ATLANTA AREA MULTIPLE MYELOMA SUPPORT GROUP INC.

Meeting Minutes

Northside Virtual MM Support Group

January 10, 2026

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Annual ASH review with the Emory Winship team:

The January meeting was our annual ASH (American Society of Hematology) review with the Emory/Winship team. Charise Gleason welcomed the attendees, which peaked at 60 screens connected. It was good that the program was virtual as the flu is spreading throughout Georgia. Charise noted that this is our 14th year doing this review and she has been with the MM program for 23 years. Charise noted that the program has grown and new physicians will be presenting. The large myeloma team includes advanced practice providers, such as nurse practitioners and physician assistants, as well as researchers and pharmacists and the clinical team. The team saw 750 new MM patients and 700 MGUS patients last year. There were 1400 new cases of MM in the state of Georgia, so Emory sees more than 50% of the new patients. Emory had 5500 patient visits for 2025 at all their locations.

A wealth of current information on numerous aspects of disease and treatment was covered. This index provides the names of the doctors and their particular topics of presentation.

Dr. Nisha Joseph Precursor Conditions, MM Definitions

Dr. Roberto Mina Updates for Newly Diagnosed Patients

Dr. Vikas Gupta	New Drugs for Relapsed Myeloma
Dr. Jonathon Kaufman	Amyloidosis Update
Dr. Ajay Nooka	Bispecific Antibodies
Dr. Nisha Shah	CAR-T Updates
Dr. Richa Parikh	Supportive Care Therapy
Dr. Sagar Lonial	Summary, Current and Future Care, Emory Update

Dr. Nisha Joseph presented first on Precursor Conditions and some definitions for newer patients. The spectrum of myeloma starts with MGUS (monoclonal gammopathy of unknown significance). These are abnormal proteins in patients, and they are monitored annually with a low risk of progression to MM. The next stage is SMM (smoldering myeloma) which has higher levels of disease with a higher risk based on certain factors. MGUS and SMM have no symptoms.

In the past, MGUS and SMM patients were just monitored. Now there are tests to determine which SMM patients are more likely to progress. At ASH, the AQUILA study in 23 countries pulled together data on clinical trials treating SMM with Dara for 36 months. This was to determine if there are clear benefits from treating high-risk patients. At the 5-year mark, the PFS (progression free survival) was 63% with Dara vs. 41% with monitoring. This is a 51% reduction in the risk of getting MM. This led to FDA approval of Dara treatment for high-risk MM. The ASH update on this study showed greater benefit for high-risk patients and that there was no issue in collecting stem cells with this treatment with minimal side effects. Given the data, Emory believes that it makes

sense to give treatment for high-risk SMM in some way. There are several clinical trials underway exploring other options to compare outcomes to the Dara study. Two of the trials include a plant-based diet. Another includes Iberdomide, which they believe may be approved by the FDA next year.

Dr. Roberto Mina joined the myeloma team about a month ago and moved here from Italy. He spent a year at Emory in 2017 and is happy to be back and to present the updates for newly diagnosed MM patients (NDMM). The current treatment approach uses four drugs: Dara-VRd (daratumumab) or Isa-VRd (Isatuximab) as induction therapy. If patients are fit enough, then they will proceed to SCT (stem cell transplant) and then maintenance therapy. For those who do not qualify for transplant, due to age or comorbidities, they will continue Dara-Rev until progression. The results show that 50-75% of patients achieve MRD (measurable residual disease) negativity. This means that the bone marrow does not show MM cells. Some of the data presented at ASH showed mathematical projections that the duration of the first remission is lasting more than ten years for patients with transplants and close to ten years for patients who do not receive a transplant. This is a significant improvement from survival of only 2-3 years twenty years ago.

What are the unmet needs and how do we improve the outcomes? First, is to prevent early progression in HRMM (high risk MM) and those patients who do not get the optimal response from their first line of treatment: they do not achieve MRD negativity. Second, treatment optimization with better and fewer drugs. Can we get better results with fewer drugs and fewer side effects? At ASH, there was some interesting data that will change the future treatment of MM.

HRMM is defined as patients who had less than a 50% reduction in their MM after the first line of standard therapy. These patients have been shown to have a shorter remission duration. There are also cytogenetic abnormalities that can predict the risk of suboptimal response. The goal is to be able to identify these patients early and tailor the treatment for maximum response. At ASH, there was interesting data to tackle this specific problem. Patients who were MRD positive after initial treatment were given 4-6 cycles of Linvoseltamab bispecific. 100% of patients converted to MRD negativity! This is proof of concept that even after a less-than-optimal response, changing the mechanisms of action can lead to the eradication of the disease. This is very encouraging.

Another important study that will help improve the approach for newly diagnosed MM is a European study of Iberdomide, which is more potent and safer than Revlimid. Iberdomide was tested as maintenance after transplant in standard risk patients as a possible replacement for Revlimid. Iberdomide was effective in reducing MM after transplant as it converted roughly 50% from MRD positive to MRD negative. But more interesting was the safety profile. With Iberdomide, there was an exceptionally low rate of fatigue and diarrhea which caused many patients to discontinue Revlimid. Also, there was a much lower risk of secondary cancers with Iberdomide. There is a Phase III study comparing Iberdomide as maintenance in an effort to replace Revlimid as a standard of care.

What about patients who are not eligible for a transplant? Now they are given four drugs or just three drugs for the frailest. The question is, can we reduce the number of drugs used? There were two studies at ASH with a different approach to the one that is used now. The Linker-MM4 trial tested the use of only one drug, which is Linvoseltamab, the bispecific antibody. With very preliminary data, it showed that the vast

majority responded to treatment. The response rate is between 70% and 86%, and 43% to 75% achieved complete remission with MRD negativity. These rates are very similar to what would be seen with three or four drugs. It was a limited number of patients, and a Phase II study has been developed to start in 2027 with Emory leading. The other study is from a French group that combined Dara with teclistamab for elderly NDMM patients. The median age was 73 (range 66-87). This was highly effective with 100% responses and high rates of MRD negative. The future may look quite different for the standard of care, especially with CAR-T therapy actively being compared now to transplant as initial treatment. Also, there are several studies at Emory with bispecifics in the maintenance setting.

