

THE BLOODLINE WITH LLS

A PODCAST FOR PATIENTS AND CAREGIVERS

Episode: 'Hopeful Advancements for Chronic Myeloid Leukemia (CML)'

Description:

Join us as we speak to Dr. Richard Larson and Dr. Anand Patel from University of Chicago Medicine about chronic myeloid leukemia (CML). In this episode, they discuss the latest advances in treatment for CML and what is on the horizon.

With decades of progress with targeted treatments, CML patients are now given more hope of achieving deep remission and potentially discontinuing medication. A cure for an incurable disease may now be in sight.

Transcript:

Elissa: Welcome to *The Bloodline with LLS*. I'm Elissa.

Jesse: And I'm Jesse. Thank you so much for joining us on this episode.

Elissa: Today we will be speaking to Drs. Anand Patel and Richard Larson about chronic myeloid leukemia, or CML. Dr. Patel is a hematologist/oncologist at the University of Chicago Medicine who specializes in the treatment of leukemia, myelodysplastic syndromes or (MDS), and myeloproliferative diseases, or MPNs.

In addition to treating his patients with standard treatment approaches, he's committed to delivering novel therapies through clinical trials that combine basic and translational research into groundbreaking advancements in medicine for better outcomes and quality of life.

You may recognize Dr. Patel from when he joined us on a previous podcast titled, *"Instilling Hope Through Progress: Myelodysplastic Syndromes"* in October of 2022.

Dr. Larson is a Professor of Medicine in Hematology/Oncology and a Director of the Hematologic Malignancies Clinical Research Program at the University of Chicago. His

research interests have included clinical trials in acute and chronic leukemias, stem cell transplantation, experimental therapeutics, and the determinants of response to therapy in leukemia and myelodysplastic syndromes and the cause of therapy, related leukemias.

Welcome Dr. Larson, and welcome back Dr. Patel.

Anand A. Patel, MD: Thanks so much for having us.

Elissa: Our topic today is on chronic myeloid leukemia, or CML. Could you tell listeners what that is?

Richard A Larson, MD: Sure. Chronic myeloid leukemia is a type of blood cancer. It's a malignant disease of bone marrow cells, which leads to the overproduction of white blood cells in particular, but sometimes also red blood cells and platelets. It's not a very common disease. There're only 5,000 or so cases diagnosed each year in the United States, but over the last 20 years because patients with CML have been living longer, the prevalence of the disease is becoming quite a bit higher so that many physicians now practicing in the community will see a number of patients with CML during their careers.

Previously, most patients with CML were referred to academic medical centers where there was expertise in the diagnosis and management of this disease. At one time, CML was the most common cause for allogeneic bone marrow transplantation, but that's rarely done these days because of the development of very effective pills that are taken by mouth.

Dr. Patel: And I'll add, CML is typified by a very specific abnormality in the chromosomes which help to house the genetic material of our cells, so there's a translocation between chromosomes 9 and 22, which leads to a fusion protein called BCR-ABL1. And as Dr. Larson had mentioned, with these very effective oral chemotherapeutics, known as tyrosine kinase inhibitors, or TKIs, we are able to take

advantage of the fact that CML uniformly has this abnormality as the medicines that we use help to target that.

Elissa: Now we hear a lot about the Philadelphia chromosome all the time. What is that?

Dr. Patel: Yeah. Great question, Elissa. The Philadelphia chromosome refers to what I was just speaking about. It's a translocation between chromosomes 9 and 22, which then leads to this BCR-ABL protein, this fusion protein that typically is not there, so it is also termed as Philadelphia chromosome; and many times you'll hear either patients or clinicians say that they have a Philadelphia chromosome positive leukemia if that is, in fact, found.

Dr. Larson: The Philadelphia chromosome is actually a smaller version of chromosome 22. It was first identified at the University of Pennsylvania in Philadelphia, and that's where it gets its name. So, for many years, cytogeneticists would look at the karyotype or the chromosome pattern in the bone marrow cells of patients with leukemia; and if they saw this small version of chromosome 22, that was sufficient to label it as a Philadelphia chromosome.

It was in the mid-70s that Janet Rowley at the University of Chicago recognized that there was no DNA lost from chromosome 22 but, rather, there was a translocation or a movement of DNA from chromosome 9 to chromosome 22. So, the naming, changed to translocation 9;22, resulting in this smaller version of chromosome 22.

