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## Management of patients with isolated acute cervical carotid artery occlusion and normal neurological exam: Technical note and case series


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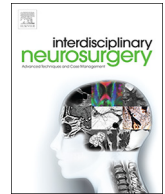
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## Technical notes &amp; surgical techniques

# Management of patients with isolated acute cervical carotid artery occlusion and normal neurological exam: Technical note and case series



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## ABSTRACT

**Objectives:** Limited data exists on the management and outcome of patients with isolated acute cervical internal carotid artery (cICA) occlusion presenting with normal neurologic exam after experiencing a period of neurological deficits. These patients are at risk for progressive neurologic deterioration but have not yet progressed to stroke. Current management is no intervention due to intervention risk of embolization. We aim to determine the optimal management of patients with isolated acute cICA occlusion presenting with a normal neurological exam after experiencing neurological deficits.

**Patients and methods:** Data was collected on 3 patients with acute cICA occlusion that presented with a normal neurological exam to our institution. Patient 1 was treated according to standard protocol, while patients 2 and 3 were treated according to the management discussed. Associations between perfusion imaging studies and clinical outcome were analyzed to determine stroke risk. A revascularization technique to minimize risk of distal embolization is described.

**Results:** A total of 3 consecutive patients with acute cICA occlusion were successfully revascularized. Patients 2 and 3 (66.67%) were neurologically intact post-operatively, while patient 1 (33.33%) had residual hemiparesis. It seems that  $MTT \geq 200\%$  or  $T_{max} > 6s$  is the optimal penumbra threshold predicting infarction and neurologic deterioration. There were no embolic complications as a result of endovascular therapy (EVT).

**Conclusion:** Cerebral perfusion imaging of patients presenting with normal neurological exam after experiencing neurological deficits is warranted to help identify patients at risk for stroke due to collateral failure. These patients should be monitored in the ICU for neurologic deterioration and given the option of intervention if mismatch is noted on CT perfusion imaging. Perfusion studies identifying penumbra and delayed  $MTT \geq 200\%$  or  $T_{max} > 6s$  are indicators for possible collateral failure. In patients undergoing intervention, we suggest a technique using proximal flow arrest to minimize risk of shower emboli. Further studies are needed to verify our findings.

## 1. Introduction

The management of patients with isolated acute cervical internal carotid artery (cICA) occlusion presenting with neurological deficits that promptly return to baseline shortly before/after admission or prior to surgery is challenging. Infarction of brain parenchyma has not yet occurred in these patients, but they are still at risk for collateral failure and delayed stroke. Patients with cross flow via an intact circle of Willis

(COW) and adequate collaterals may be asymptomatic or present with fluctuating symptoms. Direct collaterals via the anterior and posterior communicating arteries of the COW and indirect collaterals via leptomeningeal-cortical branch anastomoses are of particular importance during acute occlusions [1]. Patients with adequate collaterals may not develop neurological deficits and stroke, which is why current management is to not intervene as risks of shower emboli and subsequent iatrogenic stroke, symptomatic hemorrhage, and mortality may

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outweigh the potential benefits of intervention. However, many patients with acute internal carotid artery (ICA) occlusion have a large penumbra, with the potential to fail secondary to compromised perfusion resulting in infarcts associated with high morbidity and mortality [2].

In this study, we present a series of patients who had isolated acute cICA occlusion and normal neurological exam at risk for neurologic deterioration. It is important to note that these patients presented with ischemic penumbra and are at risk for stroke, but infarction of brain parenchyma has not yet occurred. Standard protocol at our institution is no intervention if the patient is neurologically intact and no mismatch is observed on CT perfusion studies. In the case of perfusion mismatch, surgical intervention was offered. The management of these challenging situations is discussed and a revascularization technique to minimize the risk of distal embolization is described.

2. Patients and methods

2.1. Study cohort

Patients with isolated acute cICA occlusion and neurological deficits that promptly return to baseline shortly before/after admission or prior to surgery that required emergent revascularization due to risk of progression to stroke were included in our analysis. Patient 1 was treated according to standard protocol, while patients 2 and 3 were treated according to the discussed management. The study protocol was reviewed and approved by the Institutional Review Board. Informed consent process was waived due to the retrospective design of this study.

2.2. Management and revascularization technique

Unenhanced CT was performed followed by CTP and CTA to assess perfusion and identify the site of vascular occlusion, respectively. CTP studies at presentation were evaluated for potential indications of perfusion failure based on MTT, T<sub>max</sub>, CBF, and CBV measurements. These patients were monitored in the ICU for neurologic deterioration and given the option of intervention if mismatch (MTT ≥ 200% or T<sub>max</sub> > 6 s) was noted on CT perfusion imaging.

