.... NR507 | WEEK 2

Clinical Reasoning Webinar Transcript

Anemia • Coronary Artery Disease • Heart Failure

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00:00	08:54	40:28	60:06
Overview	Anemia	CAD	Heart failure

> **TRANSCRIPTION NOTE** This AI-assisted transcript was created from the webinar audio. Punctuation and obvious medical terminology were lightly normalized; unclear audio and chat-only responses may not be exact. The recording includes an apparent internet interruption around 1:11:42-1:20:23. Near the end, the instructor states that the textbook should be treated as the course standard if the webinar and book differ.

Introduction: Oxygenation and Disease Interrelationships

Starts at 00:00

[00:01] Hello, everyone. It's great to see everyone on the call tonight. I have forgotten to press a record button here. Fortunately, very early in the presentation. So I'm going to introduce again that we are talking about some really good information tonight anemia coronary artery disease and heart failure. And all 3 of those, although they are 3 distinct diseases, they all in their own way.

[00:29] Affect a person's oxygenation and so, oxygenation as I was explaining is the ability of the tissues to be perfused, the organs and so, when oxygenated blood is released from the heart and out to the body, oxygen is dropped off by products of CO2 things like that is picked up and taken back to the lungs where it's released in the form of CO2.

[00:58] And so, so I guess my overall my overall premise tonight is that we're talking about diseases that affect an overall oxygenation status of the individuals. And so it's important to understand the similarities and how all these conditions affect oxygenation. And so we, you know, looking into pathophysiology and.

[01:23] So that alteration in the diseases is going to cause the person to have signs and symptoms. And that's where they show up to the nurse practitioner in the clinic. And you're thinking, okay, patient, let's see, let me think about what was going on at that level of patho.

[01:43] And to determine what your diagnosis is and then how we can manage you. So that is a huge relationship that I keep stressing each week in our webinars.

[01:55] Okay, and so why is it important to understand shared challenges in oxygenation among these three diseases? Well, because you think about patients, we have to have a holistic approach of managing the patient. It often takes a multidisciplinary team and face it.

[02:19] How many times have you had patients that had all of these at one time heart failure? Well, maybe they had coronary artery disease that did lead to heart failure and they have anemia on top of that. And so, yeah, so they can have so you've got three of these that is working against one individual and they're in some form. They're going to have impaired oxygenation status.

[02:43] And so it's really helpful in terms of early detection and intervention because, you know, we can screen for coronary artery disease before it even ever gets to a heart failure state stage. And so we can screening is really important and we can provide proactive treatments lifestyle management symptom management. Everything is done to improve oxygenation and reduce cardiac stress on the heart.

[03:10] And a lot of patient education, diet, nutrition, exercise, and hydration. But we're going to separate these out in three different ways in just a few minutes. We'll talk about each of them. But over the next I'm just really committed for bringing home the point about the relationship between anemia coronary artery disease and heart failure before we actually talk about them separately.

[03:36] So with, so there's a relationship between anemia and coronary artery disease. So anemia can actually exacerbate coronary artery disease by increasing myocardial o2 demand while simultaneously reducing oxygen supplies. So anemia is what? Well,

[03:57] Remember that hemoglobin carries oxygen to the tissues. And so if the person is anemic, then that's going to cause the body to...

[04:10] I'm not getting my kidneys or getting perfused. My my spleen is not getting perfused. My veins and arteries are getting perfused. And so the heart automatically starts thinking, wait a minute, I need to work harder to get the oxygen out. And so that's why there's an increased myocardial oxygen demand because it's not getting to the tissues. There's not enough hemoglobin in the person that who has anemia. And so

[04:40] Then the heart starts kicking in as a compensatory mechanism. And so it doesn't really help because their person is anemic, but the heart does try to step in and help.

[04:53] alleviate that so it can lead to a lot more frequent and severe ischemic episodes, including cardiac ischemic episodes, worsening coronary artery disease symptoms. And then actually impact there's also an impact of coronary artery disease on anemia coronary artery disease can contribute to anemia through chronic inflammation and reduced renal function which

[05:21] Impairs erythropoiesis red blood cell production. We'll talk a little bit more about erythropoiesis. I'll mention a little bit more tonight, but when we get into week 4 and going into the renal information, I'll mention it there as well.

