Complications of Mechanical Thrombectomy in Acute Ischemic Stroke

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Abstract

Multiple randomized clinical trials have supported the use of mechanical thrombectomy (MT) as standard of care in the treatment of large vessel occlusion acute ischemic stroke. Optimal outcomes depend not only on early reperfusion therapy but also on post thrombectomy care. Early recognition of post MT complications including reperfusion hemorrhage, cerebral edema and large space occupying infarcts, and access site complications can guide early initiation of lifesaving therapies that can improve neurologic outcomes. Knowledge of common complications and their management is essential for stroke neurologists and critical care providers to ensure optimal outcomes. We present a review of the available literature evaluating the common complications in patients undergoing MT with emphasis on early recognition and management.

Introduction

Stroke is the fifth leading cause of death in developed countries and the leading cause of serious long-term disability in the United States. Stroke accounts for 1 in every 20 deaths in the United States.1 Timely administration of thrombolitics and mechanical thrombectomy (MT) have been demonstrated to dramatically improve stroke outcomes. The benefits of these hyperacute stroke treatments necessitate close postprocedural monitoring in a neurocritical care or stroke unit.

Multiple positive randomized clinical trials have established MT as the standard of care for large vessel occlusion (LVO) acute ischemic stroke. Despite its efficacy, MT, like any endovascular procedure, carries small but significant risks, which have been well described in the course of these clinical trials and further observed in real-world experience. These complications can be broadly divided into 2 categories: (1) intraprocedural complications, mainly related to the access site (e.g., vessel injury, groin hematoma, dissection); (2) postprocedural complications dealing with stroke and recanalization sequelae (i.e., reperfusion injury and cerebral edema).2

We review some of the important complications after MT as well as the risk factors, underlying pathophysiology, and strategies for early recognition and treatment.

Post-MT Intracerebral Hemorrhage

Emergent reperfusion therapy is the cornerstone of treatment in acute ischemic stroke. Restoring cerebral blood flow to salvageable ischemic tissue reduces patient disability. Symptomatic intracerebral hemorrhage (sICH) is the most feared complication post-MT. The rates of sICH were reported to be 7.6% and 4.4% among pooled thrombolysis and thrombectomy trials, respectively.3 Clinically significant sICH usually develops early following reperfusion treatment with most of the fatal hemorrhages occurring within the first 24 hours of receiving MT.4 Flat-panel detector CT offers the possibility to perform cross-sectional imaging within the angiography suite and can be used to detect early intracerebral hemorrhage (ICH).5 By rapidly
visualizing hemorrhage in the treatment environment, life-saving treatments can be initiated without the need for a trip to the CT suite and an additional scan.

Post-Reperfusion Hemorrhage Classification

The National Institute of Neurological Disorders and Stroke definition of sICH includes any hemorrhagic transformation temporally related to neurologic worsening, which may be overly inclusive because it captures small petechial hemorrhages associated with minimal neurologic deterioration that are unlikely to have an effect on long-term functional outcome.6,7 In contrast, the ECASS (European Cooperative Acute Stroke Study) and SITS-MOST (Safe Implementation of Thrombolysis in Stroke–Monitoring Study) definitions include only hemorrhage associated with substantial clinical worsening of ≥4 points on the National Institutes of Health Stroke Scale (NIHSS), which may be more predictive of ICH that affects long-term outcome.7,8

Previously, the ECASS II/III classification was used to report sICH, which is divided into hemorrhagic infarction (HI) and parenchymal hematoma (PH) (Figure 1). HI is a heterogeneous hyperdensity occupying a portion of an ischemic infarct zone on CT, whereas PH refers to a more homogeneous, dense hematoma with mass effect. HI and PH are classified further into 2 subtypes. The reported rate of HI is higher than PH (9% vs 3%, respectively). PH2 with a lesion volume of >30% is the only subtype of hemorrhagic transformation (HT) that may significantly alter the clinical course of ischemic stroke and has been found to be a significant predictor of neurologic deterioration with higher mortality and poor 3-month outcome.9,10

