

Normal range women: 34-152 ug/dL

Fun Fact:

dL = deciliter

1 liter = 10 deciliters

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Serum Iron**?

Serum iron (SI) levels reflect the total amount of iron in blood, including transferrin-bound and non–transferrin-bound iron.

From: Fowler's Zoo and Wild Animal Medicine, Volume 8, 2015

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Serum Iron**?

Measurement of serum iron concentration alone provides little useful information of iron status as values show considerable variation within normal individuals. **Low concentrations are seen in iron deficiency but are also seen in the anaemia of chronic disease and after surgery.**

From: Rebecca Frewin, in Clinical Biochemistry: Metabolic and Clinical Aspects (Third Edition), 2014

Anemia
Iron Deficiency

Fun Fact:

ng = nanogram

1 g = 1 billion nanograms

Labs Findings and Interpretation:

Ferritin will tend to be **LOW**.

Normal range men: 30-400 ng/mL

Normal range women: 15-150 ng/mL

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Ferritin**?

https://pubmed.ncbi.nlm.nih.gov/18835072/

“Ferritin, a major iron storage protein, is essential to iron homeostasis and is involved in a wide range of physiologic and pathologic processes. In clinical medicine, ferritin is predominantly utilized as a serum marker of total body iron stores. In cases of iron deficiency and overload, serum ferritin serves a critical role in both diagnosis and management. **Elevated serum and tissue ferritin are linked to coronary artery disease, malignancy**, and poor outcomes following stem cell transplantation”

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Ferritin**?

https://pubmed.ncbi.nlm.nih.gov/24549403/

"Serum ferritin" presents a paradox, as the iron storage protein ferritin is not synthesised in serum yet is to be found there. Serum ferritin is also a well known inflammatory marker, but it is unclear whether serum ferritin reflects or causes inflammation, or whether it is involved in an inflammatory cycle…"

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Ferritin**?

https://pubmed.ncbi.nlm.nih.gov/24549403/

"...We argue here that serum ferritin arises from damaged cells, and is thus a marker of cellular damage. The protein in serum ferritin is considered benign, but it has lost (i.e. dumped) most of its normal complement of iron which when unliganded is highly toxic. The facts that serum ferritin levels can correlate with both disease and with body iron stores are thus expected on simple chemical kinetic grounds…"

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Ferritin**?

https://pubmed.ncbi.nlm.nih.gov/24549403/

"…Overall, this systems approach serves to explain a number of apparent paradoxes of serum ferritin, including (i) why it correlates with biomarkers of cell damage, (ii) why it correlates with biomarkers of hydroxyl radical formation (and oxidative stress) and (iii) therefore why it correlates with the presence and/or severity of numerous diseases. This leads to suggestions … of the recognition that serum ferritin levels mainly represent a consequence of cell stress and damage."

Anemia

Iron Deficiency

Labs Findings and Interpretation:

Ferritin bottom line:

Low ferritin is the best marker for iron deficiency.

Chronic inflammatory conditions can raise ferritin levels which can sometimes mask iron deficiency.

So normal ferritin does not completely rule out iron deficiency.

But high ferritin can be a sign of iron overload which can be just as problematic for the patient as low iron due to iron's tendency to cause oxidative stress.

High ferritin can also be a sign of inflammation, and not related to iron overload.

Anemia

Iron Deficiency

Labs Findings and Interpretation:

Transferrin will tend to be HIGH.

Normal range: 215-380 mg/dL

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **Transferrin**?

Transferrin and TIBC measure essentially the same thing, so usually only one or the other of these is ordered. See the next slides on TIBC for more info.

Anemia
Iron Deficiency

Fun Fact:

ug = microgram

1 g = 1 million micrograms

Labs Findings and Interpretation:

UIBC/TIBC (Unsaturated/Total iron binding capacity): will tend to be **HIGH** when iron deficiency is present

Normal range: UIBC 111-343; TIBC 250-450 ug/dL

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **UIBC/TIBC**:

https://pubmed.ncbi.nlm.nih.gov/32644545/

"Iron-binding capacity is the capacity at which transferrin binds with iron. Transferrin, previously known as siderophilin, is the principal plasma transport protein for ferric iron (Fe^{3+}) …the indirect laboratory assessment of transferrin concentration may be inferred by TIBC. TIBC may be calculated as total or unsaturated. Depleting bodily iron stores by any mechanism increases circulating levels of transferrin…

Anemia

Iron Deficiency

Labs Findings and Interpretation:

What is **UIBC/TIBC**: (Continued)

https://pubmed.ncbi.nlm.nih.gov/32644545/

… At optimal health, only one-third of transferrin is saturated with iron, and serum transferrin has an extra binding capacity of 67%, the unsaturated iron-binding capacity (UIBC). TIBC is the total serum iron and UIBC. Percentage transferrin saturation is calculated by dividing serum iron by TIBC and multiplying the result by 100.”

