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Acute Ischemic Stroke Due to Large Vessel Occlusion

Introduction

As a result of improved recognition and available treatments, death from stroke fell from the fourth to the fifth leading cause of death in the United States, behind diseases of the heart, cancer, chronic lower respiratory diseases, and unintentional injury. Despite decades of advances in prevention and treatment, and a decade of decreasing stroke mortality and morbidity in the United States, ischemic stroke remains a leading cause of death and disability worldwide. These trends have begun to reverse in the past several years. Advances in stroke systems of care and stroke therapies have increased the number of stroke patients who receive reperfusion therapies, such as intravenous (IV) alteplase and endovascular therapies, leading to improved outcomes for patients fortunate enough to receive these interventions. In nearly half of all acute ischemic stroke patients, a large artery occlusion or stenosis is identified. Large artery occlusions are caused by in situ thrombosis, artery-to-artery embolism, cardioembolic sources, or cryptogenic causes. Acute stroke reperfusion therapies have led to significant reduction in the morbidity and mortality associated with ischemic strokes due to large vessel occlusion (LVO). This article will discuss the prevalence, mechanism, diagnosis, and treatment options of acute ischemic stroke due to LVO.

Epidemiology

According to the World Health Organization, 15 million people worldwide suffer a stroke each year. Of these, 5 million die while 5 million are left permanently disabled.[1] In the United States, stroke is the fifth leading cause of death and is a leading cause of disability.[2] Each year, approximately 795,000 people in the United States suffer new or recurrent strokes.[2] Approximately 610,000 are first events and 185,000 are recurrent stroke events.[2] It is estimated that every 40 seconds someone in the United States has a stroke and every four minutes someone will die from one. The total direct and indirect cost of stroke and cerebrovascular disease in the United States for 2009 was estimated to be $312 billion, leading to a significant economic impact.[3] So, despite decades of advancement, stroke will remain a major public health issue for the foreseeable future.

Ischemic strokes account for 80% of all strokes and occur as a result of stenosis or occlusion of a small vessel or a large vessel, leading to a reduced or complete lack of blood flow to a specific vascular territory of the brain. Strokes caused by LVO have a significantly higher morbidity and mortality than strokes caused by small vessel occlusions.[4] Furthermore, standard

EXECUTIVE SUMMARY

- Large vessel strokes are caused by intracerebral atherosclerosis, artery-to-artery embolism, intracardiac embolism most often from atrial fibrillation, or cryptogenic causes. Risk factors are similar to those for other strokes, and include age, hypertension, hyperlipemia, diabetes, and smoking.
- The single most important fact in the history of a patient with a stroke is the time the symptoms started or time when the patient was last known normal.
- The National Institutes of Health Stroke Scale is widely used to assess stroke severity. However, it has some shortcomings. It overestimates stroke in the dominant hemisphere and is poor at assessing posterior circulation strokes.
- Once a patient presents within the 4.5 hour time frame, a CT is done to rule out hemorrhage. Hypoglycemia is treated if present. Alteplase is started when indicated. If the patient has signs of a large vessel stroke, the patient should have vascular imaging and then mechanical thrombectomy if indicated. Patients who present after 4.5 hours with signs of large vessel occlusion also should be evaluated for endovascular therapy potential.

of care IV alteplase is less effective with LVO because of a much larger clot burden, thus endovascular therapies for direct clot removal have been developed.

Mechanism and Risk Factors of Large Vessel Occlusion

There are four main mechanisms of acute LVO: 1) in situ occlusion due to atheromatous plaque rupture of an intracranial artery; 2) artery-to-artery embolism in which embolic fragments arise from extracranial arteries affected by stenosis, ulceration with plague rupture, or dissection; 3) embolism originating from the heart, most commonly due to atrial fibrillation; and 4) cryptogenic or unknown causes, commonly believed to be occult paroxysmal atrial fibrillation.

The incidence of LVO due to the above-mentioned mechanisms varies, with cardioembolic sources being the most common in North America, and intracranial atherosclerosis being the most common in Asia.[3,5]

Patients with LVO with underlying intracranial atherosclerotic stenosis typically have gradually progressive stenosis of one of the major cerebral arteries due to formation of atheromatous plaque.[6,7] Risk factors for intracranial atherosclerosis include advanced age, African American and Asian race, male sex, hypertension, diabetes mellitus, metabolic syndrome, and hyperlipidemia.[5] Occasionally, a plaque rupture or intraplaque hemorrhage occurs, causing a superimposed thrombosis and acute occlusion of the vessel.[8,9]

The second common mechanism of LVO is artery-to-artery embolism when an embolus originates from a proximal extracranial artery and travels distally, occluding an intracranial artery. Emboli commonly arise from the internal carotid artery (ICA), but can arise from the common carotid artery (CCA) or the aortic arch. A large vessel occlusion due to an embolus originating from the ICA accounts for 17% of all ischemic strokes.[10] Risk factors for internal carotid disease are similar to those for intracranial atherosclerosis and include advanced age, uncontrolled hypertension, hyperlipidemia, and smoking. When the embolus originates from a severely stenosed or occluded ICA, producing two areas of flow obstruction, this mechanism is labeled as a tandem arterial pathology in the Stroke Data Bank. Internal carotid artery dissection is the source of the embolus in up to 20% of strokes in young adults.[11]

Another common source of embolism causing LVO is the heart. Thrombus originating in or passing paradoxically through the left side of the heart with subsequent occlusion of an intracranial artery accounts for 20-25% of all ischemic strokes.[12] The underlying disease process associated with cardiogenic stroke is grouped according to the underlying cardiac abnormality: disease of the left atrium, the atrial septum, the left-sided heart valves (mitral or aortic), or the left ventricle. The most common source of cardioembolic stroke is atrial fibrillation. Advanced age, uncontrolled cardiovascular risk factors, and the presence of a structural heart disease are the most common risk factors for atrial fibrillation.

Clinical Presentation and Early Assessment

Although reperfusion therapies for acute ischemic stroke treatment are very effective, the degree of effectiveness is exquisitely time dependent.[13] Successful acute stroke treatment requires early recognition, rapid transportation to the nearest most appropriate stroke center, and early activation of the stroke team.