The significance of that, as Dr. Patel mentioned, is that there's a gene on chromosome 9 called ABL1 and a gene on chromosome 22 called BCR; and as a result of the translocation, there's a fusion between these two genes, which are normally on different chromosomes, now they're brought together on the Philadelphia chromosome so that every time BCR is transcribed, ABL is also transcribed. The significance of that is that ABL1 is an enzyme which stimulates the growth of blood cells. So, this new

category of medicines that Dr. Patel mentioned, the tyrosine kinase inhibitors, are actually enzyme inhibitors that inhibit the BCR-ABL1 enzyme.

Jesse: Thank you so much for that explanation. What are some common signs and symptoms that a patient could have prior to diagnosis? And how do patients usually end up being diagnosed with CML?

Dr. Larson: Interestingly, about a third of patients with CML have no symptoms at the time they're diagnosed. They see their doctor for a routine physical exam, a complete blood count is obtained, and it's noted that they have an excess number of white blood cells in their blood. Or they're seen for some completely different medical problem and, incidentally, CML is diagnosed.

On the other hand, the majority of patients do have some symptoms. Most often it's related to some tiredness or fatigue, sometimes shortness of breath if they're anemic, which means a low red blood cell count, low hemoglobin level. As the white blood cell count rises in patients with CML, the spleen often gets congested and some patients will complain of a heavy feeling in their abdomen, particularly over on the left side. And sometimes because the spleen sits on top of the stomach, they find that they're losing weight because they're not able to eat comfortably to maintain their weight. So, tiredness, weight loss, sometimes some low fever, sometimes some sweats, particularly at night, are symptoms that would take patients to the doctor.

Dr. Patel: And when thinking about diagnosis, oftentimes initially what's found on laboratory work, as Dr. Larson mentioned, is an elevated white blood cell count. And when we're thinking about white blood cell counts, just the elevation is not what we're looking for. We also look to see what is making up that white blood cell count. So, we have white blood cells that fall under a myeloid lineage and then white blood cells that fall under a lymphoid lineage. And in CML, it's usually an excess of these myeloid cells and there various types that can be seen. So that's an additional clue, in addition to the elevated white blood cell count that a patient may have CML.

Nowadays, we have a few different ways to identify the presence of that Philadelphia chromosome that we were talking about earlier. It can be done with molecular testing, which looks for the presence of this BCR-ABL1 fusion protein. You can also do a test from the blood called FISH, fluorescent in situ hybridization, to help identify that translocation 9;22. And that is kind of the first step that's oftentimes taken in making the diagnosis.

Upon confirmation that that is there, a bone marrow biopsy is oftentimes obtained to get a sense of what phase of disease CML is in. So, you can have patients that are in the chronic phase of CML, which is the vast majority of patients, and that's typified by not seeing an increase in immature cells called blasts. However, you can also have patients in the accelerated or blast phase of CML, which is the minority of patients. And, again, that's typified by the number of blasts that are present on a bone marrow biopsy that's obtained upon confirmation of the presence of that Philadelphia chromosome.

Elissa: Now what are the current treatments for CML and are they different based on what phase you were just talking about?

Dr. Patel: That's a great question. We live in an era where, thankfully, we have several FDA-approved medications for CML, the first of which is imatinib. We now have had experience with imatinib for over two decades, which is really incredible.

Imatinib is oftentimes referred to as a first-generation medicine for CML. We have three TKIs, or tyrosine kinase inhibitors, that are FDA approved that are considered second generation dasatinib, nilotinib, and bosutinib. There is a third-generation medication called ponatinib. And the most recently approved medicine for CML is one called asciminib.

And it's a great question about should the choice of TKI be dependent on kind of the phase of disease, and we often have tools available to us that can help to inform the prognosis of patients with CML. In general, we know that imatinib has excellent long-

term outcomes associated with it. And in patients with lower-risk disease, we tend to reach for imatinib first. However, other things that can inform the decision could be the prognostic scores that I alluded to and what other medical conditions patients may have.

The last piece that can inform the decision of what medication to choose is, we are increasingly trying to identify patients that may not need to be on these therapies for a lifetime, but rather can stop them after having appropriately controlled disease for several years.

