Facebook Live Event Transcript
Ask Me Anything – Featuring Dr. Richard Furman and Jeff Folloder
July 24, 2024

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This text is based on a computer-generated transcript and has been compiled and edited. However, it will not accurately capture everything that was said on the webinar. The complete recording of this webinar is available on-demand.

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Hi everyone. I'm Jeff Folloder, CLL/SLL patient and advocate.

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We are absolutely live with CLL Society's Facebook live event.

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Ask Me Anything.

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Where we spend the next 60 minutes answering your questions with a CLL expert, and we are so lucky to have Dr. Richard Furman joining us today. There are no presentations.

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We encourage you to ask your questions on the Facebook page. If this is how you're joining us or through the Zoom Platform.

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This event is dedicated to your questions, so ask them early, and make sure we get to them all.

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If we don't get to them, we'll figure out a way to follow-up.

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Before we begin,…

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I have a few important disclaimers to share. Nothing said today should be taken as medical advice. Any questions about your health and your treatment should be discussed with your healthcare provider.

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The information that you post on Facebook will be shared on a public forum. So please do not post or share confidential information.

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Without further ado, Dr. Furman, would you please introduce yourself for our audience?

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Thank you. I'm Dr. Richard Furman from Weill Cornell Medicine and I'm the Director of the CLL Research Center and...

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I'm...

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very happy to be here today.

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Fantastic.

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As you can imagine,..

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we…

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already have a ton of questions that have come in and the 1st question that I've got on my list is one of the questions that I hear very often on our support groups, and frankly, the entire community.

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I'll make it simple.

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What are the criteria for starting treatment...

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with CLL?

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I really think that it's so important for people to remember that,..

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you know, these criteria were established a very long time ago...

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and the criteria were really meant to...

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distinguish between patients who were...

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really quiescent and whose disease was not progressing...

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from patients whose disease was progressing.

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And it really wasn't meant to be an idea about how long can you delay treatment.

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You know, a lot of patients think that the longer you delay treatment...

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the more time you get at the other end.

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And I really don't think that's true. In fact,..

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some of us think there's actually...

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clonal evolution that happens.

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And that the longer you delay treatment, you may actually have...

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some bad genetic evolution occurring…

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when the disease begins to get active and intervening early, may actually help limit that.

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So waiting too long can sometimes be deleterious.

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So the idea is that when the cells...

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start…

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becoming more aggressive that's when you want to intervene.

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And that's really going to be characterized…

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by when patients start, you know, really start having a doubling of their white blood count in less than 6 months.

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And that's…

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of all of them really a softer indication.

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You know, I've certainly seen patients who might go from a white count of…

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40,000 to 80,000 to...

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you know,..

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160,000 and then just stop at 160,000 over the course of 12 months.

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But you know, that's certainly one thing that would show that someone's disease is becoming active.

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The more important ones would be the development of big, bulky…

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lymphanopathy, the development of splenomegaly...

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or the development of an anemia, and that's a hemoglobin less than 11,,.

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and the development of thrombocytopenia and that would be a platelet count of less than100,000.

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Those are really the classic criteria and remember that the anemia and thrombocytopenia...

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are really equivalent to Rai Stage 3 and 4.

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There's also, you know, B symptoms. So the development of basically...

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Fatigue, weight loss…

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fevers.

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And really, importantly, when patients do develop those symptoms, they sometimes could herald that something else is going on and it's important that those things...

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do get investigated.

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And I want to point out and I really do emphasize this, that when patients do develop fatigue or they develop sort of those other symptoms.

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It really is something that develops with disease activity.

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So when someone is first diagnosed with CLL and they have small amounts of disease,.

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now with low disease burden, you shouldn't have B symptoms.

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And the B symptoms would really...

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you know, develop with...

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either active disease or large tumor burden.

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And if you don't have that,..

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the fatigue is going to be more likely due to something else, and the…

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phrase that all my patients hear me say all the time,..

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you know, as we did a study, very tongue-in-cheek,..

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you know, 70% of patients with CLL report fatigue...

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and that 80% of New Yorkers...

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report fatigue.

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So of course,..

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you know, you can actually take that...

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at face value.

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I'm grinning because yes, I experience fatigue as a CLL patient but I also did 6.2 miles this morning at less than 14 minutes a mile, so...

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I can probably blame the exercise for the fatigue.

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Right.

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This is great information to have, the markers, if you will, for when to start treatment. Are there prognostic markers out there that might be able to predict the time someone has before they need treatment?

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So it's really important that these prognostic markers...

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are really of very little use currently in the management of patients with CLL.

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And these prognostic markers really serve to...

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provide a means for...

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people like myself…

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to get promoted.

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And that's really, you know, they serve very little purpose for patients but to generate anxiety.

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So when you have a large number of people...

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who have a large variability in their time to progression,..

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prognostic markers will give us an ability to predict how a population will do...

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but never the individual.

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So, even when you have all these different curves,..

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there'll be people progressing on the good curve before people progressing on the bad curve.

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And so that's really something that you always have to keep in mind.

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So, regardless of the prognostic markers, we're still going to look at...

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basically,..

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when someone progresses.

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Now, when I look at the markers,..

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there's really,..

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it's really most important to keep an eye on, what is the endpoint you're looking at?

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So most of our prognostic markers predict time to needing treatment.

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So, if someone needs treatment in two years versus four years…

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but everything is well in 20 years,..

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does the two years…

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of earlier treatment or earlier time to treatment make a difference, and the answer is probably not.

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So is that really an important prognostic marker? And, in my opinion, no.

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So when I look at prognostic markers, what really is important to me...

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are the ones that are going to predict how someone's going to respond to treatment long term.

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And those are the prognostic markets that really mean, you know, that are important to me.

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So...

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the currently...

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available prognostic markers universally...

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are going to be, you know, Zap 70...

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which is a protein on the inside of the cells.

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And being Zap 70 positive predicts an earlier time to needing treatment.

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CD38 which is a protein on the outside of the cells and that's being positive predicts for an earlier time to needing treatment.

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Interphase FISH which looks at...

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chromosonal changes...

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and those will be either…

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having a deletion of…

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17p…

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which is a, the “p”, the...

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short arm of the chromosomes and the “q” is the long arm.

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So chromosome 17p would be the short arm of chromosome 17.

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Deletion 11q which would be a deletion of the long arm of chromosome 11.

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Trisomy 12, which would be 3 copies of chromosome 12...

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or a deletion of 13q which would be a deletion of the long arm of chromosome 13…

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or basically normal, which really is none of the above.

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So there's really five different possibilities:..

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normal 13q deletion,..

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11q deletion, trisomy 12 and 17p.

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And those will create 5 different curves that will have different…

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predictions in terms of time to treatment.

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And then, and those are all...

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called interphase FISH, and then finally,...

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the last one is immunoglobulin gene mutational status.

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And that basically looks at the immunoglobulin genes and whether or not they're mutated or unmutated.