Anemia

Iron Deficiency

Labs Findings and Interpretation:

UIBC/TIBC:

Generally:

If either UIBC or TIBC is high then the patient likely has low iron levels.

Anemia

Iron Deficiency

Labs Findings and Interpretation:

WARNING: You cannot diagnose iron deficiency with a CBC alone. Yet I've seen many patients prescribed iron supplements without ever being tested with an iron panel or ferritin levels. ***Taking iron can cause inflammation, muscle and joint pain, and liver stress for those who are not actually low in it.***

Anemia

Iron Deficiency

Medical vs Alternative Treatment Options:

Medical: Iron sulphate pills and a stool softener to counteract the constipation the iron sulphate causes. Birth control pills to reduce menstrual bleeding, antibiotics and acid blocking drugs to heal stomach ulcer, surgically remove colon polyps, and hysterectomy or uterine ablation to cure bleeding fibroids.

Anemia

Iron Deficiency

Medical vs Alternative Treatment Options:

Alternative: Take liver concentrate pills for non-constipating iron together with vitamin C for better absorption. Take herbs to heal stomach ulcer (mastic gum, okra, DGL). Take cayenne pepper capsules to stop mild intestinal bleeding. Herbally balance hormones to reduce excess menstrual bleeding. Bioidentical progesterone topical to shrink uterine fibroids.

Type II Diabetes

Diabetes

We will cover lab tests for Type II diabetes

Type I Diabetes

Autoimmune disease

Beta cell of the pancreas are destroyed by the immune system.

Cannot make insulin and requires insulin injections.

Type II Diabetes

Lifestyle diabetes

Insulin Resistance diabetes

High insulin at first because muscle cells can't use it to let glucose in. Eventually pancreas stops producing insulin.

Obesity, junk food, sedentary lifestyle are major risks.

Type III Diabetes

AKA: Alzheimer's (AD)
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2769828/

"We conclude that the term "type 3 diabetes" accurately reflects the fact that AD represents a form of diabetes that selectively involves the brain…"

Diabetes

History of Diabetes

https://pubmed.ncbi.nlm.nih.gov/31855345/

“Diabetes mellitus is taken from the Greek word *diabetes*, meaning siphon - to pass through and the Latin word *mellitus* meaning sweet. A review of the history shows that the term "diabetes" was first used by Apollonius of Memphis around 250 to 300 BC. Ancient Greek, Indian, and Egyptian civilizations discovered the sweet nature of urine in this condition, and hence the propagation of the word Diabetes Mellitus came into being…”

Diabetes

History of Diabetes (Continued 2)
https://pubmed.ncbi.nlm.nih.gov/31855345/

"…Mering and Minkowski, in 1889, discovered the role of the pancreas in the pathogenesis of diabetes. In 1922 Banting, Best, and Collip purified the hormone insulin from the pancreas of cows at the University of Toronto, leading to the availability of an effective treatment for diabetes in 1922. Over the years, exceptional work has taken place, and multiple discoveries, as well as management strategies, have been created to tackle this growing problem…."

Diabetes

History of Diabetes (Continued 3)
https://pubmed.ncbi.nlm.nih.gov/31855345/

"…Unfortunately, even today, diabetes is one of the most common chronic diseases in the country and worldwide. In the US, it remains as the seventh leading cause of death."

Diabetes

Clinical Overview

Type I Diabetes

- AKA: Juvenile Diabetes (Because onset usually occurs in childhood, but term not used as much since now it is accepted that onset can often be in adulthood)

- Autoimmune disease

- Blood tests: the same as Type II (HA1c, fasting blood glucose) but to differentiate I from II, auto-antibodies in the blood are additionally tested, the presence of which generally indicate Type I.

