Webinar Transcript
Understanding the Numbers: A Patient's Guide to CLL/SLL Testing and Imaging
August 26, 2026

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Hello and welcome to today's webinar. I am Liza Avruch, program director with CLL Society.

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We are dedicated to bringing credible and up-to-date information to the CLL and SLL community, because we believe smart patients get smart care.

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As a reminder, you can rewatch all of our educational programs by going to the section of our website called Education on Demand.

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Prior to beginning, we'd like to mention a few pre-event items.

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All attendees in this webinar are muted, and the only people on camera are the speakers.

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We ask you to please direct all questions to the Q&A section displayed on your screen.

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Questions will be sent directly to our moderator, speakers, and staff and are not visible to the audience.

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After today's event, you will receive a very brief survey that will help us plan for future events. We greatly appreciate your feedback.

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This session will be recorded and made available to everyone on our website.

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Closed captions are available if you want to turn them on or off. Go to Captions and select Show Captions or Hide Captions.

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This program was made possible through donor support and grant support from our industry partners.

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At this time, I'd like to welcome our moderator. Thank you.

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Thank you, Liza. My name is Larry Marion. I'm a 20-year veteran of CLL, a member of the CLL Society's Patient Advisory Board, and a co-facilitator of the Boston CLL Society support group.

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Welcome to today's event.

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We are joined by our speaker, Dr. Heather Wolfe, who is Assistant Professor in the Department of Internal Medicine and the Director of the CLL Survivorship Unit at University of Texas Southwestern Medical Center.

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We will be answering audience questions at the end of this event, so please take advantage of this opportunity and type your questions in the Q&A box.

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Before we begin, I'd like to share a few important disclaimers.

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The information provided during today's webinar is for educational purposes only. It should not be considered medical advice. For any personal health or treatment questions, please consult with your healthcare team.

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Please note that while the CLL Society may have its own opinions and policies, the speaker may offer differing viewpoints, especially regarding management of CLL and its complications.

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Now it is my pleasure to welcome Dr. Heather Wolfe.

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Hi, I'm Heather Wolfe and I am an Assistant Professor of Internal Medicine and a CLL doctor at UT Southwestern in Dallas, Texas. And today, we have the opportunity to talk about…

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Understanding CLL Testing and Imaging: What Patients and Care Partners Need to Know.

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So, during our talk today, a couple things that we're going to review: how to interpret blood work…

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Testing and Imaging at diagnosis…

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What are these prognostic markers that we talk about?

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How do we monitor your disease?

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We'll briefly talk a little bit about treatment response and grading a treatment response once you start treatment. And lastly, we'll touch on measurable residual disease or MRD testing. And while this is…

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really kind of confusing and complicated material that we'll be covering. I hope to try to make this…

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approachable and…

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kind of more of a conversation, because a lot of this is things that we review at the first visit, so we'll try to make it like we are having a clinic visit together, to review and better understand a lot of this…

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Testing and imaging when it comes to CLL…

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So, to start off, and one of the first things that I talk to patients about in clinic is blood work, a complete blood count or CBC. This is something you'll get to know very well throughout your CLL journey. So, I want to hit some of the important…

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features of a complete blood count. So, first off, what is a complete blood count, or CBC? So, this is the way that we can measure all the different cells in your blood…

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So, there's 3 main types of cells that we think about in our blood…

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So, the first is the WBC or white blood cell count. These are our infection-fighting cells.

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Hemoglobin is our red blood count. Red blood is important in carrying oxygen throughout our body. And lastly is our platelet count. It's often the forgotten cell type, but these are tiny cell fragments that go to places whenever you get a cut and help…

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form a clot and stop the bleeding. So going back up to the white blood cell count, this in many patients with CLL will be one of the first abnormalities that we see.

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And to make life difficult, there's 5 different types of white blood cells, or infection-fighting cells…

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Most of our white blood cells are neutrophils and lymphocytes, and often you'll see in your CBC as a part of the differential, an absolute neutrophil and absolute lymphocyte count.

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We'll also see kind of less common types of white blood cells, monocytes, eosinophils, and basophils mentioned in the differential.

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But absolute lymphocyte count, or ALC…

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is a very important marker or lab test that we track in CLL. So, this is a way that we can measure the disease burden or the amount of CLL in your blood. And this will be something tracked…

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along the way, and we'll talk a little bit more about it in a couple slides. Another important white blood cell to monitor is the neutrophils.

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So, we think about in CLL, some of our cells are abnormal or bad. However, we want to also be sure that we have some good type of healthy white blood cells to keep our immune system strong. And that's how we can measure…

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Some of these other cells, including neutrophils. So, it is very important to make sure that you have an adequate neutrophil count along the disease course.

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So, besides the white blood cell, again, we talked about hemoglobin, which is important as a…

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measure of red blood cells, as disease can progress, you can see a drop in other cell…

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lines like the hemoglobin and platelets, just due to involvement in the bone marrow. So, here we'll monitor for anemia or low red blood cell count, and we'll also be monitoring your platelet count.

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So, just to summarize a little bit about what we talked about…

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First, white blood cells. This is a measurement of your total white blood cell count, or infection-fighting cells…

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Red blood or hemoglobin. So, hemoglobin is a way to measure your red blood cells…

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And we're monitoring for low hemoglobin or anemia, which can…

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contribute to some symptoms related to…

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CLL or disease progression. So, we watch for fatigue, shortness of breath, heart palpitations, or weakness, which could be a marker of progressive anemia.

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So, platelets, like we said, these are the tiny cell fragments that help with bleeding. A low platelet count can increase the risk of bruising or bleeding, which is why it's important to have an adequate platelet count as well.

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Absolute lymphocyte count, or ALC. This is a way that we can measure your CLL disease burden, because this is your number of circulating lymphocytes.

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And lastly, absolute neutrophil count, or ANC. This is the measurement of these good white blood cells. In patients that have a low neutrophil count can be at higher risk for infection, so we want to make sure that our good white blood cells are also adequate.

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So, when it comes to diagnosis…

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What tests are required to make a diagnosis of CLL, or what things do we expect to be drawn at diagnosis? So, a complete blood count with differential, that's the testing that we looked at on the last few slides…

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is often something that's been done before a patient is even diagnosed with CLL. That's often what trigger this initial workup. So, a CBC with differential is very important. Often, we'll also perform a comprehensive metabolic panel.

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This looks at your electrolytes as well as the health of your kidneys and liver, which can be important to monitor for any…

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side effects or…

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downstream effects related to CLL or treatment…

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Flow cytometry, we'll discuss a little bit more on the next slide, but this is often one of the first tests that's done to make the diagnosis of CLL, and then we'll spend a lot of time talking about prognostic testing or…

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CLL markers.

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Tests that we see that can be done…

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At diagnosis or during a CLL disease course, but these are not always routine or needed at diagnosis. These things include bone marrow biopsy, lymph node biopsy, and imaging. So, what I tell patients is that…

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these can be done if in case-to-case basis. However, these are not required for all patients at diagnosis.

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I often say if things are a little off or if…

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patients have unexplained symptoms…we may dig a little bit deeper with some of these other tests. But it is always important to know what you're looking for when you do perform these tests.