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And that just determines whether or not the…

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CLL cell has actually been under the influence of a T cell…

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during its development.

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And interestingly, being mutated…

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actually has a better prognosis…

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and the mutated immunoglobulin gene CLL...

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have a much more slower time or a longer time to needing treatment.

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So those are the prognostic markers that are more universally available...

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and those will predict for times to treatment.

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But you know, when we look at what's going to be important out of all of those,..

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really only the 17p deletion, which is one that actually predicts for what's called…

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The TP 53 gene, or the p53, protein function...

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really predicts for how patients do long term...

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with our current…

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and you know, our current novel therapies.

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So that's the one that really does become important because...

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you know, if you...

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have aTrisomy 12 or you don't have a Trisomy 12...

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you're going to do exceedingly well with the BTK inhibitor.

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You know, if you have a 17p deletion.

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You're going to do not as well as if you don't have a 17p deletion. So those are the things that really do matter…

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and really are things that could factor in, to how we should be treating or planning our treatments for our patients.

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That really does fill in a lot of questions and blanks for me. So, thank you for that.

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I started off this program by mentioning two acronyms,CLL and SLL.

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I would like for you to explain to our guests, one, are they the same? And if they're not quite the same, how are they different?

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And…

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what does it mean when my SLL changes to CLL? I thought this was a great question.

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So…

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no one's CLL changes to SLL and vice versa. They're exactly the same thing. And it's just so important to keep that in mind.

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The, you know, the whole idea…

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is actually in retrospect, hilarious.

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So in 1993, you know, a patient could actually have...

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A lymph node biopsy sent to the pathology department...

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and a bone marrow biopsy sent at the exact same time.

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And the pathologist would look at the lymph node and say…

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that this is a small lymphocytic lymphoma, or SLL.

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And then the same pathologist could look at the bone marrow biopsy…

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and say that this is chronic lymphocytic leukemia or Cll.

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And it's because anything in the lymph node...

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was a lymphoma...

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and anything in the blood and bone marrow was leukemia.

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But these were the exact same cells, with the exact same abnormalities…

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and they…

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terminologies and diagnoses for absolutely no good reason.

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So in 1994 when we came out with a new classification system...

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which was called the real classification system...

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for the Revised European Lymphoma Classification System.

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They just changed the name from CLL.

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or SLL or CLL/SLL.

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Not very creative...

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but that's the way they used it.

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For historical purposes,..

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some of the CLL physicians clinged to the idea that to have a diagnosis of CLL...

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you needed to have more than 5,000 circulating monoclonal B cells...

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and so in the peripheral blood.

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And so that definition still persists.

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And so you'll see some clinical trials still use it and you'll see…

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you know,..

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even the IWCLL, the international workshop on CLL, will identify...

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CLL as having...

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more than 5,000 monoclonal B cells in the peripheral blood...

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And SLL not.

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But they really are the same thing.

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And interestingly,..

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you can have CLL get treated...

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and then relapse as SLL.

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And...

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they're really the same thing,. there's no distinction, there's no difference…

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and what's really fascinating is we've never been able to really figure out...

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why there are differences.

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So interestingly, 11a deleted patients…

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do seem to have more bulky lymphadenopathy compared to other…

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interphase FISH abnormalities.

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Beyond that we really can't tease anything else out.

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And what's also really interesting is, you know, of course…

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when we take someone who has a lot of bulky lymphadenopathy and give them a BTK inhibitor…

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and we interfere with the BTK...

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enzyme, we actually interfere with the ability of these cells to hold on in the lymph nodes…

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and so they do fall out of the lymph nodes and fall into the blood.

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And you end up with basically, taking someone who might have been an SLL patient and making them a CLL patient.

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But they've always been a CLL/SLL patient all along.

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So it's an old terminology that should never be used.

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And patients are always a CLL/SLL patient.

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One of the things that's really, I think, important to keep in mind, medically speaking…

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is that when someone does get diagnosed, like I'll have a patient come in with lymphadenopathy…

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and a lymphocyte countin the peripheral blood of maybe about, you know, 4,000.

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And you know 4,000 lymphocytes in the purple blood is…

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elevated.

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And I know that there might be...

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2,000,…

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you know CLL cells in the peripheral blood...

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and I'll be able to use those 2,000 cells to make a diagnosis of CLL/SLL.

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But I might not be able to do what's called next generation sequencing and look for all the mutational profiling for prognostic purposes.

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So I can't get really a complete prognostic panel…

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from that patient.

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And so I might not be able to tell about p53 mutation or a NOTCH1 mutation…

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and all of those different things.

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And so it's important, like, if I have a negative FISH panel or a negative prognostic mutation panel…

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that I make a mental note and say, possibly negative, because insufficient cells in the peripheral blood, because there's always a sensitivity issue.

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And that's…

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where sometimes it gets a little bit difficult. And so it's always important to know…

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how many cells…

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that you're looking at really areCLL cells.

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And that's just the one caveat. And so sometimes even,

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you know,..

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that's where, when people get these prognostic markers, it's helpful to know exactly what the white count is.

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And I'll even, like after I give someone a BTK inhibitor,..

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But that's sort of the one caveat between a CLL and an SLL patient, I mean by and large…

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it's always nice to avoid doing a bone marrow biopsy and there's no reason to really do a bone marrow biopsy…

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if I can make a diagnosis off the peripheral blood.

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And you know, getting those additional markers…

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usually is not necessary.

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Speaking as someone who's had more than a couple of bone marrow biopsies. Thank you for saying that they're not always necessary.

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Usually or not, and I hate doing them.

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Good. I love hearing that. We've talked about what is CLL/SLL. We've talked about prognostic indicators or prognostic markers, and we've talked about criteria for starting treatment. One of the things that I've heard you mentioned a couple of times our BTK inhibitors. Soa two part question.

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Are BTK inhibitors the preferred frontline treatment for CLL and if so,..

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How do you decide..

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which one to give?

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So this is a, you know, it's a very...

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personal question. I guess personal is not really the best word, but it's...

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you know, everyone has their own biases. The physician and the patient.

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And that's really, you know, so,..

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there is no one best treatment.

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I do prefer BTK inhibitors for…

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frontline treatment in patients with CLL. And it's for a couple of reasons, and I think that they are more efficacious and safer and better tolerated.

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But you know the downside is that they're often continuous therapy that you need to stay on.

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But you know,..

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I think that they're much easier to use, and you know, and that's you know,..

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my own personal bias.

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A lot of people would rather just have, you know, a short duration of therapy and deal with everything else. So it's really,..

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a very personal approach that you have to take.

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So when we talk about BTK inhibitors,..

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we're talking about…

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basically,..

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we're talking about drugs that target Bruton's tyrosine kinase and Bruton's tyrosine kinase…

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is an enzyme that's present in a lot of different cells. And this is what's really interesting, it's present in B cells, and...