Emergency medical services (EMS) provide the first medical contact for most stroke patients, and therefore are in a unique position to reduce delays in stroke identification, treatment, and transportation. Several new prehospital stroke scales have been developed to aid EMS with prehospital evaluation and triage decisions for a suspected LVO. Table 1 lists prehospital stroke clinical tools for EMS providers. These scores include assessment for specific cortical signs, such as aphasia, neglect, and gaze preference, that usually indicate the presence of an LVO.

To improve acute stroke care outcomes, the American Heart Association (AHA)/American Stroke Association (ASA) developed Mission: Lifeline Stroke. This program was designed to transform acute stroke care by focusing efforts on connecting all the components of acute stroke care into an integrated system that reinforces the use of evidence-based guidelines, measures performance, and identifies gaps. These efforts resulted in the creation of the Severity-based Stroke Triage Algorithm for EMS.[14] The algorithm seeks to

Table 1. Prehospital Stroke Clinical Tools for EMS

Stroke Screens	Stroke Scores
• Cincinnati Prehospital Stroke Scale (CPSS) • Los Angeles Prehospital Stroke Screen (LAPSS) • Melbourne Ambulance Stroke Screen (MASS) • Miami Emergency Neurologic Deficit (MEND) • Recognition of Stroke in the Emergency Room (ROSIER) Score	• 3-Item Stroke Scale (3ISS) • Cincinnati Stroke Triage Assessment Tool (C-STAT) • Field Assessment Stroke Triage for Emergency Destination (FAST-ED) • Los Angeles Motor Scale (LAMS) • National Institutes of Health Stroke Scale (NIHSS) and shortened NIHSS (sNIHSS –5/8) • Rapid Arterial Occlusion Evaluation (RACE) Scale

balance the benefit of rapid, early access to endovascular thrombectomy for patients with suspected LVO with the potential harm of delayed initiation of IV alteplase.

Although both Primary Stroke Centers (PSC) and Comprehensive Stroke Centers (CSC) provide acute stroke care, CSCs are better equipped to provide a higher level of care, including endovascular therapies, for patients with severe stroke. These capabilities have been shown to be cost-effective and associated with better functional outcome at CSCs compared to PSCs.[15-19]

In light of these findings, focus recently has shifted to finding more sensitive screening tools to help EMS identify patients with potential LVO and transfer them to the nearest CSC. In patients with a high likelihood of having an LVO, there is growing evidence to suggest that it might be reasonable to bypass a nearby PSC and transfer those patients directly to a CSC, particularly if such diversion would not add more than 15 to 30 minutes.[20,21] However, it is very important to recognize that some PSCs, but not all, are capable of performing thrombectomy. Therefore, emergency physicians should be aware of which hospitals have this capability in order to make the appropriate decision about where to transfer a patient with a suspected LVO.

Additionally, The Joint Commission currently is collaborating with the AHA/ASA to offer certification for Thrombectomy-Capable Stroke Centers (TSC). These hospitals are not CSCs, but are required to meet rigorous standards for performing endovascular thrombectomy.

Stroke symptoms due to LVO share many of the same symptoms of acute ischemic stroke due to small vessel disease, including hemiparesis/hemiplegia, hemisensory disturbance, facial droop, and dysarthria. However, compared to patients with small vessel strokes, patients with acute ischemic stroke due to LVO typically present with additional cortical symptoms, including gaze deviations, visual field defects, visual and sensory extinction, aphasia, and agnosia. These symptoms indicate hypoperfusion to the cerebral cortex. LVO causes deprivation of blood supply to a larger area of the brain, impairing cortical function. The clinical presentation varies depending the location of the occlusion, the amount of tissue affected, the vascular territory involved, and the robustness of collateral perfusion. For emergency medical technicians, emergency physicians, and neurologists, it is critical to recognize the clinical signs and symptoms that indicate LVO.

Depending on the severity of stroke, patients might be able to cross midline, which is called gaze preference, or cannot cross midline, which is termed forced gaze deviation. In the context of a cerebral injury, the term gaze deviation denotes an acute inability to produce gaze contralateral to the side of the lesion. Gaze preference denotes the preference to look to the side of the stroke. It is similar to gaze deviation except that the eyes can cross the midline in patients with gaze preference. Gaze deviations and preferences are observed in patients with injury to the frontal visual eye field, which is responsible for initiating conjugate eye movements to the contralateral side. When injury to the frontal lobe occurs, the frontal visual eye field on that side becomes dysfunctional and the contralateral, unaffected frontal eye field pushes the eyes to the side of the stroke.

Visual field defects occur when there is injury to the optic tract or visual cortex. This can be a homonymous hemianopia or quadrantanopia, depending on the location of the lesion. In general, temporal lobe lesions lead to contralateral superior homonymous upper quadrant defect, whereas parietal lesions cause contralateral inferior homonymous lower quadrant defect. Occipital lobe lesions lead to contralateral homonymous hemianopia.

Extinction to double simultaneous sensory stimulation is a clinical phenomenon in which a patient perceives a unilateral sensory stimulus presented in isolation but fails to perceive the same stimulus when presented simultaneously with a second stimulus. This typically occurs in the setting of ischemia to the right parietal lobe; however, it also can be seen with left parietal lobe injury.

Sensory extinction is tested with the patient's eyes closed and by lightly stroking the dorsum of the hand or cheek on the right side, left side, and then both sides. Patients with sensory extinction consistently will fail to report contralateral touch with bilateral stimulation to light touch.

Similarly, visual extinction is tested by having the patient look at the examiner's face. Then the examiner shows one or two fingers on the right, the left, and both sides. Patients with visual extinction consistently will fail to report the visual stimulus contralateral to the affected hemisphere with bilateral stimulation.

Speech deficits, such as dysarthria and aphasia, are common symptoms observed in acute stroke patients. It is very important for clinicians treating patients with stroke to be able to assess language and know the difference between aphasia and dysarthria.

