ROUNDTABLE DISCUSSION

Exploring the Limits of Acute Ischemic Stroke Intervention Indications

Moderator James Milburn, MD, FACR, asks panelists Donald Frei, MD; Maxim Mokin, MD, PhD; and Stacey Quintero Wolfe, MD, FAANS, to describe their management approaches to five challenging clinical scenarios in stroke care.

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This article assembles an expert panel of neurointerventionalists to discuss controversial issues in stroke care. There are numerous clinical scenarios in which endovascular treatment of arterial occlusions is outside of the accepted American Heart Association/American Stroke Association guidelines.[1] Despite the lack of strong trial evidence in these controversial areas, many believe the benefit of treatment outweighs the risk in some patients. For example, many neurointerventionalists were treating emergent large vessel occlusion (ELVO) patients using mechanical thrombectomy years before trial evidence was finally available in 2015. In this article, Dr. Milburn has proposed several clinical scenarios, and the panelists discuss their approach to management. Any of these clinical situations may be encountered in a busy neurointerventional stroke practice.

CASE 1: ELVO AND NIHSS OF ZERO

A patient in his mid-50s presented to the emergency department (ED) with transient left arm and leg weakness 2 hours prior that resolved on arrival. In the ED, his National Institutes of Health Stroke Scale (NIHSS) score was 0, blood pressure was 180/100 mm Hg, and CTA showed a short-segment right M1 occlusion with good leptomeningeal collaterals.

• How would you manage this patient?

Dr. Mokin: I find this type of ELVO case to be one of the most challenging to manage, and I may choose a conservative approach depending on the following factors. First, I examine the noninvasive study very carefully, because sometimes a very severe stenosis (flow-limiting stenosis > 95%-99%) could be mistaken for a complete occlusion. I also look for signs of intracranial atherosclerosis elsewhere, which may help me identify the underlying pathophysiology of the lesion in question. Finally, I find it helpful to gently "challenge" the patient—if changing from lying flat to sitting or standing up triggers recurrent symptoms or systemic pressors are needed to keep the brain adequately perfused, it tells me that a more aggressive approach with endovascular revascularization is warranted. Perfusion imaging demonstrating a clear asymmetry between the affected territory versus normal brain tissue may also indicate the need for revascularization.

Dr. Frei: In all scenarios where patients fall outside the inclusion and exclusion criteria for published randomized clinical trials showing the efficacy of thrombectomy, frank and open communication with the patient's family is very important. This patient had symptoms before arrival and has an LVO. He is hypertensive. I think it is likely that his collaterals eventually will fail, and he will become symptomatic again. As Murphy's law is always in effect, neurologic decline will probably happen in the middle of the night and won't be noticed right away, leading to a delay in therapy and potentially a poor outcome. In our experience, before the positive thrombectomy trials and improvements in thrombectomy device safety, our stroke team had clinical equipoise in this patient group, and half of these patients were not offered thrombectomy acutely. More than 50% of the patients not treated with thrombectomy in the acute setting ended up with a completed stroke. We must remember that mechanical thrombectomy is one of the most effective treatments in medicine. There must be a very good reason to deny a patient such effective treatment. I would treat this patient immediately.

Dr. Wolfe: There are several variables I would consider. In situations in which the patient has a good clinical examination, I have to always remember the Hippocratic Oath, "First, do no harm." There is always a chance of distal embolization with thrombectomy, even with balloon guide catheters. However, there is a not infrequent occurrence of delayed stroke in situations where there is an ELVO and we are depending on collaterals. In this case, his blood pressure is elevated. Was it lower when he was still symptomatic? Is he maintaining this himself or requiring augmentation? If we have to work to augment him or he becomes at all symptomatic with a lower blood pressure, we'd pursue thrombectomy. Also, what does the perfusion imaging show? If there is a significant flow delay, that is a good indication that his collaterals may not be adequate, and we would consider performing thrombectomy using a balloon guide catheter. If he remains neurologically intact without requiring blood pressure augmentation, we would likely load with dual antiplatelet therapy (unless he received tissue plasminogen activator [tPA] while he was symptomatic) and watch closely in the intensive care unit. If there is any deterioration, we would perform thrombectomy. We have often seen M1 ELVO open up with dual antiplatelet therapy after 3 to 4 weeks.