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it's present in macrophages, and then platelets andeosinophils and really,..

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many, many other cells, many more than we actually realized.

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But it seems to be most critical to B cells. And that's what's really so striking...

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as it turns out, you know, kids who are born without this enzyme...

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have really only a B cell defect…

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and they have no B cells and they end up succumbing to infections…

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at a very young age. Now they do quite well, because they can get immunoglobulin replacement therapy.

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But in 1950 there was actually...

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Colonel Ogden Bruton...

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identify these kids as having no immunoglobulins and coined the term…

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Bruton'sagammaglobulinemia.

00:20:03.000 --> 00:20:05.000
Turns out that it's…

00:20:05.000 --> 00:20:06.000
the…

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BTK gene is on the X chromosome and that's why it became x-linked agammaglobulinemia.

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They have no protein and that's why they have a slightly different phenotype.

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Fortunately for us,..

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we're just blocking the enzyme but there's still the protein there, so it's not as severe...

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and our patients do much, much better.

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The…

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other thing that's interesting is, you know, we block BTK and platelets.

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ButTEK, which is a similar protein, can actually compensate…

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in platelets. So these kids who are missing BTK...

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have normal platelet function because the TEK protein totally compensates.

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Now…

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ibrutinib, acalabrutinib, zanubrutinib and pirtobrutinib, which are the four BTK inhibitors currently approved,..

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all block BTK fully.

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And then they all block TEK to varying degrees.

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And so, because they block TEK, they cause platelet dysfunction and that's why we get bruising and bleeding.

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Now the amount they block TEK is actually what causes the varying degrees of bruising…

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and so that's why it's sort of interesting. And we've learned now about TEK and platelets only because of what we've seen with the BTK inhibitors.

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So it's sort of this interesting aspect of it all. And what really makes all this also a little bit more complicated…

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is because we use these drugs at fixed doses…

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so…

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everyone who gets, you know, 420 milligrams of ibrutinib...

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will have BTK fully inhibited.

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But TEK won't be fully inhibited and the amount of TEK that's inhibited is going to be determined by basically how large you are.

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And so the bruising does correspond to sort of the size of the individual…

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such that someone who's small will have much more bruising than someone who's large.

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And so it's almost titratable.

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And of course, you know, these are all different things that will sort of factor out. And so it's sort of an interesting…

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scenario, and a lot of these things can help impact sort of the choice of your BTK inhibitor.

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So,..

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in general, when we talk about BTK inhibitors...

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the other big major category is going to be BCL2 inhibitor or venetoclax…

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and there's a new one calledsonrotoclax which is coming very soon.

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And the...

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BCL2 2 inhibitors work…

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differently in the sense that they actually target the BCL2 protein…

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and really cause a great deal of,..

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basically,..

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cause the mitochondria to collapse and they cause the cells to actually...

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undergo cell death very rapidly.

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So whereas the BTK inhibitors, by targeting BTK can cause…

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the way I look at it is, you know, ibrutinib, which is the 1st one,..

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causes typically, you know, bruising and diarrhea. It can cause...

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joint aches and hypertension,..

00:23:00.000 --> 00:23:03.000
atrial fibrillation…

00:23:03.000 --> 00:23:04.000

and

00:23:04.000 --> 00:23:06.000
nail and hair changes.

00:23:06.000 --> 00:23:14.000
And then you can have the second generation which actually cause fewer problems. So acalbrutinib causes basically bruising…

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and headaches.

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And you have...

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zanubrutinib which can cause really just bruising and hypertension…

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and maybe a little bit of neutropenia. And then pirobrutinib which causes pretty much just bruising.

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The tBCL2 inhibitors really just cause tumor lysis and thrombocytopenia.

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And so it's a slightly different...

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problem because the tumor lysis is something that happens very quickly and can be dangerous and just needs to be monitored very quickly.

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And so it's a matter of…

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sort of choosing sort of the...

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side effects...

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and the monitoring that's required, because the…

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venetoclax will require a lot of monitoring very early on when you administer it…

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because you have a lot of potential tumor lysis that occurs…

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whereas with the BTK inhibitors you can just give someone a pill and say, I'll see you in 3 months.

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So it's sort of those differences that allow for people to be treated very differently.

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Overall, I think that the big differences between them…

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also factor in into the sense that, besides the risk of bruising…

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and bleeding for the BTK inhibitors which don't exist with venetoclax, I think is the one big thing that helps me decide between one category and the other, and the continuous therapy for one versus the…

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typically the year duration of therapy for the...

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venetoclax,..

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you know, it's really just the, you know, the patient's desire to be on a long-term therapy or not.

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So those are really the big decisions, I think, that factor into that.

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Very good. The BTK subject, we've received a lot of questions.

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You mentioned atrial fibrillation? And speaking of someone who was literally just at the cardiologist last week, are there concerns about these drugs and cardiac patients?

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Yeah. So one comment in general, which I did want to make is, you know,..

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when we look at the package insert, and, as you see on advertised on television, you know,..

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whenever we do a clinical trial, everything that happens to a patient has to get included in the package, insert so...

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if someone got hit by a bus crossing York Avenue on the way to my office,..

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the package insert for that drug will say, taking this drug could cause you to get hit by a bus.

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And it's important to recognize that,..

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you know,..

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when we talk about these drugs, you know,..

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as the investigator, I know what is drug related and what's not drug related.

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But that's not what goes in the package insert.

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So, there's always a background incidence and it's always hard to discern what might be real and what might not be real.

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It's also important to distinguish,..

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you know,..

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a lot of these drugs have, when they're especially head-to-head studies, differences in time on treatment.

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So...

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like the bendamustine rituxamab versus…

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the,..

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you know, acalabrutinib study,..

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you know, patients were on bendamustine rituxamab for six months but on a acalabrutinib for five years.

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So…

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having five years of follow-up…

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is going to generate far more adverse events compared to just six months.

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So these are all things that get lost…

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in the follow-up, but in general…

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you know, atrial fibrillationis sort of the important one...

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because that really has a lot of issues...

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in terms of the risk of anti-coagulation and cardiac…

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problems and so forth. So ibrutinib absolutely does cause an increase...

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in atrial fibrillation and…

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you know, the numbers are, of course hard to pin down because there is always an incidence in the older population.

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You know there was a very good study done out of the Mayo clinic which…

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says, in a general population of people needing treatment for CLL...

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it's probably going to be about 5 to 7%.

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in patients on treatment foribrutinib...

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it's probably going to be about 15 to 17% so it is about a threefold increase.

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When we look at acalabrutinib and zanubrutinib,..
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it's looking like it's actually going to be around the 5 to 7% range…

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so we don't think it's increased for those...

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but it's, you know, always hard to tell for certain…

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but it's definitely increased for…

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ibrutinub.

00:27:42.000 --> 00:27:45.000
Interestingly, hypertension…

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also definitely increased, foribrutinib.

