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https://reachmd.com/programs/cme/contrasting-trop2-targeted-adcs-in-breast-cancer-therapy/33024/

Released: 07/17/2025
Valid until: 07/17/2026
Time needed to complete: 30 Minutes

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

Contrasting TROP2-Targeted ADCs in Breast Cancer Therapy

Chapter 1

Dr. Kalinsky:
Hi. Welcome to this educational activity on TROP2-targeted antibody-drug conjugates, or ADCs, and the treatment of metastatic breast cancer, also known as mBC.

In this first chapter, we'll discuss treatment strategies using TROP2-directed ADCs for hormone receptor-positive metastatic breast cancer.

This is CME on ReachMD. I'm Dr. Kevin Kalinsky at Winship Cancer Institute in Atlanta, Georgia. I am fortunate to also be joined by Dr. Jhaveri.

Dr. Jhaveri, if you want to introduce yourself?

Dr. Jhaveri:
Yeah. Thank you so much. It's a pleasure to be here today. I'm Dr. Komal Jhaveri, a breast medical oncologist at Memorial Sloan Kettering Cancer Center.

Dr. Kalinsky:
Great. Let's get started. Dr. Jhaveri, could you give us a brief overview of TROP2 in the development of TROP2-targeted therapies in breast cancer?

Dr. Jhaveri:
Absolutely. So TROP2, or trophoblast surface protein 2, is really a transmembrane glycoprotein that's involved in calcium signaling and really present on the surface of multiple cancer cells. It's very highly expressed in breast cancer, in fact, in all breast cancer subtypes, with the highest expression really in the triple-negative breast cancer subtype, which is why when we think about utilizing TROP2-directed ADCs, we don't necessarily think about choosing a tumor only if they have expression because the majority do have this expressing on their cell surface.

And in fact, now, because of this, we already have approved therapies against TROP2. So we have TROP2 ADCs that are approved in triple-negative breast cancer; we have sacituzumab govitecan. And then for hormone receptor-positive disease, we have 2 options available already, which is sacituzumab govitecan and also datopotamab deruxtecan. And a third one that is currently in late-phase development, sacituzumab tirumotecan, and we'll see how that pans out for both hormone receptor-positive and triple-negative breast cancer.

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With that, why don't we hear about what have we learned with respect to clinical data from these TROP2 ADCs? Maybe Dr. Kalinsky, you can walk us through the clinical data.

Dr. Kalinsky:
Yeah, for sure. So we have data. The first ADC that we had available for hormone receptor-positive, HER2-negative disease was sacituzumab govitecan based upon the TROPics-02 study. This was a population of patients that were a little bit more heavily pretreated than what we had seen for the population with datopotamab deruxtecan that you'd mentioned that I'll talk about in just a moment. But we saw that there was an improvement compared to physician-choice chemotherapy. It was a randomized phase 3 trial that demonstrated an improvement in progression-free survival but also an improvement in overall survival as well. And the data that we saw in terms of the tolerability of sacituzumab govitecan in this setting was similar to what we'd seen in the context of triple-negative breast cancer. That was the first approval.

Then we had the approval of datopotamab deruxtecan. This was the first approval for this particular agent. Sacituzumab govitecan is given 2 weeks on, 1 week off. Datopotamab deruxtecan is given once every 3 weeks. This also had a design, randomized phase 3, comparing Dato-DXd compared to physician choice chemotherapy. It was evaluated a little bit earlier on than what we saw for sacituzumab govitecan. And in this particular study, there was an improvement in progression-free survival. We've seen that there hasn't been an improvement of overall survival, but this study, it's a little bit different than sacituzumab govitecan, is that we had approved ADCs that we could also give at the time of tumor progression with the Dato-DXd trial, TROPION-Breast01. And so that's important to keep in mind in the context of interpreting those overall survival data.

I also just want to highlight, before I talk briefly about the additional agent that you described that's currently in development but not yet approved, that there are differences in tolerability between the 2 agents. Sacituzumab govitecan, alopecia, neutropenia, and growth factor use, and then it's important with Dato-DXd to think about stomatitis and also ocular issues as well.

Then lastly, we have TroFuse-010, which is an ongoing study which is also looking in this context of hormone receptor-positive, HER2-negative disease. It's a 3-arm study, so it's giving the agent that we talked about, which is given once every 2 weeks. This is a 3:3:2 randomization where patients get either the TROP2 ADC by itself or TROP2 plus pembro versus physician-choice chemotherapy. And this also is given a little bit earlier on.

