Blood Chemistry Intro: Blood Sugar

Transcript

Hello and welcome to our Blood Chemistry section on *Blood Sugar*. This is one of my favorite sections to teach because I love talking about blood sugar and I love the kind of results I get when we get peoples blood sugar in balance. This is not intended to be the comprehensive treatment of blood sugar obviously, we have a whole blood sugar module to do that, but this is to just pull out all the labs that we generally use to really delve into what labs are in our general blood chemistry and are expanded. Also to teach you a little bit about some of the more advanced stuff related to autoimmune diabetes and some of the levels and antibodies that you can test.

This is not intended to replace a one on one relationship with a qualified health care professional, not medical advice, it's intended to be me sharing with you, you sharing with your clients in an educational way and if they're under the care of a doctor, make sure that they run it by their doctor before they make severe decisions.

Blood sugar markers. We all know that fasting blood sugar, while it's a semi-useful indicator, it's not really useful at all when it comes to subtle signs. Fasting blood sugar is great for diagnosing or indicating that a person has diabetes. We have ranges, but it's really the last thing that changes and there are so many other things that change before that. That's where we want to be stepping in and helping people to prevent going down the diabetes route and pick up the early warning signs so that it's not after they've been diagnosed that they start to heed and take action.

The fasting blood sugar, I like it to be between 75 and 85. There's a number of studies that indicate that once it hits 90 the risk of heart disease goes up dramatically, like 10 fold in some of the studies. The higher it goes, the more that risk is. Triglycerides are related to blood sugar and we know that the triglyceride HDL ratio is one of our most important markers. The ideal is in the 50 to 100 range. I like to see it more like 75, that's a really nice ideal triglycerides.

Insulin is another marker that we don't often do. We don't do it often enough. I wish it was included on a standard blood panel. It's not that expensive to add it, like $25 or $30, something like that. If you do the insulin, there's 2 ways you want to do it in order to get some good information.

You want to measure the insulin fasting. Ideally you want it as close to 2 as possible, between 2 and 5. That's how much insulin is this person putting out when they're not eating any food.

The postprandial insulin is actually a really good number to have and it's even more important for discovering how advanced somebody is in terms of a type 2 diabetic, but also by itself without the fasting doesn't tell us a whole lot. If somebody has a really ideal fasting insulin of 2, but their after eating insulin, also called postprandial, is something like 80 or 90 you know that fasting blood glucose numbers are not going to give us a true picture and we're going to see a lot of blood sugar swings if we were to test their glucose. Their body's responding with huge amounts of insulin along the way.

Here's something I wish we could do. Insulin is too hard to measure in a little kit that we get, like the glucose strips, it has to be vacuum sealed and there's some special handling that has to happen to the blood after you draw it, so it's not something we can do at home. However, there are some insulin kits that you can take at home and they have you do some special stuff, but you have to take it off to the lab. There is a blood spot insulin test.

The ideal would be to test your fasting and then you test it postprandial. Insulin curve will be very similar to a glucose curve. We know that from all the charts that we had in our insulin resistance section. If you know that the person has a peak glucose at around the 45 minute mark, that's about when you want to take the insulin give or take 15 minutes. That way you will see what their peak insulin is. We know that the higher the insulin goes the more damage you get to the endothelial linings, the more permanent damage, the stiffening, the stuff that really puts people at a high risk. It's really good to know what that is.

Interestingly enough I was reading an article and a medical doctor was talking about this person who had all the signs of insulin resistance. She had the belly fat, she was brain fogged, she was exhausted, and every time they measured her blood glucose, no matter when they measured her glucose it was normal at all the different peaks. What happened with this woman, she had perfect blood sugars all the time, but she had all the signs of insulin resistance. Her fasting insulin was normal. They decided to do postprandials.