[05:36] But anyway, that's a relationship there and also medications used to treat coronary artery disease. We're always given the antiplatelet agents, Plavix, for instance, that can increase the risk of bleeding and cause subsequent anemia. And then we have anemia and heart failure. There's a relationship here as well. So the impact of anemia on heart failure anemia will

[06:05] Increase the cardiac workload as I was mentioning just a few seconds ago as the heart compensates for reduced

[06:13] oxygen-carrying capacity by pumping more blood. And so this leads to worsening heart failure symptoms and the progression of the disease. And then we have the impact of heart failure on anemia. So heart failure can cause anemia through, again, chronic inflammation. And remember, we talked about the hypersensitivity actions last week, and we talked about, you know, inflammation, the inflammatory process and things like that. We can't ever with all of our diseases.

[06:42] I'll always take you back to the immune system and how it's impacts just about, it's a part of just about every disease process. And I'll point that out when that happens, but this is one of them where there's a lot of chronic inflammation, renal issues, and also the use of diuretics which can help to lead to iron deficiency anemia.

[07:06] The thinking about that as well. And so there's basically a reduced cardiac output and heart failure, impairs renal function because it is not the kidneys are not being perfused. And so that's going to further contribute to the anemia.

[07:25] And then we have a relationship between coronary artery disease and heart failure. And so that impacts coronary artery disease on your well aware of this is a leading cause of heart failure. The reduced blood flow from narrowed or blocked arteries just deprives the heart muscle of oxygen, which leads to ischemia and weakening of the heart muscle.

[07:52] Over time, this can result in heart failure as the heart struggles to pump blood effectively. So that's that is the impact of coronary artery disease on heart failure. Now the impact of heart failure on coronary artery disease heart failure can actually worsen CAD by increasing the myocardial oxygen demand and reducing coronary perfusion.

[08:16] And so basically, that's a compensatory mechanism. The same thing I was just mentioning early and heart failure such as increased heart rate and blood pressure can exacerbate the progression of atherosclerosis.

[08:28] Yes, I love some of the things I'm saying coming across the chat there. All right. And so with those relationships, kind of set the stage for that. And, you know, we're talking about the impact of all this on the body's oxygenation. Let's just go on into the, you know, talk about these separately. So I'll talk a little bit about some terms here when you're trying to classify anemia.

Anemia Classification and Microcytic Anemia

Starts at 08:54

[08:54] They're either microcytic meaning the MCV stands for mean corpuscular volume and that's really the size of the of the red blood cell. And when it's micro meaning small, that's a microcytic, that's a small red blood cell. And we do have certain anemia that are considered microcytic. One of the biggest ones and most common ones is iron deficiency anemia.

[09:23] And that's where the MCV is going to be anywhere between 80 to 100 will be within normal range. The biggest example that I can think of that and we will talk about it multiple times, not only here, but when we get into the renal content in week four, but in anemia of chronic disease, right? You've got it, cognitive kidney disease, right? And people with chronic kidney disease often do have anemia of chronic disease.

[09:53] So, and then macrocytic anemia is where the blood cell is going to be larger. That way is an MCV greater than a hundred. And so our two prototype diseases for this is going to be vitamin B deficiency and folic acid deficiency. Perfect. Absolutely.

[10:16] Thank you. And so when we look at the pathophysiology of microcytic anemia, there's an impaired hemoglobin synthesis because there's insufficient iron. Now, there are different ways that people can get insufficient iron. It could be nutrition related. They're not getting enough iron in their nutrition. They could have a very slow bleed somewhere in the body. They're losing blood.

[10:44] So that is one of the couple of ways that, you know, the insufficient iron could impair hemoglobin synthesis. And then thalassemia is one of the genetic.

[10:56] One of the genetic anemia is that there's actually a defective globin chain synthesis and that's again genetically related. So that will lead to some insufficient iron and a microcytic anemia. And then anemia of chronic disease, and I know that I just said earlier that anemia of chronic disease is normocytic. It is early on. And so I will talk about that again.

[11:25] Later, it will come up if not here tonight, it will come up in our renal content, but...

[11:33] clinical And so, and McCants talks about this as well as students often get confused about this point about anemia of chronic disease because on the little table, I think that's in McCants. It says it's normocytic or microcytic and it can be so it just depends on the stage. Like, the level, the level of it's a later on with anemia of chronic disease. It can go more into a microcytic pattern. So just took that away for later.

[12:01] And then iron deficiency anemia itself again, it's microcytic. It's also hypochromic, meaning it's not it's a pale. It's a pale blood cell. And so there's issues with hemoglobin synthesis, especially iron deficiency. And I do want to bring your attention to ferritin and that's a really important measurement. Just that it's a general measurement. If we draw ferritin level, it reflects the body's total iron stores.