The advent of MT necessitated an expansion in the classification of hemorrhage after reperfusion therapy with or without the use of recombinant tissue plasminogen activator (rtPA). The IV rtPA trials were focused on parenchymal hemorrhage because ICH is the predominant event after thrombolytic therapy.7 However, after MT, subarachnoid hemorrhage (SAH) can also be observed due to penetrating or dissecting vessel injury with the device or stretch injury on perforating vessels from traction on the large arteries of the circle of Willis when removing the thrombectomy devices. The Heidelberg Bleeding Classification (HBC) for ICH was created by a consensus of stroke experts in 2015 and is now commonly employed in ongoing trials. The HBC amplifies the ECASS classification for ICHs after reperfusion therapy by including previously nonclassifiable hemorrhages and providing a formal approach for more precise anatomical description and better assessment of symptomatic ICHs (Table 1 and Figure 1).7 It is important to take into consideration the differences between these classifications when comparing the rate of sICH of different trials.

On a routine noncontrast head CT scan performed after IA or IV contrast administration, it can be difficult to differentiate hyperattenuation resulting from iodinated contrast vs that arising from intracranial hemorrhage. This distinction is especially critical when antithrombotic therapy is being considered. Dual energy CT or MRI gradient echo sequences can be helpful in distinguishing blood from retained contrast agent in the brain or subarachnoid space. If the attenuation value of an area of hyperattenuation exceeds that expected for hemorrhage, it can be assumed that there is a component of iodinated contrast material within the area of abnormality.

Pathophysiology

The fundamental underlying pathophysiology of ICH after MT is disruption to the blood-brain barrier (BBB) secondary to ischemic and mechanical endothelial injury leading to increased tissue permeability.11 The BBB is composed of endothelial cells, pericytes, astrocytes, neurons, and the extracellular matrix, collectively referred to as the neurovascular unit.12 Reperfusion injury includes activation of endothelium, excess production of oxygen free radicals, inflammatory responses and leukocyte recruitment, increase in cytokine production, and edema formation (Figure 2).13,14 There are 3 stages of paracellular permeability (reactive hyperemia, hypoperfusion, and biphasic response) after reperfusion.

Stage 1 corresponds to reactive hyperemia, which is secondary to loss of cerebral autoregulation and vasodilation leading to an increase in cerebral blood flow (CBF) and BBB permeability resulting in cytotoxic edema. Stage 2 is hypoperfusion secondary to vasoconstriction and decrease in CBF. This response leads to nutritional deficiency in brain tissue and enhances neutrophil adhesion with subsequent inflammatory activity. Stage 3 is due to vasogenic edema, which is associated
with alterations in BBB tight junctions, resulting in increased permeability to macromolecules. These alterations result in deregulated extracellular proteolysis and elevated levels of matrix metalloproteinases (MMP-9 and MMP-2), cellular fibronectin, and caveolin-1, all of which have been associated in human studies with HT.

In the setting of an LVO, IV thrombolysis with rtPA is frequently used in conjunction with MT. By interacting with the NMDA-type glutamate receptor, rtPA amplifies potentially excitotoxic calcium currents. Furthermore, rtPA increases MMP activity, which in turn increases the risks of neurovascular cell death, BBB leakage, edema, and hemorrhage. IV rtPA converts plasminogen into plasmin, which in turn degrades fibrin into fibrin split products, causing thrombolysis. While 80% of rtPA is rapidly cleared 10 minutes after cessation of the infusion, the effect of rtPA on the coagulation profile may last up to 24 hours or longer, with resultant prolongation of the prothrombin and partial thromboplastin times as well as a reduction in fibrinogen levels. The incidence of sICH may peak 6 hours after treatment when there is a depletion of fibrinogen below 200 mg/dL.