It was finally that her postprandial insulin's were going up to 90. 90. This was happening. She was like this for years. He had been working with her for years and couldn't figure this out. Postprandial insulin's of 90. He said this is the most amazing case because this is someone who just the insulin was working, it was high levels, it had to keep going higher and higher and higher to work, but her body was still able to produce those high levels. Usually what happens is the pancreas burns out. You can't produce the levels that it takes in order to get it up stream and into the blood, into the cells, and so the blood sugar starts to go up. Everybody is a little bit differently.

If you are working with somebody and you go, "Classic insulin resistance, what's going on here? Why is it not showing?" Check their postprandial insulin. Really important to do. What you do is have them eat and then go to the lab and make sure that they're at the lab to get their blood drawn right at about the 45 minute marker or whatever their peak is. The ideal would be they sit in the office with their blood sugar meter and they know what their peak usually is and they catch it at peak and then they get their blood drawn, but you can't always time it that way when you are dealing with a busy lab.

If any of these are out of balance and this is showing that there is a blood sugar problem, I always like to follow with a hemoglobin A1c or I get the person to check their glucose meter. The hemoglobin A1c is a nice number to have because that way you know what their long term glucose levels were, the average glucose. For example, if the person has a hemoglobin A1c of about 5.6, which is still considered normal before it moves into insulin resistance. 5.7 is considered moving into insulin resistance. What that means is their average blood glucose is around 119, and that's a little bit high right? We talk about not letting it go above 110. The average is 119, that means that counts in when their sleeping, in between meals, so clearly that number is off. I like to see that hemoglobin A1c between 4.5 and 5.

If fasting glucose is high and hemoglobin A1c is normal, it could be a vitamin B1 deficiency. You'll also see if that's the case that carbon dioxide, the bicarbonate less than 25, so in the acidic range, and LDH, which we'll talk about in the liver section, above 140, because that indicates a hypoglycemia situation.

Let's talk about hemoglobin A1c. It's an indirect measure of the blood sugars over a 120 day period. The glucose gets attached to the hemoglobin when the levels are high and they call it glycosylation. Glycosylated hemoglobin. It's irreversible. The good news is the blood cells will replace themselves after 120 days, so it's not like it's irreversible and irreversible damage as if that damage was to a blood vessel lining that couldn't get repaired. It's a good long-term measure. It's used in diabetes.

Oftentimes diabetics are measured and they have A1c's of like 11. A diabetic is considered controlled if it's around 7, which I think is ludicrous because that's an average blood glucose of around 140. When the glucose is high or low, you want to follow up for diabetes. Sometimes people are in a pre-diabetic state and their blood glucose is super low, like they'll eat a meal and very shortly after it goes way low because of the hypoglycemia. Either way it's a blood sugar problem, whether it's hypo or hyper, it's really the same problem it's just different stages.

Hemoglobin A1c can be depressed. If it's below 4.5 this person might be hypoglycemic but can also be hemolytic anemia. Remember we talked about that? Blood loss in pregnancy.

Going back to our anemia markers, if they have some of these anemia markers, severe iron deficiency and ferritin deficiency and they also have decreased A1c, that's the time to refer them for a consultation to see if they have some occult blood loss going on in their body.

We've got hypoglycemia, insulin resistance, metabolic syndrome, diabetes. We've got all these different stages of blood sugar imbalance. Each of these stages is going to have a different presentation in terms of their numbers. Let's look at some of those numbers.

Insulin resistance. If the glucose is above 100 but less than 120 that person is considered in insulin resistance. If they're hemoglobin A1c is greater than 5.7 it's considered insulin resistance. If they're fasting insulin is greater than 5 it's considered insulin resistance. Then the big one, the easy one to tell, which is not always accurate, I can attest to that personally, is the waist to hip ration. Oftentimes you just look at a person and you go, "Oh my God, this person is insulin resistant." Right? Because they've got the big belly and the smaller hips.