Diabetes

Clinical Overview

Type II Diabetes

- AKA: Adult Onset (term not used as much since now many children have it), Lifestyle Diabetes, “Insulin Resistance” type.

- **Blood tests:**
HgA1c, fasting blood glucose, (random blood glucose if can’t fast), Fasting Insulin

Diabetes

Clinical Overview

Type III Diabetes

- AKA: Alzheimer's Disease (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2769828/ "We conclude that the term "type 3 diabetes" accurately reflects the fact that AD represents a form of diabetes that selectively involves the brain…")

- No lab tests for this yet.

Diabetes
Type II

Causes

1) Dietary stress
2) Sedentary lifestyle
3) Obesity

Diabetes
Type II

Causes

1) Dietary stress: anything that causes insulin release.

Sugars: Desserts, soda, fruit juice. (Even most ketchup has added sugar, it's in everything. Most restaurant food has lots of added sugar not disclosed in the menu.)

Excess refined carbs: Breads, pasta, rice, crackers, chips, etc.

Diabetes
Type II

Fun Fact:
High Fructose Corn Syrup = 50% fructose

Table sugar (sucrose) = 50% fructose.

Causes

1) Dietary stress:

Fructose: (table sugar, sucrose, is 50% fructose)

https://pubmed.ncbi.nlm.nih.gov/30959577/

"Fructose intake is known to induce obesity, **insulin resistance**, metabolic syndrome, and nonalcoholic fatty liver disease…"

Diabetes
Type II

Fun Fact:
High Fructose Corn Syrup = 50% fructose

Table sugar (sucrose) = 50% fructose.

Causes

1) Dietary stress:

Fructose:

A good video discussion about the detrimental effects of excess fructose (comes from regular sugar, sucrose) in the diet.
https://www.youtube.com/watch?v=Lpsmq6S7BMQ

Diabetes
Type II

Fun Fact:
High Fructose Corn Syrup = 50% fructose

Table sugar (sucrose) = 50% fructose.

Causes

1) Dietary stress:

Fructose:

High fructose corn syrup and regular sugar have the **SAME** amount of fructose. They are equally bad.

Diabetes
Type II

Causes

2) Sedentary lifestyle:

Increased risk of high junk food diet and obesity.

Diabetes
Type II

Causes

3) Obesity:

https://pubmed.ncbi.nlm.nih.gov/33561645/

"...long-term obesity and overnutrition develop into insulin resistance…"

Diabetes
Type II

Signs and Symptoms

https://diabetes.org/about-diabetes/type-2

- Urinating often
- Feeling very thirsty
- Feeling very hungry—even though you are eating
- Extreme fatigue
- Blurry vision
- Cuts/bruises that are slow to heal
- Tingling, pain, or numbness in the hands/feet

Diabetes
Type II

Labs to order:

Hemoglobin A1c

Fasting blood glucose

(Random blood glucose)

Fasting Insulin

Diabetes
Type II

Labs Findings and Interpretation:

Hemoglobin A1c (HbA1c) will tend to be HIGH.

HbA1c **normal** range:

4.0%-5.6%

Diabetes
Type II

Labs Findings and Interpretation:

Hemoglobin A1c (HbA1c) will tend to be HIGH.

HbA1c ***pre*-diabetes** range:

5.7 - 6.4%

Diabetes
Type II

Labs Findings and Interpretation:

Hemoglobin A1c (HbA1c) will tend to be HIGH.

HbA1c **diabetes** range:

6.5% and higher

Diabetes
Type II

What is HgA1c? (1)

https://www.ccjm.org/content/83/5_suppl_1/S4

"HbA1c was first discovered in 1955, but elevated HbA1c levels in diabetes patients were not noted until 1968.[1] Another 8 years passed before HbA1c was correlated with blood glucose values in hospitalized patients with diabetes and was proposed for monitoring glycemia…"

Diabetes
Type II

What is HgA1c? (2)

"…Biochemically, HbA1c forms through a nonenzymatic reaction in which glucose attaches to the valine amino terminal of one or both beta chains of hemoglobin A. This compound can be separated out from nonglycated hemoglobin and from other glycated hemoglobin molecules through various methods, such as high performance liquid chromatography or immunoassay…"

Diabetes
Type II

What is HgA1c? (3)

"…The HbA1c level is affected by the blood glucose concentration, the duration of red blood cell (RBC) exposure to varying concentrations, and RBC quantity. **HbA1c most accurately reflects the previous 2 to 3 months of glycemic control** in the setting of the usual RBC life span of 120 days.[9] As a relatively long-term indicator of glycemic control, it may not accurately represent acute improvements or deteriorations in glycemia. Recent factors affecting glycemia must be considered, as HbA1c represents a weighted average glucose with 50% contribution from the preceding month…."