So that's really the context of these. The other thing I'll just say in terms of tolerability is that we see that the agent that's currently in development, that's not yet approved, it has a kind of a tolerability that's in between, right? Where you see some stomatitis, you see some neutropenia, and so it's kind of like if you combined sacituzumab govitecan and Dato-DXd together, like that's kind of the tolerability that we're seeing.

All right. Dr. Jhaveri, what do you think about the clinical implications of what we have just discussed and how you utilize the ADCs?

Dr. Jhaveri:
Yeah, no, I think this is really a good problem to have in clinic, right? We have more than 1 option now available to our patients. We were used to that with other agents, but now we have that with TROP2 ADCs as well—two already approved for HR+ breast cancer, as you walked us through, and then one on its way in further development.

And so the way I think about them is they are both TROP2 ADCs, yet they both have distinct administration schedules. They both have distinct side effect profiles, and I think it's good to see how they can be utilized. Now, for TROPics-02, it was a very heavily pretreated patient population, as you said, median 3 lines of prior chemotherapy that these patients had received—almost all patients had had prior CDK4/6 inhibitor, and yet we were able to show overall survival benefit. Because when TROPics-02 was designed, we necessarily did not have T-DXd or any other ADC with overall survival gain available in clinic to our patients.

In contrast, with datopotamab, we've been able to see that even earlier in the setting, with its utility, we're seeing some benefit with this drug as well. So median just 1 line of chemotherapy, and you can still utilize this agent, which is an every-3-week regimen and a different side effect profile. We haven't shown OS benefit with this drug, but again, when this study was being conducted, we already had availability for sacituzumab and T-DXd. And in fact, when we looked at the postprogression therapies, there was a higher rate of patients receiving the approved T-DXd and sacituzumab, which might have potentially impacted the OS gain that we could have proven in this study prospectively.

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So I think one thing that I have learned is that it might get trickier with drug development to show consistent OS benefit, especially if we have more and more agents with OS benefit currently available to our patients. We don't want to necessarily hold back on some drug development just because we're not going to be able to prove OS benefit, is what we need to really humbly appreciate based on these data, I think.

But it's good to have these options. I discuss both my options with my patients to see what might be appropriate for them based on the administration schedule and the side effect profile.

Dr. Kalinsky:
Yeah, I totally agree with that. I think you highlighted, really, 2 important pieces here. I think the first one being that the field changes. And when we were evaluating the phase 3 trials that had been reported, it was prior to utilization of T-DXd, right, for those patients with HER2-low and now consideration in HER2-ultralow disease. And so that also will influence, in the real world, how long patients may be on TROP2 antibody-drug conjugates, just given that the payload is a similar class of payload with both of these agents.

There were some sensitivity analyses that were done just to look at, okay, would you have seen a survival in patients if they had not received a subsequent ADC? But then you're really starting to get into some small numbers, and I just think that that's important when we're interpreting whether to utilize Dato-DXd.

Dr. Jhaveri:
Yeah, so I personally—this is my personal opinion—that I don't think that that has deterred me to think about datopotamab as a reasonable option for my patient in the clinic.

Dr. Kalinsky:
This has been great. Before we move along, can you give 1 key takeaway from this chapter?

Dr. Jhaveri:
Yeah. I think we have two TROP2 ADCs that are available for hormone receptor-positive breast cancer in the metastatic setting. We certainly want to think about the administration schedules and the side effect profile and have a good patient-physician decision-making process so that we can utilize these drugs appropriately. I do use them for our IHC 0 or truly IHC null, HER2 null tumors, given that we also have trastuzumab deruxtecan otherwise approved for our HER2-low and -ultralow breast cancer. So certainly excited about having this option for patients.

Dr. Kalinsky:
Great. Awesome. Well, this has been an excellent discussion. Please join us for Chapter 2, where we'll be discussing TROP2 ADCs in metastatic triple-negative breast cancer. Stay tuned.

Chapter 2

Dr. Kalinsky:
Hi, welcome back. In the first chapter, we went through treatment strategies using TROP2-targeted ADCs for hormone receptor-positive metastatic breast cancer, mBC. In Chapter 2, we're going to discuss treatment options using TROP2-targeted ADCs for metastatic triple-negative breast cancer, or mTNBC. All right, let's get started.