Some of the later generation medicines, such as dasatinib and nilotinib, we now have fairly significant data to help guide when and how many patients could expect to achieve that level of response to where they could potentially stop therapy. And if a patient is highly motivated to ultimately achieve the level of disease control that would allow them to stop therapy, that could potentially influence the decision to reach for a medicine in that generation as opposed to imatinib.

Dr. Larson: Well, all of these tyrosine kinase inhibitors have certain features in common. They're all oral. They tend to be quite well tolerated compared to other forms of cancer chemotherapy. They all work by inhibiting the BCR-ABL1 enzyme. And once you switch this enzyme off, the leukemic cells are not able to survive. So, many patients, within a few weeks of starting on one of these oral tyrosine kinase inhibitors, will achieve hematologic remission where their blood counts return to normal and if the spleen was enlarged, the spleen will shrink back down to the normal size and their symptoms, if they had any at the time of diagnosis, tend to improve fairly rapidly as well.

So, because the BCR-ABL1 fusion gene is only present in the malignant cells and not present in normal cells, we now have a molecular test that can track the disappearance of the malignant cells over time. It's called a quantitative RT-PCR test or quantitative reverse transcriptase polymerase chain reaction. That's why we use the abbreviation

more often. This test can actually be done on the blood, the circulating white blood cells and doesn't require repeat bone marrow biopsies. In fact, most patients with newly diagnosed CML will only need a bone marrow biopsy at the time of the initial diagnosis, and it's no longer necessary to repeat bone marrow biopsies over the course of the disease as long as patients are continuing to have a good response as monitored by this molecular test.

The test is now widely available. It's been standardized, really, across the world in both academic medical centers and commercial laboratories, so it's available to patients on TKI therapy. And it's really quite helpful. Many patients within three to six months of starting TKI therapies will have achieved an early molecular remission. And that's defined as a decline in their leukemia cells by more than 90% during those first few months. And we know from prospective trials that patients who achieve an early molecular remission regardless of which of the TKIs they initially receive for their first therapy, those who have an early molecular response tend to have an excellent long-term response and their survival appears to be the same as the normal population without CML. So, it's been a dramatic change in the natural history or the clinical outcome of patients with CML over the past 20 years since these drugs have been available.

Elissa: It's just amazing how things have changed for CML patients since the TKIs became available.

Now you mentioned very briefly the asciminib, which has been a little more talked about lately. Could you talk a more about that?

Dr. Larson: It's also a TKI, which means that it inhibits the tyrosine kinase enzyme which describes the activity of the BCR-ABL1 fusion enzyme. The difference between asciminib and the other five commercially available TKIs is that it binds to a slightly different site on the enzyme protein and inhibits its activity that way. The first five TKIs that were developed all bind to a particular pocket or site on the enzyme and

inhibit the transfer of energy to the tyrosine amino acids on various substrates required for the survival of those cells.

One of challenges with the first five tyrosine kinase inhibitors is that the BCR-ABL1 gene can undergo additional mutations so that it affects the binding, particularly of a drug like imatinib. Over 100 different mutations have been identified, which interfere with imatinib binding. And if that happens, the patient stops responding and then we switch to one of the other tyrosine kinase inhibitors which can overcome, in many cases, those new mutations. Because asciminib binds to a different site on the enzyme, it's not susceptible to the types of mutations that interfere with imatinib binding and inactivation of the enzyme. So, it's an important new medicine to have available for patients with CML.

Jesse: CAR T-cell therapy and other immunotherapies have been getting more attention over the last few years. Are CML patients eligible for any of those treatments?

Dr. Patel: That's a great question, Jesse, and I'll start by highlighting a point that Dr. Larson made earlier, which is, we've come such an incredibly long way in the treatment of CML. We have these orally available pills you can take. And for the most part, we think these patients that are receiving these therapies have a normal lifespan when compared to someone of a similar age that wouldn't have CML. So, the bar for success is very high in CML, which is a good thing.