Proximal flow arrest mechanical thrombectomy (MT) was performed on patients requiring emergent intervention. A balloon guiding catheter (BGC) flowgate 2 was placed proximal to the occlusion (mostly in the common carotid artery) and used to prevent distal showering by flow arrest. Using ACE60 catheter Penumbra over a XT27 over a synchro wire, the synchro wire was advanced. With flow arrested via flowgate balloon, the XT27 was advanced on heparinized drip followed by the ACE60 catheter with simultaneous suctioning from ACE60 catheter. If ACE 60 catheter could not be advanced at the origin of the ICA, balloon angioplasty was carried out. With suctioning applied, the clot was tracked intracranially up to bifurcation, Once flow was established, the XT27 and wire were pulled out and ACE60 catheter retracted slowly to ensure clot removal up to the flowgate catheter tip. After suctioning for 20 s and visualizing flow reversal, the balloon was deflated and antegrade ICA flow established. If severe carotid stenosis was observed, angioplasty with a distal protection device or stent placement was performed via standard techniques.

3. Results

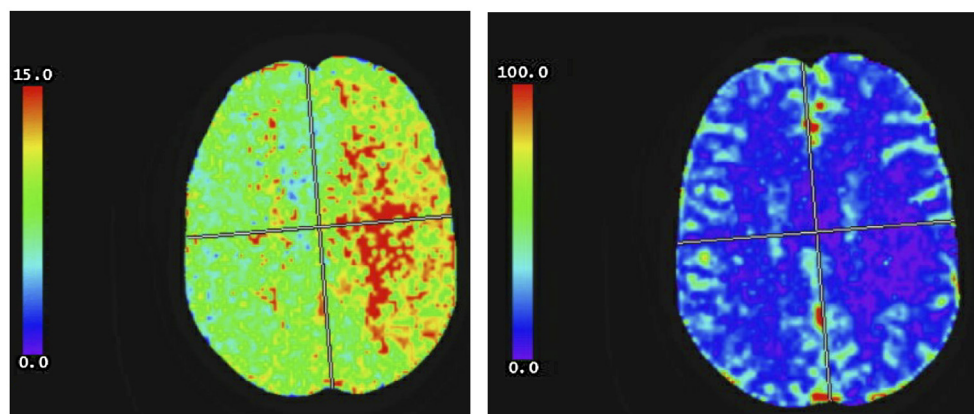
Patient summaries can also be seen in Table 1.

3.1. Patient 1

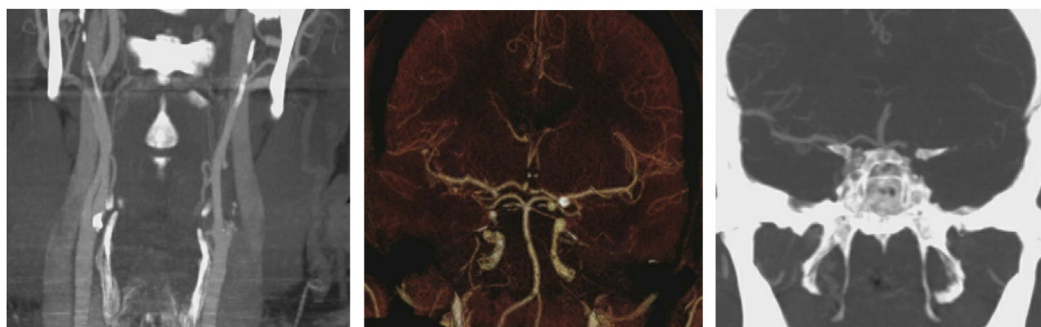
A 54-year-old patient presented with right-sided weakness and loss of sensation NIHSS 9. He had returned to baseline prior to arriving to the emergency department. CTP imaging (Fig. 1) showed MTT

Table 1  
Summaries of patient clinical courses.