Dr. Jhaveri, would you mind reviewing the clinical data on TROP2-directed ADCs in second-line metastatic TNBC and beyond, please?

Dr. Jhaveri:
Absolutely. So I think the very first exciting data that we saw for triple-negative breast cancer with an ADC was with sacituzumab govitecan in the phase 3 ASCENT trial. And really, that trial was for patients who had 2 or more lines. There was no real upper limit on that study, which was very interesting, right, with our triple-negative breast cancer patients. One of these therapies could have been in the early-stage setting. And nearly about 530 patients were then randomized to receiving sacituzumab versus physician-choice chemotherapy, where we did see not only a statistically significant improvement in PFS, but we also saw a statistically significant near doubling of OS for the very first time, which got us very excited to think about utilizing sacituzumab for patients in the second-line setting

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with metastatic triple-negative breast cancer.

Now, beyond ASCENT, we've also seen some data for datopotamab. Right now, we only have available data from the phase 1 TROPION-PanTumor01 trial. It enrolled both cohorts: triple-negative and hormone receptor-positive cohort. The triple-negative cohort, small phase 1 cohort of 44 patients, but what really caught my attention in that cohort was that if you were to look at just the PFS for the entire cohort, it was 4.4 months. But if you were to think about a topoisomerase I-naïve patient population within that cohort, it was 7.3 months. So not only obviously the activity was better if we were topoisomerase I naïve, but what was also nice to look at is, despite prior topo I payloads, there was still some activity seen, perhaps speaking to the potency of the drug or at least got us excited in the phase 1 trial. We yet have to see definitive phase 3 data with this drug.

Now, the third agent that you have discussed for hormone receptor-positive in Chapter 1 was sacituzumab tirumotecan. We have data from the OptiTROP-Breast01 trial as well. This was actually a phase 3 trial conducted in China—again, similar patient population to the ASCENT study. The distinct part about this drug is unlike sacituzumab, which is Day 1/Day 8 of a 21-day cycle, 10 mg/kg, and datopotamab, which is 6 mg/kg every 3 weeks, sacituzumab tirumotecan, or sac-TMT as we call it, is 5 mg/kg every 2 weeks. And so patients were randomized to receiving that versus physician-choice chemotherapy. And again, improvement in progression-free survival, which was significant with sac-TMT, was 6.7 months. The overall survival was not reached in the sac-TMT arm. So good data there with that drug as well.

In terms of safety profile, there are slight differences. Again, as we've learned, we know that sacituzumab gives us predominantly neutropenia and diarrhea. When we think about datopotamab, we're thinking more like nausea, stomatitis, and some dry eyes. And then when I think about sac-TMT, I feel like it's kind of in between sacituzumab and datopotamab. We do see some diarrhea; we do see some stomatitis; we do see some dry eyes.

And so I think it's an interesting way of appreciating that while we have three TROP2 ADCs with similar-ish payloads of some kind, we still have slight differences and distinct toxicity profiles. There are differences in their DAR, the drug-to-antibody ratio, there are differences in the potencies, perhaps, and there are differences in the stability of the linker. So it's not as simple as we think when we think about an ADC.

So, Dr. Kalinsky, maybe I'll take a moment here now that we were talking about this. We recently heard new, exciting data for sacituzumab at the ASCO annual meeting not too long ago. Maybe you can walk us through the first-line data that we've now seen in triple-negative breast cancer with sacituzumab.

Dr. Kalinsky:
For those of you who are just tuning in, you're listening to CME on ReachMD. I'm Dr. Kevin Kalinsky, and here with me today is Dr. Komal Jhaveri. We're discussing TROP2-targeted ADCs in the treatment of metastatic breast cancer.

Yeah, at ASCO, we saw data from ASCENT-04, and these were highly anticipated data. And I would say, of the data that we saw at ASCO, these really were, I think, practice-changing data. And we'd already seen by press release that there was an improvement in progression-free survival in this particular trial.

So to give some context, ASCENT-04 was looking at patients who were PD-L1 positive and appropriate to receive checkpoint inhibition. So it's frontline. It was essentially giving sacituzumab govitecan plus pembro versus physician-choice chemotherapy plus pembro. And ultimately saw that there was a several-month improvement in progression-free survival. Tolerability was as anticipated. We also saw that because there was crossover that was allowed in this particular study, that we are starting to see a trend toward improvement in overall survival and recognizing that the overall survival data are immature. But I do think that these were really practice-informing data, demonstrating the utility of moving up TROP2 ADC into the frontline space.