Dr. Vikas Gupta reviewed a couple of abstracts on new drugs for relapsed myeloma (RRMM). One of the goals, especially in the relapse setting, is tailoring therapy to a patient's myeloma. Even though everyone's myeloma starts off as a B cell and eventually turns into a plasma cell, how it gets there is different from person to person. There are multiple different genetic abnormalities, and all these mutations are a factor in whether someone is considered standard risk or high risk. Dr. Gupta wants to focus on how the biology of these different genetic subtypes differ and whether these genes can be targeted in a more personalized way. He presented two abstracts. The first drug is KTX-1001, which is in Phase I which tests the safety of the drug; not really looking at efficacy, but that data is studied as well. This drug targets the gene transcription that helps the cell turn into a translocation(4;14) myeloma cell and may result in myeloma regression. The trial starts out with a very low dose and slowly increases until they find a dose that is not causing too many side effects. This included not just t(4;14) patients but also patients who have had their myeloma come back after at least three different

treatments. The team closely watches side effects, mostly hematologic, such as low platelets, red cells, or white cells. There were other non-hematologic side effects, but most of them were grade 1 or 2. There was an exceptionally low number of grade 3 infections or fatigue related to this drug. It is well tolerated. In this group of 8 to 10 patients, a lot of patients did achieve a very good response. The patient with the best response didn't have t(4;14), so maybe they need a different genetic biomarker for this drug. The trial is open at Emory, so if you have t(4;14) and have had more than three lines of treatment then you might consider this option.

The next study was on Sonrotoclax. Dr. Gupta also works in the lab and is extremely interested in the subtype of myeloma with t(11;14) and how it works. This drug is in the same class as venetoclax and may be slightly more potent. This study combines Sonrotoclax with carfilzomib to see if that combination is safe and how well it works. Looking at safety, there were very few grade 3 or 4 toxicities, with most being grade 1 or 2. The response rates were exceptional with response rates at least 82% and up to 50% complete response in some patients. Responses occurred quickly within the first month and deep responses within a couple of months. At one year, 80% of those were still responding, so durable responses as well. T(11;14) occurs in about one fifth of MM patients so this trial is something to consider if you have this marker.

Q: Can you please explain how you identify the patients for these studies?

A: When we do a bone marrow biopsy, we do a FISH test that helps us identify a subset of these translocations. However, there is a new technology that is coming that helps us identify all the translocations that are present as well as new mutations in common genes that are

mutated in myeloma. It also helps identify some of the targets of the new bispecifics, like BCMA and GPRC5D. This could really help us in the future to further tailor our treatment to your particular myeloma as we start doing these tests on all patients. The test is called PlasmaSeq and we have done it on about 60 patients. In the next month or two, we will eventually switch over to doing it on almost everybody. We need to review the data, but we are getting close to applying it across the board. PlasmaSeq is one of two or three different technologies, and it is FDA approved and commercially available. It is done from the bone marrow because there are usually very few plasma cells in the blood. If you are in remission, it may be hard to get any data since there are not enough plasma cells from which to get the genetics. For NDMM and RRMM, there is a much better chance of getting the data. Also, genetics change over time. For example, if you get treated with a BCMA drug, we have been finding patients whose BCMA becomes mutated when their MM progresses and that tells us that we should not do a second BCMA treatment. This can help us decide with better precision what will work for our patient population.

Q: Is the bone marrow biopsy the best way to get this data and only from the hip?

A: That is a fair question. Right now, it is the best way because that is where the myeloma traditionally lives. Very few patients have plasma cells in the blood and that is usually a bad sign. Plasma cells in the fluid around the lungs or in the belly are usually indications that the myeloma is more aggressive. For the average patient, it really needs to be from bone marrow. As to where we do the biopsy, the iliac crest, as we call it, is really the safest location. They used to do bone marrow biopsies from the sternum, but that is not a big bone, and you run the risk of going through into the chest because it is not that deep of a

bone. There is not going to be a change in where we do the biopsies in the future.

Dr. Jonathan Kaufman gave updates on amyloidosis. What is the amyloidosis relationship to myeloma? Amyloidosis is a disease like MM that comes from abnormal plasma cells in the bone marrow. The plasma cell's job in life is to make antibodies to help fight and prevent infections. The antibodies are made up of two paired pieces. The longer piece is called the heavy chain, and the smaller piece is the light chain. Usually patients with MM, who do not have amyloidosis, have an M-spike or elevated light chains which do not cause any problems. But in amyloidosis, those light chains are misshapen, for reasons we do not fully understand. Those misshapen light chains misfold and aggregate, and become sheets of misfolded light chains, otherwise known as amyloid. Then they get stuck in organs. The two most common areas where amyloid gets stuck are the heart and kidneys. They can be in the liver or in other areas of the GI tract as well as in the nerves and soft tissue. Patients with amyloidosis, in contrast to MM patients with bone disease or anemia, have acute kidney problems and problems with their heart or other organs. It is harder to treat, and we have taken the therapy for MM to treat amyloidosis because we are still trying to get rid of abnormal plasma cells. But we must be much more careful with dosing, so it has taken us a while to move all these treatments to amyloidosis until recently. We are really excited about the first study looking at CAR-T cells in relapsed amyloidosis: NEXICART-2. There are selective criteria for entering the trial: at least one prior line of therapy and measurable disease, but not MM. It is open at Emory where one patient was treated, and the next patient is ready for T cells to be collected. In the study, 95% of the patients had normalization of their disease markers. More importantly, 70% of the patients had organ response where the impact on their organs was reduced and they got

better and it happened quickly. If this study continues to show really good improvement, the investigators and the company are working with regulatory agencies to get this treatment approved.

What about bispecifics? Another study uses elranatamab and the first eight patients all responded and 75% had a complete response with no serious side effects. This study should open shortly at Emory.