Figure 1A. Complete Occlusion of the Proximal Segment of the Right Middle Cerebral Artery

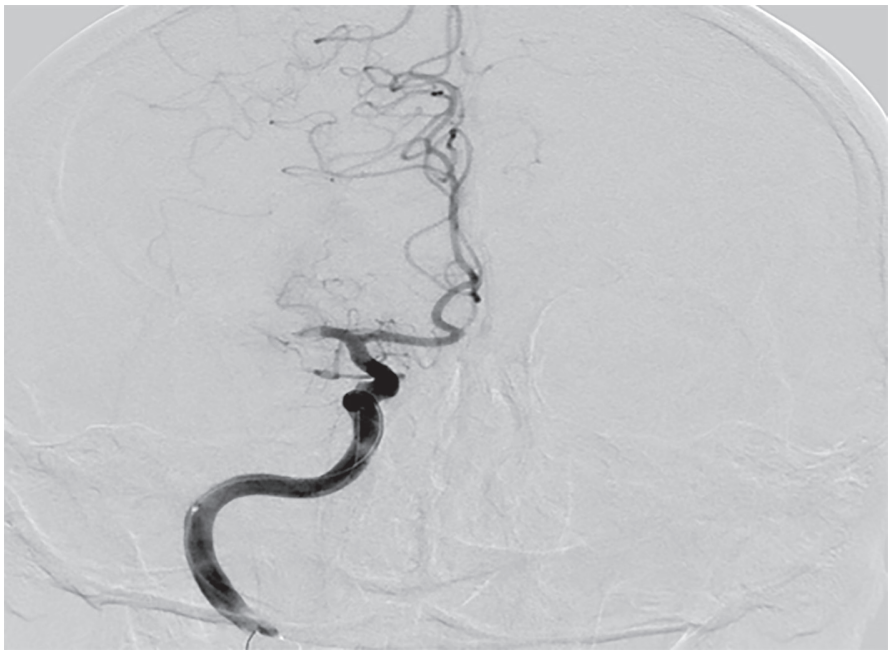
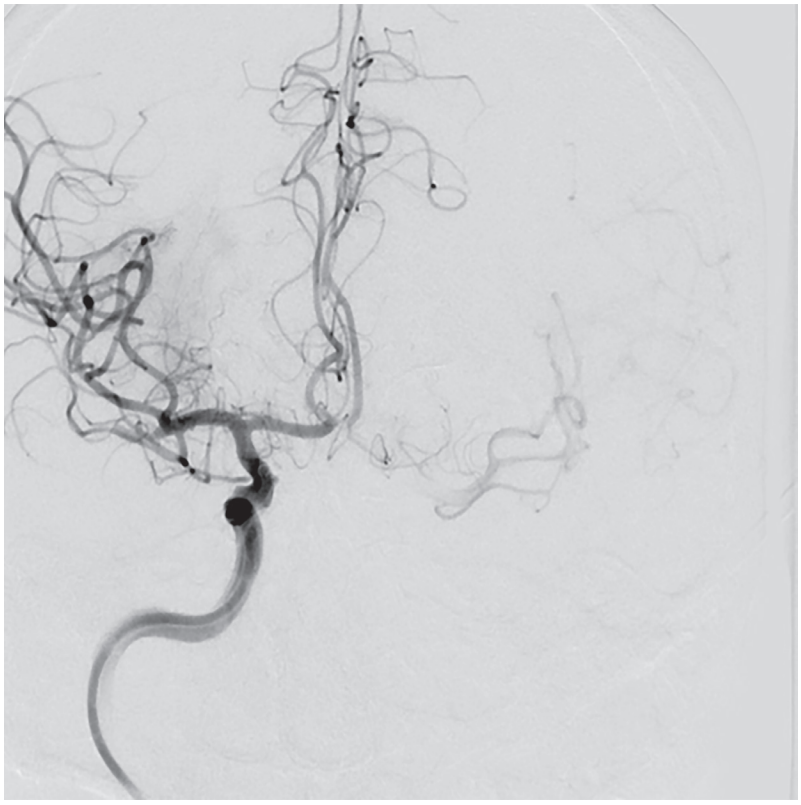


Figure 1B. Revascularization of the Previously Occluded Proximal Segment of the Right Middle Cerebral Artery



Dysarthria is a motor speech disorder. It results from impaired movements of the muscles used for speech production, including the face, lips, vocal cords, and diaphragm. Patients with dysarthria will have slurred or mumbled speech and will be difficult to understand. Commonly, dysarthria is associated with dysphagia due to oral and pharyngeal muscle weakness. Patients with dysarthria should be screened routinely for dysphagia prior to receiving anything by mouth.

Aphasia indicates a loss of the ability to produce or understand language. It happens when there is injury to the language center in the dominant cerebral cortex. Commonly tested language components are comprehension, fluency, repetition, naming, reading, and writing.

Agnosia is another important cortical sign seen in stroke, although it is tested less commonly in the acute setting. Agnosia indicates a loss of the ability to interpret sensations and, therefore, to recognize objects. There are different types of agnosia. One commonly tested in the emergency setting is finger agnosia, in which patients are asked to show the examiner their ring or index finger. Patients with agnosia lose their ability to recognize their index or ring fingers despite intact understanding and vision.

Another form of agnosia is agraphesthesia, which denotes difficulty recognizing a written number or letter traced on the skin. This is tested with the patient's eyes closed and by asking the patient to identify letters or numbers that are being traced onto the patient's palm.

The National Institutes of Health Stroke Scale (NIHSS) is widely used to quantify the degree of neurologic impairment in acute ischemic stroke.[22] It was developed to assess neurologic signs in the distribution of each of the major arteries of the brain and has been used in many clinical trials. It is also a valid tool to predict stroke outcome. The scale is designed to be performed quickly and easily at the bedside, providing a rapid and reliable assessment of the neurological function in patients with acute stroke. The NIHSS is not a neurologic exam but was designed to predict stroke severity. Currently, the NIHSS is used routinely to assess stroke severity in most stroke centers.[23-27]

The NIHSS has some limitations. The scale is more biased to the dominant hemisphere, as seven points are given to measure language function,

and only two points are given for neglect. Additionally, stroke arising in the brainstem and posterior circulation may not be characterized adequately by the NIHSS, as cranial nerves are not assessed fully in the scale. Therefore, possible life-threatening posterior circulation strokes may have low NIHSS scores. It is very important always to be alert to the signs and symptoms of posterior circulation large vessel occlusions, which include gaze palsy, ataxia, dysphasia, dysarthria, crossed neurologic deficits, and decreased level of consciousness.

Emergent Evaluation and Diagnostic Studies

The initial evaluation of a patient with a potential LVO stroke is similar to that of other critically ill patients and includes emergent evaluation of airway stabilization, breathing, and circulation, quickly followed by an assessment of the neurologic function with the NIHSS. In evaluating patients with acute ischemic stroke, the single most important piece of patient history is the time of symptom onset, as defined by the time when the patient was last at his or her previous baseline. This is important because it will define treatment options, such as intravenous thrombolysis and mechanical thrombectomy.