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So, a little bit about flow cytometry. So, I tell patients that…

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our cells in our blood have little flags or markers on the outside of them, and it's the pattern of flags which help me identify what these cells are.

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So lucky for us, we have a machine called a flow cytometry machine that helps us read these flags on the outside of cells.

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So, whenever we see this high white blood cell count, we can send your blood through this machine, through this flow cytometry, which will read the outside of these flags.

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And there's a specific pattern that's consistent with CLL that we'll often see on flow cytometry. So, CLL is one of these diseases that we can often make a diagnosis alone with flow cytometry.

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The other thing that flow cytometry does is it's able to tell us what percentage of your white blood cells or lymphocytes are this atypical pattern consistent with CLL. So, it can sometimes tell us the amount of disease that we see in the blood.

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So, imaging, as we talked about before, not all patients with CLL need imaging at diagnosis. However, there's a number of different modalities or types of images, imaging studies that we can do.

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So, first up and probably one of the most common is CT scans or CAT scans. And these are black and white images that can be performed sometimes of the neck, chest…

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abdomen, belly, pelvis, to help us look at the anatomy. So here we can see different organs, we can also look at size and number of lymph nodes…

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Oftentimes when looking at lymph nodes, we'll use things like intravenous contrast to better kind of describe organ structure, size and get details and size and shape of lymph nodes.

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The one thing about CT scans is you cannot determine kind of activity of these lymph nodes from a CT scan alone…

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Which leads me to PET scan. So, this is actually the same…

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patient on a CT scan and a PET scan, so you can see the difference between these two imaging studies. Over on a PET scan, we get anatomy. However, we also get metabolic…

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activity. So, with a PET scan, patients will get a radioactive tracer that gets taken up in cells that have…

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increased turnover…

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and…

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with this, you'll see kind of bright spots that usually are a sign or marker of…

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significant activity.

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We don't use PET scans in all patients, but one thing that this can be very helpful is because it can measure metabolic activity, this can be very useful if there's concern for Richter's transformation, or that…

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transformation from slow-growing CLL to a higher-grade disease or lymphoma. This helps us kind of target for a biopsy and pick out areas that seem to be growing faster than what we would expect.

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However, I would say CT scans are probably most commonly used. We use PET scans occasionally to look…

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for metabolic, increased metabolic activity. And the last imaging modality that we sometimes use is MRI scans. So, MRI is the one positive is we don't use ionizing radiation to perform an MRI…

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which is, of course, is a concern for many patients who do get lots of imaging. However, there are also downsides to MRI imaging, which includes the cost. Oftentimes, MRIs will be twice the cost of a regular CT scan.

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You also have significant increase in time with these scans. Oftentimes, CT scans can be performed in a couple minutes, whereas MRIs can take 30 minutes up to a few hours, which of course with a two-hour scan there can be tolerance issues with that.

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It's hard to lay still for an extended period of time. So, I would say MRI is not a common imaging modality. However, we have it in special circumstances.

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So, next up we have the prognostic markers, and this is very important…

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testing that should be done in all CLL/SLL patients.

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There have been 3 different tests that help us predict how a patient's CLL may behave, when they may need treatment, and which treatment may be…

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a better option for each individual patient. So, first step is CLL-FISH. So, this is a test that helps us look for common chromosomal changes in CLL. These are different changes that have been found in multiple studies…

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to have prognostic significance. That means help us predict how your CLL may behave.

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Many different changes that we see here, like deletion 17p, deletion 11q, deletion 13q, and trisomy 12.

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Next up is IGHV.

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This is a test that looks for a mutation in the immunoglobulin variable heavy chain.

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Results will show up as either mutated or unmutated, with mutated typically being consistent with a more favorable disease course.

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And last but not least, TP53 mutation. So, this is a test that looks for a mutation in the TP53 gene. And TP53 normally acts like a quality control for a cell. So, as you can predict…

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if you lose this protein through a mutation in the gene, you don't have that quality control and cells go on to divide and grow without any control, often going on to cause a malignancy or cancer.

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And TP53 mutations are often the highest risk kind of category for CLL. And again, the results will either be no mutation or mutated, which means that you have a mutation.

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The one thing that I always talk about is deletion 17p often encodes for a TP53 mutation. However, both FISH and TP53 mutation testing should be performed because you can have a TP53 mutation…

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outside of this deletion 17p, so just doing the FISH, you'll miss about 10% of cases. So, it's important to get all 3 of these studies to complete the comprehensive prognostic marker panel.

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Two other kind of prognostic tests that can be performed is chromosome analysis, which looks for additional chromosomal changes outside the traditional CLL-FISH panel. And last but not least, next-generation sequencing, which we'll discuss a little bit more in a few slides…

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which, again, looks for other commonly seen mutations in CLL

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So, CLL Society also feels strongly about sending these prognostic tests in all patients, so there is a campaign called Test Before You Treat, which tells patients and also informs…

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physicians about what testing that should be done in all patients, either at diagnosis or before starting any treatment.

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Again, I typically recommend FISH, TP53, and IGHV testing at diagnosis or before…

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frontline treatment, but we do also repeat both FISH and TP53 testing at relapse because some of these can change. IGHV…

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status does not typically change throughout a CLL patient's disease course. And this is important for a number of different factors, like we said, not only prognosis, but it does affect recommendations when it comes to treatment. So, chemotherapy should not be considered for…

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high-risk patients, and the more we're kind of learning and the better treatments we have, potentially no CLL patients at this time. But there is a QR code to learn a little bit more about the Test Before You Treat campaign.

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So, here's the next generation sequencing. So, this is a study that can look for genetic mutations seen in CLL patients. So, of course we'll see that TP53 mutation testing, but again, we'll get information on additional other mutations. So, often…

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when you send NGS test, you're getting a panel of hundreds of different genes.

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And some of these mutations can have prognostic significance or impact treatment decisions. However, this is not standard of care in all patients with CLL, and especially at diagnosis. So, I feel like it likely has more limited utility at diagnosis…

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but I do often use next generation sequencing, particularly in relapse, because I do want to know about other mutations like BTK mutations, which will help me kind of predict what would be the best next treatment in a patient with CLL.

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So, next generation sequencing is available. It can provide useful information. However, not standard of care, especially at diagnosis.

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So, after you're diagnosed, what should you expect?

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So, disease monitoring and active surveillance will often be the recommendation for a number of different patients. So, what goes on during this active surveillance?

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So, things that I'm watching at these surveillance appointments with patients. So, one, we'll perform a very thorough history. So, we'll see how patients have been feeling, we'll see if there are any new B symptoms, like the fevers, the night sweats, the weight loss…

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any new lymph nodes or bumps that they have felt, are there any new infections that have been going on?

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Then, we'll perform a detailed physical exam. So, this includes listening to the heart and lung, but also a very in-depth lymph node exam. Also, can we feel for a liver and spleen? So, these are ways that we can monitor disease activity.

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Again, here comes the complete blood count with differential. This will be performed at every visit. Because I want to see what's your red blood count doing, what's your platelet count doing? What are your lymphocytes? Are you having a doubling of the lymphocytes? Some of these things that may…

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contribute to us recommending treatment. And then again, we want to see the health of your immune system and your neutrophil count.