**Predictors for Post-MT ICH**

There are many predictors for good outcome post MT but no clear predictors for post-MT ICH incidence. The following discussion is based on the best available data from retrospective meta-analysis of randomized data and pooled data from retrospective single and multicenter studies. Preprocedural arterial hypertension, worse initial NIHSS score, and poor collaterals are some factors associated with post-procedural ICH. Approximately 75% of patients present with elevated blood pressure (BP) at the time of ischemic stroke. Several studies have confirmed significantly higher rates of sICH with elevated pretreatment BP readings (systolic BP [SBP] >185 mm Hg and diastolic BP [DBP] >110 mm Hg). The 2018 American Heart Association/American Stroke Association guidelines recommend the BP to be at or below 185 mm Hg systolic and 110 mm Hg diastolic before administering rtPA and must be maintained below 180/105 mm Hg within 24 hours following rtPA without MT. Postprocedural BP variability (BPV) in the first 24 hours after MT has been associated with worse outcome at 3 months. This relationship is stronger with SBP than with DBP. Increased BPV can further impair the cerebral autoregulation and enhance BBB permeability.

SBP reduction post-MT is thought to be beneficial in cases with complete reperfusion; however, it can be detrimental in cases of incomplete reperfusion, as these patients may be dependent on higher SBP to support retrograde collateral supply to the territory that remains occluded. Owing to lack of randomized control trials, current practice is variable across institutions, but most institutions favor intensive SBP reduction to <140 or <160 mm Hg after successful reperfusion, defined as modified Thrombolysis in Cerebral Infarction score of 2b or higher. ASPECTS (Alberta Stroke Program Early CT Score) is a quantitative score that measures the extent of early ischemic changes in anterior circulation hyperacute middle cerebral artery ischemic stroke. Lower ASPECTS is associated with worse outcomes post MT, ICH post MT, and higher mortality. Patients with poor collaterals are at higher risk of developing sICH; thus, collateral status can help predict the
Table 1  Heidelberg Bleeding Classification (HBC) and European Cooperative Acute Stroke Study (ECASS) Radiologic Classification of Post Thrombolysis and Post-Mechanical Thrombectomy Intracerebral Hemorrhage

<table>
<thead>
<tr>
<th>ECASS II–III</th>
<th>Definition</th>
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<tr>
<td>1</td>
<td>Hemorrhagic transformation of infarcted tissue</td>
</tr>
<tr>
<td>1a</td>
<td>HI 1: Small petechial hemorrhage without space-occupying effect</td>
</tr>
<tr>
<td>1b</td>
<td>HI 2: More confluent petechial hemorrhage without space-occupying effect</td>
</tr>
<tr>
<td>1c</td>
<td>PH 1: Hematoma within infarcted tissue, occupying &lt;30%, no substantive mass effect</td>
</tr>
<tr>
<td>2</td>
<td>PH 2: Intracerebral hemorrhage within and beyond infarcted tissue; hematoma occupying &gt;30% of the infarcted area with significant space-occupying effect</td>
</tr>
<tr>
<td>3</td>
<td>Intracerebral hemorrhage outside the infarcted brain tissue or intracranial-extracerebral hemorrhage</td>
</tr>
<tr>
<td>3a</td>
<td>Parenchymal hematoma remote from infarcted brain tissue</td>
</tr>
<tr>
<td>3b</td>
<td>Intraventricular hemorrhage</td>
</tr>
<tr>
<td>3c</td>
<td>Subarachnoid hemorrhage</td>
</tr>
<tr>
<td>3d</td>
<td>Subdural hemorrhage</td>
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Abbreviations: HI = hemorrhagic infarction; PH = parenchymal hematoma.

risk of reperfusion injury after revascularization. Increased neutrophil/lymphocyte ratio is an independent predictor of sICH in patients receiving MT. Neutrophil granulocytes are an important source of MMP-9 that may further contribute to early disruption of the BBB in ischemic stroke and cause sICH.14

Patients who experience an ICH after recanalization have a higher mean peak systolic velocity ratio by transcranial Doppler (TCD) immediately after MT in the recanalized middle cerebral artery (MCA) compared to those who did not have ICH. These results suggest that hyperperfusion after MT might have deleterious effects on several elements of the BBB in ischemic tissue due to reperfusion mediated injury.12,26 Thus, TCD evaluation post MT, though not standard of care, can provide valuable prognostic information regarding patients exhibiting hyperperfusion or loss of vaso-motor reactivity, which may portend a higher risk of reperfusion hemorrhage. This information may help identify patients at risk of sICH and influence clinical care decisions like more intensive BP reduction.