When thinking about CAR T-cells, they've made a significant presence, again, going back to thinking about white blood cells and specifically whether they're myeloid cancers or lymphoid cancers. There have been significant inroads with CAR T-cell therapy on the lymphoid side. So, that would be things like acute lymphoblastic leukemia [ALL], certain kinds of lymphomas and multiple myeloma. However, in the myeloid side of things, so CML and other diseases in that realm would be things like

myelodysplastic syndromes [MDS] and acute myeloid leukemia [AML], we haven't yet had these same sort of successes with CAR T-cell therapies.

Now referring to immunotherapy, which is a much broader bucket, allogeneic stem cell transplant can actually be thought of as an immunotherapy.

So, when you are receiving cells from a donor after receiving a conditioning regimen that would wipe out your own native bone marrow. And pre-TKIs, as Dr. Larson had mentioned earlier, allogeneic stem cell transplant was considered much more frequently in patients with CML. Nowadays, thankfully, with the effectiveness of these oral medicines, for most patients, we are able to achieve the sort of response that we would term an optimal response or a molecular response that would be associated with excellent long-term outcomes.

Elissa: Now we know that currently CML is not considered curable even though patients are having a longer lifespan, which is wonderful. But are patients ever able to go off medication?

Dr. Larson: You're right. When we first had the TKIs developed, we thought that they would be excellent medicines to suppress CML, but perhaps not eradicate it. And we made efforts to tell patients that they would probably need to take these medicines life-long in order to keep their disease in remission.

It was somewhat surprising then about ten years after the development of imatinib that the French first, but later many other groups, showed that for patients who had a deep molecular remission where the persistence of cells with the BCR-ABL1 transcripts by this molecular test that we mentioned, if you took those patients who had been in a deep molecular remission for several years' time, you could actually stop imatinib and monitor those patients over the next six to nine months' time. And, surprisingly, many of those patients stayed in a deep molecular remission. So, we've had to rethink the idea of whether over time, at least in some patients, CML may actually be cured by these drugs.

It does seem to take a while for that to happen, however, even with the more potent second-generation drugs. It seems like three, or four or five years of continuous daily treatment with these tyrosine kinase inhibitors is required to suppress the disease sufficiently that it is less likely to recur.

Dr. Patel mentioned earlier the clinical scoring systems that we use at the time of diagnosis sometimes to select the optimal drug for a particular patient depending upon the phase of their disease. And, interestingly, low-risk patients have a much greater likelihood of success with what's called prospective discontinuation, that is after four or five years, being able to discontinue their tyrosine kinase inhibitor and not have the disease recur. It's a little more challenging for patients who fall into a high-risk category. So, using these scoring systems at the time of diagnosis seems to provide important information over the course of their disease.

I should caution that not every patient is able to successfully discontinue their tyrosine kinase inhibitor. In fact, about half of patients within the first 6 to 12 months after stopping their treatment, will have evidence by the molecular test on their circulating blood cells that the disease is starting to reoccur. Usually, it comes back slowly as a chronic phase disease. And if that's recognized within the first month or two and patients resume the same TKI that they had previously discontinued, almost all those patients will go back into a deep molecular remission. Whether they're ever able to successfully discontinue later really hasn't been proven yet, but many patients, perhaps about half, after four or five years of treatment, seem to be able to discontinue their tyrosine kinase inhibitor without having a molecular recurrence.

Elissa: That's wonderful.

Jesse: That is wonderful. Now a concern from a lot of our listeners are side effects from treatment. We know these can vary between patients. Can you please share some of the side effects a CML patient can experience as a result of treatment and how are they managed?

Dr. Patel: Yeah. That's a great question, Jesse. Oftentimes, when a TKI is first started, patients may notice things like muscle aches or cramps, maybe even a little bit of mild nausea with initially taking it. Thankfully, those tend to be symptoms that improve with time as you're on the medications. Sometimes, patients may need an anti-nausea medicine before taking their CML medicine initially, and with time, they oftentimes are able to no longer require that anti-nausea medicine.

Other things that we can see with initiation of TKIs is as we start treating the CML initially, blood counts may actually get a little worse before they get better. And as the response or remission becomes deeper, those blood counts will start to improve.

Now the TKIs that we mentioned, they all have their own specific side effects that we may keep an extra close eye out for, and I'll mention a few illustrative examples. So, nilotinib, in patients who have underlying diabetes, sometimes can worsen that diabetes. Dasatinib can, in rare instances, lead to accumulation of fluid in the lungs or places where it shouldn't be, so what we call a pleural effusion. Bosutinib can lead to diarrhea or loose stools. And ponatinib, asciminib, both of those are ones that we use with caution in patients who have a history of cardiac disease or high blood pressure.