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Again, looking at the complete metabolic panel to see the health of your liver, kidneys, things like that. And lastly, immunoglobulins. I may not check this at every visit; however, immunoglobulins are your antibodies, and we know patients with CLL…

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can have a drop in the production of antibodies, leaving them at higher risk for infectious complications.

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So, if patients have been having recurrent infections, immunoglobulins will be something that I'm also monitoring to see who may need other supportive care strategies to help boost their immune system.

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So, I would put under the “sometimes helpful” category: imaging and biopsies. I do not think for normal disease monitoring that we need routine CT scans or PET scans.

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I do use these in some patients, particularly those that don't have a lot of disease in their blood and have sneaky lymph nodes kind of deep down in their belly that we're monitoring. And again, I don't think we need repeat bone marrow biopsies or lymph node biopsies for routine monitoring.

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However, if something does come up during the history exam labs, we may order these tests too.

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And the one other thing I want to mention with disease monitoring is how often are we doing these disease monitoring appointments? It actually often depends…

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to me on what your prognostic markers look like. So, for instance, if a patient's very low risk, I may only see them once or twice a year if things are very stable. However, higher risk disease, I may see them much more frequently…

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just because disease can change in shorter periods of time.

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So, moving forward a little bit more with disease monitoring, I like to tell patients that a single value does not tell the whole story. So, oftentimes, whenever a patient will come to clinic, they see that their white blood cell count is high. It's understandable to be concerned. However,…

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we don't make decisions or jump to conclusions based on one number. The trend and kind of pace of change is way more important. So, this is an example of two different patients who come in with the same white blood cell count of 100.

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So, if you can see in patient A,…

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kind of over a period of time, right? White blood cell count is maybe trending up, but it's been pretty stable over this year. However, this other patient has had a dramatic

increase in the white blood cell count, right? Same absolute value, however, very different story.

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So, it's important to keep symptoms, exam findings, trends into account when assessing patients. So, as we say, treat the patient, not the number.

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The CLL Society provides lots of good resources when it comes to lab results, how to interpret lab results, how to track your lab results. So, two good resources include normal lab values, how do your labs compare to normal lab values?

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And then also there's a really good Excel sheet which we'll look at on the next slide, which can be a helpful way to track your results. So, here's a little QR code on the bottom, which will link to these resources.

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So, here is the keeping track of the lab results Excel sheet, which allows you to track your values over a period of time. Of course, a lot of our different kind of MyCharts or electronic health records will help you do this too…

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but this is a way that you can do it at home as well.

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So, next up is response criteria. So, how do we know how we're doing after we start a treatment? So, there are different criteria that we use when assessing a patient's disease response. And again, just remember that this is only…

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pertinent or only useful after you've started treatment. So, there's a number of different factors that come in to determining your response. Some of these include lymphocyte count. What is your white blood cell count doing?

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Platelet count, hemoglobin, lymph node size, and then the assessing your liver and spleen. So, the easiest category is the complete response category. So, this is a full response to treatment.

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So, this means that your lymphocyte count normalizes. This means that your platelet count and red blood have also…

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become normal.

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We don't have on imaging any lymph nodes over a centimeter and a half, your spleen size has also returned to normal. So, the way that I think about for this is oftentimes all of these kind of abnormalities that we saw at baseline…

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have since returned to normal.

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On the other end of the spectrum, progressive disease is from baseline or before you started treatment, you're having an increase in your white blood cell count, a drop in the platelet count, drop in the red blood count, and an increase in lymph nodes and or spleen.

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So, if you're having…

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negative changes from baseline, that's classically more consistent with progressive disease.

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Stable disease means things are quite stable from your baseline, either imaging or laboratory data before you started treatment. And then this partial response means somewhere in between stable disease and a complete response, meaning…

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things are getting better, but they're not totally back to normal. So, this can be confusing. Don't get caught too much in the weeds, but it can be helpful just to understand where…

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your response is after initiation of treatment, and I do also want to note that the kind of expectation or where we expect our disease to be after starting treatment depends on which treatment we've chosen to go. So,…

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make sure to discuss with your doctor and provider team about kind of the expectations after treatment and where you may be in this kind of spectrum after starting treatment.

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And lastly, measurable residual disease or MRD. It's kind of a hot topic in the world of CLL right now and in many different parts of oncology. So, MRD testing looks for very small amount of cancer or CLL cells…

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in a sample. So often in CLL, we're either measuring this in blood or sometimes in bone marrow. So, this is looking for a small amount of disease below what we normally would do as a part of the response criteria.

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So, in 2026, we're often looking in for one in a million or one cancer cell in a million cells.

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So, there's lots of different ways than we can do MRD testing. Kind of the first that came onto the scene was flow cytometry. This is where we'd be able to detect 1 in 10,000 cells and then more kind of in-depth…

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testing like PCR testing, or next generation sequencing looks for one in a million cells. One of the common forms of MRD testing that we're using now is clonoSEQ, which uses and looks for one in a million cells that could be affected with CLL.

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What I will say is that how we're using MRD and CLL is evolving very quickly. So, even if we're doing MRD testing, which may not be applicable in all patients or all treatments, this also may not change kind of our recommendations with observation or treatment, but often it does have prognostic significance at the end of a fixed-duration treatment.

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There are newer treatments and regimens from trials that help us use MRD as a way to be able to stop treatment. So, what I tell patients is this is kind of evolving, but we are currently using this in my practice…

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with fixed-duration treatment strategies. So, a lot of times we'll use this at the end of the venetoclax combinations, like venetoclax and obinutuzumab, or venetoclax BTK inhibitors, and obinutuzumab.

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If you're being considered for fixed duration, often we will use MRD, but it's less well established than continuous therapy. But, what I would say is more to come as we're learning, and hopefully we'll have more information about how to best use MRD testing in CLL in years to come.

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So, some big picture takeaways from this talk. So, 1., often a CL diagnosis can be made on peripheral blood draw alone. So, that's that flow cytometry and other tests that we talked about.

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Other tests like bone marrow, CT scans are not always necessary at diagnosis.

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So, 2. test before you treat, know your markers. This is very important when it comes to treatment decisions and prognosis.

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So, 3.…

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Scans and bone marrow biopsies should be used only to answer a specific question, so I don't think all patients with CLL need scans or bone marrows at diagnosis.

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These sometimes may be recommended, but I think they should only be used in certain…

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situations.

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Follow count trends, symptoms, and findings, not one isolated number. Remember, one value does not make your story, so oftentimes we'll need a trend or a complete picture to really have ways to recommend… to make recommendations…

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or to initiate treatment.

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And lastly, we often have time to watch and reevaluate. Most cases of CLL is a slow evolving condition, so kind of getting back to the trend is oftentimes we have time to see kind of how things will evolve over a period of time.

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With watch and wait….

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sometimes this may mean coming back for a closer lab draw, or just continuing to watch things, so we have time in the world of CLL.

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And with that…

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thank you for coming to this talk, and we will be talking a little bit more in the form of question and answers, so feel free to submit questions if you want to talk a little bit more about any of these topics…

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in detail. Thank you!