Perfusion-weighted MRI (PWI) can be used to predict successful recanalization as well as reperfusion hemorrhage. PWI can potentially identify favorable and unfavorable patterns of blood flow in patients with LVO who undergo thrombectomy. Patients with a target mismatch profile as described in DEFUSE 2 with a substantial volume of salvageable tissue (small diffusion-weighted MRI lesions relative to large perfusion-weighted MRI lesions) responded favorably to recanalization. In addition, patients with a nontarget mismatch profile who received reperfusion had higher rates of parenchymal hematoma and reperfusion-related edema.28

Recognition and Management of Post-MT ICH

There are no current established guidelines for post MT imaging for stable patients. However, it is reasonable to obtain a CT or MRI brain within 24 hours of treatment to assess for any hemorrhage in order to guide initiation of an antithrombotic regimen. sICH should be suspected in any patient who develops sudden deterioration in level of consciousness, new headache, nausea and vomiting, or a sudden rise in BP after revascularization therapy (especially within the first 24 hours of treatment). Emergent CT imaging to confirm ICH is indicated after rapid assessment of respiratory and hemodynamic stability is performed. In cases of ICH on CT scan, aggressive BP control is often recommended beyond current post thrombectomy guidelines to prevent further ICH expansion. At our center, we recommend keeping the systolic BP between 140 and 160 mm Hg and diastolic pressure less than 90 mm Hg in patients with sICH. Although tPA has a short half-life of 5 minutes, in cases of PH1 and PH2 hemorrhage, reversal of tPA with cryoprecipitate is indicated, especially if fibrinogen is <100 mg/dL.

Urgent neurosurgical consultation may be indicated. Surgical intervention to reverse or prevent herniation can be lifesaving. Neurosurgeons may be reluctant to perform a ventriculostomy or craniotomy owing to the difficulty in achieving hemostasis in patients who have been anticoagulated or received thrombolytics. Although the half-life of tPA is very short, approximately 5 minutes for initial half-life and 72 minutes for the terminal half-life, a prolonged antiplatelet effect may persist for hours and reversal of its effects are reasonable in the setting of intracranial hemorrhage beyond its expected half-life. To that end, it is essential to have written protocols that can assure emergent reversal of anticoagulants and thrombolytics. The recommendations for antithrombotic and fibrinolytic reversal per neurocritical care society are summarized in Table 2.29

Vessel Reocclusion Post MT

The strongest predictor of good outcome for patients undergoing MT for LVO is successful recanalization of the occluded cerebral vessel. However, despite successful recanalization, reocclusion of the target vessel occurs in 3% of patients. Vessel reocclusion most often occurs from 0 to 48 hours after successful recanalization. Predictors of reocclusion include elevated platelet counts at admission >220 g/L, residual thrombus or stenosis on the last angiographic run after MT, and stroke of undetermined cause. Underlying stenosis or residual embolic fragments at the thrombectomy site act as a
nidus to which the higher concentration of circulating platelets adhere, leading to the formation of a new occlusive thrombus in the same location. Despite the clear benefit of MT for LVO, it is important to recognize that vessel wall injury with the use of stent retrievers or the use of distal aspiration catheters can occur, and that intimal injury may contribute to reocclusion.33

Once the patient is in the intensive care unit (ICU), reocclusion should be suspected in any patient who clinically deteriorates after initial post-thrombectomy improvement. Serial neurologic examinations for the first 24 hours are a fundamental part of postprocedure care of all patients undergoing any reperfusion therapy. In cases of suspected reocclusion, emergent neuroimaging of the brain parenchyma in addition to vascular imaging is indicated. If vessel reocclusion is noted and the core infarct remains small, strategies to salvage the tissue at risk should immediately be implemented. These may include induced hypertension and return to angiography for definitive revascularization.