If in a male you see that the waist to hip ration is greater than 1, meaning that the waist is greater than the hips, then for sure you suspect insulin resistance. In a woman if it is more than 80% or the waist is greater than 35 inches, then you definitely consider it a very high risk of insulin resistance, and it doesn't always happen that way. I discovered I had insulin resistance, or at least pre-insulin resistance maybe. Yeah, I had pre-insulin resistance. Without the waist to hip ratio off and without the high glucose, without going above 100. I have a very, very good control of my first morning, my fasting glucose, but I was all over the map afterword's. I guess we would call that pre-insulin resistance, which is really where we want to catch people. I can get that under control before it ends up, you know like my grandmother with the diabetes.

Metabolic syndrome is really insulin resistance with some lipid abnormalities. Really it puts people at a much higher risk of cardiovascular disease. Generally speaking we're going to see their triglycerides greater than 110, and for those of you outside the U.S. that converts to 1.24 mmol/L, total cholesterol of greater than 220 or 5.69 in the SI system, HDL cholesterol of less than 55 or 1.42, glucose of greater than 100, fasting insulin of greater than 5, high blood pressure, hemoglobin A1c 5.7, and the waist/ hip ratio is off. This is classic for metabolic syndrome.

If they have most of these symptoms and not all of them or most of these markers, that's... you just kind of pull it all together, right? Especially if you see that belly like in this picture for sure there is some insulin resistance going on and if all those lipid numbers are off, we call it metabolic syndrome.

Let's look at how we assess it. Generally for medicine it has to be greater than 120 on 2 tests to assess as diabetes and hemoglobin A1c has to be greater than 6.5.

Cholesterol greater than 220, triglycerides greater than 110, HDL less than 55, and blood pressure increased. Really the only ones that we really need, that they actually use is the first one and possibly the second one as well. The glucose above 120 on 2 tests and hemoglobin A1c greater than 6.5.

Normally after a meal glucose goes up, insulin gets secreted by the pancreas and that causes the insulin to bind to the cell membranes, triggers those glucose receptors, says, "Open up, I got some sweets for you," dumps the glucose in and also amino acids, fats, magnesium, and other nutrients all gets transported into the cell through this thing right here, which is your receptor. Then once the nutrients are cleared from the blood, the pancreas stops secreting insulin. That's normal. That's the way it's supposed to be.

When the insulin binds to the cell it causes that cell to prefer the use of glucose over fat as fuel. Even if you've got some fats in the bloodstream that have been released from your fats, those are going to go back if you're eating a bunch of sugar and it triggers this insulin response. The other effect of insulin binding also inhibits the fat burning by the cells. When the insulin binds to the cells they can't burn fat. Why not? Because why burn fat, we've got all the sugar that we need.

It also inhibits growth hormone. As you recall growth hormone is important for growth and repair. For putting down lean muscle mass and burning fat. Insulin and growth hormone in this regard are polar opposites. Whenever the insulin goes up, the growth hormone it just goes away, it goes down again. The other important part of these insulin binding is it's slightly depresses thyroid effects by blunting conversion from T4, to T3. If we're constantly in this state of high insulin, we're going to have sluggish thyroid which is going to further contribute to the weight issue.

What happens when we have insulin resistance? Again, this should all be review, but it might not be. Either the circulating insulin doesn't bind to the cells or it binds but the effects are deficient because it doesn't have the right nutrients to get through. Say it goes and binds, but there's not enough chromium to secrete it through or magnesium. The pancreas just keeps secreting more insulin as long as that blood sugar stays up it says, "OK more, OK more, OK more." What that does is leaves your insulin up high for a long time which causes damage to the blood vessel linings, it causes excessive blood pressure, and is a very big risk factor for cardiovascular disease. Then any cells that are insulin resistant are going to have deficient function. Your liver cells, your untrained muscle, fat cells, they're all going to be point problems.

Let's look at the negative effects of insulin resistance, deficient function in the insulin resistant cells. We have cells that there's all this sugar and there's all this extra fat being stored, but the cells are not getting enough energy to actually do an efficient job. Excess anabolic effects meaning increased body fat, especially around the middle.

