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<https://reachmd.com/programs/cme/osteoporosis-pivotal-role-orthopedic-surgeons-can-play-optimizing-bone-health-their-postmenopausal-post-fracture-patients/12949/>

Released: 12/01/2022

Valid until: 12/01/2023

Time needed to complete: 15 minutes

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Osteoporosis: The Pivotal Role That Orthopedic Surgeons Can Play in Optimizing the Bone Health of Their Postmenopausal Post-Fracture Patients

### Announcer:

Welcome to CME on ReachMD. This activity, entitled "Osteoporosis: The Pivotal Role That Orthopedic Surgeons Can Play in Optimizing the Bone Health of Their Postmenopausal Post-fracture Patients" is provided by Omnia Education.

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### Dr. Anderson:

A fracture in anyone over the age of 50 should be considered suspicious for an underlying osteoporosis and be properly evaluated as such. A wrist fracture or foot and ankle fracture are just harbingers of more major fractures, like a hip or pelvis fracture later on in life, in maybe 5 or 10 years. So orthopedic surgeons in particular need to pick up on the fact that these more minor fractures are just really harbingers of bad things to come because that gives us a great opportunity to intervene.

### Dr. Bukata:

It's also really important for people not to be afraid to start the treatments right away. There has been some concern in the orthopedic community that osteoporosis medications will interfere with healing, and we definitely have data that they do not interfere with healing.

### Dr. Singer:

Studies have shown that it is only a small minority of patients who have had an osteoporosis-related fracture that are actually evaluated, diagnosed with osteoporosis, or treated for this underlying condition. While there's growing awareness among orthopedic surgeons that osteoporosis is the underlying cause of fractures, there is still often a disconnect between recognition and the need to take action to prevent subsequent fractures. In this program, we will identify strategies to address postfracture identification and evaluation and the need for osteoporosis treatment, including pharmacotherapy. Ultimately, we hope that a fracture will be recognized as a sentinel event and a call to action to address osteoporosis and reduce the risk for future fracture.

This is CME on ReachMD, and I'm Dr. Andrea Singer.

### Dr. Bukata:

I'm Dr. Susan Bukata.

### Dr. Anderson:

I'm Dr. Paul Anderson.

### Dr. Singer:

Welcome, Susan and Paul. So let's dive right in. Susan, why do you think there is a disconnect between fracture and osteoporosis as the underlying cause and follow-through beyond attending to the acute fracture? What are we missing?

### Dr. Bukata:

Well, some of it is actually knowledge gap, both on the part of the patients and the physicians, and some of it is actually fear, I think, on the part of the orthopedic services, of not being able to feel like they can comfortably take care of it all by themselves. People don't recognize a fall from standing height or less as being a fragility fracture. My patients will often say to me, "But you don't know how hard I fell." And there's a big difference between slipping on a sidewalk, falling and breaking something, and falling out of a tree or getting hit by a car. And even surgeons and other orthopedic providers don't always recognize that difference. In addition, some orthopedic surgeons do take care of this, and some don't. And we need to let people understand that it's just about the identification and making sure that you have a smooth handoff to someone who is comfortable handling it. If you want to handle it within your practice – within orthopedics – you can, but you don't necessarily have to do it all. You just have to identify it, make the referral, and make the appropriate handoff. So it's really about translating the information to a group that can take care of this.

**Dr. Singer:**

Paul, any thoughts?

**Dr. Anderson:**

Yeah. I find, for orthopedic surgeons, and other practitioners, that there's a lot of confusion regarding recommendations for who should be treated medically, when diagnostic testing should be performed, what are the indications for the various medications – and this leads to a lot of inertia, where the patient is left not being treated, and there's also a tremendous burden on whoever is going to prescribe medicine, on the preauthorization process, and this has really blocked the care for patients after fragility-type fractures.

**Dr. Singer:**

I think those are all great points, and I just wanted to add one thing, because there was a recent paper that was published that actually looked at women in the women's health initiative who had fractured. And it didn't really matter whether it was what we would typically call a low-trauma or fragility fracture or whether someone had a more traumatic fracture. All of those women were at increased risk for future fracture down the line, which is ultimately what we're trying to prevent, so that arbitrary distinction we make may or may not be so important. We really need to recognize fractures, in general.