Another bispecific study in relapsed amyloidosis used Etentamig. What is interesting about this dosing is that there is minimal step-up dosing and it has been done every four weeks, and they planned on a fixed number of cycles, not treatment until progression. They had a low rate of CRS, under 10%, and all the events were grade 1(mild) and no neurologic side effects. The overall response was 100% and the complete response rate was 82%. The response rates seem higher in amyloidosis because the disease burden is lower. They are moving quickly to bring these new therapies to patients. This has changed how relapsed amyloidosis is treated and it will change how newly diagnosed patients are treated. Another study looked at Venetoclax in relapsed amyloidosis patients with t(11;14) markers. Approximately 50% of patients have the 11;14 translocation. Eleven of the 12 patients had a response and complete response was 50%. Dr. Kaufman said that they are excited about the research in amyloidosis that has been borrowed from myeloma treatments. There are multiple trials open at Emory including underrepresented populations.

Q: When are myeloma patients tested for amyloidosis?

A: We typically test a myeloma patient for amyloidosis when there are symptoms of amyloidosis such as heart failure, enlarged liver, a lot of protein in the urine, or other signs. If a patient has a particularly hard time with treatment that we cannot otherwise explain, that is also a clue that the patient may have amyloidosis, and we will seek that out. It

is very rare for a patient to have symptoms of both myeloma and amyloidosis. Some patients have myeloma for a long time, and they have a small light chain in their blood for a long time, so those patients can then develop amyloidosis. This is a rare event, but Dr. Kaufman has such patients.

Dr. Ajay Nooka talked more about bispecific antibodies. Dr. Mina and Dr. Kaufman discussed the usage in the frontline setting and in the amyloidosis patients. He started with a brief review about what bispecifics are, what is currently approved, and what is available for treatment on a regular basis, as well as what was presented at ASH. Think about bispecifics antibodies as having two arms. One is targeting CD3 on the T-cell and the other is looking for a target antigen on the myeloma cell. Once connected, the drug brings the T-cell and MM cell together, creating an immune synapse so that the MM cells are killed by various mechanisms. The target that it hooks onto the MM cells varies and this is the difference between each of these bispecific antibodies. We currently have four bispecific antibodies that are approved for MM, with three of them targeting BCMA on the MM cell: Elranatamab, Teclistamab, and Linvoseltamab. Talquetamab is the one that targets GPRC5D and Linvoseltamab was just approved in July of last year. All these approvals are for patients who have received more than four prior lines of therapy from three different drug classes. These are not the only bispecific antibodies that are approved. There are close to 160 bispecific antibodies that are in clinical trials across the spectrum of oncology at this time. In lymphoma, there are four other bispecific antibodies approved. These drugs are very easy to administer, but each has its own nuances, and you have to understand just how to give them. Half of our time goes to educating the community oncology

teams on how to administer these drugs so that it can be given closer to home and benefit more patients.

Teclistamab was the first bispecific antibody approved, and it targets BCMA on the MM cells. In the Majestec-1 clinical trial, the overall response rate was 63%. If we treat three people, two of them get a response. In the trials, the patients had five or more lines of therapy (induction, transplant, then maintenance is one line of therapy) which is considered heavy pre-treatment before getting a bispecific. And even among these patients two of the three were responding. Prior to this, what did we have? We had Selinexor, where the response rate was around 30%. Belantamab as a single agent had a response rate around 30%. Even daratumumab, which most of you have seen, has a single agent activity of 30%. So, now looking at these bispecifics, you see very high responses of around 60% to 70%. This is a class of drugs that has a potential in combination with other agents to increase those response rates. Even as a single agent, they have high rates of responses compared to what we have historically seen. And now, with the new study with Linvoseltamab, we are seeing the median PFS significantly better than the previous BCMA bispecific antibodies. Nancy was asking earlier about what is the biggest difference between the three BCMA bispecifics: Elranatamab, Teclistamab, and Linvoseltamab? There are no clear visible differences, but the studies did not happen at the same time. They were done sequentially, so our learning curve for giving these bispecific antibodies improved significantly. By the time we were given Linvoseltamab, we knew what the side effects are and how to monitor those so that we do not lose any patients in any possible way. We saw overall responses to Linvoseltamab at 70%. We do not have any preference of one over the other and all of them work with a similar mechanism of action for the similar indication and it is given IV. The step-up doses are given once a week with Linvoseltamab.

Moving on to the other target, Talquetamab targets GPRC5D on the MM cells. GPRC5D exists more on the myeloma cells than on the normal plasma cells and it has a unique toxicity for those of you that have experienced this treatment. GPRC5D is expressed on the salivary glands, on the oral mucus, and on the keratinized tissues which are the hand, nails, and skin. The response rates are around 70%, so this is not a drug to throw away. We need to figure out how to give this in a safer way so that patients can tolerate it.

With this background, what is new at ASH? There is a new study named CAMMA3. This drug is targeting a new antigen on the MM cells, FcRH5. It has the same mechanism of action (two arms with each looking for targets on MM cells and T cells), and similar side effects as the other bispecifics. In this Phase I trial, the responses reached 50%. So, the more bispecifics that we have targeting different antigens on the MM cell, the more options that we have as sequential treatments. Also, there are opportunities to combine agents in the future. They are looking at the toxicity profile and the safety profile when agents are combined: Is it higher toxicity or is it a manageable safety profile? A question at ASH was: What is the safety of combining a bispecific antibody with another agent? Let us start with Cevostamab plus pomalidomide and dex. The overall response rates are close to 90% and these were safe. They also saw that the MRD negative rates were significantly higher with this combination. Now, can you combine antibodies? The MajesTEC-3 trial was reported at ASH for the combination of teclistamab and Dara, a monoclonal antibody. The biggest concern was the increased risk of infections. All the BCMA bispecific antibodies increase the risk of infection significantly. Would the infection profile with these combinations be prohibitive for their usage? We were all skeptical in the initial days. The Dara is targeting the regulatory T cells, you are knocking off the immunosuppressive