CASE 2: DISTAL OCCLUSION WITH APHASIA

A woman in her mid-60s presented at an outside hospital with acute-onset right arm weakness and aphasia. NIHSS was 18. She received intravenous (IV) tPA at the outside facility, and her arm weakness resolved during transfer to our hospital. Noncontrast CT showed no infarct, but CTA revealed a superior division left M3 branch occlusion. The patient is 5 hours from symptom onset, NIHSS is 6 with continued aphasia, and blood pressure is 160/96 mm Hg.

• How might you proceed in this scenario?

Dr. Wolfe: Given a significant functional deficit of aphasia in a younger patient without serious comorbidities, I would absolutely perform digital subtraction angiography and likely thrombectomy. In the M3 territory, I use a distal access catheter into the M2 and a 3MAX (Penumbra, Inc.) for ADAPT (a direct aspiration first pass technique). The risk of branch avulsion is higher in the distal vasculature with tortuosity, so I aim to create as straight a segment as possible if a stentriever is needed. However, if this was someone with a premorbid modified Rankin scale > 2, I would be less likely to attempt thrombectomy.

Dr. Frei: The question to ask yourself is: Would the presenting symptoms be disabling? My answer here is yes. There are endovascular tools specifically developed to treat distal occlusions. The published thrombectomy trials only evaluated internal carotid artery and M1 occlusions, but it is a logical extension to assume benefit in treating posterior circulation LVO (basilar artery occlusion) and M2 middle cerebral artery (MCA) occlusion. There are also no published randomized clinical trials showing efficacy of thrombectomy for distal occlusions. Many neurointerventionalists would also intervene in this scenario, because they believe they can safely recanalize the occlusion. I am one of these physicians.

Dr. Mokin: I consider NIHSS of 6 with aphasia a disabling deficit. With additional time, IV tPA may or may not lyse the more distal clot. For such distal occlusions, I favor gentle aspiration thrombectomy with a 0.035-inch aspiration catheter or sometimes a 0.025- to 0.027-inch microcatheter with low threshold to abandon the procedure if the intervention becomes too technically challenging. I also find the use of general anesthesia helpful for these cases.

CASE 3: BASILAR OCCLUSION WITH SIGNIFICANT BRAINSTEM STROKE

A man in his early 50s presented with nausea, vomiting, and slurred speech and worsened clinically in the ED, requiring intubation. He is 8 hours from symptom onset and had a poor physical examination, only withdrawing from pain on the left side. CT showed several moderate-size infarcts in the pons, right midbrain, and right cerebellum. CTA showed occlusion of the basilar artery, which begins just beyond the anterior inferior cerebellar artery. He has small posterior communicating arteries, which supply the posterior cerebral arteries (PCAs).

• What is your approach to this patient?

Dr. Mokin: The information provided suggests that there is low likelihood that this patient will regain independence with revascularization. One important caveat is that noncontrast CT could be subject to error for the posterior fossa structures such as the brainstem. Careful history review sometimes shows that the first symptoms occurred even earlier than originally thought. On the other hand, the natural history of basilar artery occlusion is very grim if untreated. I would offer endovascular treatment in this case, but I would make sure to explain to the family that the likelihood of the procedure helping is low.

Dr. Wolfe: With the young patient age and relatively short time from symptom onset, I would err on the side of taking the patient for thrombectomy with ADAPT, usually with a transradial approach for a basilar ELVO. Use of a stentriever in this territory has a higher risk of snowplowing clot into the perforators, as we've seen in the STAR data. CT is a relatively nonspecific way to look at the brainstem, so I would question the veracity of hypodensities. If there is no improvement, it is an easy answer to withdraw care. If there is a delayed time to arrival with a similar situation and a poor examination (> 12 hours is our usual cutoff), I would take the time to get an MRI to assess the burden of infarct. However, at our institution, even an emergent MRI will add at least 1 to 2 hours, and at an 8-hour presentation, I would rather give the patient every chance, especially at age 50.