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Definitely increased for zanubrutinib as well, but not as much for aibrutinib and not increased for acalabrutinib. And so that's also a...

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cardiac issue that…

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you know, is something that should be accounted for as well.

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Pirobrutinib...

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does not look like it causes either…

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atrial fibrillation or hypertension...

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which is also something nice to see.

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And it's something that should be considered as well.

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So, it sounds to me like once again we have to remind patients and their caregivers…

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to be candid with your care team.

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Let your medical professionals know what's going on. If you have a history of hypertension, if you have a history of cardiac issues…

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that information needs to go into your matrix to help make better decisions. Is that a fair statement?

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Absolutely. And I really think you know, a lot of physicians…

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we'll get comfortable with something and not switch. And I think that's really a bad thing to do.

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And you know,..

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I actually, you know, started...

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prescribing ibrutinib in 2009 or 2008 even.

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And I have not prescribed ibrutinib since…

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2019 now.

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You know, it's important to evolve…

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and we have better agents now, and it's important to recognize that.

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And I think that, you know, a lot of patients who are on ibrutinib might do better on some new drugs and...

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you know people are afraid to…

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try different things, and you know, listen,..

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a lot of the things that's important also is a lot of these adverse events happen early on. So if you've been on a drug for five years and you're fine,..

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you very well, may just be fine, and that's important, too right So it's hard to know.

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We do know that when we look at studies, the adverse events, cardiac wise withibrutinib...

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we're seeing predominantly in people over the age of 70.

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So that's another thing to take into account as well. So there's a lot of differences and it's,..

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I know. the pragmatic issues related to some insurances, only wanting to pay for a ibrutinib, you know versus zanubrutinib,...

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you know, so it's always hard to sort of…

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figure out which battles you have to fight.

00:30:07.000 --> 00:30:08.000
It's nice to have choices.

00:30:08.000 --> 00:30:13.000
And you know we'll have more choices in the not too distant future.

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I'm going to hold you to that one of the things that I…

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have witnessed in my…

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14, almost 15 years of CLL...

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is that when I started there wasn't a whole lot to choose from, and now there's…

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so much to choose from, and hearing doctors like yourself say, and there's even more coming. So I'm holding you to that because I,..

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I personally want…

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better treatments with less toxicity that are more effective. I want it all and I want it now.

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So I think I heard that song, but the,..

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you know, the one thing that I just want to add to what we were talking about earlier…

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and I think that's really more important…

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is, you know, not just whether it's a BTK inhibitor or whether or not it's a BCL2 inhibitor…

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but these combinations, you know, a lot of like, you know, my worldview is that 80% of patients with CLL...

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have genomically stable disease and will do exceedingly well with just a single agent BTK inhibitor, and not need anything else.

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20% might have the ability to evolve and behave a little bit more difficult,..

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a little bit more aggressively…

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and they will actually, will be well-controlled with just using a combination of agents.

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Now I do think the anti-CD20 monoclonal antibodies...

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are,..

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you know, they're a little tough on the immune system because they knock out the normal B cells and they kill your immunity and they'll…

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actually destroy your, you know, responses to prior vaccinations.

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But you know wha,t if you were to use instead of a venetoclax plus obinutuzumab, venetoclax and if you were to use plus a BTK inhibitor,..

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all of a sudden you preserve your prior immunity. You get the two agents that are synergistic and,..

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you know, take care of what you need to. And so it's sort of like, you know, we've done the figuring out and we have the tools available to us. And that's really the important thing.

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So you know, we have what we need. And it's really sort of a very,..

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you know,..

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and I think you know, I'm very risk adverse, and that's why I sort of…

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like avoiding the anti-CD20s and…

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taking advantage of all these great new agents.

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It sounds great. I want to shift…

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because…

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I'll share with you, coming up on Friday, . I am headed off to a birthday party for my uncle.

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He is the last uncle that I have on my father's side, and we're all looking forward to celebrating time with him.

00:32:45.000 --> 00:32:49.000
My uncle also has CLL.

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What's the current research on genetic risk factors for CLL and is it familial? Is it just chance? Is there something going on here?

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So CLL is a fascinating disease and,..

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you know, the one thing that we know about…

00:33:07.000 --> 00:33:10.000
it has a tremendous ethnic predilection.

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So there's no CLL

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in Japanese and Native Americans.

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And as you move...

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west across Asia into Northern Europe, the incidence increases.,..

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You know, the incidence is probably about 400,000 in Northern Europeans.

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But what's interesting is the incidence actually is double in Ashkenazi Jews.

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So...

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it really does…

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follow ethnic,..

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it segregates along ethnic lines...

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which is really quite interesting, because, you know, ethnicities segregated about the same time the human leukocyte antigens...

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evolved.

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And those are the proteins that determine how the immune system sort of recognizes self and non-self.

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And it's sort of like, you know, when we do bone marrow transplants in patients,..

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we always look in the same ethnic group and that's because we need to match those HLA proteins.

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And so I've always taken this to sort of imply that the HLA proteins…

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are probably sort of a predecessor, or a predictor, or a necessary...

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risk factor for the development of CLL.

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And so you know, all the Ashkenazi Jews are going to have closer HLA proteins…

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to each other, then they will to, you know then, people who are, you know,..

00:34:29.000 --> 00:34:32.000
of a different ethnic group. And so it's sort of,..

00:34:32.000 --> 00:34:38.000
you know, that similarity, that sort of enables everyone to sort of have that same risk factor for developing CLL.

00:34:38.000 --> 00:34:40.000
Of course, an uncle…

00:34:40.000 --> 00:34:41.000
and his nephew…

00:34:41.000 --> 00:34:45.000
are going to be far closer in their HLA match…

00:34:45.000 --> 00:34:48.000
then, you know, too distant Ashenazi Jews. So it's,..

00:34:48.000 --> 00:34:51.000
you know, that sort of thing as well, so…

00:34:51.000 --> 00:34:58.000
that's sort of how I put together the genetics, you know. It's not a gene like blue eyes where you have,..

00:34:58.000 --> 00:35:02.000
you know, a couple of different choices, and you either get it or you don't get it.

00:35:02.000 --> 00:35:03.000
You know,..

00:35:03.000 --> 00:35:05.000
HLAis probably a,..

00:35:05.000 --> 00:35:07.000
you know, 45 different…

00:35:07.000 --> 00:35:09.000
gene...

00:35:09.000 --> 00:35:11.000
process and, you…

00:35:11.000 --> 00:35:15.000
know, inherit all these different genes…

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and then you need to have…

00:35:17.000 --> 00:35:20.000
all those genes get turned on in a certain manner…

00:35:20.000 --> 00:35:22.000
and then a certain...

00:35:22.000 --> 00:35:25.000
stimuli to that cell has to occur.

00:35:25.000 --> 00:35:28.000
So it's a lot of different things that must occur.

00:35:28.000 --> 00:35:31.000
And since you have billions of lymphocytes,..