molecules so that the combination of TEC and Dara can enhance the immune synergy. This combination was compared to the standard of care, either Dara/Pom/dex or Dara/Vel/dex. The results showed that almost 90% of the patients received a response on the antibody combination while the standard of care had a 75% response. Looking at the PFS, there were some of the most impressive results that Dr. Nooka has seen in all his time at Emory. At the three-year mark, only 17% of the patients on the antibody combo progressed while in the standard of care group, 70% of the patients progressed in three years. This is a huge difference and will change how we treat myeloma by offering more of teclistamab with Dara in early relapse. These results are as good as CAR-T. Not saying that CAR-T should not be done, but we have other options for effective therapy other than CAR-T or no CAR-T. Emory participated in this clinical trial and all the patients who were on the Tec/Dara group are still in remission. Dr. Nooka is pointing this out because if there is an opportunity for a clinical trial, please do not ignore it. There are so many opportunities to think proactively about advancing the level of results. There was also an overall survival difference as well. With Tec/Dara, the survival at three years was 83% compared with 65% in the other group. The safety profile (risk of infection) was similar in both groups. With such great results in early relapse, what happens when they combine Tec and Dara with the newly diagnosed patients? There was a study presented by the French group and they took two cohorts of NDMM patients who were not eligible for transplant and were over age 65. One group had Tec and Dara, the other with Tec and Revlimid as frontline therapy. The overall response rate was 100% and VGPR was close to 79%. MRD negative rates are being watched and the first 27 patients on Tec/Dara have 100% MTD negative. Not to put the cart before the horse, but this is the time where we are thinking about and discussing what the cure looks

like in myeloma and the first step is to achieve MRD negative at 10 to the minus 6. With 100% of these patients reaching MRD negative, these bispecific antibodies will have a huge role in the frontline setting, based on the available data.

Dr. Nooka went on to other clinical trials presented at ASH. Elranatamab in combination with Iberdomide in RRMM was reported from the MagnetisM-30 trial. So, what is Iberdomide? It is the next generation of Revlimid and Pomalidomide known as CELMoDs. Dr. Joseph briefly mentioned this, and Dr. Mina talked about their usage in the maintenance setting, inducing significantly higher MRD negative rates. Dr. Kaufman was leading the MagnetisM-30 trial at Emory, and a lot of patients were on this, getting significantly better responses. The overall response rate is 100%, even in patients with high-risk disease, and they continue to have this response. They did not see an increased significance in terms of the toxicity when they combined bispecifics with CELMoDs. This is a continuation of combinations with bispecifics, but can we combine two bispecific antibodies? Talquetamab and Teclistamab (Tal/Tec) were combined in a trial for RRMM and extramedullary disease (disease outside the bone marrow). For standard risk patients in the initial study, the response rates are beyond the 90% mark. In the recent update, they looked at the patients who had disease outside the bone marrow. That means the myeloma is occurring in masses that people can feel under the skin. This type of myeloma is very difficult to treat, and patients have a reduced survival rate. Across all bispecific antibodies, instead of 60-70% response rates, this group has 30-40% response. So, when the bispecifics are combined, we see a significantly higher response rate for extramedullary disease. Dr. Nooka thanked all the myeloma team for the great research. If someone on the Emory team approaches you asking for a blood or marrow sample, it would be used for us to understand more about the

disease than anything else and to try to improve how we treat myeloma. We have a great clinical team, not just the physicians, but the huge Advanced Practice Providers (APP) and everyone is dedicated to your care. There is also the clinical research team that works with all the labs to understand the disease.

Dr. Nisha Shah will try to summarize some of the updates from ASH about CAR-T. There are two CAR-T products approved for RRMM patients: Carvykti and Abecma. Both are approved for patients in their second line of therapy, as well as patients who have had more lines of therapy. The update on CARTITUDE showed that for standard risk patients at 30 months, 80% are doing well with overall survival at 87%. The high-risk patients are the ones where we need to work harder to improve outcomes for their CAR-T therapy. When a bone marrow biopsy is done, we look at the newer definitions of cytogenetic features and try to understand the flavor of MM that a patient has. This might help to tailor treatment accordingly to what we call a risk-adapted treatment based on a patient's profile. They looked at data available for cytogenetic features in close to 600 patients and did not see any features concerning high-risk disease. However, we still see that a portion of patients are relapsing early, within 18 months of their initial treatment. Those are patients who we call functional high-risk. Based on everything that we see prior to going for treatment, we would not have predicted that they would progress so fast. And then we also looked at patients who have the genomic features of high-risk myeloma and the outcomes for all these patients. This is a real-world study, and it helps us to understand that we need to work harder in terms of bridging therapy as well as the therapy that they received prior to going for CAR-T. These are the patients where we should be using CAR-T therapy earlier than later. Also looking at the real-world data, we know that there are some early toxicities such as cytokine release syndrome

(CRS) and neurotoxicity. There are also some delayed toxicities like cytopenias (low blood counts), low hemoglobin, low platelets, or low neutrophils. Some patients have neurotoxicities that develop late after CAR-T or secondary malignancies and infections are something that we worry about for the first six months after CAR-T. In the real-world data, the delayed neurotoxicity occurs in about 10-12% of patients. This was also shown in the trials that led to FDA approval. There are three different types of neurotoxicities that doctors look for. One is cranial nerve palsies, and one is Parkinsonism-like features that some patients can develop. The third is ascending neuropathies. Parkinsonism is seen in about 2-5% of patients who get CAR-T. About half of these delayed neurotoxicities are reversible with prompt intervention, best to prevent these from occurring. Using real world data, doctors can see which patients are developing these delayed neurotoxicities. Studies have shown that patients who have a rapid expansion of their CAR-T infusion are potentially at risk of developing these delayed neurotoxicities later, in one to three months. The test for CAR-T expansion is not an easily available test, so we found a surrogate, which is looking at the absolute count of a particular subset of white cells called lymphocytes (ALC). The provider keeps tract of the lymphocyte count for patients after their CAR-T infusion. The real-world data shows a good correlation between the CAR-T expansion and the lymphocyte count. This can identify patients where we need to intervene early to stop this expansion of CAR-T cells early. To stop the expansion of CAR-T cells for now steroids are administered to patients who have a rapid expansion of their lymphocyte count. There are some studies at sites, including Emory, which will look at this going forward to see whether this early intervention is preventing delayed neurotoxicity.