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Thank you very much, Dr. Wolfe, for a clear and detailed presentation. A lot of information for people to absorb. And fortunately, with CLL, we have time.

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We'll begin answering your questions, the audience questions. We'll try to get to as many as possible. There are, you know, dozens and dozens of questions that we've received today. We've already received dozens more before, so we're going to get to as many as we can.

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If we don't get to your question, there is a Trusted Care Exchange email address at the end of this event that you could use to actually send questions to and the CLL Society will address them later.

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First question, Dr. Wolfe, is about something called, you know, there's a comprehensive metabolical panel that has lots of different substances: calcium, sodium, magnesium…

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Yeah, alkinate, both phosphatase, etc. Can you explain the importance, what these are, related to CLL, and why they're important?

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Good question. I know there's a lot of tests that always come through at routine visits, but the CMP or Comprehensive Metabolic Panel looks at a number of different things kind of up at the top, we first think of the electrolytes, things like sodium, potassium. Those can be important at baseline just to…

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to do with kind of nutritional status, are you getting enough kind of in? It's also really important when we're thinking of electrolytes after starting treatment, because as we're talking about some of our treatments, some of them can mess with our electrolytes. So, these are things that we really closely watch.

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But other important parts of the comprehensive metabolic panel include your kidney function, right? How kidneys and other organs function are really important at baseline when we're talking about involvement with CLL, but also as how we're tolerating treatment.

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The comprehensive metabolic panel includes kidney function and kidney tests, as well as liver function tests. So, these can be important at looking at different organ function, electrolytes, and things like that, whether it's related to CLL involvement or kind of…

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kind of the status of your disease or also how these organs are doing after initiation of treatment.

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We've got a lot of questions about LDH, lactate dehydrinase is typically done by my doc, my hematologist, as, you know, and others have asked about this.

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Good.

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What is it, when is it done? Why is it done? Why should we care about LDH?

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Good question. It's also a hot topic in my clinic as well. So, LDH is something that I also send in every patient whenever we're seeing them. So, the way I think about it is it's a marker of cells turning over. So, cells growing and cells dying.

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So, it can be helpful in CLL because if CLL gets more active, sometimes you can see elevations in that LD or LDH marker. And oftentimes patients with active CLL might have a mildly elevated LDH…

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which is stable, nothing to worry about. This is just a way that we can track it. However, you can see more significant elevations if the disease is significantly active or in things in Richter's transformation. So, let's say a patient we follow, your LDH has been running around…

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200. One day you come into clinic and it's 600, 700. That may prompt me to get a little bit more evaluation. However, the one thing I, in caveat, I always say is a lot of things, it's not a perfect test, a lot of things can also elevate the LDH.

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Infections. I have one patient who loves to run marathons, and comes running 15 miles before seeing me in clinic, and right, there's a lot of tissue breakdown with vigorous exercise. So, his LDH is always elevated. However,…

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what I would say is small changes, nothing to worry about. However, if you do see big swings in the LDH in terms of elevation, that might be something to investigate a little bit further.

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When it comes to when someone is in treatment, either with a BTK or venetoclax, whatever, are you watching the LDH then as a sign that the drug is working?

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Good question. So, I tell people that when we get started on treatment, so for instance, let's say we're using venetoclax as an example, LDH can be helpful because…

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when we're talking about watching for things like tumor lysis. So again, you can see elevations in that, so we don't want to see too much, you know, elevations that could mean that we're breaking down cells too fast. When we get started on treatment, I'll often see the LDH rise just a little bit.

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Right, because we're starting to kill off some of these cells. But after a period of time, you often will see the LDH normalize. Does it happen in everyone? No. So, I would say I would not be…

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panicked if the LDH does not always return back to normal quickly after starting treatment, but you can often see that and will often see that, but it's something that we're also monitoring in terms of…

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you know, side effect profile and everything else when starting treatment.

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Thank you, Dr. Wolfe. Very helpful. There have been a number of questions about what is complex karyotype and why should I care?

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So, complex karyotype, that gets back to these prognostic markers that we were talking about. We talked about the big three, right? FISH, IGHV, and TP53. One thing that is also sent that I end up sending on most of my patients is the karyotype or chromosome analysis.

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And what that is, is looking for other structural changes in the DNA of the CLL cells. So, you'll find changes outside of the FISH, kind of the panel, or the changes that are sent on the FISH panel.

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And one of the things that we can find on the chromosome analysis is this thing called complex karyotype. And that… the definition of complex karyotype is three or more structural changes that we can see…

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in the CLL cells, and the moral of the story is the more changes, the more…

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mutations, more…

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things that happen to the DNA and the CLL cells, the kind of more complicated the CLL picture is. So, we often think about…

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complex karyotype is higher risk disease. There's some patients that come in, have never had treatment and have a complex karyotype at diagnosis. We've seen in studies

that patient population doesn't necessarily carry the same prognosis as those who develop…

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complex karyotype after treatment, so that's often when we will see the development of complex karyotype after the stress on the CLL cells from treatment. So, the one thing, like I said, we are looking for the complex karyotype. The more changes in the DNA,…

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potentially higher risk. However,…

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patients that have this at diagnosis may not have the same kind of inferior prognosis or inferior outcomes compared to those who develop complex karyotype later in their disease course.

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Thank you, Dr. Wolfe. It's a complex subject, haha.

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It sure is.

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We've had a number of questions that are basically a drill down in math, specifically when we get our test results, it'll typically have a percentage for some of these markers.

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Does a low percentage mean it's not a major factor in the disease and therefore less likely I'll need treatment? Alternatively, if the percentage is relatively high, according to the range…

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included in the report. Does that mean a higher disease burden, and therefore less favorable outcome? One example, IGHV is mutated at 7%, does that mean I'm mostly unmutated, and therefore at higher risk? Can you sort of do an explanation of these percentages, what they mean?

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Yeah, and this can be very complicated because it actually depends on the test. So, I was curious to see kind of what examples people are noticing these in. But IGHV is a great example. So, oftentimes you'll see on your report and we think about IGHV is either mutated or unmutated. We try to make it a little bit more simple just because…

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this is a little bit easier to kind of wrap your head around. However, there is a line in the sand at 2% that really kind of defines whether you're mutated or unmutated. And actually, any mutation burden over that 2% is considered mutated.

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So, if you're at 7%, that's actually significantly over that 2% line, and you would be considered mutated. So, I would not expect you to kind of behave like an unmutated CLL. However, if you're less than 2%...

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classically, we characterize you as unmutated. However, I'll have patients who show up as 2.1%, right? And…

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we drew this line in the sand at 2, because this is what was found to be clinically significant in trials, so…

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2.1 versus 1.9%. It's hard to tell, but the 2% is the line in the sand when we… what we use to kind of define mutated versus unmutated.

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Thank you. I've been dealing with this for 20 years and now I know.

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We've had a number of questions about MRD. So, first part of that is using flow cytometry to determine MRD versus clonoSEQ or some other NGS?

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Could you sort of explain to us when they should be used? Every year, every month, after treatment, before treatment? What is the… and which one is flow cytometry fine and just do clonoSEQ once a year? Sort of take us through what makes sense.