Immediately following the initial assessment, the patient should be taken for brain imaging. Ideally, the initial assessment should be performed on the way to and in the scanner room to avoid treatment delays. The goal of the brain scan is to rule out intracerebral hemorrhage and evaluate the extent of the ischemic stroke. Computed tomography (CT) commonly is used for the initial scan; however, some centers have magnetic resonance imaging (MRI) capability in or near the emergency department. Only non-contrasted imaging studies are required to determine eligibility for IV alteplase in potential patients. Additional brain imaging should not delay initiation of IV alteplase.

Minimal emergent laboratory studies are required before determining eligibility for reperfusion approaches for ischemic stroke. Serum glucose assessment typically is performed in the prehospital setting, and correction of hypoglycemia often leads to rapid symptom resolution. For patients taking anticoagulation medications, INR assessment for patients taking warfarin is indicated. Anticoagulation studies do not accurately reflect the effect of the newer novel anticoagulation agents, so timing of the last dose is critical if alteplase administration is to be considered.

In patients with a suspected LVO, additional vascular imaging of the intracranial and extracranial vessels may be performed to determine the

Figure 2A. Near Complete Occlusion of the Proximal Basilar Artery

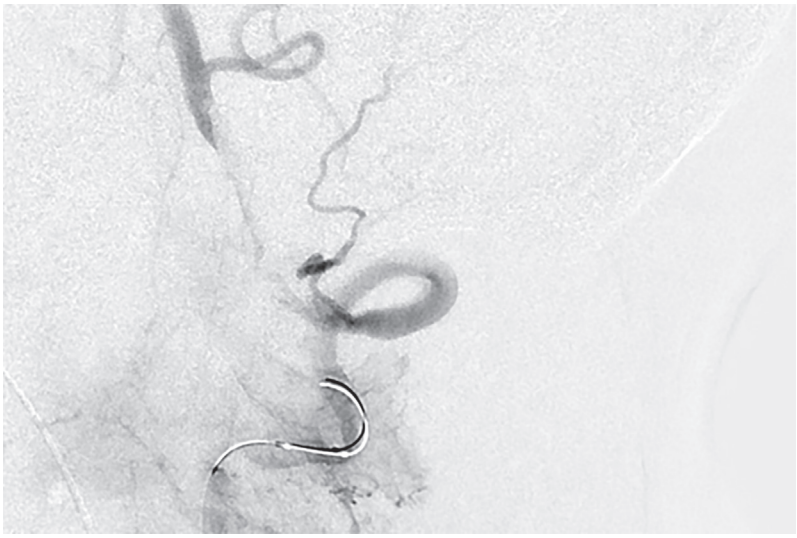


Figure 2B. Complete Revascularization of the Previously Occluded Proximal Basilar Artery

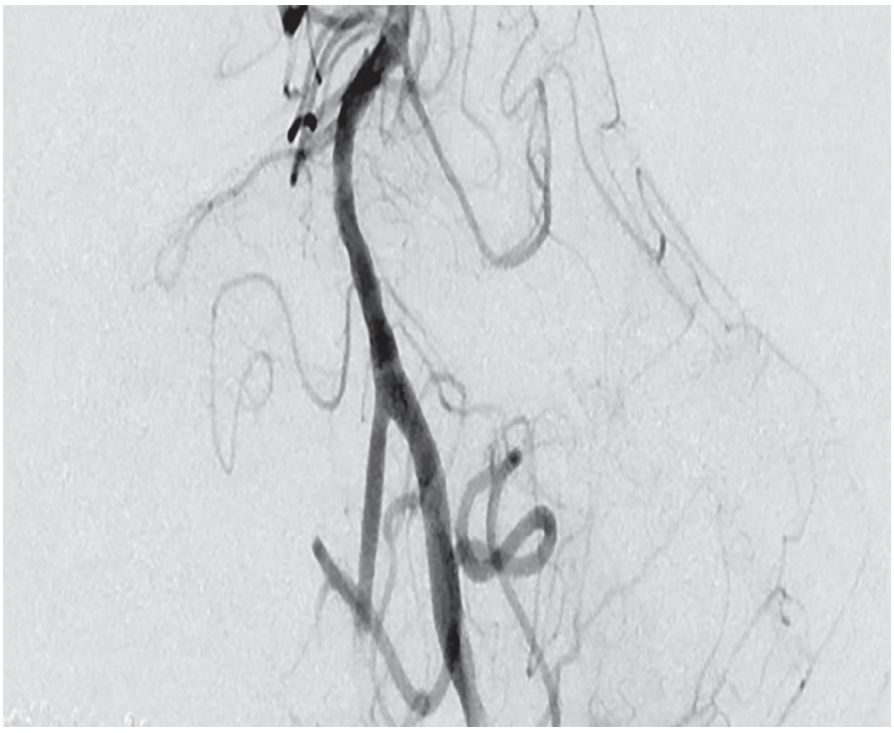


Figure 3A. Complete Occlusion of the Distal M1 Segment of the Left Middle Cerebral Artery