What are some other things that we can do to mitigate delayed neurotoxicity for patients with CAR-T? Another abstract presented data for over 700 patients who received CAR-T and out of those, Parkinsonism, or delayed neurotoxicity in about 10% and about 3% of those patients have Parkinsonism. We have about 22 cases of Parkinsonism in a data set of over 700 patients getting CAR-T therapy. Of these 22, 21 of those were patients who did not respond to bridging therapy. Bridging therapy is what we use after the patient has undergone apheresis to collect their T cells and wait to get their CAR-T infusion. This is something that we are doing routinely in our clinic to control the myeloma while waiting for the CAR-T infusion. Potentially that lowers the risk tenfold for patients developing Parkinsonism in the future. There are two interventions that we can do. One is to give some bridging therapy that is working to control the myeloma. The other is to monitor lymphocyte count and if it rises very rapidly, we give them steroids. A lymphocyte count over 3000 is potentially when we need to intervene with steroids, as confirmed with the real-world data. At ASH, these were both validated that these are effective strategies and we should be continuing over time.

Another side effect that we see after CAR-T is cytopenia: developing low blood counts in any cell line, like white cells, hemoglobin, or platelets. We have seen this and is there a way to predict this? There was another scoring system that was used that not only looked at the platelet count prior to going for their CAR-T apheresis but also looked at some other features that are concerning for DNA damage in the bone marrow. Now these patients have gotten several treatments for their myeloma, and all these treatments potentially could lead to some damage to their stem cells or bone marrow cells and putting them at risk for developing other cancers in the future. What they are doing in this study is looking at some of the DNA mutations that are present in a

patient prior to going for CAR-T apheresis and whether that can be used to predict who would be at risk of developing cytopenia in the future. Using the risk scoring system, they see that patients who had intermediate or high scores on the scoring system are at risk of developing delayed cytopenias one year after Car-T therapy. This is a very easily adaptable approach that can be done for patients and could be used to predict who could have delayed cytopenias in the future. Looking at some of the new CAR-T products, is there something in the process in which the CAR-T cells are produced that could mitigate these side effects? Looking at a new CAR-T which still targets BCMA, but has a new manufacturing process where preclinical data is suggesting that because of this new structure, these patients will have slightly less side effects because it doesn't cause as much inflammation inside the patient's body once CAR-T cells are infused. One study showed that this was a highly effective CAR-T therapy compared to the data from the trials of the currently approved CAR-T therapies. Anito-cel, which is the name of this product, was administered the same as current products and the outcomes are as good as Carvykti with a response rate of 96%. It also seems like anito-cel has minimal CRS at grade 1 or 2. A very few proportion of the patients developed neurotoxicities, about 7% had early neurotoxicities. No patients who got this product developed delayed neurotoxicities, including Parkinsonism or cranial nerve parenthesis or any sort of ascending neuropathy. This is with long term follow-up of this group of patients. This is very reassuring that we have new products in the pipeline that are not causing the same delayed neurotoxicities, which typically happen in the first six months. This product is not FDA approved, but potentially this year. There is also a new CAR-T product that is targeting both BCMA and CD19 and it is produced within three days. When it is produced so fast, we think that the T cells are younger and fitter and potentially more effective in

controlling the myeloma. Also at ASH, they revealed a new technology where instead of manufacturing CAR-T outside the body, they are infusing these particles inside the patient's body. There is no need for removing any white cells from the patient or giving them lymphodepleting chemotherapy. We now wait about six weeks to manufacture CAR-T, so this new product eliminates all these steps for the patient. This is still very early in the testing process, but a very interesting strategy. This is a trial that we are hoping to open at Emory soon. Current trials at Emory include: anito-cel which had no incidence of delayed neurotoxicity; the dual targeted CAR-T that was mentioned; allogenic CAR-T which uses donor T- cells to make the CAR-T product; CAR-T that seeks an alternate target on the MM cells; and several more trial coming!

Dr. Richa Parikh just started last year with the Emory Myeloma Group. There were so many exciting studies to discuss from ASH and that is great news for all our patients. She presented updates in supportive care therapy. The big study discussed was the evaluation of safety and efficacy of denosumab, also known as Xgeva, in patients with both myeloma and severe kidney impairment. Myeloma patients often present with bone disease and that predisposes our patients to developing skeletal-related events. Almost 50% of patients at diagnosis have a pathological fracture or need radiation therapy or surgery. Renal impairment, which is also a characteristic feature of MM further complicates treatment strategies. The medications used to prevent bone-related events, commonly called bone-strengthening agents (like bisphosphonates), are cleared through the kidneys. That is why we must pay attention to patients who have kidney impairment or kidney injury because of the myeloma. Zometa is one of the major bisphosphonates that we use in the management of bone disease in

myeloma patients. It acts by inhibiting bone resorption, or bone breakdown, and thereby helps treat hypercalcemia, which is high levels of calcium in the blood. Zometa is cleared through the kidneys and thus it is contraindicated (not advised as a course of treatment) in patients with severe kidney impairment. Xgeva is a bone-directed monoclonal antibody, which, unlike Zometa, is cleared by the reticuloendothelial system (in the blood) which makes it safe for patients with kidney disease. Back in 2018, a group published an article comparing Xgeva to Zometa in bone disease treatment of newly diagnosed myeloma patients. But this study enrolled only patients with normal kidney function because patients that have severe kidney impairment, should not receive Zometa, so they were excluded. All the clinical trials that looked at Xgeva for safety and efficacy in preventing bone disease excluded patients with severe kidney impairment. These trials were not just for myeloma, but other cancers like breast cancer and prostate cancer. A retrospective study that took place in nine centers was reported at ASH. This study was conducted by the IMWG (International Myeloma Working Group) and looked at the efficacy and safety of Xgeva in patients with myeloma and severe kidney impairment. It looked at myeloma patients with bone disease and are on active myeloma treatment and have kidney impairment. The primary endpoint was to see if Xgeva was able to prevent new skeletal-related events at two years. Secondary outcomes were assessing the safety and making sure that it was not interfering with kidney response or myeloma response in these patients. The study included 118 patients with a median age of 70 years, and it was a good split between NDMM and RRMM. About one quarter of the patients were on dialysis. The primary endpoint was looking at the incidence of new skeletal-related events at two years. It was astonishingly low with only one event, which is only 0.8% of the patients. In terms of safety, the major side effect