Diabetes
Type II

What is HgA1c? (4)

"…HbA1c must be interpreted with caution. In non-pregnant adults, HbA1c is often falsely low in conditions that reduce the number of glycosylated RBCs, such as hemolysis, splenomegaly, chronic kidney disease, cirrhosis, hemorrhage, blood transfusions, use of erythropoiesis-stimulating agents, and certain hemoglobinopathies (ie, HbS, HbC, HbF). Alternately, HbA1c is elevated in other hemoglobinopathies and in conditions that result in decreased RBC turnover such as iron or vitamin B12-deficiency anemia."

Diabetes
Type II

Fun Fact:

dL = deciliter

1 liter = 10 deciliters

Labs Findings and Interpretation:

Fasting Blood Glucose will tend to be HIGH.

Normal range:

70-99 mg/dL

Diabetes
Type II

Labs Findings and Interpretation:

Fasting Blood Glucose will tend to be HIGH.

Pre-Diabetes range:

100-125 mg/dL

Diabetes
Type II

Labs Findings and Interpretation:

Fasting Blood Glucose will tend to be HIGH.

Diabetes:

126 mg/dL or higher

Diabetes
Type II

Labs Findings and Interpretation:

Random **Blood Glucose** will tend to be HIGH.

Diabetes = **200 mg/dL or higher**

Diabetes
Type II

Discussion

https://academic.oup.com/clinchem/article/55/5/850/5631768

“...changes in the way blood samples are handled before laboratory measurement of glucose need to be strongly considered. Universal adoption of methods that inhibit glycolysis would be expected to improve the precision and utility of glucose measurements, but it might substantially increase diagnoses of diabetes unless compensatory changes in diagnostic cutpoints were made.”

Diabetes
Type II

Discussion

To avoid laboratory error and random stress events in the patient leading to false positive diagnosis, make sure to see a high glucose blood test on at least two separate occasions before diagnosing diabetes.

Diabetes
Type II

Fun Fact:

uIU = micro-international unit

1 IU of insulin = 0.0347 mg

Labs Findings and Interpretation:

Fasting Insulin will tend to be HIGH.

Normal = 2.6-24.9 uIU/mL (Labcorp)

Optimal = <10 uIU/L

Extra credit = <5 uIU/mL

Diabetes
Type II

Labs Findings and Interpretation:

Fasting Insulin why's it important?

According to Dr. Robert Lustig, MD, this lab is the single most important lab to order because high insulin is correlated not only with insulin resistance and Type II diabetes, but also with heart disease, cancer, and dementia.
https://youtu.be/Lpsmq6S7BMQ?si=6j3U53l2anZfEiSe

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (1)

Metformin: aims to lower glucose production by liver and make body more sensitive to insulin.

Possible side effects: Can cause B12 deficiency.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (2)

Sulfonylureas and Glinides: aims to increase pancreas secretion of insulin.

Possible side effects: Can cause low blood sugar and weight gain.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (3)

Thiazolidinediones: aims to make the body more sensitive to insulin.

Possible side effects: Increased risk of congestive heart failure, bladder cancer, **bone fractures**, weight gain.

Attention Chiropractors!

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (4)

Attention Chiropractors!

DPP-4 Inhibitors: aims to reduce glucagon release which in turn would increase insulin secretion.

Possible side effects: Increased risk of pancreatitis and **joint pain**.

Diabetes
Type II

Attention Chiropractors!

Medical vs Alternative Treatment Options:

Medical: (5.1)

Glucagon-Like Peptide-1 (GLP-1) Receptor Agonist: (ie. **Ozympic**) delays gastric emptying and inhibits glucagon production from pancreatic α-cells.