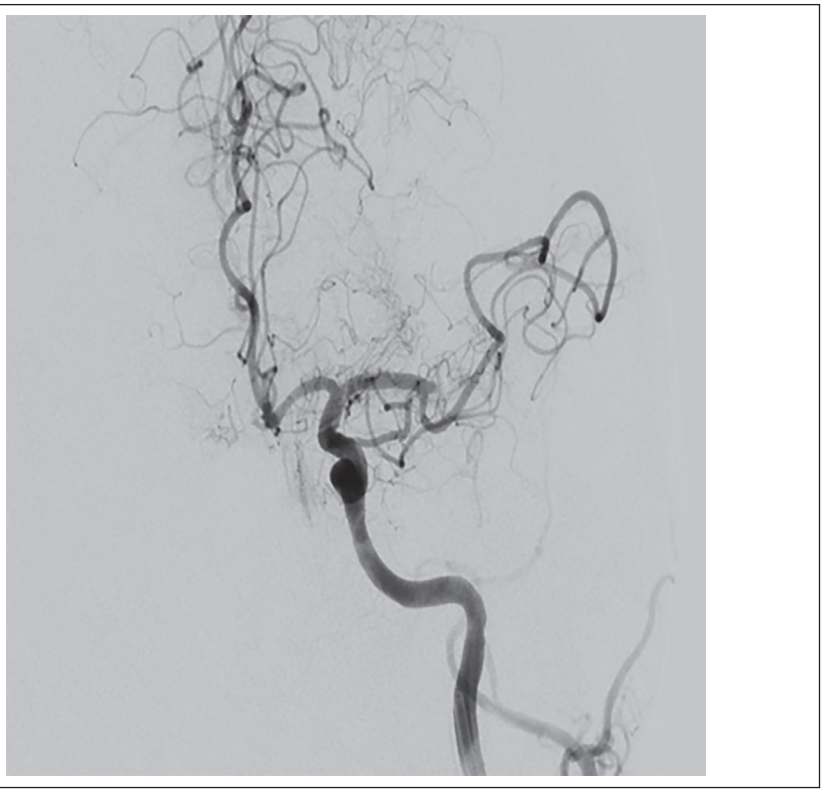
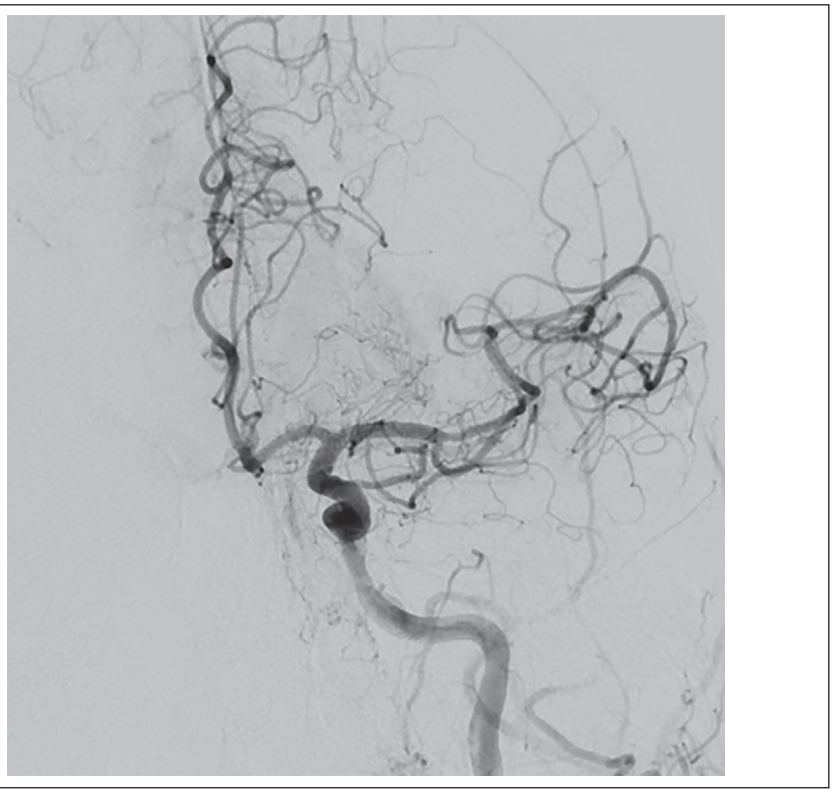


Figure 3B. Near Complete Revascularization of the Previously Occluded Segment of the Left Middle Cerebral Artery



presence and location of an LVO, as well as extracranial vessel findings that may identify tandem lesions or severe stenosis. Vessel imaging allows for the appropriate planning for mechanical thrombectomy. Both CT angiography and MR angiography are appropriate modalities for vessel imaging. For patients beyond current time windows for endovascular therapy, penumbral imaging with either CT perfusion of MRI perfusion may be performed to determine salvageable penumbra.

Management

Patients with acute ischemic stroke should be evaluated for candidacy for reperfusion therapies, including IV alteplase and mechanical thrombectomy. The figures in this article show angiographic evidence of occlusion and revascularization. The results of several prospective, randomized clinical trials over the past two decades confirm the safety and efficacy of alteplase for acute ischemic stroke.[28,29] IV alteplase is recommended for acute ischemic stroke within 4.5 hours of symptom onset.[28-30] A significant limitation to the use of alteplase is the time window during which it can be administered. Although IV alteplase is recommended within 4.5 hours of last known well time, the exclusion criteria are different for 0 to 3 and 3 to 4.5 hours because of different exclusion criteria in the NINDS and ECAS III trials.[31] Soon after the introduction of alteplase for stroke, studies revealed that only a small proportion of patients with acute ischemic stroke received alteplase.[32] This occurred for several reasons, including delay in presentation, delay in diagnosis, and presence of a contraindication.

Importantly, the degree of improvement following alteplase is strongly correlated with the time from last known well or symptom onset to alteplase administration. Previous studies and pooled data from previous trials of alteplase found that the odds of a favorable clinical outcome at three months increased as the onset to treatment time decreased.[31,33,34]

Another limitation of alteplase is its efficacy in LVO. Previous studies have shown that in patients with intracranial ICA or middle cerebral artery occlusion,

IV alteplase results in reperfusion in only 13-50% of patients.[35-37]

Once initial brain imaging is performed, blood glucose is measured, and the patient is deemed to be a candidate for IV thrombolysis, alteplase should be administered without delay. Although the current AHA/ASA guidelines recommend that alteplase be initiated within 60 minutes of the patient's arrival in the emergency department,[30] treatment should be initiated as quickly as possible after arrival. In an effort to reduce door-to-needle (DTN) times nationally, the AHA/ASA developed Target: Stroke Phases I and II. The goal of Target: Stroke Phase I was to treat 50% or more of IV alteplase-eligible patients. To reach this goal, 10 key strategies were recommended to the 1,200 hospitals that enrolled in the program. These time-saving strategies included EMS pre-notification; stroke team-based approach to stroke care with early, single-call stroke team notification; rapid acquisition and interpretation of brain imaging; acute stroke protocol development and implementation; and IV alteplase premixing performance data feedback. (*See Table 2.*)

Target: Stroke Phase II set more aggressive DTN goals, including a primary goal of 60 minutes in 75% or more of IV alteplase-eligible patients and a secondary goal of 45 minutes in 50% or more of IV alteplase-eligible patients. Once alteplase is initiated, noninvasive intracranial vascular imaging should be performed whenever there is a concern for an LVO based on history and exam.