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Yeah, while MRD is complicated, I know I talked a little bit about it in our talk, but I feel like…

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MRD was started to be used in these clinical trials, and now we're kind of bringing it out to the clinical setting, and we're still trying to figure out the best…

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way to do this. So, when we're talking about methods for MRD, right? There's lots of different ones. So, the first kind of MRD assay that we were using was flow cytometry, because that's the best that we had. So, however, kind of the depth…

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of MRD with flow cytometry is actually less sensitive than some of our newer tests like clonoSEQ. So, clonoSEQ is a next generation sequencing-based test…

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that gives us a little bit more sensitivity and specificity at one in a million CLL cells. So, we can find, right, very deep levels of CLL. So, oftentimes in my practice, if I'm going to be doing MRD testing, because we have it widely available…

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I'm using tests like clonoSEQ just to get that depth of response. However, there's still some places that it's hard to get some of these…

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PCR-based testing and they will be using flow cytometry. However, to get good MRD flow cytometry, it really takes a very specialized…

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pathologist who runs these tests. So, oftentimes I'll be using clonoSEQ to to monitor the MRD just because you're getting really deep levels of of information. So, when do we do it? So, I saw a couple questions go into the Q&A. I'll just talk about kind of how I use clonoSEQ…

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right or wrong. This is the way that we use it in our clinic. Oftentimes, I will send it. So, the first thing that you'll typically do, you have to get something called clonoSEQ ID, and this is kind of a baseline. I tell patients this is…

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finding your fingerprint of your CLL. So, what we'll do is I'll often do one at baseline or before we start talking about treatment to get the kind of fingerprint of your CLL, because this is how we're going to track it down the road.

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This is also helpful in patients that have not had IGHV testing because this is actually what clonoSEQ is using. So, you kind of can kill two birds with one stone, sending this clonoSEQ in patients, because you'll get the fingerprint to track for MRD.

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But, also the IGHV mutation status. So, for patients who are not on treatment, there is no reason to follow MRD. Just because we would not expect you to be MRD negative or things like that without treatment. So, when do I think that we need to use this? Again, this is when we're talking about…

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Fixed-duration treatment. So, if we're planning on stopping a treatment, this is when we should be using MRD. So, for instance, again, it depends on the regimen. It depends on the line of treatment. So, of course, talk to your doctor about how they should be using it or…

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how they're planning to use it. But for instance, frontline CLL treatment with something like venetoclax or obinutuzumab, or even the newer combination therapies. I'll typically do an MRD test after…

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at the end of treatment. Again, this is really helpful when it comes to kind of telling patients how long to expect with remissions, right? We like to know how to prepare, and it also tells me kind of how we're going to be monitoring them. So,

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for patients who are MRD negative after frontline treatment…

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I'll typically be monitoring it based on their test maybe every six months to a year. What am I doing with that information? Am I going to be starting on treat patients on treatment who are MRD positive? I'm not. So, I actually feel like it's a patient to doctor conversation.

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I have some patients who MRD testing makes them crazy nervous. They don't want to see what this level is because they know themselves well enough to know…

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they're going to worry, they're going to want to start on treatment when we don't have data to support that. And in those patients, we may not be doing MRD testing, or we may be doing it very rarely versus some patients that like the information and kind of understand…

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what we're talking about in the data, just for another kind of modality to monitor. So, we, there are different clinical trials that we are using MRD…

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and also kind of stopping criteria, which is conversation for another day when we talk about these combination therapies. But sometimes some combinations and some treatment strategies are using MRD to stop, which is kind of a whole other…

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can of worms, but we're working to figure out how to best use MRD, but I do send clonoSEQ, you know, for ID at baseline, and then after fixed-duration treatment.

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Thank you, Dr. Wolfe. You know, before you had noted to treat the patient, not the numbers. And I think an MRD of X is a great example. You don't immediately what you're saying basically is you don't immediately start treatment just because MRD goes…

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to 4 or 5 after it had been at 0 or 1. It's all about the patient's situation, and if the patient is not exhibiting any symptoms, I assume that's a reason to not start treatment.

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Exactly. No, exactly. And again, some people, and I've even seen levels that go from five to four. And then the next one is five. So again, kind of what we talked about, we don't panic over one number. There's some people that you'll see rising MRD…

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maybe I'm going to be clinically monitoring them a little bit closer, particularly those with high-risk disease versus someone who's 3 years out MRD negative, right? So, I use it more for kind of how we're going to clinically monitor them…

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not necessarily kind of when to restart treatment.

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Thank you. You had talked about CT scans as being something that is sometimes done. I suspect sometimes in relation to getting insurance coverage or on a medication, you know, based on the size of the, you know, the spleen, etc.

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Let's talk about the risk of scans. Too many, you know, you get more risk flying in an airplane. How many scans are too many? How would you address that concern?

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Wow, that is tough. And I think you talked about something earlier with the fact that…

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you have a long time to learn about CLL, and we'll also have a long time to learn about your CLL. So that means that, as you'll have CLL for a period of time…

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for blessing and a curse. But,

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we have a long time to be exposed to radiation and other kind of downside effects of scans. So, the one thing I do think about is the amount of radiation a CLL patient will have during their lifespan.

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The other fact is we know patients with CLL at baseline have a higher risk of secondary malignancies, right? So, we're kind of weighing all of these different factors when thinking about…

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who to scan, when to scan, how often to scan. And unfortunately, there's not crystal clear guidelines for oncologists to follow up how close to do scans, because everyone's disease is different. So, what I often say is a patient who is…

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asymptomatic, we can monitor their blood, their blood counts, and we can track their disease in their blood. Maybe we're not getting scans.

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However, if a patient who we've tracked for a while develops abdominal pain, you know, left upper quadrant, you know, left-sided abdominal pain…

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kind of weight loss. Other symptoms that are kind of red flag symptoms, and that in my mind is when the benefit of looking inside would outweigh the risk of the radiation. So, when we get scans, if we get scans, that also gives you a baseline of…

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maybe how long to do scans or how often to, so I will typically do scans at initiation of treatment just because we need a baseline, right, because we want to see where we're starting. Often at the end of treatment, for instance, that 1 year after ven + obin or whatever, I will do scans just to see how things are changing.

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Am I going to be scanning that patient every 6 to 12 months after that? No. Especially if they do not have any other evidence of disease or they're MRD negative, they don't need to have scans because again…

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the risk of scans and radiation may outweigh the benefit. The one thing is I don't want patients to be scared of scans, right? Because it's the cumulative…

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dose of radiation that you're getting with these different imaging modalities. So, we think about, for instance, a chest X-ray when we're thinking about radiation dosages. So, a chest X-ray is about the equivalent of…

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one domestic airline flight. However, so, a chest X-ray…

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Kind of the same amount that we're getting often in daily life. However, when we think about CT scans or PET scans. That level goes up way, way higher to typically where it's 100, the equivalent of hundreds of chest x-rays.

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Again, for a patient that gets a CT scan, the risk of getting a malignancy related to a single or a couple scans…

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is low typically percentage of a percentage. However,…

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if you've had 100 CT scans, you know in your life, that that risk goes up. So, in my mind, I think it's…

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pros and cons. If you're having symptoms or other evidence of disease progression. I think it's okay to get some scans. If there's a concern for disease transformation, I think the benefit of getting a PET scan outweighs the risk.