00:35:31.000 --> 00:35:33.000
you know, it probably can occur…

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if you live long enough.

00:35:34.000 --> 00:35:36.000
And that's the other thing is,..

00:35:36.000 --> 00:35:42.000
you know, they say 3 to 5% of the, you know, US population is walking around…

00:35:42.000 --> 00:35:47.000
with CLL cells at extraordinarily low levels in their blood.

00:35:47.000 --> 00:35:50.000

We just, you know, need special techniques to detect them.

00:35:50.000 --> 00:35:57.000
And so it's sort of that type of thing that if we all live to 120, you know, CLL would be quite common.

00:35:57.000 --> 00:35:58.000
Indeed.

00:35:58.000 --> 00:36:02.000
And so it's sort of that type of scenario. And that's sort of the genetic connection. So I don't want people to panic.

00:36:02.000 --> 00:36:07.000
I think there's just two more things to say this, you know, when they look in Mexico,..

00:36:07.000 --> 00:36:12.000
where there's still a large Native American population. The incidence of CLL is 0.

00:36:12.000 --> 00:36:16.000
And there's a still, a large, pure Spanish population...

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and the incidence of CLL is…

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equal to that in Spain.

00:36:20.000 --> 00:36:22.000
And then there's all those people who are…

00:36:22.000 --> 00:36:23.000
interbred.,.

00:36:23.000 --> 00:36:26.000
and the incidence there is actually right in the middle.

00:36:26.000 --> 00:36:29.000
So it's sort of a blendable trait.

00:36:29.000 --> 00:36:34.000
And then, when we look at environmental factors like radiation,..

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so after World War II there was no increase in...

00:36:37.000 --> 00:36:41.000
Japan after the atomic bombs.

00:36:41.000 --> 00:36:49.000
And so they assume that radiation was not a risk factor for CLL. But, of course, that was on a non-permissive population.

00:36:49.000 --> 00:36:53.000
When they looked around Chernobyl, of course there was a spike in CLL.

00:36:53.000 --> 00:36:57.000
So radiation on top of a permissive...

00:36:57.000 --> 00:37:00.000
ethnic population did lead to an increase in CLL.

00:37:00.000 --> 00:37:03.000
So, it's sort of a very interesting…

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interplay of a lot of different factors.

00:37:06.000 --> 00:37:16.000
There's lots of layers to that onion, as it were. I'm going to ask a question in regard to supplements.

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A lot of people are very enthusiastic about adding supplements to their regimen. Some of them can have...

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great effect. Some of them can have no effect, and some of them can actually be hazardous to not just CLL patients, but people in general.

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Let's start off…

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with one that I hear all the time.

00:37:35.000 --> 00:37:40.000
Does vitamin D play a role in CLL outcome?

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So the vitamin D is a very fascinating topic, and I'm actually,..

00:37:44.000 --> 00:37:48.000
you know, a very big believer in vitamin D...

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because,..

00:37:49.000 --> 00:37:53.000
you know, when we look at the morbidity in our population,..

00:37:53.000 --> 00:37:59.000
you know, osteoporosis is probably the number one cause of morbidity…

00:37:59.000 --> 00:38:01.000
and,..

00:38:01.000 --> 00:38:10.000
you know, I don't know if vitamin D is the answer to osteoporosis. But right now, it's the one thing we can impact upon. So they did some studies and they showed…

00:38:10.000 --> 00:38:11.000
in…

00:38:11.000 --> 00:38:12.000
CLL patients…

00:38:12.000 --> 00:38:13.000
That,..

00:38:13.000 --> 00:38:20.000
you know, those who had lower levels of vitamin D did have more aggressive courses and worse outcomes.

00:38:20.000 --> 00:38:28.000
And so originally, I thought that maybe vitamin D may predict, you know, lower vitamin D levels may predict for more, you know, for a worse outcome.

00:38:28.000 --> 00:38:32.000

But you know it's hard to supplement, because it then, of course...

00:38:32.000 --> 00:38:35.000
everyone was taking vitamin D supplementation.

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Because, you know, this was actually, then everyone figured out about vitamin. You know, what happened is for so long we couldn't measure vitamin D levels.

00:38:43.000 --> 00:38:48.000
And so there was this period of five years when they then started measuring vitamin D levels.

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And the other thing about this study that was done, and this was done at the Mayo Clinic, in Rochester, Minnesota.

00:38:55.000 --> 00:39:02.000
I mean, the impressive part of the study was, they found that 70% of the people they assessed were vitamin D deficient.

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So we're talking about massive amounts of vitamin D deficiency.

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So all of a sudden, you know, oh, my God, everyone's vitamin D deficient.

00:39:10.000 --> 00:39:12.000
So,..

00:39:12.000 --> 00:39:16.000
you know, it's a real problem, because we just got to repeat everybody anyway.

00:39:16.000 --> 00:39:17.000
So,..

00:39:17.000 --> 00:39:18.000
you know,..

00:39:18.000 --> 00:39:21.000
it's sort of the type of situation where…

00:39:21.000 --> 00:39:28.000

alright, so let's replete everyone to what would be considered to be, you know, 30, and I think it's nanograms per…

00:39:28.000 --> 00:39:29.000
ml...

00:39:29.000 --> 00:39:31.000
became the target…

00:39:31.000 --> 00:39:35.000
for shutting off PTH and helping to prevent osteoporosis…

00:39:35.000 --> 00:39:36.000
and,..

00:39:36.000 --> 00:39:38.000
you know, that should actually help…

00:39:38.000 --> 00:39:39.000
prevent,..

00:39:39.000 --> 00:39:42.000
less worse, you know, osteoperosis.

00:39:42.000 --> 00:39:46.000
But it didn't really seem to have any of the other benefits that people started touting about,..

00:39:46.000 --> 00:39:51.000
you know, decreased cancer risk and all those other things. So...

00:39:51.000 --> 00:39:57.000
I advocate for vitamin D, getting people's levels over 30 only because of the prevention of osteoporosis.

00:39:57.000 --> 00:39:59.000
Nothing to do with CLL but that's how it got really…

00:39:59.000 --> 00:40:02.000
stuck in everyone's minds about CLL.

00:40:02.000 --> 00:40:07.000
The Mayo Clinic did try to do an intervention study where they gave people vitamin D...

00:40:07.000 --> 00:40:09.000
versus placebo...

00:40:09.000 --> 00:40:10.000
but…

00:40:10.000 --> 00:40:16.000
the study actually never got done because they found everyone was just taking vitamin D.

00:40:16.000 --> 00:40:18.000
Wow!

00:40:18.000 --> 00:40:23.000
I believe in vitamin D and getting it naturally.

00:40:23.000 --> 00:40:28.000
But I also know that as a CLL patient, I have a higher risk of skin cancer. So,..

00:40:28.000 --> 00:40:35.000
I'm the one wearing the floppy hat and the long sleeve shirt and hoping that I get enough sunlight to help me, but not...