In patients receiving tPA with or without MT, antiplatelets are routinely started at 24 hours. However, in patients with residual thrombus seen during MT, IV infusions of heparin or IIb/IIIa inhibitors such as eptifibatide may be considered during or immediately post MT to prevent reocclusion followed by dual antiplatelets. Although there are no evidence-based data to guide this practice, early cross-sectional imaging with CT or MRI to determine the extent

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**Table 2 Recommendations for Reversal of Antithrombotics, Anticoagulants, and Fibrinolytics in Patients With Intracerebral Hemorrhage**

<table>
<thead>
<tr>
<th>Antithrombotic</th>
<th>Reversal agent</th>
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<tr>
<td>Vitamin K antagonists</td>
<td>If INR &gt;1.4: vitamin K 10 mg IV, plus 3- or 4-factor PCC IV (dosing based on weight, INR, and PCC type) or FFP 10–15 mL/kg IV if PCC not available.</td>
</tr>
<tr>
<td>Direct factor Xa inhibitors</td>
<td>Activated charcoal (50 g) within 2 hours of ingestion, activated PCC (FEIBA) 50 units/kg IV, or 4-factor PCC 50 units/kg IV</td>
</tr>
<tr>
<td>Thrombolytic agents</td>
<td>Cryoprecipitate 10 units IV or antifibrinolytics (tranexamic acid 10–15 mg/kg IV over 20 minutes or e-aminocaproic acid 4–5 g IV) if cryoprecipitate is contraindicated</td>
</tr>
<tr>
<td>(plasminogen activators)</td>
<td></td>
</tr>
<tr>
<td>Antiplatelet agents</td>
<td>DDAVP 0.4 μg/kg IV ×1; if neurosurgical intervention, platelet transfusion (1 apheresis unit)</td>
</tr>
<tr>
<td>Unfractionated heparin</td>
<td>Protamine 1 mg IV for every 100 units of heparin administered in the previous 2–3 hours (up to 50 mg in a single dose)</td>
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</tbody>
</table>

Abbreviations: INR = international normalized ratio; PCC = prothrombin complex concentrate.
of infarct and presence of reperfusion hemorrhage is often useful to tailor therapy and mitigate risk of hemorrhagic transformation.

**Post-MT Cerebral Edema**

The natural course of ischemic stroke includes cerebral edema. A great deal of research has been directed at more fully understanding the pathophysiology of cerebral edema. A complicated cascade of events is precipitated by the initial ischemia and neuronal hypoxia, both cytotoxic and vaso-genic processes, contribute to an eventual breakdown of the BBB.34,35

Cytotoxic edema occurs in the immediate period after brain ischemia. Decreased oxygen availability causes a shutdown of the electron transport chain, which results in decreased ATP production necessary for cellular metabolism; ultimately the sodium-potassium pump stops functioning properly and intracellular sodium increases. This is followed by an increase in intracellular water.36 Throughout this initial process, the BBB is intact, and generally no significant increase in intracranial pressure (ICP) is observed; however, individual neurons are lysed secondary to impaired cell membrane activity.37 Vaso-genic edema exacts its influence on increased ICP primarily by destruction of the BBB. This process is poorly understood but involves pinocytosis of endothelial cells and injury to the blood vessels’ tight junctions.38 Multiple intermediaries have been suggested to facilitate the actual breakdown of the BBB.39

The extent, time course, and location of cerebral edema relates directly to its clinical consequences. Poststroke edema can be divided into 2 distinct categories—nonmalignant and malignant forms—both of which can result in major secondary injury via increased intracranial pressure. Both categories differ principally in the time course of edema formation. Nonmalignant edema typically begins as early as 6 hours postinsult and progresses slowly and peaks at 3–5 days.40 In contrast, the malignant MCA syndrome is defined by space-occupying edema resulting in more rapid and significant neurologic worsening and can occur within the first 24 hours.35,41 Between the brain parenchyma and CSF in the subarachnoid regions lie the major water membrane channels, which may have a significant effect on brain edema; these membrane channels, called aquaporin 4 (AQP4), are organized within astrocytes and perivascular end feet and are suspected to have a key function in CNS osmoregulation and subsequent malignant edema. Upregulation of AQP4 attenuates edema formation in
laboratory studies and may decrease BBB disruption; as a result, manipulation of AQP4 is an area of ongoing research.42 Cerebral edema may also be triggered or exacerbated by MT itself. Although the goal of MT is to prevent stroke, recanalization of infarcted tissue can result in edema, which may further contribute to the secondary brain injury. Malignant infarctions involve a greater percentage of the cerebral hemisphere (i.e., the middle MCA territory) and are caused by intracranial internal carotid artery occlusion or MCA occlusion.35