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So, I think it's just saying we're not going to get scans just to get scans, but we're going to get scans to answer a question. And that's when. So again, I think it's shared decision-making and talking to the doctor about why are we getting these images?

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Thank you very much for your perspective on imaging and radiation, etc. We have had a number of questions about SLL in terms of which of those markers are relevant for SLL, which kinds of testing, etc.

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Could you give us your perspective on testing for SLL?

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Absolutely, and just to kind of answer, we did see some questions popping in about are things with CLL relevant to SLL? And yes, it's hard. We were talking before starting that even in the literature, we say CLL…

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but oftentimes that refers to SLL kind of just…

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you know, as well. So, whenever I see patients, I kind of explain a little bit about CLL and SLL. I think about it as kind of a continuum or kind of a spectrum. So, where CLL…

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is often a more kind of leukemic presentation of the disease because it's the same exact disease, it just depends on the disease presentation. So often, patients with a more CLL-like presentation will present with that classic elevation in the white blood cell count,…

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the high lymphocyte counts, where on the other end of the spectrum, patients presenting with more SLL will present with more lymph nodes and may not have that kind of high white blood cell count. But it's actually the same exact disease process.

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I often do tell patients too is many patients actually are between the two. So, where I call them CLL/SLL, meaning that they have the high white blood cell count, the high lymphocyte, but the lymphocyte count, but they also have some lymph nodes or a big spleen. So, characteristics of both of these disease processes.

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However, whenever we're thinking about tracking a disease, it's very important to think about this patient's disease presentation. So, in a patient with CLL…

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Their labs are going to be very important, right? Seeing what their white blood cell count is doing, seeing what the red blood count is doing, seeing what the platelets are doing, and maybe the scans are less important. On the other hand, a patient without a lot of…

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blood disease, so they don't have the high white blood cell count…

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that may be a patient that I need to get at least baseline scans on, right, to see what the lymph nodes look like. So, in patients that have SLL, I will rely more on scans than a patient with CLL.

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The other thing is all of the same prognostic tests apply. So, the FISH, the IGHV, the TP53, these are tests that patients with both CLL and SLL should get. The one thing to note…

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we talked a little bit that you can send the FISH, the IGHV or clonoSEQ and the TP53 off peripheral blood.

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So, in a patient with SLL, you may not have disease in your blood. So, it's actually really important to get these tests off the lymph node biopsy. So, it's important to kind of think about where we're sending these tests because we don't want to get a false negative because there's no…

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disease in the blood and then… oh, what test to kind of follow. So, when thinking after treatment, for instance, scans again may be a little bit more important in a patient with SLL, particularly when we're thinking about MRD testing because…

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peripheral blood MRD may not determine or kind of make the whole clinical picture and is not perfect for looking at lymph node or lymph node-based disease. So that's why at the end of treatment…

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to kind of get your entire response assessment, I do scans and kind of MRD tests to get the full picture.

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We've gotten a lot of questions about the immune system. So, we're going to have to do a bit of a deep dive here. This is one of my favorite topics because I have a really damaged immune system.

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So, a number of folks ask about IgA, IgM, IgG, T-cells, etc. Can you take us through the immunoglobulin panel?

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How frequently is it done? What are the docs looking for? What triggers treatment?

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This is perfect. Now, we were talking about this with a patient yesterday because this is actually a hot topic. And oftentimes…

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in a new patient, I will send immunoglobulins. Again, it's kind of interesting because I've been learning that insurance companies sometimes will only allow IgG testing to be sent, so I'm kind of working to adjust my practice too.

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But classically, I would send the whole panel. But when a panel with… when we're looking at immunoglobulins, we're looking at your antibody production. We know that patients with CLL, 90% of patients will develop…

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low antibody production during their course of disease. Often, the longer you have CLL, the more treatments you've been on, the higher risk of low antibodies. However, I do find patients that have had fairly early disease presentations that do struggle with…

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frequent infections and the low antibody levels. The one interesting thing when we think about the immune system, low antibody levels are just part…

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of the immune system, so we think about kind of B cell function, antibody production, which are affected by the CLL cells, but there's a whole other arm of the immune system, which is the T cell function of the immune system.

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We do know that CLL affects kind of both arms of the immune system. However,…

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kind of antibody production is something that we can at least intervene on. So, in patients that have had frequent infections…

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I will be getting immunoglobulins kind of frequently during the year. So, for instance,…

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Let's say you have a patient that comes in with recurrent sinus infections. They've had 5 sinus infections and have been on antibiotics five times during the course of the year. That patient needs immunoglobulins. And the reason why is what I'm looking for…

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is an IgG level classically less than 400. It's classically kind of our…

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target. So, if it's less than 400 and patients have had 2 or more infectious complications in a year that are clinically significant, whether you have to go to the hospital or getting antibiotics.

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Typically, we will recommend IVIG, and this is antibody replacement therapy. Classically, it's given once every month, and if patients are getting the IVIG, we'll be monitoring the IgG, usually once a month when they come.

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However, on the other end of the spectrum, if a patient has never had an infection…

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I may check this once a year. So, it really depends on a patient's infectious history and kind of if they're on IVIG and other things like that. When it comes to T cells subsets and other testing…

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I would say it's more rarely done. Sometimes you'll see people, and it's kind of interesting. I see some patients with an IgG level of 100, 200, and they never get infections. And you look at their T cell subset, and the T cells are nice and healthy. And then on the other hand, you'll see people that have an IgG level of 800…

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but are getting frequent infections, but the T cell subset is… is down. Unfortunately, there's not a…

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something I can give necessarily or prescribe that helps support T cell function, but it's often kind of limiting other immunosuppression or…

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kind of other lifestyle factors that we can work on to try to kind of boost other immunity as well.

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I think you're muted, Larry.

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Yeah, yeah, sorry, sorry. There's a cat whining for dinner in the background. So, I'm trying to avoid subjecting everyone to him. We have a bunch of questions about what when watch and wait becomes active…

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treatment, you know, which of the numbers, you know, you've talked about the, you know, the, the big five in terms of ALC, etc. And, you know, the iwCLL, there's usually a range involved here, and it's a combination real judgment call. Perhaps you could talk about a couple of hypothetical patients…

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And, you know, construction areas further when they're no longer Watch and Wait.

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That sounds good. And I typically tell patients there's three buckets that I think of. This is the way that I think about it. So, the first bucket is a patient who comes into clinic and is having unexplained fevers. They're having night sweats that are waking themselves and their spouse up. They're having to change the sheets, they're having to change their clothes…

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Or, you know, they've lost 20 pounds in the last 3 months. So, these are B symptoms. These are symptoms related to CLL. And if I see a patient that has progressive B symptoms, that would be, in my opinion…

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reason to kind of dig a little bit more and potentially start treatment. The other bucket is lymph nodes and spleen. I often tell patients just having scattered lymph nodes or having a little bit big spleen does not mean you need treatment, but it's when these become problematic.