Possible side effects: nausea, vomiting, and diarrhea, dizziness, mild tachycardia, infections, **headaches**, and dyspepsia.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (5.2)

Glucagon-Like Peptide-1 (GLP-1) Receptor Agonist: (ie. **Ozympic**)

Video discussion about the pros and cons of these drugs:

https://www.youtube.com/watch?v=zcBrvJzPiWU

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (6)

Sodium-Glucose cotransporter-2 (SGLT2) Inhibitors: aims to cause the kidney to spill blood glucose into the urine.

Possible side effects: Increased risk of yeast infections, urinary tract infections, low BP, high cholesterol, and gangrene.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (7.1)

Insulin Therapy: injections that aim to replace the body's normal insulin levels when the pancreas stops producing enough.

Possible side effects: Hypothetically none if perfectly administered in the exact amount needed at the exact time needed.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Medical: (7.2)

Attention Chiropractors!

Insulin Therapy:

Symptoms of Overdose: **Headache**, fatigue, brain fog, tremors, sweating, insomnia, seizure, disorientation, coma.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Alternative: (1) To be safe, alternative treatments for diabetes should be done with close cooperation with a medical practitioner who can prescribe and manage necessary medications until the Type II diabetes has been improved enough to warrant medical reduction or cessation of diabetic medications.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Alternative: (2) Reduce or remove concentrated sugars and refined carbohydrates like bread, rice, and pasta from the diet.

Find the patient's carbohydrate tolerance with the Two Week Test: https://philmaffetone.com/2-week-test/

Diabetes
Type II

Medical vs Alternative Treatment Options:

Alternative: (3.1) Encourage the patient's muscle fibers to convert from white to red fibers.

White muscle fiber = Type II anaerobic, fast twitch, or *sugar burning fibers*.

Red muscle fiber = Type I aerobic, slow twitch, or *fat burning*.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Alternative: (3.2)

White muscle fibers are white because they have few blood vessels, and thus mainly use anaerobic **sugar burning**.

Red muscle fibers are red because they have many blood vessels that supply ample oxygen for aerobic **fat burning**.

Diabetes
Type II

Medical vs Alternative Treatment Options:

Alternative: (3.3) Encourage patient's white muscle fibers to convert into red muscle fibers via **maximal aerobic function exercise.**

Find their optimal aerobic zone via VO2 max testing equipment: https://korr.com/products/vo2-max-testing-system/

Diabetes
Type II

Medical vs Alternative Treatment Options:

Alternative: (3.4) Encourage patient's white muscle fibers to convert into red muscle fibers via maximal aerobic function exercise.

Or, find a close estimation of it via Dr. Phil Maffetone's 180 Formula: https://philmaffetone.com/180-formula/

Hyperlipidemia

Hyperlipidemia

What is hyperlipidemia?

-Hyperlipidemia is when a patient has elevated levels of cholesterol (usually assumed when the lipo-protein LDL is elevated) or elevated levels of triglycerides in their blood.

-Hyperlipidemia can be a risk factor for cardiovascular disease.

Hyperlipidemia

Lipo-proteins

High Density (HDL)

Low Density (LDL)

Very Low Density (VLDL)

Intermediate Density (IDL)

Lipoprotein a {Lp (a)}

Chylomicrons (CM)

Triglycerides

Dietary fats absorbed by small intestine.

Fats produced by liver from excess dietary calories or from liver glycogen stores.

Fats released from adipocytes.

A source of energy for cells.

Cholesterol

80% is made by the liver 20% comes from diet.

Precursor to steroid hormones and vitamin D.

Strengthens cell membranes.

Needed to make bile.

Involved in various immune system functions.

Hyperlipidemia
Clinical Overview

Lipo-proteins

- Triglycerides and cholesterol are hydrophobic (“afraid of water” or not water soluble) and so they cannot travel through the bloodstream safely without being transported by lipo-proteins.

- There are 8 types of lipoproteins but usually only 2 of them are tested routinely on blood panels (HDL and LDL) with 2 others being tested occasionally {VLDL and Lp(a)}

Hyperlipidemia

Clinical Overview

Triglycerides

- Three fatty acids + one glycerol backbone = one triglyceride

- A source of energy for cells.

- LDL carries triglycerides from the liver to the adipocytes for storage.

- HDL carries triglycerides from the adipocytes to the body tissues to burn for energy.