Identification of patients at higher risk for developing a malignant MCA syndrome is important to assist in post MT management, and a number of clinical and radiologic risk factors have been identified. Factors associated with malignant MCA syndrome and severe brain edema include large stroke, predicted by higher initial NIHSS, presence of the hyperdense MCA sign, and low ASPECTS; and the extent of collateral supply.36 Other predictors include elevated serum glucose levels and large ischemic core on initial CT head.38,43

There are a number of radiographic predictors for the development of cerebral edema. First, involvement of a greater volume of brain parenchyma is associated with worsened edema. An infarct affecting more than 50% of the MCA distribution territory or an initial preintervention perfusion deficit of greater than 66% on CT and CT perfusion, respectively, have been shown in multiple studies to be the best predictors of ensuing brain edema. A small penumbra or a large core predicts worse outcomes29,35,44 and the presence of sulcal effacement has a higher association with worsened edema when seen on initial noncontrast CT. By convention, CT imaging can be performed in the early time window (0–6 hours) or late time window (6–24 hours). In the late time window, CT head may be a more precise tool for judging the extent of irreversible infarct. MRI studies have identified that an ischemic lesion volume of greater than 145 mL on diffusion-weighted imaging at 14 hours reliably predicts worsened edema, as does apparent diffusion coefficient lesion >82 mL at 6 hours.29,35,45

Treatment of cerebral edema hinges on discriminating malignant and nonmalignant edema because they have different outcomes. Hallmarks of the former include swelling that worsens over the first 48 hours and often results in significant increased ICP and rapid clinical worsening resulting in significant morbidity including coma or mortality.36,37 Medical treatment begins with the basics of neurocritical care, which includes proper patient positioning with the head of bed between 30° and 45°. Adequate ventilation must be maintained to prevent deleterious compensatory cerebral vasodilation in response to hypercarbia.
Hyperosmolar therapy with mannitol or hypertonic saline can also be used to help mitigate cerebral edema. Mannitol should be administered in a weight-based strategy with bolus dosing of 1 g/kg for impending herniation and serial monitoring of serum osmolarity when it is continued throughout the course of cerebral edema to ensure adequate mannitol clearance prior to the next dose. Serum osmolarity of less than 320–340 is generally an accepted cutoff for redosing of mannitol. In addition, when the osmolarity exceeds these levels, an osmolar gap may be calculated, which is the difference between measured and calculated osmolarity (calculated osmolarity = 2 × [Na, nm] +1.15 × ([glucose/18 + urea/2.8])). An osmolar gap >10 indicates that there is circulating mannitol yet to be cleared and that additional doses should be withheld. Intermittent scheduled mannitol boluses are often employed at lower doses (0.25–0.5 g/kg) to continuously manage cerebral edema. Mannitol works as an osmotic diuretic and thus its major complications include volume depletion and renal failure, both of which can be effectively mitigated by measuring daily weights, intake and output, and avoiding dosing when mannitol has not been cleared by the kidneys.