We are awaiting data from ASCENT-03. ASCENT-03 is a similarly designed study, except for it's for patients who are not appropriate to receive a checkpoint inhibitor, ie, those patients who are PD-L1 negative. There are like 60% of patients or so who are PD-L1 negative in that frontline space. And we've also seen by press release that there's an improvement in progression-free survival. We haven't seen those exact data quite yet, but we saw that sacituzumab govitecan compared to physician-choice chemotherapy was better for that particular endpoint.

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And then, just to also kind of round out this discussion, there's also TROPION-Breast02, which is a frontline study which really mirrors ASCENT-03, right? So there are those patients who have PD-L1 negative tumors. We've already talked about Dato-DXd and its once-every-3-week dosing. In this particular study, it's comparing that agent to physician-choice chemotherapy. Also just highlighting that the chemotherapy options are a little bit different. It's a nuance that we'll see as those data report out, because in that study, eribulin was allowed as one of the physician-choice options.

So those are the data that we have seen. And I anticipate in the upcoming year that we will see some additional data in this space, particularly ASCENT-03, given that we have a press release, and I suspect it will be reported at a symposium soon.

So, Dr. Jhaveri, I was hoping maybe you could just put all of this into context for us and just thinking through how to think about these agents and patients who have metastatic TNBC.

Dr. Jhaveri:
Yeah, no, I think this is such an important dataset that you walked us through because this is where the field is evolving, right? We started with HER2-positive breast cancer and ADCs. We had T-DM1 that got replaced with trastuzumab deruxtecan, showing superiority. And we also heard data for, now, trastuzumab deruxtecan as well in the first-line metastatic setting.

So certainly, the trend that we keep applying in our ways of questioning or understanding how can we make patient outcomes better, is trying to utilize effective therapy early on. And so that's what these strategies are looking at. Right? We saw that there was benefit with ADCs, including OS benefit in the second- and third-line or beyond setting. Can we then use them in the first-line setting and further improve outcomes for our patients? Which is what at least the ASCENT-04 showed, that compared to chemotherapy and pembro, we did show benefit with sacituzumab and pembro.

I think what is very interesting now that we will have to focus on: What do we do after patients' tumors progress with these effective therapies upfront? What will they receive at the back end, and how will that continue to impact their patient outcomes?

And the other thing that I feel like, which is going to be very, very important with all these exciting new data that I'm very excited about, honestly, for triple-negative breast cancer, is also that in early-stage setting, we are utilizing the KEYNOTE-522 regimen, which is comprising of pembrolizumab. So checkpoint inhibitor is being utilized there.

So if these tumors really act like bad actors, and we do see recurrence despite those really important therapies, the KEYNOTE-522 really is like you have everything—you've given the patient everything that you could up front, right? And if despite that there is recurrence, how do we put these data in the first-line metastatic setting into context? Would we expect a similar benefit in that setting? Would we offer the same therapies?

How do you think about that, Dr. Kalinsky? Maybe if you could share what thought process you have there.

Dr. Kalinsky:
Yeah. I mean, I think – well, one, I will say you're really highlighting an important point of just the field is moving forward so fast. And I was just thinking about this is where we are with CDK4/6 inhibitors in ER+ disease as we move them into the frontline and then in the early-stage setting. So then what do we do if a patient has a tumor that then recurred despite receiving adjuvant therapy? And so that is a world that I suspect we will get to relatively soon. And I would say this is the standard of care for right now.

And I do think that this question about sequencing the currently available ADC after ADC—and we'll talk about some of these data—but there's limited activity, and I think it really speaks to the need that we need new ADCs with new targets and new payloads to continue to move the field forward.

So, Dr. Jhaveri, if you were to take away one key point from what we've talked about, I'd like to hear what you think.

Dr. Jhaveri:
Yeah, no, I think ADCs have been very exciting. We are now bringing them up front in the metastatic and also in early-stage settings to further impact outcomes for our patients. I think we will need to focus on research efforts to look at what do we do once patients' tumors progress on these effective therapies? What do we give them at the back end? And as you pointed out, we really have been blessed

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with topoisomerase I payloads, which have been game changers across all subtypes. But we do need novel payloads, bispecific antibodies, novel combinations to continue to further improve outcomes for patients and utilize that in the treatment paradigms.