Hyperlipidemia
Clinical Overview

Cholesterol

- The body makes vitamin D with the help of UVB from sunlight from cholesterol. Steroid hormones (cortisol, testosterone, estrogen, progesterone, aldosterone, DHEA) are made from cholesterol.

- Cholesterol is a required ingredient in the production of bile by the liver which helps emulsify dietary fats which is required for their absorption including the fat soluble vitamins like A, D, E, and omega 3.

- Cholesterol is carried in the blood by lipoproteins.

Hyperlipidemia

Causes

1) Dietary stress
2) Hereditary factors

Hyperlipidemia

Causes

1) Dietary stress: There has been confusion around which foods cause hyperlipidemia. The CDC website says:

“Limit foods high in saturated fat. Saturated fats come from animal products (such as cheese, fatty meats, and dairy desserts) and tropical oils (such as palm oil). Foods that are higher in saturated fat may be high in cholesterol.”

Hyperlipidemia

Causes

1) Dietary stress: Essentially the CDC hypothesis goes like this:
Saturated fats tend to be in foods that are high in cholesterol.
Eating cholesterol increases cholesterol.
The trouble with this hypothesis is that 80% of the cholesterol in your blood was made by your liver. High cholesterol blood levels are usually because your liver made too much. Why would it do that?

Hyperlipidemia

Causes

1) Dietary stress:

"When you ingest fructose in high amounts without the associated fiber found in whole fruit, it turns on the cholesterol-producing factory in your liver called *lipogenesis* which makes super dangerous small LDL particles, jacks up your triglycerides, and lowers the HDL (or good) cholesterol. So does sugar in any form, including flour and refined carbs."

https://drhyman.com/blogs/content/7-ways-to-optimize-cholesterol

Hyperlipidemia

Causes

1) Dietary stress:

So in other words there is a fight between competing hypotheses:

A) Eating cholesterol rich foods causes hyperlipidemia,

OR

A) Eating sugar, especially fructose, causes the liver to overproduce cholesterol, causing hyperlipidemia.

Hyperlipidemia

Causes

2) Hereditary Factors:
If hyperlipidemia runs in your family (Familial Hyperlipidemia or FH) then it is presumed to be caused by a genetic disorder, but often patients are not tested for whether they actually have the gene or not. **True cases of Familial Hyperlipidemia often don't respond well to dietary treatments (eating low fat foods or eating low sugar and low carb foods).**

Hyperlipidemia

Fun Fact:
ASCVD = Atherosclerotic cardiovascular disease

Causes

2) Hereditary Factors:
https://testdirectory.questdiagnostics.com/test/test-guides/TS_FH/familial-hypercholesterolemia
"According to International Atherosclerosis Society guidelines, clinical diagnostic criteria for FH include personal or **family history of premature ASCVD**, physical findings of **tendon xanthomas** (yellowish patches or lumps of cholesterol buildup in the tendons of the hands, feet, and heel) or **corneal arcus** (opaque ring in the corneal margin), and **elevated LDL-C**."

Hyperlipidemia

Fun Fact:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3070150/

“Low-density lipoprotein (LDL) is conventionally quantified in terms of the mass of cholesterol carried by these particles. LDL cholesterol (LDL-C) has been the standard measure of LDL and LDL-attributable cardiovascular disease (CVD) risk for so long that “LDL” and “LDL-C” tend to be used interchangeably.”

Hyperlipidemia

Fun Fact:

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3070150/

"The amount of cholesterol per LDL particle is variable and related in part to particle size, with smaller particles carrying less cholesterol. This variability causes concentrations of LDL cholesterol (LDL-C) and LDL particles (LDL-P) to be discordant in many individuals."

Hyperlipidemia

Fun Fact:
FH = Familial Hyperlipidemia

Causes

2) **Hereditary Factors**:
https://testdirectory.questdiagnostics.com/test/test-guides/TS_FH/familial-hypercholesterolemia
"...**genetic testing** alone can provide a definitive diagnosis. An international expert panel convened by the FH Foundation recommends testing for variants in the 3 genes most commonly associated with FH: ***LDLR***, ***APOB***, and ***PCSK9***. Loss-of-function variants of *LDLR* are the most common (79% to 88% of FH cases), followed by loss-of-function variants of *APOB* (5% to 13%) and gain-of-function variants of *PCSK9* (<1%)."