Hypertonic saline is also effective in controlling cerebral edema. It is available in multiple concentrations and can be customized by pharmacy. The most commonly used formulations include 3% NaCl and 23.4% NaCl preparations. The 23.4% preparation is used in boluses of 30 mL and must be administered slowly through a central line over several minutes to avoid venous sclerosis and arrhythmia. It has the advantage of being a small volume and is used in a similar manner to 1 mg/kg dosing of mannitol to reverse impending or active herniation syndromes. Continuous infusions of 3% sodium are used to maintain a hypertonic state that will minimize cerebral edema by the osmotic movement of water out of the brain. In contrast to mannitol, hypertonic saline is not a diuretic and can lead to volume overload and hyperchloremia with metabolic acidosis and renal failure. There are no high-quality data comparing the efficacy of these 2 therapies and as such the choice of agent is often guided by the patient’s comorbid conditions, particularly cardiac and lung function.

Hyperosmolar therapy is a key component to managing cerebral edema, but it is essential to recognize patients who are manifesting a malignant syndrome as they are much more likely to herniate and die despite medical therapies.36 Approximately 80% of patients with malignant edema will die if treated with medical therapy alone.37 Definitive treatment via decompressive craniectomy is the standard of care treatment for malignant MCA infarcts. Multiple randomized controlled trials have demonstrated increased survival and decreased morbidity when craniectomy is performed within up to 96 hours of initial injury in malignant MCA in patients 60 years and younger. A prespecified meta-analysis of the pivotal randomized trials HeADDFIRST, DESTINY, DECIMAL, and HAMLET confirmed the aforementioned improvements in outcome. Numbers needed to treat (NNTs) varied: to prevent death, NNT was 2; to prevent modified Rankin Scale (mRS) >4, NNT was 4.46 It is essential for patients who have been selected to have hemicraniectomy to receive aggressive medical treatment while awaiting surgery. It is customary at our center to electively intubate these patients and maintain them paralyzed, sedated, and mildly hyperventilated as a bridge to surgery. Craniectomy in patients >60 years old has in general been shown to be deleterious, with increased morbidity and mortality; however, the data are for all-comers and patients >60 years of age with low baseline mRS scores should be evaluated individually.37

Infratentorial infarctions involving the cerebellum have unique challenges associated with them and are classified as a separate subgroup. There are no large clinical trials to inform treatment; rather, management of these injuries is driven by accumulated experience and understanding of the pathophysiology of acute obstructive hydrocephalus. The development of cerebellar edema and obliteration of the fourth ventricle will result in devastating increases in ICP and rapid decerebration, which culminates in tonsillar herniation.48 As opposed to the previously discussed volume predictors in supratentorial strokes, cerebellar infarction volumes and their respective predictive capacities as pertaining to decompressive suboccipital craniectomy have not been well established.49 However, given the clear dismal outcome when not decompressed and the often excellent clinical outcome of patients who survive large cerebellar strokes without much brainstem involvement, there is a clear standard of care and strong corresponding multisociety recommendations in terms of aggressive surgical treatment of these infarcts with prompt suboccipital decompression. Suboccipital decompressive craniectomies have been shown to be beneficial in multiple nonrandomized controlled studies and accumulated experience; concomitant brainstem infarcts or bilaterality of the cerebellar stroke portend higher morbidity and mortality.50

Management with ventriculostomy alone is not recommended as definitive management for infra- and supratentorial stroke for multiple reasons. First, herniation syndromes can occur with normal ICP51 due to the local nature of a ventriculostomy monitor. As such, waiting for ICPs to be elevated prior to proceeding with surgery violates the principle of decompressive surgery to relieve the mass effect prior to the patient being in extremis and unsalvageable. Recent retrospective studies demonstrate that some patients who meet criteria for supratentorial decompressive surgery according to randomized clinical trials do not end up requiring surgery and can be managed medically.52 However, these patients are not identifiable at 24 hours based on prospectively available data, so decompression is often pursued to avoid herniation progression. When patients present 4–5 days into the stroke, it is reasonable to manage the patient with hyperosmolar therapy based on the assumption that the peak edema has subsided.
Access Site Complications Post-MT