Dr. Kalinsky:
Yeah, completely agree. Thank you. In Chapter 3, we'll be discussing the comparison and positioning of TROP2 ADCs in metastatic breast cancer. Stay tuned.

Chapter 3

Dr. Kalinsky:
Welcome back. In the second chapter, we discussed treatment options using TROP2-targeted ADCs for metastatic triple-negative breast cancer. In Chapter 3, we are looking at the comparison and positioning of TROP2-targeted ADCs in metastatic disease. All right, let's get started.

Dr. Jhaveri, can you take us through a molecular structure comparison of the TROP2 ADCs and then their clinical implications, please?

Dr. Jhaveri:
Absolutely. So while we have three TROP2 ADCs, there are slight differences in their molecular structures. Let's start with sacituzumab govitecan, where the linker is a hydrolyzable linker, and certainly, obviously, the antibody is again the TROP2, and the payload is an SN-38 payload with a drug-to-antibody ratio of nearly 8:1. So for every antibody, 8 molecules of chemotherapy are delivered, or payload are delivered.

When it comes to datopotamab deruxtecan, this is a deruxtecan payload with a tetrapeptide-based cleavable linker with a drug-to-antibody ratio of 4:1.

And last but not least, we have sacituzumab tirumotecan, and here it's a belotecan-derived topoisomerase I inhibitor payload, again, a humanized TROP2 IgG1 antibody, and a pyrimidine-thiol linker with a drug-to-antibody ratio of nearly 7.4:1.

So they all are TROP2 ADCs, they all have the 3 components, but with some slight distinct differences for all of them and differences in the linkers and the drug-to-antibody ratios, which is why we also see some slight differences in their toxicity profiles and their administration schedules.

Sacituzumab is delivered Day 1/Day 8 of a 21-day cycle. Datopotamab is every 3 weeks 6 mg/kg, and sacituzumab govitecan was 10 mg/kg, and sac-TMT is 5 mg/kg every 2 weeks. So certainly, various options, various administration schedules.

With respect to toxicities again, as we said, sacituzumab govitecan, 2 most common toxicities, neutropenia and diarrhea. With datopotamab, I think more about nausea, stomatitis really being the most important side effect, and some dry eyes. And then with sac-TMT, I kind of think it's somewhere in between the 2, where I think about some neutropenia, some stomatitis, some dry eyes as well, some diarrhea, a little bit, but not too much.

And so there are differences across these 3 antibody-drug conjugates and differences in those administration schedules, which will play in mind when we think about how do we offer these therapies when all 3 are available to our patients across all subtypes? And how do we think about what might work? I think it's going to be a decision that we'll make with our patients based on their preferences as well for the administration schedule and the side effect profile as well, unless there are distinct differences in efficacy, which so far, we've not been able to see—at least HR+ breast cancer.

Dr. Kalinsky, maybe how would you position these TROP2 ADCs in metastatic breast cancer? Maybe you can kick us off for the positioning in HR+ versus HR- disease. How do you think about utilizing them and using them?

Dr. Kalinsky:
Yeah, Dr. Jhaveri, I agree with the sentiment that you had shared when we were talking a bit about this just in Chapter 1, about the idea that if you have a patient who is no longer responding to endocrine-based treatment for hormone receptor-positive disease, if a patient has HER2-negative, 0, or null, in that sort of circumstance, I would think about trying to utilize a TROP2 ADC as soon as I can, meaning after endocrine-based therapy. If you have a patient who has HER2-ultralow or -low disease, in that sort of circumstance, I would think

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about utilizing T-DXd based upon the data that we've seen from the DESTINY studies.

Then the question becomes, if a patient has received T-DXd, what is the role for a TROP2 ADC? And some of the information that we've been seeing has really been from real-world data. And we're seeing that there does seem to be, in the context of what we're seeing in the real world, that the response of one ADC after the next ADC, that that second ADC ,for the most part, doesn't respond as well as the first ADC. Not always, but most of the time.

And then the question comes up, well, what about sequencing a TROP2 after another TROP2? And so we do have data from a very small number of patients from the phase 1 study, where there were few patients who received Dato-DXd after SG, and there were some patients that benefited, others not. But I would tell you now that we're utilizing T-DXd for the majority of these patients, I am not sequencing a TROP2 after another TROP2. I just think that there are limited efficacy with what we have right now.