Until recently, alteplase was the only proven treatment widely available for acute ischemic stroke. Intra-arterial alteplase was recommended for a very narrow cohort of patients, but treatment was recommended only within six hours from symptom onset, and this approach suffered similar limitations in reperfusion success as IV alteplase.[30,38] However, in 2014 and 2015, five randomized clinical trials showed safety and efficacy of standard of care medical management, including IV alteplase plus mechanical thrombectomy over medical management alone in patients presenting with proximal anterior circulation occlusions within six hours of symptom onset.[39-43] A subsequent meta-analysis of these trials confirmed that mechanical thrombectomy with the current generation of stent-retriever devices increased the likelihood of complete reperfusion and improved three-month functional independence. The number needed to treat with endovascular thrombectomy to reduce disability by at least one level on the modified Rankin Scale (a measure of functional outcome routinely used in clinical trials) for one patient was 2.6.[44]

Following these pivotal trials, the AHA/ASA issued a Focused Update of the 2013 Acute Ischemic Stroke Guidelines regarding mechanical thrombectomy. The guidelines now recommend mechanical thrombectomy for patients with anterior circulation LVO presenting within six hours who meet other eligibility criteria (Class I; Level of Evidence A).[45] (*See Table 3.*)

Importantly, the focused updated guidelines recommend against waiting after IV alteplase to observe for clinical improvement before pursuing endovascular therapy (Class III; Level of Evidence B-R). This is because of the lower likelihood of LVO strokes to respond to IV alteplase and the higher chances of a better functional outcome with faster endovascular recanalization in these patients.[35,36]

Data from the recently published DAWN (Clinical Mismatch in the Triage of Wake Up and Late Presenting Strokes Undergoing Neurointervention With Trevo) trial provided strong evidence that more patients with acute LVO may benefit from endovascular therapy. While the previously mentioned clinical trials have proven the benefit and efficacy of endovascular therapy within six hours of symptom onset, the DAWN trial showed endovascular therapy significantly improved functional outcome when performed on patients with anterior circulation LVO within 24 hours of symptom onset, with clinical imaging mismatch demonstrating significant salvageable penumbra (patients with severe symptoms but small core infarct on imaging).[46] In DAWN, the rate of functional independence at 90 days was 49% in the thrombectomy group compared with 13% in the best medical management group. Mortality at 90 days was similar in both groups.

Similarly, DEFUSE 3 (Endovascular Therapy Following Imaging Evaluation for Ischemic Stroke 3), a prospective, randomized, Phase III, multicenter, controlled trial evaluated patients with acute ischemic anterior circulation stroke due to LVO treated between six

Table 2. Stroke Best Practices

- Prehospital stroke tools and tool kits
- Team-based approach
- Emergency medical services prehospital notification
- Single-call activation of entire team
- Rapid stroke triage in the emergency department
- Direct EMS transfer to computed tomography
- Rapid CT and interpretation
- Rapid laboratory testing/point of care
- Mix alteplase early
- Rapid alteplase administration
- Prompt feedback to all parties
- Early notification for possible endovascular therapy

Based in part on AHA Target: Stroke strategies (Available at: http://bit.ly/2ArnBzl)

Best Practices for Rapid Endovascular Therapies

- Continue team-based approach
- Prestock and prepare neurointerventional radiology suite for next case
- Utilize prehospital large vessel occlusion scores and triage protocols
- Early notification of NIR team of possible LVO, especially in transfers
- Early notification of interhospital transport personnel
- Prepare CT/MRI prior to arrival if needed
- Prepare need for sedation/anesthesia
- Consider direct transfer to neurointerventional suite upon transfer arrival

Table 3. Summary of the AHA/ASA Recommendations for the Use of Endovascular Therapy

1. Patients being considered or transferred for endovascular treatment should receive thrombolytics if they are candidates.
2. Criteria for endovascular treatment are all of the following:
   a. Prestroke mRS score 0 to 1 *
   b. Receiving thrombolytics within 4.5 hours of onset of acute ischemic stroke
   c. Occlusion of ICA or proximal MCA (M1) causing symptoms
   d. Age greater than or equal to 18 years
   e. National Institutes of Health Stroke Scale score greater than or equal to 6
   f. ASPECTS (Alberta Stroke Program Early CT Score) greater than or equal to 6**
   g. Able to start procedure within 6 hours of the onset of symptoms
3. Better outcome is associated with shorter time between treatment and onset of symptoms; therefore, endovascular treatment with return to TICI (thrombolysis in cerebral infarction) grade 2b/3 should be as rapid as possible and ideally within 6 hours of the start of symptoms.
4. Endovascular treatment (internal carotid artery or proximal middle cerebral artery M1) beyond 6 hours is of uncertain effectiveness.
5. Consider endovascular treatment for patients with anterior circulation occlusion who cannot receive thrombolytics. (Limited evidence)
6. Consider endovascular treatment for patients with stroke due to occlusion of the M2 or M3 branch of the MCA, anterior arteries, vertebral arteries, basilar artery, or posterior arteries. Benefits are uncertain based on limited evidence.
7. Although none of the studies included children, it may be reasonable to consider endovascular treatment in selected pediatric patients.
8. Consider use of endovascular treatment in patients with a prestroke mRS score greater than 1.

*Modified Rankin scale (mRS), where 0 is no symptoms and 1 is mild disability but still able to independently carry out the activities of daily living.
**ASPECTS is calculated from the changes seen on CT, where the score starts at 10 and 1 point is deducted for each region involved. This score generally is calculated by the radiologist.

Adapted from: Powers WJ, Derdeyn CP, Biller J, et al. 2015 American Heart Association/American Stroke Association focused update of the 2013 guidelines for the early management of patients with acute ischemic stroke regarding endovascular treatment: A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2015;46:3020-3035.

and 16 hours of stroke onset with endovascular therapy along with best medical management vs. best medical management alone. The trial was terminated early because of the high likelihood of benefit in the endovascular group.[47] Results from DEFUSE 3 will be presented at the upcoming International Stroke Conference in January 2018. The results of these two trials likely will lead to a revision of current guidelines for endovascular therapies.