[12:29] That's a good way to just find out if you think your patient is anemic. It is often very

[12:36] It is often low in iron deficiency anemia, but if you just want to draw a ferritin level to get an eye, just to get a general idea of the person's total body iron stores, that is the, that's the lab to draw. And so just in a general sense, the low ferritin does reflect anemia, but it can't really specifically determine which type, but oftentimes I will say the caveat with it,

[13:05] And there's ethylosemia again and so with theylosemia again this one of the inherited blood disorders that causes decreased circulating hemoglobin. There's so many genetic mutations of this but when you read about it what you're often hearing about is alpha and beta thalassemia and I won't get too deep into these but I will just say with alpha.

[13:30] thalassemia it's it's just caused by mutations in the alpha globin genes which which causes and reduced or absent production of the alpha globin chains and this is the reality of it really is based on the number of the genes that are affected. Now beta thalassemia is caused by mutations in the beta globin genes which leads to a reduced absent production of beta globin chains and the severity

[13:58] Is also based on the nature of the type of mutations there are and I know that you've heard of beta thalassemia minor, which is where one gene is affected and the person's basically asymptomatic or this might be a very mild anemia.

[14:14] Or major the beta thalassemia major, which there's much more severe anemia, which this is the one that requires the person to get regular blood transfusions. And so their symptoms will basically be just what we think about the typical symptoms of anemia like fatigue and weakness. And we could see

[14:38] Peller in their mucus membranes skin jaundice yellowing of the skin in the eyes splenomegaly where they have a large in large spleen and then also some bone deformities especially in the skull in the face due to there's an increased erythropoiesis as part of this and and so regular blood transfusions

[15:00] Always are of the what we have to do with those patients and there are other treatments, but we won't get to we don't have to get into a deep dive on that at this point. Okay. And then.

[15:15] Once again, here is the enemy of chronic disease. I did say I would mention it again, and here it is. So that's associated with this one. People have chronic infections, chronic inflammation, people with malignancies. And so basically, with this one, it's there's a moderate decrease in red blood cell production, and it's very distinct from

[15:40] Other types of anemia because of its underlying causes and and again, this one's relay just think inflammation and the immune response and basically with this one and I and I just put some just some some bullets here to just tell you about the path of this one. And so with there is an inflammation inside a kind production.

[16:04] Chronic diseases like people who have autoimmune disorders, chronic infections, cancer, cancer,

[16:11] Anything like this that triggers an inflammatory response in the body. This is mediated by the cytokines and you've heard of it in which are the interleukins, particularly interleukins 6 is the one involved in this and also tumor necrosis factor and an interleukin one as well. And so basically these cytokines will alter iron metabolism and

[16:36] And also alter erythropoiesis, which we have to have erythropoiesis for the production of our red blood cells. And so there's that process. And then along with the impaired erythropoiesis that's basically just in response to the inflammation. So that so so the kidneys are are producing.

[17:02] Less erythropoietin which is, and by the way, erythropoietin is a hormone and that does stimulate red blood self-production in the bone marrow. And then there is some inflammatory cytokines can directly suppress the.

[17:21] The proliferation and differentiation of these cells in the bone marrow that would that would eventually become red blood cells, which is going to reduce the overall production of the red blood cells. And then, and so with the decrease production of the red blood cells, then there's going to be iron metabolism changes with there's a term that you'll read in your in your McCants book.

[17:51] Hep sedin production HEP CIDIN and this is where we've got those again those inflammatory cytokines will stimulate the liver to produce hepcidin and that is a peptide that actually regulates iron homeostasis and hepcidin basically what it does is inhibit

[18:15] Iron absorption from the intestine and then it just basically traps the iron in the macrophages and liver cells, which makes it less available for a erythropoiesis. And then, and then, then we end up with a functional iron deficiency. So, and it need me of chronic disease, even, and this is where it's normocytic. Okay. Even though the body may have sufficient iron stores.

[18:42] We've got this increased hepcidin levels and then the impaired iron release from the macrophages that leads to this functional iron deficiency. So that so based. So this means that while the iron is present in the body, it's not available. That's the problem with it. And it's not available for use in erythropoiesis with the name of chronic disease. And so this is why you'll end up seeing this is why.

[19:11] The cells will still look normal and that's why that's the rationale for it being enormous cytic anemia.

[19:20] Which means again, normal size red blood cells, or then at some point we could see microcytic smaller red blood cells. It just really depends on the duration and severity of the chronic disease. And so the M. C. V. actually could be either normal or low while the hemoglobin levels are.

[19:44] Are decreased and so I guess overall, if we're going to say that overall anemia of chronic disease is a normocytic anemia. However, it could turn into microcytic depending on the level of duration of the disease. But for all practical purposes, we're gonna think about this one as as being normocytic. Okay. Now.