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Clinical Conundrums in ARIA: Differential Diagnoses and Potential Pitfalls in ARIA Evaluation

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Dr. Bateman:
Welcome to Clinical Conundrums: Navigating Case Scenarios in Your Own Practice Setting, where we will cover quick and challenging cases related to amyloid-related imaging abnormalities, or ARIA, management. I'm Dr. Trey Bateman, and here with me today is Dr. Jerry Barakos. Let's dive into our case.

So our case is James, a 74-year-old patient on lecanemab who presents with an acute onset of a headache. His MRI shows there is a new flare sulcal hyperintensity along the right frontal convexity with susceptibility artifact along the right frontal cortex. Findings are consistent with either recent subarachnoid hemorrhage, or a combination of ARIA-E and ARIA-H with sulcal exudate and superficial siderosis. Given that the differential includes recent subarachnoid hemorrhage, this patient went to an outside emergency department and got a non-contrast head, CT, and serial follow-up CT imaging was recommended to document stability.

Jerry, can you walk us through some key considerations when making a differential for a patient on anti-amyloid therapies, and some potential pitfalls to keep in mind?

Dr. Barakos:
Very nice. And this is a very important case. It highlights how imaging findings, by definition, are non-specific. So if there's a pathologic process in the brain, whether it's ischemic, traumatic, infectious, neoplastic, etc., will tend to have an edematous response. And that's the strength of MRI, is to find that edematous response. But it doesn't tell us necessarily what it is, and that is where clinical history combined with the morphologic features of the imaging findings provide insight.

Now, in other words, we have a patient who presents, as you've outlined, with the focus of increased signal in the right frontal lobe. This could be a focal cerebritis. This could be a subarachnoid hemorrhage. This could be an infectious This could be many things. But what is it? Well, as radiologists, we're physicians, you have to know why you're doing what you're doing. You have to know why the patient's being imaged, what the clinical history is, what you're looking for. By definition, if you don't know what you're doing, you're lost. So if we understand that this is a patient who's undergoing anti-amyloid agent, they have no significant symptomatology, and they come in for routine serial monitoring and we find this abnormality, is this likely to be a subarachnoid hemorrhage? Well, obviously not. If this patient doesn't have nuchal rigidity, worst headache of her life, and this is also a very unusual location for a subarachnoid hemorrhage. So we have to use the clinical information available to us, and knowing that this is routine surveillance imaging, this is classic for incidental ARIA-E findings.

Now, what is the role of, let's say, computed tomography in this setting? Well, it really doesn't play any role. We have the diagnosis here. Now, if this patient had other symptomatology, yes, you would have to consider other findings in the differential diagnosis. But this

finding here, given the clinical information available to us, is very straightforward.

Now, let's talk about potential ARIA-E interpretation pitfalls, these ARIA-E changes can be very subtle. And when they develop and present on a follow-up scan, they're most easily identified if we have a good quality baseline study to compare. Now, in the first case, we have a patient who actually does have a left occipital ARIA-E, and there was a small little infarct as well. Well, it turns out that ARIA-E does not show restricted diffusion, and of course, an infarct will show restricted diffusion, so that's one means by which we can differentiate a stroke from ARIA-E.

Now, differences between scanners, if the patient is scanned at one facility as compared to their baseline scan, you can get subtle changes in the water suppression, especially around the parietal/occipital lobes that can give you some subtle artifactual changes that can mimic ARIA-E. Again, that's tricky, and it takes an understanding of what the ARIA-E typically looks like, because that is the most common location for ARIA-E in the first place.

But long story short, it makes the point when you see these changes on, let's say, a T2 flare sequence, not all changes you see are ARIA and you do need a differential, and that has to be kind of spoken to from the perspective of knowing your clinical history and being aware of potential artifacts that may serve as mimics.

Dr. Bateman:
Those are great points. And ultimately, this case highlights one of the challenges that we face in clinical practice. This is someone who had symptoms that had onset far away from the academic medical center that they were receiving their anti-amyloid therapy at. And so there was no baseline to compare against, and there was no immediate communication between the prescriber and the emergency department. This patient ultimately was put back in contact with the prescriber. It was felt that this most likely represented ARIA-H and not a traumatic or atraumatic subarachnoid hemorrhage, and the patient ultimately did go back on therapy and continued to receive anti-amyloid therapy successfully.

Thank you for your insight with this conversation, Jerry. It was a great conversation and great case. To our viewers, be sure to explore our other episodes for more in-depth insights into the nuances of ARIA management. Thank you for joining us.

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