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So, whether they become symptomatic, like you have a lymph node in your neck, and it's causing trouble swallowing or hoarseness, that patient may need treatment. Or if they get big and bulky, we classically say over 7 to 10 centimeters of lymph nodes…

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typically require treatment because we want to treat you before we run into problems. And of course, if you have a 7 centimeter lymph node in your neck, you would get some symptoms way before it gets kind of close to that size.

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The last bucket is the labs…

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And this is often probably the most common indication for treatment in my clinic as to when we start treatment. This is when we think about kind of the big 3. So, red blood count, so a decline in your red blood count…

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Typically, we say less than 10.

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A drop in your platelet count, typically less than 100, or a doubling of the lymphocyte count. So, the one caveat I would put to the doubling time is the clock of the doubling typically starts when your lymphocyte count is around 20.

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So, let's say a doubling of 4 to 8 may not kind of set off the treatment compared to a lymphocyte count of 30 to 75 and less than six months. So, kind of the absolute number does matter a little bit when we are thinking about the doubling time. And classically, the doubling occurs with other things too.

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So, I typically think of the doubling as kind of like a yellow light, right? That means caution. We're closely monitoring things. What are the lymph nodes doing? What are the B symptoms doing? Just as we're evaluating patients. However, like we talked a little bit about.

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One value doesn't make the game, right? So, oftentimes, if I do see patients who have been stable, then all of a sudden kind of doubled. I'm looking for infection. I'm looking for other things going on. Sometimes I may bring them back in two to four weeks just to see what the labs look like.

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Because you'll be surprised how many times it actually goes down. So, doubling, I think, yellow light, cautious, maybe we start talking about treatment, but the other things, like we said, the anemia, the low platelet count are often things where we do need to start treatment.

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Thank you. For those of us who are metric system impaired, good rule of thumb, two centimeters equals an inch. For all of us on this side of the pond…

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Set a good measure.

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That is, I know we often use I'll often people like what is two centimeters look like? So, we try to do get a little ruler to just get an estimate. But yeah, around an inch.

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Great. Thank you. You know, you were talking about low red blood cell counts and some of those other numbers. Number of people have asked after treatment, whether it's obinutuzumab, venetoclax, what happens to those numbers?

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Do they get back to normal over six months, a year? You know, red blood cells recover when if you have obinutuzumab, you're not going to see B cells, you're not going to see IgG for years. Take us through the numbers and what's going to happen after treatment in terms of…

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those numbers?

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Yeah, no, that makes…that's a good question. And right, it depends on the treatment you're getting. So, there are some people with BTK inhibitors, right, who are on continuous therapy, who, of course, the first thing that we see, right, you'll…

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you'll see that rise in the white blood cell count and the lymphocyte count, and it typically comes down. You'll also see kind of improvements in anemia if that was present. However, I have some people who are on BTK inhibitors and have a very mild low platelet count…

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running somewhere in the 130s to 150s, so just below the normal range, and this is where they stay when they're on the BTK inhibitors, and that's okay, because they're otherwise clinically doing well.

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The, when we think about the fixed-duration treatments, that is sometimes where we see these kind of prolonged low blood counts. So, I do see people who, after treatment with venetoclax or obinutuzumab, will have slightly low blood counts for a period of time, whether it's low red blood count, low platelets, or even the low white blood cells.

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You hit the nail on the head. The lymphocytes take a long time to recover, potentially years. However, I do expect, like, neutrophils and other things to recover, particularly at the 6-month mark or so.

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Again, it's not a perfect thing. I actually have one patient who did get frontline venetoclax and obinutuzumab, and her neutrophils and platelets took a long time to recover, so long that we actually did recommend a bone marrow biopsy, but…

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Guess what? She had no disease in her bone marrow. It was literally just recovery from the venetoclax. So, some of these treatments do affect the bone marrow and can just take a long time to recover. So, if you do have prolonged low blood counts, a little bit longer than what we would expect, sometimes we will…

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request these other tests just to make sure there's nothing else going on. However, many times it is still just your body and your bone marrow recovering from what we put it through.

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Thank you, yeah. So, it seems that CLL, the chronic is chronic recovery as well as chronic disease progression.

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Absolutely.

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There's some interesting questions about interpretation of these results. If you're going to, you know, if you have a primary care physician, can you rely on him or her to be doing these tests and looking at the results?

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Or is it a great, better idea to have a combination with a CLL specialist looking at these numbers once a year, once every six months? For people who have to drive or fly a distance to get to a CLL specialist.

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Yeah.

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How much can you rely on your primary care physician to know these numbers or know what to do about these numbers?

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No, and this is actually a big problem, right? Especially here in Texas. So, I this hits very close to home where people are driving hours and hours and hours to come see us.

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I… well, I would say the diagnosis and kind of initial prognostic markers, I would recommend seeing an oncologist for that, right? These are not routine tests that most primary care doctors send. However, the monitoring…

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maybe something that you can work and kind of collaborate with a primary care and oncologist. It does depend a little bit about kind of your risk status, how stable your disease, and things like that. However, I do have patients who their disease is…

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favorable risk, or has been stable for a long time, where maybe they're checking in with me once or twice a year, but then in between those visits are getting labs close to home. They often will send the primary care, or the patient will send the labs to me.

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Just for me to check in, because these primary care doctors will want…

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they want my input. They want to make sure everything's okay. So, I would say…

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A good primary care can absolutely work with and collaborate with an oncologist to help…

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kind of with the convenience factor, right? Not everyone has a CLL doctor down the street that they can go to. So, I often tell people it's wonderful to have a great CLL doctor, but the number one most important part of your team is a good primary care, because it's very important with this collaboration and making sure you have good support close to home.

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So, it absolutely, I think, a primary care can be extremely helpful in finding a way for having them work with your oncologist is…

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I think the best thing, if you can get it set up that way.

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Amen. Amen to that. We have a question about one of those other blood factors. Trisomy 12. What is it? Why should we care?

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So, kind of loaded topic there. So, we talk about these different kind of important FISH findings that we can see. I think some of the most common ones that we talked about, right? Deletion 17p is one that we kind of often think about

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We typically associated with the TP53 mutation. We also think about deletion 13q, which is classically a more favorable marker on FISH. Trisomy 12 is somewhere in between. It's classically considered…

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intermediate risk disease, the one thing that people do ask when they are reading on trisomy 12 is sometimes it can be enriched with Notch 1 mutations. So, Notch 1 mutations can…

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be seen in patients who do go on to develop Richter's. I think it's always confusing because some patients with trisomy…

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12 say, I bet I have Notch. I bet I'm going to have Richter's and that's not that's not always the case. I don't say that to scare people. I actually hesitate when I talk about this with patients because fear is not what I want people to come away with.

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It's classically an intermediate risk marker. Some patients can have Notch one mutations. Do we monitor trisomy 12 different than the other ones? Not necessarily, because again, most of our trisomy 12 patients will not develop Richter's.

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But there is…

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like a risk, just like other kind of higher risk markers that patients can go on to develop kind of more aggressive disease. However, rare occasion we monitor for it, but…

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classically considered intermediate risk disease. I hope that answers it without…

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putting too much worry into anyone.

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Thank you. A number of folks have asked about the infiltration. You know, should I be worried if I'm 95% infiltrated? I'm only 5% infiltrated, hooray. Could you explain what this is all about?