Dr. Frei: If not recanalized, basilar artery occlusion is fatal in most patients. When evaluating a patient for thrombectomy, one should always consider whether the treatment will be futile. If the patient's stroke is too extensive at presentation, a good recovery is unlikely, even if there is rapid, complete recanalization. Moderate to large core infarct at presentation is currently being evaluated in clinical trials, but I think the patient population with basilar artery occlusion is too small to be studied in a randomized study. I have been personally surprised more than once in patients with moderate to large core infarcts at presentation who have recovered well after thrombectomy. In our experience, with careful blood pressure control, symptomatic hemorrhage rates are low in this patient population. In patients with basilar artery occlusion, where the natural history is so poor and in a young patient like this one, thrombectomy is their only chance to recover. Communication with the family should be clear. Successful recanalization is very likely, but the patient may not improve; however, if the artery is not recanalized, the patient won't recover. Most families would want you to give their loved one the best chance for recovery. I would offer this patient treatment.

CASE 4: LATE-PRESENTING ELVO WITH SIGNIFICANT PENUMBRA

A patient in their early 70s with a proximal left M1 occlusion with early infarct in the caudate, putamen, internal capsule, and insula presented with an NIHSS of 20 at 25 hours from symptom onset. CT perfusion showed mismatch volume of 60 mL in the left MCA territory.

• What is your management approach for this patient?

Dr. Frei: In my opinion, time from last known normal is not important. If a patient has a normal head CT, a severe stroke syndrome, and an M1 occlusion, I will treat that patient at 24, 48, 72, or infinite hours from last known well. This patient has an ASPECTS of 6 with a large mismatch. Cortical areas show no infarct. The most eloquent territory is the internal capsule. If the M1 is not recanalized, there is a lot more to lose. Clinical trials are currently evaluating patient populations with larger core infarcts at presentation. Published trials tell us that ELVO patients rarely do well if not recanalized. I would offer this patient thrombectomy.

Dr. Mokin: I would feel comfortable offering endovascular therapy in this case. The evidence to support revascularization beyond 24 hours is not the strongest, but several retrospective publications have shown that thrombectomy could be performed safely and effectively.[2,3] Especially in a scenario where the time of onset is unclear and instead the time of last known well is used, the stroke could have happened within the 24-hour window.

Dr. Wolfe: If the volume of core infarct is < 70 mL, I would offer thrombectomy. I have performed thrombectomy in carefully selected patients with late-presenting ELVO with good success up to 34 hours. If there is a large core, the risk of reperfusion hemorrhage is likely too high, but if not and there are reasonable collaterals on CTA, it is worth giving them the chance. It is likely that we will see data from pending trials that corroborate this approach.

CASE 5: P2 OCCLUSIONS RESULTING IN DEBILITATING VISUAL DISTURBANCES

• What is your approach to a 60-year-old airline pilot with a PCA occlusion at the P2 segment with homonymous hemianopsia and no infarct on CT? What about an 80-year-old with the same P2 occlusion who reads voraciously and completes *The New York Times* crossword puzzle every day?

Dr. Wolfe: I would perform thrombectomy for the 60-year-old airline pilot via a transradial approach to give them every chance of normal recovery. I would likely use the same approach for the 80-year-old, as a hemianopsia can be quite disabling and lead to falls, but I would assess the tortuosity first. With a transradial approach, I have had good success getting a distal access catheter into the distal basilar artery followed by a stentriever, but sometimes tortuosity precludes vertebral access (even left vertebral artery from the femoral approach), and you spend hours trying to get into the vertebral artery with little to show for it except a dissection!

Dr. Mokin: I approach isolated P2 occlusions with great respect. Similar to more distal anterior circulation occlusions, I would likely use general anesthesia and aspiration in these cases. The 60-year-old pilot with CT showing no infarct signs likely warrants consideration for thrombectomy. On the other hand, performing a P2 thrombectomy in an 80-year-old patient could be considered "too risky," especially if vertebral artery access is challenging or there is major intracranial atherosclerosis.

Dr. Frei: Both patients present with clinical deficits that would be disabling. The PCA diameter can be similar to the M1 or certainly the M2 MCA segment. I have treated many patients with this occlusion with the largest-diameter aspiration catheter because the catheter size matched the diameter of the occluded artery. The artery is occluded, and the deficit would be disabling. We have the tools to open the artery safely, and it should be treated with thrombectomy. ■

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