00:40:35.000 --> 00:40:38.000
hurt me, wuestion mark?

00:40:38.000 --> 00:40:40.000
Well, but you probably couldn't…

00:40:40.000 --> 00:40:43.000
ever get enough vitamin D from sunlight.

00:40:43.000 --> 00:40:44.000
Period.

00:40:44.000 --> 00:40:45.000
I mean.

00:40:45.000 --> 00:40:47.000
So drink more milk and consider a supplement right?

00:40:47.000 --> 00:40:55.000

Well, you know, it's interesting. The lowest vitamin D level I've ever seen was in probably one of the healthiest…

00:40:55.000 --> 00:40:56.000
women,..

00:40:56.000 --> 00:40:59.000
you know, I imagine I mean she ran,..

00:40:59.000 --> 00:41:00.000
you know,..

00:41:00.000 --> 00:41:08.000
you know, 75 miles a week. She only ate organic foods. She, you know, I mean she had no supplement, you know, nothing she ate was...

00:41:08.000 --> 00:41:09.000
processed…

00:41:09.000 --> 00:41:14.000
and the only way vitamin D gets into our diet is by processed foods.

00:41:14.000 --> 00:41:19.000
And you know, and there's also a famous New England Journal paper about…

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a woman who,..

00:41:20.000 --> 00:41:23.000
you know, ended up with a disease called…

00:41:23.000 --> 00:41:26.000
Wernicke encephalopathy...

00:41:26.000 --> 00:41:32.000
because she had no thiamine in her diet and she developed cholecystitis or a gall bladder attack.

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And you know,..

00:41:34.000 --> 00:41:35.000
she was put on IV....

00:41:35.000 --> 00:41:38.000
sugar or dextrose…

00:41:38.000 --> 00:41:40.000
and ended up...

00:41:40.000 --> 00:41:45.000
developing this because she had, you know, only alcoholics get Wernickes...

00:41:45.000 --> 00:41:49.000
because they can't absorb thiamine, because of the alcohol. But…

00:41:49.000 --> 00:41:55.000
because she was eating no processed food, she had no thiamine in her diet because it's only put in processed foods.

00:41:55.000 --> 00:41:58.000
You know it, it's funny, I mean, you know,..

00:41:58.000 --> 00:42:01.000
have to be careful. There's a lot of…

00:42:01.000 --> 00:42:04.000
things that get into our food because we put them there…

00:42:04.000 --> 00:42:05.000
because,..

00:42:05.000 --> 00:42:06.000
you know,..

00:42:06.000 --> 00:42:12.000
evolution never thought about the fact that we needed to have, you know, bone health into our 70s.

00:42:12.000 --> 00:42:13.000
And that's,...

00:42:13.000 --> 00:42:15.000
that's the problem.

00:42:15.000 --> 00:42:19.000

Indeed! While we're talking about supplements,..

00:42:19.000 --> 00:42:28.000
I've got a list that's been added here, things like, EGCG, olive leaf, reishi, resveratrol…

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and other supplements.

00:42:29.000 --> 00:42:37.000
I know that there's a lot of enthusiasm for adding the extras to diet. Is, is there...

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any tangible benefit,. in your opinion?

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None.

00:42:41.000 --> 00:42:50.000
And I really have to emphasize that. And I really have seen harm in quite a few circumstances. And I really can't emphasize that. I mean…

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enough.

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You know,..

00:42:52.000 --> 00:42:55.000
they did a study from ECGC...

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and the amount of…

00:42:57.000 --> 00:43:02.000
basically green tea that you would need to take to get to the doses that they used…

00:43:02.000 --> 00:43:03.000
is extraordinary.

00:43:03.000 --> 00:43:08.000
And actually, there are a lot of problems with LFT abnormalities and so forth.

00:43:08.000 --> 00:43:09.000

And…

00:43:09.000 --> 00:43:11.000
certainly,..

00:43:11.000 --> 00:43:16.000
you know the treatment was far less well-tolerated than any BTK inhibitor…

00:43:16.000 --> 00:43:17.000
But,..

00:43:17.000 --> 00:43:23.000
you know, the benefits were actually far lower too. So why not just take the BTK inhibitor.

00:43:23.000 --> 00:43:29.000
But with all the others, as well, like resveratrol, which is in red wine, and all these other things,..

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you know, they work in the test tube.

00:43:30.000 --> 00:43:31.000
But of course,..

00:43:31.000 --> 00:43:33.000
they don't work in the body…

00:43:33.000 --> 00:43:34.000
and…

00:43:34.000 --> 00:43:35.000
it's...

00:43:35.000 --> 00:43:42.000
you know, important to recognize that you know all these supplements. There's a change in the laws in the 1990s…

00:43:42.000 --> 00:43:45.000
where anything that's considered to be a supplement…

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didn't have to substantiate their claims just like...

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Tony the Tiger can say that frosted flakes are great…

00:43:51.000 --> 00:43:54.000
without proving it in a randomized, controlled trial.

00:43:54.000 --> 00:43:58.000
So these supplements can actually make these same claims.

00:43:58.000 --> 00:44:01.000
And that's what spawned this whole industry.

00:44:01.000 --> 00:44:04.000
And the problem, of course, now is that people have,..

00:44:04.000 --> 00:44:05.000
you know,..

00:44:05.000 --> 00:44:09.000
been fooled into thinking that these are true claims.

00:44:09.000 --> 00:44:10.000
And…

00:44:10.000 --> 00:44:14.000
they also don't have to market or list what really is in them.

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And there's some reports of people who have…

00:44:17.000 --> 00:44:22.000
been taking roots for energy that have been laced with amphetamine additives and…

00:44:22.000 --> 00:44:26.000
caffeine, and you know other stuff, too, that can be dangerous. But…

00:44:26.000 --> 00:44:31.000
none of these have actually been have been shown to have any benefit whatsoever, and...

00:44:31.000 --> 00:44:40.000

I've seen people, you know. St. John's wart actually degrades the BTK inhibitors by activating the Zip34 enzyme…

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so anyone who doesn't respond to a BTK inhibitor,..

00:44:44.000 --> 00:44:46.000
the first question I ask is, you know,..

00:44:46.000 --> 00:44:50.000
you've got to stop your, you know, what supplements are you taking.

00:44:50.000 --> 00:44:57.000
Because, you know, a lot of these supplements will have St. John's and then that will destroy the BTK inhibitors.

00:44:57.000 --> 00:44:59.000
Wow!

00:44:59.000 --> 00:45:09.000
We've kind of danced around the very large bear in the room. A couple of years ago, this unwelcome guest showed up...

00:45:09.000 --> 00:45:10.000
to…

00:45:10.000 --> 00:45:12.000
planet Earth by the name of Covid.

00:45:12.000 --> 00:45:14.000
And Covid,..

00:45:14.000 --> 00:45:18.000
absolutely is a serious...