So with that sort of said, Susan, let's take a look at some key data. What can you tell us about the likelihood of subsequent fractures in postmenopausal women who have suffered an initial or prevalent fracture?

**Dr. Bukata:**

Well, this is the biggest issue as to why we have to act now, right at the time of the fracture. We can't push this decision down the road. It's not only that patients who have a fragility fracture are at an 86% higher risk of a second fracture, it's that 10%-15% of them will have that next fracture in the next 1 year, and up to 20% of them in 2 years. It's a 1 in 5 chance that at 2 years they're going to have another fracture. Can you imagine if you had a 1 in 5 chance that you were going to be hit by a car? You would change your behavior. You would cross the street in a different way. And yet, we aren't getting people to change their behavior in response to recognition of these fractures. It's an urgency. It's not something we can wait 10 or 15 years, because 20% of the patients, we need to act immediately. And in fact, right now we can't pick out who that 20% is, so we really need to act for everyone.

**Dr. Singer:**

I want to build on something you said, because I often talk about that urgency or emergency, but there aren't very many osteoporosis emergencies. People don't become osteoporotic overnight, but if ever there is an emergency or that urgency, as you mentioned, it's in that patient who has had the first fracture, because they are most likely to fracture again in a very short time frame and are – what we often hear people talk about is imminent risk for additional fracture. It's also interesting to think about the greater the number of fractures, the greater the risk. And from the GLOW data, the Global Longitudinal Osteoporosis in Women study, fracture at essentially any site – 9 out of the 10 sites that they looked at – increased the risk for fracture at multiple other sites. So a fracture is a sentinel event, and we need to recognize that.

**Dr. Bukata:**

And that's been a big challenge for a very long time, making that connection and getting things lined up so we can make sure we complete the transaction for the patient. Identify the disease, fix their fracture, get them connected with osteoporosis care, make sure they get diagnosed and that they get some sort of treatment given to them. And unfortunately, we fall apart along the way. By doing this, we are keeping people out of nursing homes. A second fracture is devastating to the quality of life for a patient and really is the key for independence for patients. We don't always think about it in that way, but by missing these important windows to act quickly, we often change the destiny of someone's function in their life.

**Dr. Singer:**

We know from multiple surveys and one that the National Osteoporosis Foundation did a couple of years ago, that if you ask patients what they care most about and what they worry most about, it's independence and mobility. So I agree with you.

Paul, in your seeing patients with fractures, we focus a lot on the hip fracture, and that's some of the data that I've mentioned, but thoughts in terms of how we should be approaching patients and other fractures?

**Dr. Anderson:**

I think a fracture in anyone over the age of 50 should be considered suspicious for an underlying osteoporosis and be properly evaluated as such. Like a wrist fracture or foot and ankle fracture, are just harbingers of more major fractures, like a hip or pelvis fracture later on in life, in maybe 5 or 10 years. So orthopedic surgeons in particular need to pick up on the fact that these more minor fractures are just really harbingers of bad things to come because that gives us a great opportunity to intervene and prevent that fracture later on in life that's going to require surgery and hospitalization and end up with a lot of disability.

**Dr. Singer:**

Susan, can you give us a brief overview of the pathophysiology of osteoporosis?

**Dr. Bukata:**

Well, really, I will tell patients and I explain to providers, you develop the disease of osteoporosis because you're a bad bone builder, a big bone loser, or a combination of the two. And there are many things that can contribute to that – age, medications, menopause. But really, it's a balance between 3 cells. You have your bone-building cell – the osteoblast; your bone-cutting cell – the osteoclast; and your bone-sitting cell – the osteocyte. And it's the osteocyte that manages the mechanical forces that come across the bone, and of course the blasts and the clasts are very important for the building and the constant remodeling of bone that we have. It's when there's an imbalance between the formation and the resorption – the activity of the osteoblast and the osteoclast – that we begin to lose bone. And not only do we lose bone, we begin to get selective holes and weak spots in the bone, and that makes the bone more prone to fracture. So it's not just about bone quantity; it's bone quality and it's bone architecture that all come together to give the disease of osteoporosis.