Although mechanical thrombectomy has been shown to be effective in patients with LVO up to 24 hours from last-seen-well time, analyses from the DAWN trial found that the odds of good reperfusion and clinical outcome are significantly higher when thrombectomy is performed within 12 hours vs. 12 to 24 hours. Post-hoc analyses from the SWIFT PRIME (Solitaire with the intention for Thrombectomy as Primary Endovascular Treatment for Acute Ischemic Stroke) trial showed that symptom onset to reperfusion time of 150 minutes led to 91% probability of functional independence. This decreased by 10% over the next hour and by 20% with every subsequent hour.[48] Another analysis of the SWIFT PRIME trial evaluating the impact of recanalization time on functional outcome found that every six minutes of delay in reperfusion caused one more out of 100 treated patients to not achieve functional independence.[49]

In addition to improving the prehospital triage system, every effort should be made to reduce DTN times, discussed previously, as well as door-to-groin-puncture and vessel recanalization. Parallel processing is key in reducing door-to-recanalization rates at TSCs and CSCs. Strategies to reduce door-to-vessel-recanalization include endovascular suite prestroke preparation, stroke triage protocols, and early notification to neurointerventionalists of a potential endovascular candidate. (*See Table 2.*)

The focus for non-CSCs is reducing the door-in-door-out (DIDO) time. As demonstrated in the cardiac literature, patients with ST-elevation myocardial infarction and longer DIDO times not only had longer door-to-balloon times, but also had higher rates of mortality.[50] Strategies to reduce DIDO times include reducing DTN times, calling medical transport as soon as an LVO is suspected, and having a prespecified LVO patient transfer plan in place. Currently, it is not recommended that patients at non-CSCs or TSCs undergo perfusion imaging prior to transport. Concerns about performing vessel imaging at low-volume centers include the length of time to complete the study, length of time to scan interpretation, poor imaging quality, and the potential need to repeat imaging at the CSC should the images not be transferred. The use of air transfer has been shown to improve transfer time to CSC following IV alteplase.[51,52]

Future Directions

Future research should focus on improving patient selection criteria for mechanical thrombectomy. The current available evidence supports the use of the Alberta Stroke Program Early CT Score (ASPECTS) on CT scan for patients with LVO presenting within six hours; however, beyond six hours, evidence only exists for the use of perfusion imaging to identify salvageable penumbra (CT or MRI perfusion scan). ASPECTS was developed to offer a reliable way to assess early ischemic changes on non-contrast CT of the head.[43] Efforts also should be made to determine which patients should be transferred to a CSC for mechanical thrombectomy, given that some PSCs lack the ability to perform perfusion scans and the ability

to perform mechanical thrombectomy. Therefore, they are unable to assess the infarct volume, which, along with stroke severity, is the main determinant for mechanical thrombectomy candidacy beyond six hours.

Additional studies are underway to make endovascular therapies for LVO stroke safer and more effective. The ESCAPE-NA1 trial is investigating endovascular therapy along with a novel neuroprotective agent, NA1. Other studies are investigating the efficacy of distally trapping small thrombi dislodged during technical thrombectomy. Additional studies are evaluating the efficacy of endovascular therapy for posterior circulation LVO strokes. Mechanical thrombectomy strategies will continue to evolve to become even more efficacious than current approaches.

Given that most of the thrombectomy trials evaluated the safety and efficacy of mechanical thrombectomy of the ICA or the M1 segment of the middle cerebral artery, future research should focus on the feasibility and safety of mechanical thrombectomy of distal branches, such as M2 or M3, of the middle cerebral arteries. A recent meta-analysis that evaluated the safety and efficacy of M2 thrombectomy using a stent retriever or aspiration showed that M2 mechanical thrombectomy is associated with high recanalization rates and functional outcomes, but it was associated with an increased risk of symptomatic hemorrhage.[53]

Additional areas for research include improving the current techniques to increase the safety and efficacy of mechanical thrombectomy. Recently, the use of an aspiration technique (ADAPT) has emerged as a safe and effective method, with results comparable to the stent retriever.[54] However, this needs to be assessed in the setting of a clinical trial.

Conclusion

Acute ischemic stroke due to LVO is associated with poor functional outcome if not treated. Emergency physicians play a critical role in evaluating and managing patients with LVO. Early recognition and treatment with IV alteplase and mechanical thrombectomy lead to rapid revascularization, which is a strong predictor of good functional outcome. Future research should focus on improving door-to-needle and door-to-revascularization times.

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CME/CE Questions

1. A patient presents with aphasia and hemiparesis. What is the very first step in your assessment?
   a. Assess airway, breathing, and circulation
   b. MRI
   c. CT angiogram
   d. Serum sodium
2. Which test is suggested prior to a CT for a patient who presents with a stroke?
   a. Blood urea nitrogen
   b. Sodium
   c. Glucose
   d. Drug screen
3. A patient presents with right gaze deviation, left facial droop, left hemiplegia, left-sided sensory loss, and neglect. His NIH Stroke Scale score is 17. Which of the following is recommended in the ED?
   a. Aspirin
   b. Heparin
   c. Head CT
   d. Anti-epileptic

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4. The patient with a severe stroke (NIHSS 17) has a CT that shows no hemorrhage, but the radiologist says there are signs of a possible large vessel stroke. The patient has no contraindications to surgery or thrombolytics. It is now three hours since the onset of this stroke. The hospital across town (45 minutes away) does mechanical thrombectomy. The family understands the risks and wants everything done. Which of the following is the best course of action?
   a. Administer alteplase and transfer
   b. Admit the patient for an MRI/MRA in the morning
   c. Transfer the patient immediately
   d. Electroencephalogram
5. Which of the following is the most common cause of stroke in the United States?
   a. Patent foramen ovale
   b. Atrial fibrillation
   c. Intracranial atherosclerosis
   d. Embolism from aortic arch
6. Which of the following statements is *not true* about the NIH Stroke Scale?
   a. The larger the number, the more severe the stroke is.
   b. The scale is more biased to the dominant hemisphere.
   c. It is an excellent tool to evaluate posterior circulation strokes.
   d. It can predict stroke outcome.
7. Which of the following might indicate a posterior circulation stroke?
   a. A right hemiparesis and aphasia
   b. A sudden severe headache with normal neurologic exam
   c. A seizure followed by 30 minutes of unresponsiveness
   d. Ataxia, dysphasia, and gaze palsy
8. What is the longest recommended time after a stroke that thrombolytics can be given?
   a. 2 hours
   b. 3 hours
   c. 4.5 hours
   d. 6 hours
9. Although it is recommended that mechanical thrombectomy be performed within six hours, recent evidence suggests that it can be attempted as late as:
   a. eight hours.
   b. 16 hours.
   c. 24 hours.
   d. four days.
10. A 10-year-old with sickle cell disease presents with aphasia and a dense hemiparesis. CT angiogram shows complete vascular occlusion. The family includes several physicians who want the patient to undergo thrombolysis and mechanical thrombectomy. Which of the following statements (based on the AHA/ASA recommendations) is true?
    a. The patient is a good candidate if the symptoms started within six hours of presentation.
    b. Endovascular thrombectomy may be reasonable, but firm evidence is lacking for this age group.
    c. The patient can receive thrombolytics, but mechanical thrombectomy is contraindicated in pediatric patients.
    d. The Sickle Cell Stroke Study showed no benefit when patients were treated with thrombolytics or mechanical thrombectomy.