00:45:18.000 --> 00:45:19.000
health…

00:45:19.000 --> 00:45:27.000
situation that has affected millions and millions of people, especially those who are immunocompromised.

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Let's take a few moments to talk a bit about Covid. We've got things like, is there a role for half-dose Covid? How worried should I be about traveling? Should I mask?

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I'd like you to address the Covid situation from both...

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a clinical standpoint and a practical standpoint.

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I mean, I really feel that Covid has evolved into so much more of a...

00:45:55.000 --> 00:45:58.000
an innocuous disease now that…

00:45:58.000 --> 00:46:01.000
people could resume normalcy. And I, you know,...

00:46:01.000 --> 00:46:03.000
life has to go on…

00:46:03.000 --> 00:46:09.000
and we also have Paxlovid now. And so I, I do advocate for people to just rejoin society…

00:46:09.000 --> 00:46:12.000
is the expression that I use.

00:46:12.000 --> 00:46:17.000
You know, so someone does get Covid and we need to do something, we have Paxlovid.

00:46:17.000 --> 00:46:24.000
Yes, I'm agreeing with you, because for some strange reason I managed to avoid Covid...

00:46:24.000 --> 00:46:29.000
for a very long time until just this past January.

00:46:29.000 --> 00:46:44.000
I got Covid. I asked for Paxlovid. I got Paxlovid, and I got better, and I'm living my life. I know that there are people that are still very concerned. Is there...

00:46:44.000 --> 00:46:49.000
any situation where they should take extra precautions?

00:46:49.000 --> 00:46:53.000
I really don't think so. And I really think that a lot of people are very...

00:46:53.000 --> 00:46:57.000
worried and the truth is,..

00:46:57.000 --> 00:46:58.000
you know,..

00:46:58.000 --> 00:47:02.000
everyone has to decide for themselves. But you know,..

00:47:02.000 --> 00:47:07.000
I think by and large, you know we're not losing people to Covid.

00:47:07.000 --> 00:47:12.000
And that's sort of the important thing to keep in mind. You know, I've had a few patients who have developed…

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long term complications like a bronchiolitis obliterans, where it's an inflammation of the lungs. But we get people through that as well…

00:47:20.000 --> 00:47:21.000
and…

00:47:21.000 --> 00:47:24.000
so at this point in time, you know,..

00:47:24.000 --> 00:47:26.000
people could resume normalcy.

00:47:26.000 --> 00:47:31.000
Now I'm a big advocate for vaccines and I do believe the Covid vaccines are safe.

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And anytime you vaccinate 350 million people simultaneously, you're going to have issues develop.

00:47:39.000 --> 00:47:42.000

I do recommend getting vaccines every 6 months.

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And you know, just continue to vaccine people. I don't recommend half dose vaccines because I think obviously, you know, the vaccines are only tested at full dose.

00:47:54.000 --> 00:47:57.000
And we don't know if CLL patients are going to respond...

00:47:57.000 --> 00:48:02.000
to the full dose. Even so, why use a lesser dose.

00:48:02.000 --> 00:48:06.000
You know, measuring antibodies is always an important question and we...

00:48:06.000 --> 00:48:13.000
know that the T cell response is probably the more important part of the vaccine.

00:48:13.000 --> 00:48:17.000
So antibodies never really even predict for protection. So...

00:48:17.000 --> 00:48:23.000
I don't even bother measuring the antibody response. So I just recommend vaccinations every 6 months.

00:48:23.000 --> 00:48:29.000
Good handwashing, you know, not touching your eyes, nose, and mouth is always the best answer…

00:48:29.000 --> 00:48:32.000
because that's how most people are getting infected.

00:48:32.000 --> 00:48:36.000
And you know, and it's just about being smart in those regards. I mean, I,

00:48:36.000 --> 00:48:40.000
you know, travel on a plane is fine, you know, when people travel,..

00:48:40.000 --> 00:48:42.000
you know, it's usually the jetway…

00:48:42.000 --> 00:48:49.000

and it's usually the small rooms that people are getting infected in. You know the plane itself, the air exchange...

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is actually quite good.

00:48:51.000 --> 00:48:53.000
That's the virus accumulating in the room...

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and then you, breathing in that air that causes the problem.

00:48:57.000 --> 00:49:02.000
The air circulation on the plane is actually good enough that you're not getting sick on the plane.

00:49:02.000 --> 00:49:05.000
So, you know, it's stuff like that. I think it,.

00:49:05.000 --> 00:49:07.000
you know,..

00:49:07.000 --> 00:49:13.000
it's okay, just to, you know, go out there,.

00:49:13.000 --> 00:49:18.000
You know, and you know, listen, there's the RSV vaccine which I think everyone should also be takin, and,..

00:49:18.000 --> 00:49:21.000
you know, I do recommend doing Prevnar 20...

00:49:21.000 --> 00:49:26.000
Pneumovax 23, and Shingrix, and all those vaccines, but,..

00:49:26.000 --> 00:49:34.000
you know, people can do really quite good with everything. Now, the one other thing everyone always worries about is, you know, this Paxlovid…

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or Covid rebound or Paxlovid rebound.

00:49:37.000 --> 00:49:38.000

And I don't think that…

00:49:38.000 --> 00:49:45.000
is really as bad as people make it out to be. And if you remember in the beginning, we used to always talk about how people would sort of…

00:49:45.000 --> 00:49:48.000
Putter along for two weeks, and then get really sick.

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So what most of the Paxlovid rebound is really the people who took the Paxlovid...

00:49:53.000 --> 00:49:54.000
during, that…

00:49:54.000 --> 00:49:56.000
first two weeks…

00:49:56.000 --> 00:49:59.000
and then got sick really, at the two week mark.

00:49:59.000 --> 00:50:01.000
And you know,..

00:50:01.000 --> 00:50:06.000
as it turns out, only about 5 to 7% of patients have a real rebound…

00:50:06.000 --> 00:50:10.000
when you do it, you know, looking at the actual viral proliferation.

00:50:10.000 --> 00:50:13.000
And the worst case scenario is, you just take two,...

00:50:13.000 --> 00:50:16.000
two courses of Paxlovid….

00:50:16.000 --> 00:50:18.000
so it's not really a big deal.

00:50:18.000 --> 00:50:21.000
It tastes a little nasty, but it's not a big deal.

00:50:21.000 --> 00:50:22.000

Right.

00:50:22.000 --> 00:50:25.000
So tell me about Pemgarda.

00:50:25.000 --> 00:50:33.000
So, you know, it's interesting. I mean, it's another monoclonal antibody. It's administered intravenously every three months.

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It's meant to help protect against symptomatic Covid, it's not meant to be used in people who have Covid or have been exposed to Covid. So it's not a…

00:50:42.000 --> 00:50:45.000
pre-exposure, prophylaxis.
[Correction, Pemgarda is a pre-exposure prophylaxis for Covid]

00:50:45.000 --> 00:50:46.000
I'm actually,..

