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RAS Across Tumors: Who to Test When

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Dr. O'Reilly:
This is CE on ReachMD, and my name is Dr. Eileen O'Reilly, and I'm delighted to be joined today with Dr. Kathryn Arbour.

So we're going to get started here and talk about RAS. RAS is a key oncogene that's the cause of much badness in cancer, right? Mutated about 20%, 25% of cancers, sort of integral to signaling, to growth, to metastasis, to unfavorable biology.

So, Dr. Arbour, what molecular techniques are used to detect KRAS point mutations to help inform treatment selection?

Dr. Arbour:
So I treat non-small cell lung cancer primarily, and as a thoracic oncologist, we see that approximately 30% of patients diagnosed with lung cancer will have a KRAS mutation identified in the tumor. And typically, we identify this as a part of broad-based next-generation sequencing testing that tests for numerous oncogenic drivers. And for lung cancer, it is critical that this molecular testing is performed typically at the time of diagnosis because first-line treatment decisions are based on driver oncogene status.

Initial therapy, for example, is fundamentally different for those patients with other driver oncogenes like EGFR and ALK, than those patients with KRAS mutations. We typically do this testing on tissue-based testing. That's still our gold standard. But increasingly, we're using liquid biopsies, especially when tissue is insufficient or time is of the essence. And either role to identify a KRAS mutation or other types of driver oncogenes are crucial in this aspect.

So I'm wondering in terms of pancreas cancer, the roles of molecular testing in that setting, Dr. O'Reilly.

Dr. O'Reilly:
Thank you so much. I think you summarized where the field is in non-small cell lung cancer. And I have to say we're a little bit behind in pancreas cancer, but we're rapidly catching up here. And testing is evolving rapidly. Traditionally, we do next-generation sequencing in advanced disease where there's treatment implications, but now trying to move this to every patient who's diagnosed with this disease. And yes, largely tissue-based for somatic alterations, but just a reminder that we check, and this is a difference with non-small cell lung cancer, we check for germline alterations routinely.

And as you mentioned, we're also integrating circulating DNA. When tumor burden is high, the yield is high, it's a struggle in earlier-stage disease and in localized disease sometimes to detect RAS, and RAS being the critical oncogene that we're looking for for implications for treatment, now as investigational therapy, but hopefully soon as standard of care options.

Dr. Arbour, do you want to just outline an algorithm? When you meet a person in the clinic, what do you do?

Dr. Arbour:

Absolutely. So when we're meeting a non-small cell lung cancer patient who's diagnosed with metastatic disease at the time of diagnosis, we perform molecular testing to understand what their driver oncogene is. And then we also look at what their PD-L1 expression is because we think immunotherapy is foundational in lung cancer as well as chemotherapy and other targeted therapies.

So if a patient has a driver oncogene in lung cancers that we commonly associate it with never-smokers or rare-smokers, such as EGFR, ALK, and there are a number of others, including RET, BRAF V600E, met exon 14 alterations, ROS1 mutations—the list is growing longer and longer by the day. Those patients typically receive targeted therapy as part of initial treatment.

For our patients with KRAS mutations, this is much more an evolving space. There are no first-line targeted therapies, but it certainly does help us identify patients who would be appropriate for clinical trials. And increasingly, that's a part of what we think about in the first-line setting and can help us identify that.

For patients with KRAS-mutant lung cancer currently, we think about either immunotherapy by itself in certain patient populations, but for the vast majority of patients, chemotherapy and immunotherapy. So we use this broad array of molecular testing to identify which treatment option is going to be the best for patients in that setting and which is going to enrich in responses.

Do you wait for these molecular testing results to make your treatment decisions, Dr. O'Reilly, or are they kind of results-in-hand for subsequent decisions?

Dr. O'Reilly:
Yeah, thank you so much. You summarized it so nicely for non-small cell lung cancer. For pancreas cancer, we would love to have this information in real time. Mostly now, it's for trials, with the exception of HRD signals in the setting of BRCA1, BRCA2, or PALB2. We can sometimes get those results pretty quickly. In pancreas cancer, we're also looking whether RAS mutated or RAS wild-type. And in the wild-type setting, there are other actionable drivers, some of which have disease-specific, some disease-agnostic indications.

And then testing there can be very important, looking for those rare fusions with RNA testing as well as IHC as complementary tools to tissue and liquid biopsy where we know there are limitations there.

Well, I think we've had a great discussion on how we test and how the field is changing. Thank you all so much for listening.

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