**Dr. Anderson:**

One analogy we use here in Wisconsin, which patients really understand, about the different types of problems is about potholes. Everybody knows potholes are due to resorption of asphalt and concrete, and then ultimately you could try to patch them, or you can call in the crew to bring in more asphalt and more concrete, and really do the bone-building. And that's what we're really trying to do with these medications is repair that skeleton; that's going to make it less fragile.

**Dr. Singer:**

For those just tuning in, you're listening to CME on ReachMD. I'm Dr. Andrea Singer, and here with me today are two of my colleagues and experts in the field of osteoporosis and orthopedics, Dr. Susan Bukata and Dr. Paul Anderson.

We're just about to delve deeper into the AOA's Own The Bone – Quality Improvement Post-Fracture Prevention Initiative, and osteoporosis guidelines and treatment, as well.

Paul, could you tell us a little more about the American Orthopaedic Association's Own the Bone program?

**Dr. Anderson:**

Yeah, this program started because of the lack of follow-up care after fragility fractures. At the time, in 2005, orthopedic surgeons would refer patients back to primary care, but nothing really got done in terms of preventing it. So this initiative tried to educate orthopedic surgeons to become involved in osteoporosis care, and it really turned into a major quality improvement program. The model is really based on something called fracture liaison service, or secondary fracture prevention, which is a multidisciplinary, coordinated care approach to a patient after a fragility fracture, with the goal to try to improve the bone health and overall health of the patient so that they're less likely to fall again and less likely to fracture. And this has been ongoing since 2009, and we have over 150 sites around the country and a database that includes over about 70,000 patients enrolled into the program.

**Dr. Bukata:**

So Own The Bone has been a wonderful program within orthopedics because it gives a structure that helps you to bring like-minded providers and physicians together who are interested in helping to care for osteoporosis in our patients. And that's what's great about it is there are many different physicians who can provide this type of care, and each community, each practice, each hospital – even each group of partnerships and referring physicians – can come together with that common mission, using that model of the fracture liaison service to set up a process so that patients can get taken care of and we're not dropping so many patients. I'm sometimes doing 20 hip fractures in a week, and when we see patients in follow-up, I realize there are some of them who have fallen through the cracks – patients where we missed giving them a prescription in the hospital on discharge or we missed getting them to the bone density scan. Involvement of these fracture liaison services is fantastic because it's a secondary check mechanism, and it doesn't put the responsibility fully on the surgeon. It builds a team around the provider to help get patients proper osteoporosis care.

**Dr. Singer:**

I think a multidisciplinary team approach is key to really being successful in terms of identifying patients, making sure they get appropriately evaluated, and treated as needed.

And all of the newer osteoporosis guidelines – and there are a number of them from different societies and organizations – talk about this structured approach to secondary fracture prevention. In addition, the core message of the guidelines is consistent: after a fracture, patients need to get treated. Now that may be a little bit of an oversimplification; we obviously individualize evaluation and treatment, but in general, patients need treatment. We need to recognize that the recency of a fracture, as well as other risk factors that we've talked about – including older age, high-risk medications, underlying conditions – make someone high risk. And the AACE guidelines – American Association of Clinical Endocrinologists – the Endocrine Society guidelines, North American Menopause Society, which just released new guidelines, the National Osteoporosis Foundation Clinician's Guide – new version is about to come out. All of these guidelines talk about assessing baseline risk to help make decisions about treatment, which brings us to talk a little bit about the pharmacologic agents that we have available and how we approach treatment.

Two major umbrellas, in terms of pharmacologic agents are anti-remodeling or anti-resorptive agents, which work primarily to inhibit bone turnover, and the classic medications that fall under that category are the bisphosphonates, as well as a RANK ligand inhibitor, denosumab. The other umbrella being our osteoanabolic agents, which work primarily to build new bone. Two categories there are remodeling stimulators, which increase formation and resorption – those are the parathyroid hormone receptor activators, teriparatide and abaloparatide, and a modeling stimulator, which increases bone formation and, to a lesser degree, decreases resorption, and that's the sclerostin inhibitor, romosozumab. Many different things to consider when we think about these different medications. Just a couple of brief words, because I think it's beyond the scope to go into them in depth, but all of the guidelines talk about risk-stratifying patients for those patients that are at the highest level of risk, and a recent fracture is one of the things that elevates that level of risk. Our goal is to reduce future fracture risk rapidly, to build new bone, and to do this quickly. And that's really where our osteoanabolic agents come in, and indeed, many of the recommendations are to think about starting with an anabolic agent in the highest-risk group of patients and then following that with an antiresorptive agent, as compared to the other way around, because we know that the sequence in which we use medications matters. And people have a more robust response, in general, to an anabolic agent if it's used first, as opposed to as follow-on therapy.