Oxidation in non-insulin resistant cells, because these cells are exposed to the excess insulin. You get damage to the blood vessel linings.

Systemic inflammation. It increases inflammatory markers like CRP. Hypertension is one of the negative effects because the blood thickens, you retain water and the fat burning is suppressed. Then a lot of adverse systematic effects like blunted growth hormones and thyroid hormones which leads to body-wide effects. We all know that it's really important to get this resolved.

We're going to look at some other things, some other markers. This is outside the realm of blood chemistry, but there are definitely things that we can be looking at. What causes the insulin resistance is super important. When you see these numbers, you're really looking for what other things might be causing it. There's the genetics, there's omega 3 deficiency's, the omega 6/3 ratio, deficiencies of our specific nutrients we talk about, chromium, magnesium, zinc, B vitamins, possibly boron, lithium. Lack of resistance exercise, manual labor in trained muscle mass, sugar, process food starches, fruit juices and sodas, stress, insufficient protein, or protein malabsorption.

All of these things contribute to insulin resistance. We know how to test it. You get a blood sugar meter, there's a lot of them. You can also get your own home hemoglobin A1c test. I will warn you, if you are going to be having people do that, it is a complicated test. Don't give it to the people who are having trouble following directions. I ruined 2 of them and they are like $12 apiece. I ruined 2 of them because I didn't read the instructions well. They are not easy like the glucose. There are some things you have to do.

Measuring blood glucose at home is super amazing when you can ... You know that it's expensive and it takes time to go to the labs, well they measure their fasting blood sugar, write down the test meal, measure the glucose immediately after the meal, measure it every half hour, measure it an hour after the meal and then every hour at 2, 3, 4, 5, 6 hours. Now even better is to get people to measure it every 15 minutes for the first 90 minutes.

Glucose tolerance test results. When you do that, the max should be 110. The ideal peak is actually 99 to 100. You never have the sugar dip below the starting point. If any of these is not happening, then you're talking to the person about how to get their glucose and their insulin under control.

Consequences of insulin resistance. We've got cardiovascular disease, hypertension, leptin resistance, weight loss resistance, fatty liver, impaired detoxification. Here's a biggy, insulin resistance can lead to androgen dominance, meaning male sex hormones and lead to PCOS. We see this a lot. We see it a lot in teenagers who are having difficulties getting their period, a lot of acne, they're gaining weight.

When you look they have PCOS, which is polycystic ovarian syndrome. The funny thing about it, polycystic ovarian syndrome you'd think they have multiple cysts on their ovaries and that's not always the case.

The other thing it can cause is a glutathione reduction, and that decreases phase 2 liver detox. We have a backup of toxins which then further increases the weight because those toxins have to be stored somewhere. It has actually been linked to cancers: Pancreatic, colon, and breast. There are some medications that hinder the insulin regulation, so you always want to do a good history on a person to see that they're not taking any of these drugs: Anti-seizures, cortical steroids, birth control pills, estrogen replacements, diuretics, hypoglycemia that is caused by alcohol, insulin, hypertension medications, and oral diabetes medications. There's medications that will get in the way.

Here's my favorite part. This is stuff no body is talking about. Latent autoimmune diabetes in adults, LADA. I came upon this and started studying it as I was seeing people who they'd come to see me and they'd explained that they were type 2 diabetes and they were on their medication and the medication wasn't working. I'd look at them and they didn't look like type 2 diabetics. They weren't overweight, they didn't look it. They looked more like a type 1 diabetic. The fact that their medications weren't working and they were saying they had to go on insulin, that doesn't make sense for type 2 diabetes.

Usually the person that that's the case or they have worn their pancreas out and they go from type 2 to have to be dependent on insulin. They are usually overweight, they usually show the signs. These people were not showing the signs. I'm like, "What's going on here?" I started looking into it and came across LADA. *Latent Autoimmune Diabetes in Adults*. There's a number of things that you can measure if you're suspecting this. If you have somebody who is actually doing some of the protocols, you know they come in and they're not on a terrible diet and they are just not being able to make it with their medications.