Hyperlipidemia

Signs and Symptoms

Mild to moderate and acute severe hyperlipidemia:

- ***there are no signs or symptoms.*** This is why lipid panels are used routinely on most medical patients.

Chronic severe hyperlipidemia cases may have:

- **tendon xanthomas**

(yellowish patches or lumps of cholesterol buildup in the tendons of the hands, feet, and heel)

- **corneal arcus**

(opaque ring in the corneal margin)

Hyperlipidemia

We will cover the basic panel in this course!

Labs to order:

- **Basic Lipid Panel (Total Cholesterol, HDL, LDL, Triglycerides)**
- **Advanced Lipid Panels (VLDL, lipoprotein (a), apolipoprotein B, LDL Particle Number, LDL Small, LDL Medium, HDL Large, LDL Pattern, LDL Peak Size, LDL P, Small LDL P, LDL Size, HDL P, Large HDL P, HDL Size, Large VLDL P, VLDL Size)**

Hyperlipidemia

Fun Fact:

dL = deciliter

1 liter = 10 deciliters

Labs Findings and Interpretation:

Total Cholesterol will tend to be HIGH.

Total Cholesterol **normal** range:

100-199 mg/dL (Labcorp)

Hyperlipidemia

Labs Findings and Interpretation:

LDL will tend to be HIGH.

LDL **normal** range:

0-99 mg/dL (Labcorp)

Hyperlipidemia

Labs Findings and Interpretation:

HDL will tend to be LOW.

HDL **normal** range:

>39 mg/dL (Labcorp)

HDL **optimal** range:

>60 mg/dL (Functional Medicine)
https://drhyman.com/blogs/content/podcast-ep856

Hyperlipidemia

Labs Findings and Interpretation:

Triglycerides will tend to be HIGH.

Triglycerides **normal** range:

0-149 mg/dL (Labcorp)

Triglycerides **optimal** range:

<70 mg/dL (Functional Medicine)

https://drhyman.com/blogs/content/podcast-ep856

Hyperlipidemia

Labs Findings and Interpretation:

Triglycerides to HDL ratio will tend to be HIGH.

Triglycerides/HDL ratio **normal** range: **<2.0**

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10001260/

Hyperlipidemia

Labs Findings and Interpretation:

Triglycerides/HDL ratio below 2.0 is a much better marker for cardiovascular disease and metabolic syndrome than is total cholesterol and LDL levels.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10001260/

Hyperlipidemia

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

It's not a specific lab to order, it is a calculation performed by the practitioner.

Triglycerides divided by HDL:

$$\frac{\textbf{Triglycerides}}{\textbf{HDL}} = \textbf{TG/HDL ratio}$$

Hyperlipidemia

RED = Flagged by the lab as out of range

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

Example 1:

Total Cholesterol = 228 (normal <199)

HDL = 83 (normal >39)

LDL = 145 (normal <99)

Triglycerides = 86 (normal <149)

TG/HDL ratio =
86 divided by 83 = 1.04

Since a ratio below 2.0 is correlated with low risk for cardiovascular disease, this patient's high total cholesterol and high LDL numbers are very likely of no concern.

Hyperlipidemia

RED = Flagged by the lab as out of range

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

Example 2:

Total Cholesterol = 195 (normal <199)

HDL = 35 (normal > 39)

LDL = 160 (normal <99)

Triglycerdies = 137 (normal <149)

Before going to the next slide, try calculating the TG/HDL ratio and decide whether the high LDL in this example is of concern or not.

Hyperlipidemia

Answer!

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

Example 2:

Total Cholesterol = 195 (normal <199)

HDL = 35 (normal > 39)

LDL = 160 (normal <99)

Triglycerides = 137 (normal <149)

TG/HDL ratio =

137 divided by 35 = 3.91

Since a 3.91 ratio is higher than 2.0, then likely the high LDL is of concern and treatment to either lower the LDL or to lower the TG/HDL ratio is warranted.

Hyperlipidemia

RED = Flagged by the lab as out of range

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

Example 3:

Total Cholesterol = 245 (normal <199)

HDL = 65 (normal > 39)

LDL = 180 (normal <99)

Triglycerides = 110 (normal <149)

Before going to the next slide, try calculating the TG/HDL ratio and decide whether the high LDL and high Total Cholesterol in this example is of concern or not.

Hyperlipidemia

Answer!