And you had already highlighted this, and I completely agree with you: The conversation about which TROP2 ADC to use is a conversation with the patient. It's talking about the administration; it's talking about the side effect profile, what we know and what we don't know, and then making an informed decision.

And then, if we're looking in the triple-negative setting, if patients have PD-L1-positive tumors, I think it's clear that we would be using sacituzumab govitecan plus pembro in those patients. And we don't have Dato-DXd approved quite yet for metastatic TNBC, but we'll see how data reports out, including from TROPION-Breast02.

I also just want to make the comment that there are some ongoing studies, including in TBCRC. Reshma Mahtani has a study that's looking at sequencing T-DXd followed by SG in patients with hormone receptor-positive disease. So we will get some of these data reported out, but most of what we have now are just real-world studies.

Now, I'm curious about your interpretation of these data and if you agree or disagree.

Dr. Jhaveri:
Yeah, no, I think it's such an important question about how do we best utilize all of these drugs if we have them available. How do we best sequence them? Should we sequence them, and how do we sequence them?

I think, as you said, we have these real-world datasets, which have been intriguing. What we've learned, at least so far, is that regardless of which particular ADC you use first, it seems like the benefit you derive on the first one is definitely better and longer than the benefit you derive with the second one. And it doesn't matter whether you use T-DXd first or sacituzumab first; it just feels like the first one does better.

In fact, the most recent real-world dataset that got presented as well at the ESMO Breast meeting was looking at patients who got T-DXd first and then received treatments at the back end. And in the hormone receptor-positive cohort, which was nearly about 270 patients, patients who had already had T-DXd, chemotherapy did better than sacituzumab at the back end. We didn't have the reverse. We didn't have saci first and T-DXd after. But this was, again, a real-world dataset. That's what we learned there. From other real-world datasets, we've learned the first one does better than the other.

In fact, this new one that I was referring to, even if you use this—what I call as a sandwich chemotherapy between two ADCs, it didn't matter. I think chemotherapy in general did better than sacituzumab after T-DXd.

So I think there's a lot more work needs to be done. We need to understand a little bit better, is there a problem with the target? Is there a problem with the payload, which has developed a mutation or resistance mechanism? Can we use them? And as you pointed out, we have the TRADE DXd study to look at T-DXd and Dato in both sequences—so this is both deruxtecan payloads—which one to give first and later, and will that matter? We have the TBCRC registry study, and the Reshma Mahtani study looking at T-DXd and sacituzumab. Or as we both have highlighted, nothing of that is going to be as great as maybe having completely different payloads, completely different targets, completely different combinations and bispecific molecules. And I think that's where the field is probably going to be.

I mean, we do that in the metastatic setting with chemotherapy drugs, I think, right? We really strive hard to figure out mechanisms of resistance to chemo, but we've never been able to show that successfully necessarily, and we just change the chemotherapy drug

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because we think they're distinct mechanisms, and we use them in the metastatic setting. And I think that's what's going to happen with ADCs; we might not necessarily uncover all the resistance mechanisms, but we might just need to use different kinds to continue to derive benefit with a treatment.

So I think a lot needs to be done more. We are happy with what we have, but we need a lot more to do.

Dr. Kalinsky:
Yeah, I completely agree. And I like the fact we can think of different agents that are being evaluated in these settings, like B7-H4 antibody-drug conjugates. Those seem to have some interesting efficacy, including after a TROP2 ADC. We saw some of those data at ESMO Breast. And then the bispecifics, I think there's a lot of interest with these drugs. And then those are moving forward in phase 3 trials, so we'll see how that pans out.

So, Dr. Jhaveri, 1 key takeaway that our audience should focus on from this chapter.

Dr. Jhaveri:
I would say that while ADCs have been so efficacious and so important and we absolutely want to offer that to our patients, we cannot lose sight. They are not perfect. They still have some toxicities. We need to be very careful about what toxicities to expect, educate our patients, act on them promptly, and management is truly critical so that we can continue to keep them on these drugs and so that they can have best, optimal outcomes. So I think management of toxicities is key.

Dr. Kalinsky:
I agree. And that's a great way to wrap this up. That's all the time that we have for today. I want to thank the audience for listening, and thank you, Dr. Komal Jhaveri, for joining me and for sharing all of your valuable insights. It was great speaking with you today, as usual.

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