was hypocalcemia (low calcium levels). The calcium can drop too much. A total of 55% of patients developed hypocalcemia. Most of them were grade 1 or 2, but almost one-third of patients also had grade 3 hypocalcemia. This incidence is much higher as compared to when it is used in patients with normal kidney function. This is something to be aware of. In terms of measures of response, 9% achieved complete renal response and 13% achieved partial renal response. This drug did not interfere with renal recovery while the patients are getting myeloma treatment. The other side effect that we all may have heard from oncologists is ONJ (osteonecrosis of the jaw). This is a rare side effect, and the incidence is similar with less than 5% in patients, even with normal kidney function. In conclusion, Xgeva is highly effective at preventing new bone-related events in myeloma patients, even in patients with kidney impairment. It does come with a high risk of hypocalcemia, so we advise our patients to be on calcium and vitamin d supplements while on this therapy. Currently we give bone strengthening agents every month for the first year to patients who have bone disease, after making sure that they have dental clearance. Then we continue it every three months for the next two years and discontinue if the myeloma is in remission at that point in time.

The other study that she wanted to highlight was a study that looked at the safety of MMR (measles, mumps, and rubella) vaccines for patients with MM receiving Dara after SCT. MMR is a live vaccine, so we worry about using this in immunocompromised patients, especially those who are on Dara as maintenance. This is especially important, in current times because the rates of measles are rising in the US as people are turning away from vaccines. This was a multi-center retrospective study looking at the safety of MMR vaccines. There were 41 patients with a median age of 65 and MMR vaccines were administered at the discretion of the oncologist. Most of them received the MMR2

formulation. We looked at safety outcomes, meaning looking for anybody that developed infectious complications, confirmed infection traits, or rash. Most of the patients had a median IgG level of 482, which is above our threshold of starting IVIG therapy, and about a quarter of the patients were receiving IVIG when they received the MMR vaccination. 52% were on Dara maintenance in combination with Revlimid or other combination with Dara. One-third of patients were on Dara alone. Median time to MMR vaccination was 768 days, which is more than two years from receiving the stem cell transplant and about two years from starting Dara. The study showed that 5% of patients had sinusitis and another 5% who developed COVID-19 infection after eight days. We do not know if this was related to the MMR vaccine. And there was one patient that reported skin rash, headaches, and arthritis. In summary, no patients developed acute measles, mumps, or rubella and there were no hospitalizations or deaths reported after this vaccination. The conclusion of this study was the MMR vaccine was safe in MM patients receiving Dara post-transplant. However, this is a small study with only 41 patients, and the study did not have vaccine titers to check for the efficacy of the vaccine. The preliminary data from this study is helpful.

Dr. Sagar Lonial wrapped up this session with a couple of really interesting and thought-provoking abstracts. As I reflect on all that you have heard today from our outstanding faculty, I hope you have the same sense of pride that I do, having watched them present really up-to-standard data and realizing that living in our area, you have access to probably the best program in the world. That is exciting and for all the studies shown, we have either had our hands on, been a part of, or we are developing version two of those studies to try and make things even better.

What I want to highlight is the power of the size of our program. We do roughly 100 CAR-T cell treatments a year. We do over 300 auto-transplants a year. Just to give you some perspective, there are very few programs, if any, globally, that have that kind of volume on cell therapy. We presented data at ASH on outcomes following CAR-T. The Carvykti arm, with about 150 patients, our outcomes are as good as the Cartitude study with standard risk patients. The patients in our data were not all standard risk, yet we had the same outcomes. That speaks to what you heard from Dr. Shah about trying to optimize how we give CAR-T, how we reduce toxicity, and how we improve outcomes. That does not mean you will not have side effects, but the kinds of approaches that our team takes substantially improves the results for the patients. There are other places in town and even in the state that give the same therapies, but they do not have the know-how and expertise to make sure that you get the optimal experience. We know that they do not because we have had to clean up some of their messes. It is important to realize that you do have a pretty significant resource, and it may be up to you to make a referral to see us. Many programs, particularly in the state, will not refer to us simply for competitive reasons. We do not think that is good for the patient. You may gain benefit from coming to see us, at least for a visit or a second opinion, to make sure that you are on the right path overall.

From some late breaking news, I want to show you two trials that we think are going to revolutionize treatment in myeloma. The first is after a CAR-T cell therapy targeting BCMA, they gave Cevostamab as maintenance with the idea of what we call “switch therapy” that you hit with one target and then you come in with another target. This trial is our T-rex trial. For the foundations and donors to the program, we are excited to announce we have had our first call with the FDA that went very well in December. We have a final version of the protocol