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

Example 3:

Total Cholesterol = 245 (normal <199)

HDL = 65 (normal >39)

LDL = 180 (normal <99)

Triglycerides = 110 (normal <149)

TG/HDL ratio =

110 divided by 65 = 1.69

Since a 1.69 ratio is lower than 2.0, then likely the high LDL and high total cholesterol is NOT of concern. But an advanced lipid panel should be ordered if unsure.

Hyperlipidemia

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

The way to remember the formula for math nerds: We know that triglycerides are BAD and HDL is GOOD which means we want the lowest possible triglycerides and the highest possible HDL. When we divide two numbers the lower the number on top (numerator), the lower the answer. The higher the number on the bottom (denominator) the lower the answer. We want the ratio to be as low as possible (< 2.0) so we must put the triglycerides on the top and divide by the HDL.

Hyperlipidemia

Labs Findings and Interpretation:

What is Triglycerides/HDL ratio?

Triglycerides divided by HDL.

The way to remember the formula for normal people:

“Glide to the top to get high on the down low.”

Tri-GL-ycer-IDE-s go on top and HDL goes on the bottom.

Glide to the top to get high on the down low.

Hyperlipidemia

Labs Findings and Interpretation:

Advanced Lipid Panels:

These mainly test particle sizes.

Generally you want large fluffy particles.

The small dense ones are the bad ones.

Hyperlipidemia

Medical vs Alternative Treatment Options:

Medical: (1)

Statins: Block cholesterol production in the liver.

Possible side effects: **Muscle aches and pains**, low energy (due to interference with mitochondrial function.)

Attention Chiropractors!

Hyperlipidemia

Medical vs Alternative Treatment Options:

Medical: (2)

Ezetimibe: Prevents cholesterol from being absorbed in the small intestine.

Possible side effects: Sinus infection, diarrhea, **joint pain**, **back pain, muscle pain, arm and leg pain**.

Attention Chiropractors!

Hyperlipidemia

Medical vs Alternative Treatment Options:

Medical: (3)

Bile Acids Sequestrants: Cause the intestines to reabsorb less bile and thus pass more cholesterol in the stool.

Possible side effects: Bloating, abdominal pain, constipation, interferes with absorption of other medication.

Hyperlipidemia

Medical vs Alternative Treatment Options:

Medical: (4)

PCSK9 Inhibitors: Blocks PCSK9 proteins from breaking down LDL receptors in liver, effectively increasing their number. These receptors bind to LDL so that the liver can break down the LDL.

Attention Chiropractors!

Possible side effects: Fatigue and **muscle pain**.

Hyperlipidemia

Attention Chiropractors!

Medical vs Alternative Treatment Options:

Medical: (5)

ACLY Inhibitors: Blocks the production of cholesterol and fatty acids in the liver. Have only been FDA approved since 2020.

Possible side effects: **Tendon pain, back pain**, stomach pain, cold or flu like symptoms.

Hyperlipidemia

Medical vs Alternative Treatment Options:

Medical: (6)

Fibrates: Activates peroxisome proliferator-activated receptors (PPARs), mainly lowers triglycerides.

Possible side effects:Abdominal pain, constipation, diarrhea, dizziness, leg cramps, **headaches**.

Attention Chiropractors!

Hyperlipidemia

Medical vs Alternative Treatment Options:

Alternative: (1) Eliminate sugar from the diet and reduce refined carbohydrates (bread, rice, pasta, crackers, etc).

Find the patient's carbohydrate tolerance with the Two Week Test: https://philmaffetone.com/2-week-test/

Hyperlipidemia

Medical vs Alternative Treatment Options:

Alternative: (2) Encourage patient's white muscle fibers to convert into red muscle fibers via maximal aerobic function exercise.

Dr. Phil Maffetone's 180 Formula:
https://philmaffetone.com/180-formula/

Hyperlipidemia

Sucrose is 50% Fructose

Medical vs Alternative Treatment Options:

Alternative: (3) “When you ingest fructose in high amounts without the associated fiber found in whole fruit, it turns on the cholesterol-producing factory in your liver called *lipogenesis* which makes super dangerous small LDL particles, jacks up your triglycerides, and lowers the HDL (or good) cholesterol. So does sugar in any form, including flour and refined carbs.”

https://drhyman.com/blogs/content/7-ways-to-optimize-cholesterol

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