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Going Beyond Today's Assessments to Confirm COPD, Are Biomarkers Key?

Announcer:
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Dr. Bhatt:
This is CME on ReachMD, and I'm Dr. Surya Bhatt. Here with me today is Dr. Meilan Han. So let's get right into today's topic. Dr. Han, will you explain the role of biomarkers in COPD, what does the research currently available tell us?

Dr. Han:
This is a great question. So I think we've got a mix of things that are being used right now in clinical practice, as well as other biomarkers that we're sort of transitioning into use for COPD as we recognize the importance of type 2 inflammation as an important phenotype of disease. So probably the most well-used biomarker right now is eosinophils. But other things that have also been looked at include things like IgE as well as FeNO, or the fractional exhaled nitric oxide. I think there's pros and cons, and different ways in which each of these are being used.

But certainly, I think eosinophil count is probably the most widely available. It's available in the electronic medical record for many, many patients, even without the physician necessarily having to order it; at some point, often someone else has ordered it. And we know that eosinophil counts themselves are associated with higher rates of exacerbations, but also, importantly, associated with response to inhaled steroids. So particularly, patients who have an eosinophil count of 100 or more, I think are potentially a candidate for inhaled steroids. The data suggests that we see reduction in exacerbations in this particular subgroup population. And this is something that GOLD recommends that we look at.

There's also emerging data, though, on other biomarkers. This includes IgE, another marker of type 2 inflammation. And there is a relationship between IgE levels and exacerbations, as well as all-cause mortality in COPD.

Now, I also mentioned FeNO, or F-e-N-O; this is not something that is quite as readily available in, I would say, community practices. Most academic centers have them. And traditionally, it's been a marker of inflammation for asthma. But as we begin to understand the importance of type 2 inflammation in COPD, I think we're going to start seeing more use of FeNO in COPD. But there are some caveats. For one, there are lower levels in active smokers, so that's something that needs to be taken into account when interpreting the data. We also see lower levels associated with lower levels of FEV1 percent predicted. But it does appear to correlate with blood eosinophils, inhaled corticosteroid response, as well as predicting more frequent exacerbations. There's also some initial evidence for predicted value for response to inhaled corticosteroids.

Dr. Bhatt:
Thank you, Dr. Han. I think it sounds like these biomarkers may have utility in clinical practice. How do you apply this in your practice?

Dr. Han:

So that's a good question. Right now, I am definitely using eosinophils. And that's really outlined in the GOLD strategy. So for initial treatment, for patients who are on no therapy, they recommend considering ICS if the patient has an eosinophil count of 300 or more. For patients who are already on treatment but still exacerbating, if they are not on an inhaled steroid yet, consideration should be made if the EOs are 100 or more. That would be a good place to add ICS on top of dual bronchodilator therapy to really ramp those patients up to triple therapy.

Dr. Bhatt:
Thank you, Dr. Han. I think that was a great overview, and I agree with you that there is a lot of value for checking these biomarkers, especially blood eosinophils are easy to measure. They're important for identifying type 2 inflammation in patients with COPD, and they can help guide our treatment selection. Other biomarkers such as FeNO are perhaps a little more tricky, given the impact of active cigarette smoking on their levels, but you can certainly use them if they are elevated, irrespective of smoking. So that's perhaps one way you can use elevated FeNO levels. And then there is IgE, which is often neglected in COPD, but I think there is increasing evidence that you can use that for predicting exacerbation risk.

So this has been a great micro-discussion. Unfortunately, our time is up. Thank you for listening.

Announcer:
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