that Dr. Joseph is wrapping up, and we hope to be able to start this study in the first quarter of 2026. It is total immune therapy, which is using every immune target in myeloma for two years in newly diagnosed patients, and then we stop. We walk away and see whether we have cured patients. That is ultimately our question. It used to be called the CURE trial. That was a little presumptuous, we thought, so we changed it to Total Immune Therapy and so now we call it T-Rex. You will see version 2 of this trial continuing the dinosaur theory. We start off with something relatively simple, Isatuximab, carfilzomib, lenalidomide, and dex. The goal here is that we just cool down myeloma before we come into the second phase, which is elranatamab and Iberdomide, so a BCMA-directed therapy plus the new CELMoD. We then give a GPRC5D CAR T cell, and after that we come in with FCRH5 directed maintenance with Cevostamab. At the end of two years, we stop. We are done. We presume that patients will achieve sustained MRD negativity after months 12 and 24. And the real question is, can you stop? And if you stop, what percentage of patients never need therapy again? As you may imagine, there is a lot of correlative testing that goes into this. It took us 18 months to get the five companies involved to be on the same page and to be in the same room. But between Dr. Nooka, Dr. Kaufman, Dr. Joseph, and me, we were able to get all the companies to agree to give us their drugs. They did not give us any funding, and that is where the donors and the foundation have been so critical to us being able to do this trial. To run this trial will cost roughly $5 million, and that includes the science as well as the clinical care, and we're just incredibly grateful that we have people that believe in the concept and believe in our program, and are willing to help fund us to be able to do this. So, this is T-rex. This is the first trial that we hope to open relatively soon.

The second trial is called Triceratops, and it is really, again, continuing

the dinosaur theme for our curative intent trials. This one is called TRI because we are going to use the J&J tri-specific for patients with first relapse. This is not a newly diagnosed trial; this is a first relapse trial. The patients get daratumumab, carfilzomib, and dex times 2. They then go on to talquetamab and mezigdomide (another CELMoD). They then get Carvykti as consolidation, which is the BCMA-directed CAR-T therapy. Then they get the J&J tri-specific as maintenance again for just a year and then stop therapy. So again, the concept here is limited duration, hit multiple targets, and then walk away. And again, our intent here is a cure. There is the whole science and correlative laboratory studies that are applicable to this one. This trial will cost around $4 million. We have gotten two different companies to play in the sandbox together for this one, and we got J&J to give us access to their tri-specific, which they are very picky about who and when they give this to. We did not get funding for this. They just gave us the drugs, and this is, again, about a $4 million trial. So, in total the two trials together are about $9 million. Between foundations and donors, we think we have what we need, we hope we have what we need, to be able to get these two trials started in 2026.

I want to tell you that this is not the Emory program, this is our program together. And we are grateful for the support you all give us. Having 60 people show up on a Saturday morning to hear results and updates from our stellar faculty is just so gratifying. I hope this time was useful for you, and we are here to help however we can, even if it is just seeing a patient for a one-time second opinion to make sure that they are on the right track.

Q: What is the future of talquetamab (Talvey) in myeloma treatment? Note: Teclistamab is Tecvayli.
A: So, I think what talquetamab really brings to the table is a new target, and we always want new immune targets, because again, they

are clearly highly active. There, again, I am sure you saw that today the data combining TAL and TEC for patients with extramedullary disease, the redirect trial. It is extremely exciting that that combination can result in sustained responses in a very high-risk cohort. So, in my mind, the question is, what is the right dose and schedule for talquetamab? We have patients on this at what many would argue is a homeopathic dose of once a month, and they do not have the skin and gut toxicity. I think understanding the right dose and schedule is the key to how we use talquetamab down the road.

Q: What follows CAR-T for those that are heavily pre-treated, you know, the veterans of the myeloma game?
A: It so much as what follows CAR-T, in terms of therapy, but what targets are still available. And the first question is, can you retreat? If you had a BCMA CAR-T, can you retreat with BCMA-directed therapy again? Now we have the ability to sequence BCMA that we get back within a week. So, we understand whether there have been mutations or loss of BCMA expression that allow us to say, should we retreat with BCMA? If we want to switch targets, then GPRC5D becomes an option. Cevostamab, with FCRH5 target, becomes an option. I think about it more like switching targets or going back to the previous target if we think it is still expressed, as opposed to a specific therapeutic modality that we would go to after CAR-T. And it is good to have the options of multi-targeted treatment.

Nancy wrapped up the meeting: This has been awesome! Thank you so much to the Emory/Winship team for taking their Saturday morning as well, and we really appreciate it.

Submitted by Nancy B

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Meeting Minutes

Southside MM Support Group

January 24, 2026

Meeting Minutes

Southside Virtual MM Support Group

January 24, 2026

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Next Meeting Dates

Southside group general meeting. Saturday, February 28, 2026, 10:30 AM-12:30 PM. VIRTUAL ONLY. Discussion: Patient and Care Partner Voices.

For Men (with Myeloma) Only. Tuesday, February 24 @ 6:00 PM. VIRTUAL ONLY will reconvene after missing over the holidays. Please join us – let's catch up!

Upcoming Topics: March -"All about your labs" (Emory Winship) and April -IMF Resources (Katie Atkins).

Business and News

Our January meeting started with a centering exercise – focused on deep breathing, relaxation, and preparation for participation in our meeting. Gail shared her recent experience with putting the "breathing" to use. During the monthly blood draw, a new lab tech drew Gail's tubes of blood. Since her finicky veins roll, she asked for her 'special' lab tech, Tan. They proceeded but the first and second attempts (creating a medium sized hematoma) were unsuccessful. Both sticks were painful because it called for 'digging' around trying to follow the rolling vein. Deep breathing can

help us through these experiences – mind, body, sprit connections, and taking us through difficult moments or periods of time.

Southside welcomed one new member, Moses, referred to our support group by Dr. Sarah Myers of the VA Hospital. He was diagnosed in November 2025 and treatment is going well. Moses initially went in for chronic kidney disease and his doctor called for further evaluation and found myeloma based on lab results. Moses was encouraged to also join the monthly Men's Only group on Tuesday. Deborah T., retired VA nurse for over 42 years assisted in his referral.

It has been an unusually cold winter with upcoming power outages and ice forecasts. Members shared their strategies for minimizing the negative impact. Freezing gallon (or smaller) jugs of water for blocks of ice in your freezer, using rainwater to freeze in coolers, and to protect food in the fridge. Others included small generators that can power electric blankets, lamps, and phones. Top off gas tanks – potentially for heat, charging phones, and so gas lines will not freeze. Good luck to all!