The first thing is glutamic acid decarboxylase (GAD) antibodies. What the heck is that? Glutamic acid decarboxylase is an enzyme that converts glutamate to GABA. GABA is a neurotransmitter that's calming, it's like a bedtime neurotransmitter. Glutamate is excitatory. What the heck does this have to do with diabetes and the pancreas and ... I was on a mission to find this out. What I found out was the mechanism is really not clear after going through a whole bunch of research papers. Basically there is a production of GABA in the pancreas. Who would have known, right? GABA is a neurotransmitter. You expect it to be produced in the brain. It's actually produced in the pancreas. The glutamic acid decarboxylase will convert the glutamate to GABA.

We weren't able to tell from the studies how GABA affects it and how glutamate [affects it]. Is it the buildup of the glutamate due to these antibodies that causes a problem, or is it the decreased GABA? I couldn't find anything that was real clear about that.

Suffice it to say, if a person has glutamic acid decarboxylase antibodies, they're going to have ... That's one of the best signs of autoimmune diabetes.

The other signs can be real obvious, we think this is the most common one, insulin antibodies. Antibodies that attack the person's own insulin. That's commonly associated with LADA too. Islet cell antibodies. Islet cells are those little cells within the pancreas that make the insulin. What if we attack these cells and can't make insulin anymore? Voila, we have elevated blood sugar, we have diabetes.

There's another set of antibodies called the zinc transporter autoantibodies (ZnT8). They attack our protein that's responsible for the uptake of zinc in the membrane of the insulin secretory granules. That causes a problem if there's antibodies to that. Tyrosine phosphatase antibodies attack a protein that regulate the cytokine-induced pancreatic beta-cell apoptosis. What the heck does that mean? Cytokines are inflammatory chemicals. The pancreatic has beta-cells produce the insulin. Apoptosis is programmed cell death. We may end up with this regulation of apoptosis being off, so too much apoptosis or not enough apoptosis. Not enough apoptosis means that cells live way beyond their useful life and they're taking up space, and you don't get a good response that way.

And then you have something call C-Peptide. C-Peptide mainly measures the residual beta-cell function by determining how much insulin is being secreted. This one is a good one to do and is especially going to be low in a long-term diabetic who has had this problem for a while. If any of these are positive, it's suspicion for LADA, the *Latent Autoimmune Diabetes in Adults*. If 2 or more, or 3 or more, or 4 or more, you see you get the idea it's more likely. The glutamic acid decarboxylase and the C-Peptide tend to be the ones that are most commonly done. All the rest of them can be done and are often done. A lot of them are done in research. If you have never seen this before, this is something to look out for.

Finally, we've got a blood sugar case, let's take a look at it. You've got somebody with a blood glucose of 74. Glucose of 74 I don't think it's terribly low as a fasting glucose, but it depends on how the person is feeling. If I'm doing a juice cleanse or a fast my fasting blood sugars can be in the 60's and sometimes even lower and I still feel fine. The TSH in this person is high, 7.17 and their Free T3 is low. Remember we said that with the insulin resistance we can get a decrease in the T4 to T3 conversion. There LDH is very high. LDH that high can be indicative of a liver problem, but it can also be indicative of a glucose regulation problem in terms of hypoglycemia.

That concludes our *Blood Sugar*. I hope I gave you enough information about how to detect the typical blood sugar imbalances, how to detect blood sugar balances long before the medical profession will do that for your client, because they're just not aware of all these subtle nuances.

And how you can help people prevent getting into diabetes, predict that they might be moving towards it well in advance, and distinguish between a Type-2 diabetes, and a Type-1 that's a latent adult onset. Those are critical things because you are going to see people that come across your office that are not the textbook classic cases and you have to be prepared and aware.