So our two parathyroid hormone analogs work quickly. With teriparatide, there has been demonstration in head-to-head fracture trials that it is superior to risedronate. There is cost attached to it; it's a daily injection. And with both of the parathyroid hormone analogs, there is a boxed warning regarding rat osteosarcoma, which is probably not so much of an issue in humans. With the sclerostin inhibitor romosozumab, again, unique mechanism of action – dual mechanism of action – increasing bone formation and, to a lesser degree, decreasing bone resorption. Been shown in head-to-head clinical trials to be superior to alendronate, and also showed hip fracture reduction compared to alendronate. Cost may still be somewhat of an issue. It's a monthly injection, given for a total of 12 doses, and one needs to consider what's in the label, which is a possible increased cardiovascular risk or increased risk of major adverse cardiovascular events, and certainly balance risks and benefits with both categories of medicines.

Our antiresorptive agents, not to make them second tier or second rate. Again, there are some patients in whom we might start with these medications. There are also excellent follow-on therapies. Bisphosphonates, both oral and intravenous, have been the mainstay of treatment for many years and still clearly play a role. They're well tolerated in many patients, available in weekly, monthly, or with zoledronate, the IV formulation, yearly or less frequent dosing. Can be, certainly, some side effects that are associated, but again, in the right setting, benefits generally outweigh risks.

And then, our RANK ligand inhibitor, denosumab – every-6-month dosing, so there's a long dosing interval. Studied out to 10 years, so long-term safety and tolerability data, with progressive bone mineral density increases throughout that 10-year duration. Rapid reversal of effect after stopping, which is what we see with most medications, except for the bisphosphonates. So I think the most important thing is that we have a large armamentarium with lots of choices. The most important thing is that we individualize therapy and find something that is right for everyone, so that these high-risk patients can be treated.

**Dr. Bukata:**

So it's also really important for people not to be afraid to start the treatments right away. There has been some concern in the orthopedic community that osteoporosis medications will interfere with healing, and we definitely have data that they do not interfere with healing. The pattern of healing is different when you age, when you have lower estrogen, when you have normal estrogen, when you have changes in testosterone, when you're on a variety of medications, including the osteoporosis medications. But the time to healing and the point that the bone heals does not change. So we do not have to wait for a period of time for healing to occur before we start patients on osteoporosis medications. And that has been a big issue, because the longer we are away from the time of the event – away from the time of the fracture, the easier it is for us to let the patient fall through the cracks and not complete the whole process of diagnosis of

osteoporosis, discussion of treatments, and choosing of a treatment plan. There are challenges that come, also, for us with hardware in the bone. In these weaker bones, in osteoporotic bone, the bone is often not as strong as the hardware, and we all know in orthopedics, it's a race between the healing of the bone and the hardware. In younger patients, it's the hardware that breaks. In older patients, because the bone is weaker, the hardware actually pulls out of the bone. So we need to think about osteoporosis early on because we want to optimize our results from these big surgeries that we put patients through. We don't want them failing. We don't want the patient falling 6 months later and breaking next to our plates, so we really do need to balance risk and outcomes and think of this as an urgency that we want to address right away in order to get the optimal outcome for our patients, not only from the fracture healing, but also in terms of their function.

**Dr. Singer:**

Unfortunately, that's all the time we have today. I want to thank our audience for listening, and I really want to thank both of you, Dr. Susan Bukata and Dr. Paul Anderson, for joining me and for sharing all of your valuable insights and expertise. It was great speaking with you both today.

**Dr. Bukata:**

Thank you, Dr. Singer.

**Dr. Anderson:**

Thank you, I really enjoyed the conversation.

**Announcer:**

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