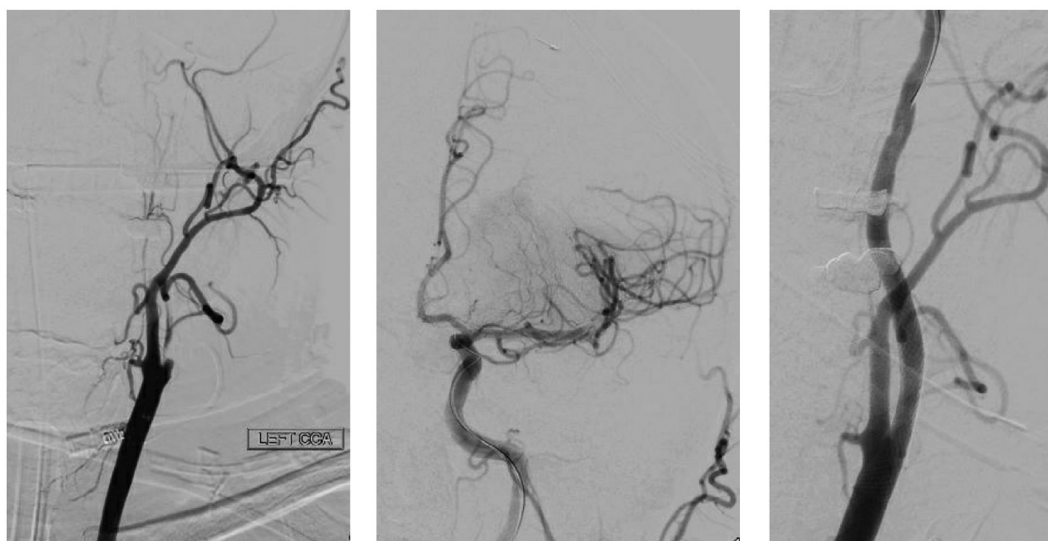
Patient No	Clinical Description	NIH at presentation	Later At ED	Outcome	CTA	CTP	TICI	Outcome	Age
1	Patient presented with right-sided weakness and loss of sensation, but returned to baseline prior to arriving to the emergency department. He was initially observed in the intensive care unit with permissive hypertension according to standard protocol, but deteriorated neurologically 8 h later	9	0	6	Isolated left cICA occlusion at presentation but progressed to MCA M1 branch on repeat CTA (after neurological deterioration in ICU)	MTT asymmetry of approximately 2	3	Severe right hemiparesis	54
2	Patient presented with intermittent transient vision loss in his right eye over the last few weeks	Vision loss	0	0	Isolated right cICA occlusion	Mismatch	3	Neurologically intact (mRS 0)	67
3	Patient with past medical history of atrial fibrillation initially presented with right upper extremity weakness and word finding difficulties. In the emergency department he did experience complete resolution of symptoms at times	8	0	0	Isolated left cICA occlusion	Mismatch	3	Neurologically intact (mRS 0)	80



**Fig. 1.** CTP imaging. (A) MTT – asymmetry observed; (B) CBF.



**Fig. 2.** CTA. (A) Left cICA occlusion on initial CTA; (B) No intracranial occlusion present; (C) Left ICA and M1 branch occlusion 8 h later.



**Fig. 3.** Emergent revascularization. (A) Left cICA occlusion; (B) Left ICA recanalization; (C) Left cICA stent.

asymmetry of approximately 2. Acute left cICA occlusion was noted on the initial CTA (Fig. 2A). He was initially observed in the intensive care unit with permissive hypertension according to standard protocol but deteriorated neurologically 8 h later. Repeat CTA (Fig. 2C) revealed progressive occlusion of the left middle cerebral artery (MCA) M1 branch; he was taken emergently for successful mechanical thrombectomy and carotid revascularization (Fig. 3). Recanalization TICI 3 was achieved but was left with severe right hemiparesis with partial improvement at time of discharge.

### 3.2. Patient 2

A 67-year-old male presented to emergency department due to a 2 week history of intermittent vision loss in his right eye that promptly returned to normal. The patient experienced 3 episodes of temporary vision loss prior to seeking medical attention. He was noted to have right cICA occlusion on CTA, and CTP showed decreased CBF and increased MTT (Fig. 4). The patient was taken emergently for revascularization and stenting (Fig. 5). The patient remained neurologically intact.