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CME/CE Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

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Acute Ischemic Stroke Due to Large Vessel Occlusion

Prehospital Stroke Clinical Tools for EMS

Stroke Screens	Stroke Scores
• Cincinnati Prehospital Stroke Scale (CPSS) • Los Angeles Prehospital Stroke Screen (LAPSS) • Melbourne Ambulance Stroke Screen (MASS) • Miami Emergency Neurologic Deficit (MEND) • Recognition of Stroke in the Emergency Room (ROSIER) Score	• 3-Item Stroke Scale (3ISS) • Cincinnati Stroke Triage Assessment Tool (C-STAT) • Field Assessment Stroke Triage for Emergency Destination (FAST-ED) • Los Angeles Motor Scale (LAMS) • National Institutes of Health Stroke Scale (NIHSS) and shortened NIHSS (sNIHSS –5/8) • Rapid Arterial Occlusion Evaluation (RACE) Scale

Stroke Best Practices

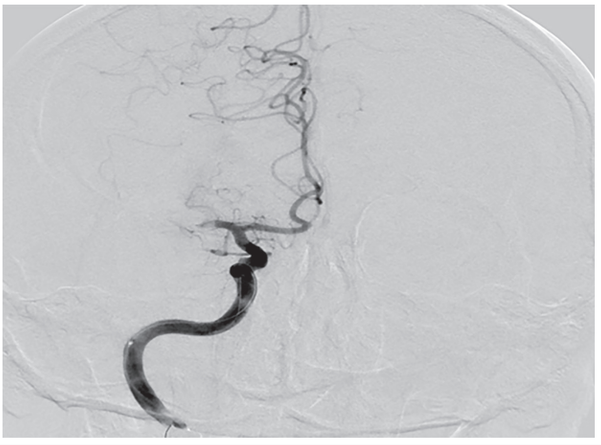
- Prehospital stroke tools and tool kits
- Team-based approach
- Emergency medical services prehospital notification
- Single-call activation of entire team
- Rapid stroke triage in the emergency department
- Direct EMS transfer to computed tomography
- Rapid CT and interpretation
- Rapid laboratory testing/point of care
- Mix alteplase early
- Rapid alteplase administration
- Prompt feedback to all parties
- Early notification for possible endovascular therapy

Based in part on AHA Target Stroke strategies (Available at: http://bit.ly/2ArnBzl)

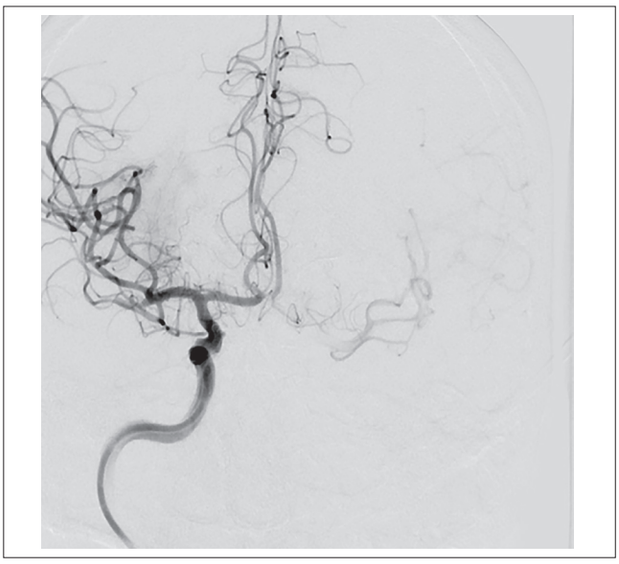
Best Practices for Rapid Endovascular Therapies

- Continue team-based approach
- Prestock and prepare neurointerventional radiology suite for next case
- Utilize prehospital large vessel occlusion scores and triage protocols
- Early notification of NIR team of possible LVO, especially in transfers
- Early notification of interhospital transport personnel
- Prepare CT/MRI prior to arrival if needed
- Prepare need for sedation/anesthesia
- Consider direct transfer to neurointerventional suite upon transfer arrival

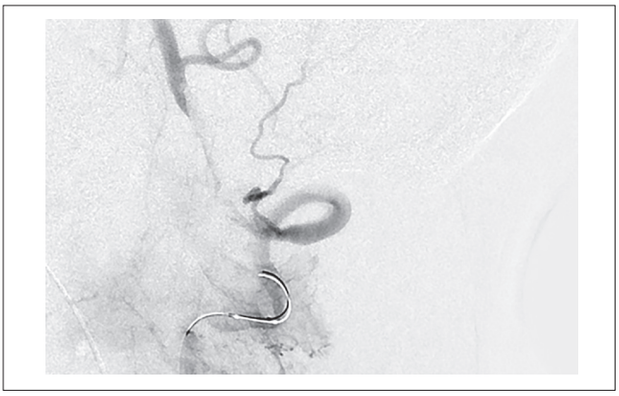
Complete Occlusion of the Proximal Segment of the Right Middle Cerebral Artery



Revascularization of the Previously Occluded Proximal Segment of the Right Middle Cerebral Artery



Near Complete Occlusion of the Proximal Basilar Artery



Complete Revascularization of the Previously Occluded Proximal Basilar Artery

Supplement to *Emergency Medicine Reports*, January 15, 2018: "Acute Ischemic Stroke Due to Large Vessel Occlusion." Authors: Sami Al Kasab, MD, Department of Neurology, University of Iowa Hospitals and Clinics, Iowa City, IA; Christine A. Holmstedt, DO, Department of Neurology, Medical University of South Carolina, Charleston; Edward C. Jauch, MD, Department of Emergency Medicine, Medical University of South Carolina, Charleston.

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