Guest Speaker

Our guest speaker was Kim Nickels, Blood Cancer United/LLS, Senior Manager, Patient & Community Outreach and is familiar with its blood cancer programs. Kim's service area includes Georgia and South Carolina. She spoke of the rebranding of LLS to Blood Cancer United to be more inclusive of all the blood cancers, including myeloma. She shared many resources and demonstrated navigation of the website.

Overview. Blood Cancer United funds research, seeks to increase access to quality and affordable care, empowers over 50,000 volunteers across the country, helps to advocate for patients/families/care partners, and provides funding for blood cancers. The re-branding of LLS to Blood Cancer United is to be more inclusive of ALL blood cancers.

· Medical Debt Case Management Program specialists include Oncology Social Workers. Contact: 800-955-4572/833-507-8036 or apply online bloodcancerunited.org/medicaldebt

Counsels on existing and upcoming medical debt from pre-diagnosis through survivorship. Call this resource early in the day – there is a limited number of cases open each day. Hours from 9 AM – 9 PM EST.

· Clinical Trials Support Service include Master level nurse practitioners and Physician Assistants. They assist with informed decision-making regarding the availability of clinical trials, eligibility criteria, and removing participation barriers. Only 7% of eligible patients participate in CTs; only 6% of those are Black or Hispanic/Latino. 20% of all CTs fail due to insufficient enrollment. Most frequent barriers include geography and insurance constraints.

· Nutrition Education Services Center. Free resources for all cancers, not just blood cancers. Services available to care partners as well. Consult is a one-to-one. Contact: 877-467-1936.

· Financial Assistance Program includes patient financial aid and co-pay assistance. Supplements private, Medicare, Medicaid, and TRICARE insurance, general travel assistance, and CAR T travel assistance. Urgent *needs* – all different buckets of funding. Announced funding runs out quickly. So, keep checking back if the program you are seeking is closed. Look for a green indicator on funds when they are available. HINT: Funds usually open in the afternoon.

· Peer Support Programs include First connection, Blood Cancer United Community, Online chats, Support Groups and more. There is training for volunteers. Peer support connections are available for those newly diagnosed, wanting to share treatment plans, and for care partners. Two Support Groups meet in ATL (virtual only) for all blood cancers.

· For Caregivers. Free tote, notebook, pen, and organizer.

· Veterans. Dental services, Support Groups, and more.

Two conferences in 2026

· Spring- Community Link, which focuses on education and outreach for Black and Hispanic communities. Blacks are diagnosed with myeloma twice as often as whites.

· Fall- annual Southern Blood Cancer Conference (more to come). There is also a conference in Charleston in March, if anyone is interested.

On the website

· Resources for patients, care partners, and healthcare professionals (CEs/CMEs available).

· LLS Blog: Podcasts (called Bloodline); Educational videos; Fact sheets; and a Newsroom.

· Information booklets are available for downloads or to have mailed to you. You can get 50 copies of materials – all are free shipping.

· Interactive Library with 3-D models and simulations of procedures like bone marrow biopsies/aspiration and scans (MRIs, etc.)

· Health Manager App. Track your medication, get recipes, develop grocery lists, questions for providers, more. https://bloodcancerunited.org/educationalresources/healthmanager-app.

· Advocacy. Support patients and research at state and national level. Sign up and advocate from the comfort of home.

· Bold Goal. By 2040, Blood cancer United will enable blood cancer patients to gain more than 1 million years of life – more celebrations, birthdays, anniversaries, graduations – Life.

<u>Voices of Patients/Care Partners</u>

Sandy B., who retired from HP, had her insurance changed to ALIGHT as of January 1, 2026. Her premiums will be cheaper now and will be taken from her social security. She says she no longer needs the LLS co-pay

assistance and would like to let them know. Further, she was told that her Revlimid, through ALIGHT, would cost 33% of retail cost. Kaiser sent her a letter saying that they will provide her a discount of 100% of the cost – so $0. Fantastic news! Regarding her dental issues, ALIGHT dental surgeon reported that the dentures were ill fitting because other surgeons went 'around' a bone in her upper mouth versus removing the bone. At this point, the procedure would be too risky, which could result in a stroke or heart condition. Zometa and Revlimid could be the source of her dental problems. The half-life of the bone strengthener (Zometa) is 10-15 years. Nancy shared the newer guidelines for using bisphosphonates is about two years with different frequencies – monthly, quarterly. Take note to discuss bisphosphonate use with your doctors. Schedule regular dental visits and remind your dentist that you are a myeloma patient. Joyce shared that she has gone through three major oral surgeries for gum infections. Her problems started with abscess and gum infection. Her last Zometa was about 10 years ago. Her last surgery was in October 2025. Flora also reports dental issues. Gloria's myeloma has mutated, so now her chromosomes 11 and 14 have translocated (t11;14) which changes her myeloma to high risk. She is not on any medications now, but Venetoclax has been proposed. We will connect Gloria with Jeff W, who was on Venetoclax (oral medication) for about five ½ years. He recently had CAR T and at last report is free of myeloma.

Myeloma in the news. David shared that FDA released a Draft Guidance for Minimal Residual Disease/MRD and Complete Response/CR: Use as Endpoints to Support Accelerated Approval for Myeloma Drugs. Also, there was a cautionary, one-study report that showed CARVYKTI as the CAR T cell therapy produced less toxicity than ABECMA. However, there were procedures that could lower that toxicity. Again – one study. Other CT CAR T drugs show similar results. We need to look closer at the CAR T experiences of Marcia and Wanda. They were both told they were boring patients – no fever, no CRS. Both adhere to a plant-based diet. Wanda had her CAR T last week and did very well. She tried to send her caretaker home. She was cooking, washing loads of clothes, and walked two miles

versus the one recommended by the staff while in the hospital. Marcia was told the CRS was due to inflammation. She was told the whole food plant-based diet (WFPB) diet helps to reduce inflammation. Morehouse School of Medicine physician (Dr. Jennifer Rooke) offers lifestyle Medicine (WFPB) webinars each Wednesday, 6 PM-8 PM.

Respectfully submitted,

Gail