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Yes.

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Is that referring to the flow cytometry? Percentage? Okay, so on the flow cytometry, you will often get a percentage of atypical cells. And that is important when it comes to even the definition of CLL.

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So, oftentimes, to meet the criteria for CLL, you have to have over 5,000 atypical B cells. So, the flow cytometry will tell your doctor what percentage of lymphocytes…

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are affected by the CLL, and that will help me come up with kind of your total abnormal lymphocyte count. Does that mean that…

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if you have a higher percentage, you're going to need treatment sooner? Not necessarily. I actually rely more on the prognostic factors than just the percentage. However, it does help me classify kind of this pre-CLL condition.

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The monoclonal B cell lymphocytosis versus CLL. Again, if you have more disease, may you have more symptoms or kind of more cytopenia? Maybe, but it's not cut and dry, meaning 90% it's bad…

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and 40% is good. So, there's more to it, and kind of more complexity.

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Thank you. Thank you. When…

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people are being treated…

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and they're going, they're having blood work done weekly, monthly, you know, may depend on the treatment. What is typical for your patients in terms of frequency of blood work once they're in treatment?

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Yeah.

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So, once they're on treatment. So, depends on the treatment. So, typically for a patient on, let's say, continuous BTK inhibitors, typically for the first cycle, that first month, they'll get labs once a week, just as we're monitoring things.

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As things stabilize, we may see them every four weeks initially, or get, like, labs every four weeks, but…

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typically, patients who are on stable dose of treatment, doing well, no problems, will be getting labs maybe every three to four months for a patient on continuous BTK inhibitor.

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For other patients, for instance, venetoclax or fixed-duration, right? Patients initially as you're getting started on venetoclax are getting labs…

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potentially twice a week right as we're kind of ramping up the venetoclax. Typically, after that, I'll be able to space out after they ramp up to venetoclax doing well, I'll space out to

labs once a month while they're getting their obinutuzumab or other infusions, right? They're coming anyways for the infusions. And then after that…

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I may do every 8 weeks, every 2 months, and then oftentimes patients will get to every 3 months as they're finishing up kind of venetoclax by itself for that kind of the rest of the treatment. So, I think the moral of the story is getting started on treatment…

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there's a lot going on, right? We'll probably be monitoring things closely. But towards the end of treatment, regardless of treatment, maybe once every three months just to monitor.

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Thank you. We haven't talked about Richter's numbers. When you're looking at the big five indicators, etc., what are the numbers you're looking at…

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when it's time to start worrying about Richter's?

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Oh, Richter's is a tough thing, in general, a tough thing to talk about. So, there's nothing perfect when we're looking at labs or thinking about when to be concerned about Richter's. Actually, it's more clinical…

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than laboratory. There can be some things that we're kind of feeding into this too. But when do I kind of worry about when does it pop into my mind? In a patient who has been very clinically stable that comes to clinic and say, “Oh my goodness, I have this new lymph node under my arm that's grown…

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You know

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into a baseball in no time flat.”

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They're also having fevers, night sweats, they're having weight loss, a lot of kind of constitutional symptoms with rapid kind of lymph nodes or rapid symptoms, rapid abdominal pain, rapid, you know, growth of the spleen. Those clinical constellations often will tell me to say…

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“Okay, you gotta come in, we gotta get labs, probably scans too.” So, what things can you see on labs that may suggest…

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Richter's as well, that you know significant elevation in the LD. Oftentimes, Richter's is not kind of, you know, you've been chugging along at 200. It's 200. You'll see a kind of increase in the LDH, where you're seeing 4, 5, 6, 700, you know when you've been previously normal. And again…

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If Richter's pops into your doctor's mind, the kind of first thing that we'll do is we'll order a PET scan because I want to see what's going on internally. Are there any, you know, kind of rapidly progressive lymph nodes, and then that will often prompt a biopsy.

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But I would say it's a lot of clinical factors that actually kind of make me think about it because I may have a patient with a normal LDH, however, clinically it's just not fitting that I'll ask to get scans and vice versa. So,…

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I think it's kind of a number of factors that we put together to really give us the full picture.

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Okay, we have time for one more question and answer, and it's going to be about resistance. What is it in BTK or even a venetoclax context, how do you measure it? How do you find it? How do you measure it? And what does it mean?

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Great question. I'm glad we did not miss this one. So, whenever we think of these oral targeted treatments, right, all of these treatments target different proteins in the cell. Sometimes when your body or cells have seen these treatments, particularly for a long period of time…

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the CLL cells get really smart and kind of work around the way that treatment works. One mechanism that they can do is develop mutations in that protein so the drugs cannot work anymore. And we commonly think in the United States, because we've been using BTK inhibitors for longer…

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about BTK mutations, so drugs like ibrutinib, acalabrutinib, zanubrutinib, people that have had these treatments, particularly for long periods of time, can go on to develop mutations in that BTK protein, causing these drugs not to work anymore.

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And the way that we check if I'm starting to see progression on these treatments, we actually often will send next generation sequencing looking for BTK mutations. You will see, we can see a number of different types of BTK mutation. There's one classically that we…

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look for C481S…

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And when we see that, that makes us kind of think things like acalabrutinib and zanubrutinib won't work for that patient. We go on to think about kind of newer non-covalent BTK inhibitors, which have been found to be effective…

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even in with certain BTK mutations. BCL2 mutations can also develop when you've seen other treatments like venetoclax. In the United States, we see less of that because we've been using BCL2s less.

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The Australians have had a lot more experience with BCL2 inhibitors and kind of own the game when it comes to these mutations and how and how we track them. But oftentimes, if I'm seeing disease progression…

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on these treatments, I will send next generation sequencing looking for mutations in these proteins. That will help me kind of know which drug not to use and which drugs to consider in the future.

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Wonderful.

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Thank you so much. Like I said before, I've been dealing with this for 20 years, and I learned a lot today. So, on behalf of everyone, thank you. This has been great. Do you have any closing thoughts for us?

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No, I would just say thank you to everyone for joining. We had great questions. Thank you to Larry and the CLL Society who put this on. I think the CLL Society is a wonderful resource for patients. It's…

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a wonderful opportunity to get to know more about the disease. And I think, again, the more you know, the better you feel and the better treatments and care you can get. So, thank you all for coming today.

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Yes. Thank you to the… we had over 800 people registered, more than 300 actually attend.

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And as you can see in this slide, we'd like to thank our generous donors and the grant support from BeOne and Lilly for making the event possible. And again, thank you for all… thanks to all of you for attending and the great questions, things we hadn't thought about. So, really helpful.

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In your emails, you're going to get a request to fill out a survey about this event. We really appreciate you filling it out so we can learn more and make it even better. As you've noted, the event was recorded. It'll be available on the CLL Society website…

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along with the deck, the slides, and a transcript.

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If your question wasn't answered, you can see there's an email address there, trustedcare@cllsociety.org. We'll find the answer. And as you can see, we're CLL Society is trying to help us, so let's help the CLL Society. And there's a link to a donation.

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We're all in this together, as Dr. Koffman, the founder says. Thank you all very much.