Now, these are not common side effects that are seen with these medicines, but they are ones that we keep a close eye out for. And, again, when coming back to that decision of how do we arrive at which TKI to use when treating a patient, many times their other medical conditions help to influence that treatment choice because of the side effect profiles that can be seen with these medications.

Dr. Larson: I would add that probably the most common side effect across all the tyrosine kinase inhibitors is low-grade fatigue. The fatigue can be relatively mild, at least as physicians judge it, but it can be difficult for patients, in part, because it seems to be there day after day, even though it's relatively mild.

Sometimes, taking a brief drug holiday, that is discontinuing the treatment for three or four days, can reset the clock so that the fatigue does not recur. But there's more art

than science to helping patients tolerate these tyrosine kinase inhibitors; and, for that reason, it's important that they be under the care of hematologists or oncologists who have had some expertise in the modern use of these medicines.

Elissa: Yes. We've definitely learned that we need to educate patients on how to deal with these side effects so that they can continue to feel good.

Now let's move on to treatments on the horizon. What treatments are in clinical trials or potentially coming up? Is there anything that you're really excited about?

Dr. Larson: Well, asciminib has been the most recently approved tyrosine kinase inhibitor for CML. It was approved for patients who had previously tried at least two other TKIs and either couldn't tolerate them because of side effects or found that they weren't very effective. So, we call that approval in the third line as a third or greater TKI, and it was shown to be more effective than bosutinib in a large, randomized clinical trial.

Having proved its value in the third line, it's now been moved into a frontline clinical trial, a randomized trial where half of patients received asciminib as their initial treatment and the other half received one of the other commercially available TKIs. That trial was done as a global study and rapidly completed its accrual, but the results haven't been reported yet. But if asciminib is at least as good as the other TKIs commonly used in the first line or frontline treatment of CML, then it's likely that it'll become available as a first-line drug.

Elissa: That's great.

Dr. Patel: I will add with regards to asciminib, there are also trials ongoing to see whether a very low dose of asciminib added to, for example, imatinib may be able to deepen responses in patients who've had disease control but maybe have not yet hit the landmark for what we think of as a major molecular response or a deep molecular response. So, again, I think that the fact that the drugs we are speaking about are

ones that we are already in use in CML, speaks to the fact that we have lots of very effective options to choose from, and now it's more a question of how can we identify the optimal doses with retaining efficacy or how do we sequence these very effective medicines?

Dr. Larson: It's interesting, even though we currently have these six very effective and generally well tolerated medicines, at the last meeting of the American Society of Hematology [ASH] in December, there were clinical data presented on four new molecules that are moving their way through clinical trials. Whether they'll prove to be better than the currently available drugs or whether they'll be drugs that have similar efficacy but may be a little better tolerated in some patients than in others, remains to be seen. But there continues to be considerable investment in developing effective drugs in CML.

Elissa: Wow, that is so exciting. Now I'd like to point out that when you were talking about clinical trials, you were talking about, potentially combining TKIs or medications versus some people think that they might get on a placebo and that is never the case, correct?

Dr. Larson: Yeah. The placebo-controlled trials are generally not done in cancer clinical trials. Sometimes with combination therapy, patients will be randomly assigned to get a standard treatment plus either an experimental therapy or placebo. But using placebos as a single intervention by itself is not done.

Dr. Patel: Yeah. And to use the example of the trial that Dr. Larson was talking about earlier where patients with newly diagnosed CML are randomized to either receive asciminib or a standard therapy. The standard therapy implies use of one of these many other TKIs that are highly effective in CML. A standard therapy would not be no treatment at all, which is what a placebo can be if that's, as Dr. Larson mentioned, the only intervention that is done.

As Dr. Larson mentioned, doesn't just apply to CML but to cancer broadly, if it is a randomized trial where a group of patients received one medicine, the medicine under investigation, and the other group of patients received something else, that something else has to be aligned with what our current standard of care is for whatever that disease may be.