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not sure it's yet commercially available. Last time,..

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you know, it was still, you know, it's approved. It's not fully approved. It's just an emergency use…

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authorization.

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So it hasn't gotten full approval yet. So it's still considered to be experimental.

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So obtaining it is still hard. I mean, there is on the website, you can actually go to find out where it's being administered.

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But it's not something that's being universally distributed.

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I'm not so sure…

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that…

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you know, it's benefits are there. Remember, a lot of the benefits are going to be measured in its ability to prevent people to be hospitalized.

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But there's so few people being hospitalized…

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that, you know, it's sort of hard to measure a benefit.

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Interesting, interesting. I've got a question here that a lot of the questions have hit me rather personally.

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For patients with chronic sinusitis, what interventions are likely to be of the most help?

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And I say that is someone who had the balloon sinuplasty and the deviated septum fixed and for the first time in 35 years, I can actually breathe through my nose well. So, I feel the pain of chronic sinusitis.

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Can you help us?

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Absolutely, so...

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common things occur commonly, that is always the first rule of medicine.

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And the most common cause of a chronic sinusitis in any patient, even a CLL patient with...

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hypogammaglobulinemia…

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is going to be, you know, a deviated septum,...

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you know, it’s going to be polyps, it's going to be, you know, just crud blocking the flow of…

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sinuses.

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And so,..

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you know, it's basically the sinuses are channels inside the skull bones.

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And when,, you know the mucus...

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basically can't flow, the bacteria basically grow and cause inflammation and pain and infection…

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So,..

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you know, antibiotics are going to kill the bacteria, but if the bacteria can't get out, they're just going to regrow again.

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And the immunoglobulins do play a role in helping suppress…

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those infections but…

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if,..

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you know, you’re hypergammaglobulinemic, you're certainly more sensitive to having those bacteria take hold.

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But even if you have antibodies, and you still have…

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obstruction, you're just going to get stuck with chronic sinusitis. So...

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my general rule in CLL patients...

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who are hypergammaglobulinemic are definitely more at risk of having…

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chronic sinusitis, chronic bronchitis recurrent pneumonias, and even life threatening infections.

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But the idea is that the first step in helping anyone who's having any of those issues is to make sure…

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using through an ENT is that you address the anatomical abnormalities.

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And so I always have, you know, make sure the channels are open. Make sure there's not a polyp. Make sure there isn't a deviated septum.

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Making sure that there isn't an allergic component. So if there is an allergic component, you know, something like a daily antihistamine or nasal steroids…

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could sufficiently calm the inflammation to help…

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the flow get started.

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The next thing is to make sure something like a Nettie pot or some nasal irrigation…

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just to,

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keep, you know, the channels open...

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is also a very good start.

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And those are things that can really,..

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by keeping the

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mucus down…

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will help prevent the infections from building.

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Now, if someone's continuing to have recurrent symptomatic sinusitis,..

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you know,...

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IVig can be very helpful, and you know IVIg is extremely well-tolerated. The only downside to the IVIg is it's a hassle.

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So it's typically a three hour infusion every four weeks.

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It's well-tolerated. It's just…

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you have to sit there for three hours every four weeks.

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It only works while you're getting it.

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But sometimes…

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patients can get it for six months and…

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you'll have like a healing of the sinuses and maybe then you'll be able to stay free and clear.

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Some people really just need it during,..

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like the spring when their allergies really kick in…

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and it sort of will protect them when things are worse. So there's ways that we can try to...

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make it as,..

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you know,..

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not as cumbersome for the patient.

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And that's something that can be really helpful for patients.

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I have some patients who really run into trouble and we do things like add antibiotics to their nasal rinses…

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or we'll give them nebulizers. With the nebulizers you basically

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aerosolize the antibiotics and they breath it into their sinuses to try to...

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reduce the bacterial load. So there's a lot of things that we can do to try to minimize it.

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But for patients who have real bronchitis or pneumonias…

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or life threatening infections, you know, they'll have low antibody levels and just need to have those antibodies systemically. You know, they're ones who are just getting the IVIg anyway, and for them it's,..

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obviously,..

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could be life saving.

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Fantastic Dr. Furman. This has been great.

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I started off the presentation by saying, “we're going to get to all the questions”. And as it turns out, I wasn't 100% candid. We did not get to all the questions.

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I hope that you'll come back and join us again before we…

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My pleasure. I would love to.

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Before we close the program. Do you have some closing thoughts for our audience?

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Yeah, you know, I really think you know,.

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from my perspective, we have so many great…

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treatment options for CLL patients…

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and I really think, you know,..

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in 2024,..

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no one should die from CLL...

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and that everyone should hopefully, be able to enjoy normal longevity.

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And just avoiding things that are going to impact upon quality of life is the most important thing.

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You know, I, no one should ever get chemotherapy, and I do know there's some physicians out there still using chemotherapy, and I really have to,..

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you know, argue, or try to persuade people to make sure you avoid it.

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You know I do have a huge bias against…

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anti-CD20 antibodies, and I know that's not something…

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shared by all my colleagues, and I definitely know that I,..

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I know enough to know, I don't know everything.

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And you know,..

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that's my bias. But it's sort of the idea that,..

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you know, preserve the immune system and to keep people as healthy as possible.

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But it's, you know, we're not trying to cure people, we're just trying to get people to live…

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to 100 and just have…

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wonderful lives and,..

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you know,..

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sometimes, you know,..

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just making it simple...

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is really all that's necessary, and I think we can do that.

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Those are great words.

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Pleasure.

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Thank you so much for your time and for your expertise. We are very grateful. Yes, we are grateful for your participation.

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I'd also like to thank…

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everyone who joined us today.

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I'd like to thank our generous donors to CLL Society and grant support from AstraZeneca, BeiGene, and Genentech just for making this event possible.

00:58:16.000 --> 00:58:25.000
A few brief reminders. If you're a Facebook user, please remember to like and subscribe to the CLL Society Facebook page.

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This is an important part for me. Please complete the short event survey linked in the comments section on Facebook…

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and will be shared with everyone who registered. We really want to hear your feedback. If we don't hear from you, we don't know how to make this even better.

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Please join us on August 7th for CLL Society's next webinar; A Brighter Future for CLL. Learn How Your Legacy Can Have a Lasting Impact.

00:58:55.000 --> 00:59:02.000
If your question wasn't answered today, please send it to our Ask the Expert email service.

00:59:02.000 --> 00:59:09.000
This is a free service and can be found on the CLL Society website under programs and support.

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Please remember to follow CLL society on Facebook and on other social media platforms.

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Lastly,..

00:59:17.000 --> 00:59:22.000
CLL Society is truly invested in your long life.

00:59:22.000 --> 00:59:28.000
And you can invest in the long life of CLL Society by supporting our work.

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Thank you very much for your participation, and remember what I tell everyone;..

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people who take an active role in their own care,..

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have better outcomes.

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Thank you.