The majority of MT procedures are performed via the femoral artery using a modified Seldinger technique for access and then introduction of large 6–8 Fr sheaths. Aside from the potential complications from the thrombectomy and revascularization procedure itself, patients are also at risk for adverse events related to the access site. Access site complications have been well described in both the coronary and neurovascular literature. When all potential complications are included, there is an overall risk of developing at least one of the following (listed generally in descending order of relative frequency): ecchymoses, groin hematoma, access site bleeding, pseudoaneurysm, arteriovenous fistula, access site occlusion, retroperitoneal or rectus sheath hematomas, femoral neuropathies, and infection.53,54 Depending upon which literature is cited, complication rates vary greatly; consensus across a broad number of sources puts the percentage of patients with any groin complication at 1%–11%.53,55,56 Investigators of the SWIFT trial (Solitaire FR With Intention for Thrombectomy) data reported a major groin complication rate of 2.8%.57 The ESCAPE trial (Endovascular Treatment for Small Core and Anterior Circulation Proximal Occlusion With Emphasis on Minimizing CT to Recanlization Times) reported a 7.2% groin hematoma rate; EXTEND IA (Extending the Time for Thrombolysis in Emergency Neurologic Deficits—Intra-Arterial) reported 2.9%; REVASCAT (Randomized Trial of Revascularization With Solitaire FR Device vs Best Medical Therapy in the Treatment of Acute Stroke Due to Anterior Circulation Large Vessel Occlusion Presenting Within Eight Hours of Symptom Onset) reported 10.7%.58

The association of a high needle stick with retroperitoneal hematoma has been well documented. This complication corresponds with the anatomy of the external iliac artery, which courses from a retroperitoneal origin and travels beneath the inguinal ligament to become the extraperitoneal femoral artery (see Figures 3–5).59 High needle sticks above the inguinal ligament can result in bleeding in the retroperitoneum (Figure 6) due to the difficulty in achieving adequate hemostasis when performing manual compression above the femoral head, against which the artery is generally compressed. More rarely, high access may cause a direct injury to the external iliac or epigastric artery itself, from which bleeding is difficult to control for the anatomic reason mentioned above.59

Retroperitoneal bleeding can be a rapidly fatal complication. This diagnosis should be suspected in any patient post femoral access who has unexplained hypotension, back pain with or without ecchymosis, or groin hematoma. Many patients who are postthrombectomy may be sedated, intubated, encephalopathic, or otherwise unable to report back pain. In these patients, the clinician must rely on physical examination findings. Stat hemoglobin and hematocrit should be drawn and immediate fluid resuscitation should be initiated. Adequate IV access with 2 large-bore IVs should be secured if not already present and red blood cell transfusion should be ordered as the patient is transported for pelvic and abdominal imaging with CT. Concurrent CT angiogram of the abdomen and pelvis can also be obtained to help determine the etiology of the retroperitoneal bleeding (e.g., pseudoaneurysm). Vascular surgery consultation should be obtained once the diagnosis is confirmed.

As the femoral artery gets smaller and progresses distally, it is directly on top of the femoral vein, and as such low needle sticks that go through the back wall of the artery causing a communication with the femoral vein are associated with pseudoaneurysms and arteriovenous fistula.60 These abnormalities usually present in a delayed fashion but can be seen as a growing and pulsatile groin mass that is often painful. Vascular surgery consultation for management of these complications is recommended.

When the patient arrives in the ICU, it is essential to obtain a clear history on the size and location of the access and the method of hemostasis that was employed—manual compression or with a vascular closure device. Preoperative pulses should be charted and compared to the initial examination in the ICU. Careful neurovascular examination of the site including distal pulses should be performed at least every 15 minutes for the first hour, then every 30 minutes for the next hour, and then hourly for at least 1 or 2 hours with the leg straight. If a hematoma is noted, the dressing should be removed, and, while holding direct pressure above the puncture site, the hematoma should be expressed. Any residual hematoma should be marked on the skin to monitor it for further expansion.

Discussion

MT is a lifesaving procedure that has revolutionized acute ischemic stroke care by significantly improving the natural history of this otherwise devastating illness. It is widely recognized that the success of these procedures is due in large part to the speed and efficacy of revascularization. Post-MT care is also essential to ensuring excellent outcomes and, with proper preparation, complications can be avoided; when complications are encountered and recognized quickly, they can often be mitigated effectively.

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