Elissa: That's always good to know. I think the patients need to understand that they will still get the standard of care, which is very good and effective medications, but they could enter a trial and potentially get something that will get them off medication in the future or work even better. It's good for our patients to hear this.

Dr. Larson: I'd like to go back to a point that Dr. Patel made earlier about the early drop in blood counts when people first start on treatment because it can be widely misunderstood. It's important to recognize during the months or maybe a year or longer that CML has been developing in a patient's bone marrow, it overwhelms the production of normal blood cells, so by the time that people are diagnosed with CML, probably 98 or 99% of all of their blood cells actually are derived from the Philadelphia chromosome positive malignant stem cell. And that includes their red blood cells or platelets and their myeloid cells, their white blood cells.

So, when you start a very effective treatment that not only suppresses the proliferation of those cells but actually causes a rapid disappearance of those cells in the body, you're left with only the 1 or 2% normal cells that were present. So, very common for the blood counts, white blood cells, red blood cells, and platelets all to fall to a low level during the first few weeks when patients first start on TKI therapy.

That's been managed in various ways. During clinical trials often physician investigators were required to stop the TKI because it was uncertain at that time whether these drugs suppressed normal blood cell production, what we call myelosuppression. But it's been shown now that these drugs don't suppress normal

cells, they only suppress the cells with the BCR-ABL1 fusion chain and this abnormal enzyme present.

So, it's probably important to continue to take the TKI everyday even though the blood counts are low; and in rare cases, a patient might need a transfusion to support their red blood cells and their hemoglobin level. We also use normal bone marrow growth factors to support the production of normal myeloid cells and normal platelets, but we're very hesitant to stop the TKI during the early phases just because the blood counts have dipped down. And as Dr. Patel said, as we clear out the leukemia and patients go into remission, then their normal bone marrow function returns and typically within four or five weeks, the complete blood count is back to normal.

Elissa: That's good to know. Now, on our patient podcast Home Page, we have a quote that says, "After diagnosis comes hope." What would each of you say to patients and their families to give them hope after a CML diagnosis?

Dr. Patel: I would start by saying, again, it can't be understated how far we've come in the treatment of CML over the last couple of decades. Oftentimes, the first time I meet a patient after a diagnosis of CML, I try to emphasize that, "We expect you to live as long and, hopefully, as well as someone your age with similar other medical conditions that doesn't have CML and that we have several therapies to help us achieve that goal."

I think the other part that we discuss early on with patients now is many patients are worried about the fact that they may need to take a new medicine for the duration of their lifetime. So, we do talk about the fact that with close monitoring of disease and achieving a deep remission, there's a chance that you may be able to come off therapy in the future.

The last point I'll make is in those patients that do need to be on medicines, and it can't be stopped for one reason or another for their CML, that their physicians are very invested in not just treating their CML but also helping to guide them through the side

effects that may come with those medicines kind of like the fatigue that Dr. Larson was talking about earlier.

I'd say those are the three points that I try to emphasize with any patient that I meet with a new diagnosis of CML. I think those three points all point towards the hope that someone can have despite receiving a diagnosis that can seem somewhat scary at first.

Dr. Larson: I would agree with that. Having just been told that you have leukemia is a pretty scary discovery. But this is one blood cancer, one cancer in general that we understand very well at a molecular level, the genetic level, at the biological level; and, for that reason, we have now developed targeted therapies, these TKIs, that have a very focused effect on malignant cells and avoid many of the side effects that are seen with other types of cancer treatment.

We're hopeful that every patient now will achieve a clinically meaningful remission, at least a deep molecular remission, and in many cases be able to stop their treatment because they've been cured of their CML. We have great hopes that these patients will be able to live out their normal lifespan.

Jesse: Wonderful.

Elissa: That's great. Thank you so very much, Dr. Patel, Dr. Larson, for joining us today. You've given such great and hopeful information to patients. And we never know, there might be a cure in the future. So that is wonderful and thank you so much. We really appreciate you joining us today.

Jesse: Yes, thank you.

Dr. Larson: Thank you.

Dr. Patel: Thanks so much for having us, it was our pleasure.



Elissa: We would like to give a special thank you to University of Chicago Medicine for supporting this episode. And thank you to everyone listening today. *The Bloodline with LLS* is one part of the mission of The Leukemia & Lymphoma Society to improve the quality of lives of patients and their families.

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