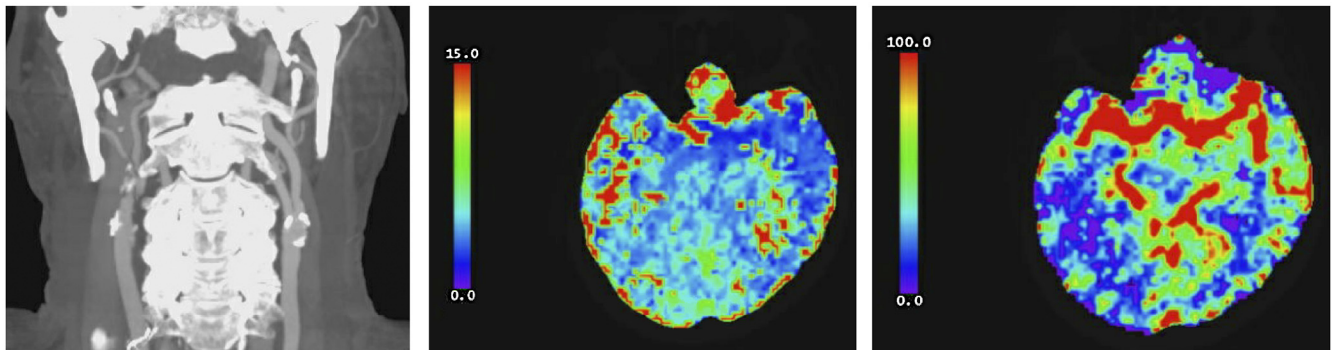


Fig. 4. (A) CTA – right cICA occlusion; (B) CTP – increased MTT; (C) CTP – decreased CBF.

### 3.3. Patient 3

An 80-year-old male with past medical history of atrial fibrillation initially presented with right upper extremity weakness and word finding difficulties NIHSS 8. In the emergency department, the patient experienced complete resolution of his symptoms. CTA revealed left cICA occlusion, with obvious delayed perfusion on CTP (Fig. 6). He was taken emergently for recanalization (Fig. 7) and remained neurologically intact post-operatively.

## 4. Discussion

In the present study, we aimed to demonstrate a potential role of CTP and EVT in the management of patients with acute cICA occlusion presenting with normal neurological exam after a period of neurological deficits. These patients are at risk for progressive neurologic deterioration but have not yet progressed to stroke. The role of EVT in acute ICA occlusion is unclear and current guidelines recommend MT only for patients with NIHSS 6 or higher [3,4], yet studies have shown better functional outcomes at 3 months in patients with large vessel occlusion (LVO) and low NIHSS treated with MT compared to best medical therapy (BMT) [5]. Current general criteria for MT require all of the following: neuroimaging displays a small or no infarct core and excludes hemorrhage, proximal LVO in the anterior circulation noted on angiography, persistent and disabling neurologic deficits, and intervention can be started within 24 h of last known normal. Common exclusion criteria for treatment is low NIHSS or rapidly improving stroke symptoms (RISS) [6], yet patients with acute ICA occlusion and the aforementioned clinical presentation are still at risk for collateral failure. This also holds true for patients with intracranial occlusion of the MCA. In our case series, patient 1 presented with right-sided weakness and loss of sensation before returning to baseline. Patient 1 was managed according to standard protocol and proceeded to

neurologically deteriorate. Despite TICI 3 recanalization, progression to stroke left patient 1 with severe residual hemiparesis. Patients 2 and 3 also presented with neurological deficits symptoms that included complete resolution within the emergency department. CTP of patients 2 and 3 revealed obvious mismatch indicating ischemic penumbra but had not yet progressed to infarction, and they were taken emergently for revascularization. Both patients 2 and 3 were discharged neurologically intact.

Clinical presentations of patients with acute ICA occlusion range from asymptomatic to transient ischemic attacks to severe stroke dependent on vascular factors such as collateral flow, autoregulation, hemodynamic alterations, and occlusive patterns [1]. Cerebral blood flow (CBF) is regulated by metabolic demands of the brain, which vary by region and with neuronal activity [7]. It is dependent on cerebrovascular resistance, cerebral perfusion pressure (CPP), and autoregulation. Cerebrovascular autoregulation maintains perfusion at a relatively constant rate within mean arterial pressures (MAP) ranging from approximately 50–170 mmHg. Autoregulatory mechanisms involve neurogenic, myogenic, and metabolic regulations in response to alterations in CPP and CBF. In the context of acute ICA occlusion, direct collaterals immediately provide antegrade flow distal to the occlusion assuming an intact COW (subject to substantial anatomic variability). CPP decreases distal to the ICA occlusion establishing a pressure gradient across the total vascular bed resulting in retrograde flow via leptomeningeal anastomoses with cortical branches (indirect collaterals). The decrease in intraluminal pressure initially causes vasodilation and decreased vascular resistance via smooth muscle relaxation, which further promotes collateral blood flow to ischemic parenchyma provided systolic blood pressure (SBP) is maintained at 50 mmHg or greater. Small cortical arterioles (microcirculation) are primarily responsible for these variations in cerebrovascular resistance [2].

Collateral or hemodynamic failure, which may result in a significant

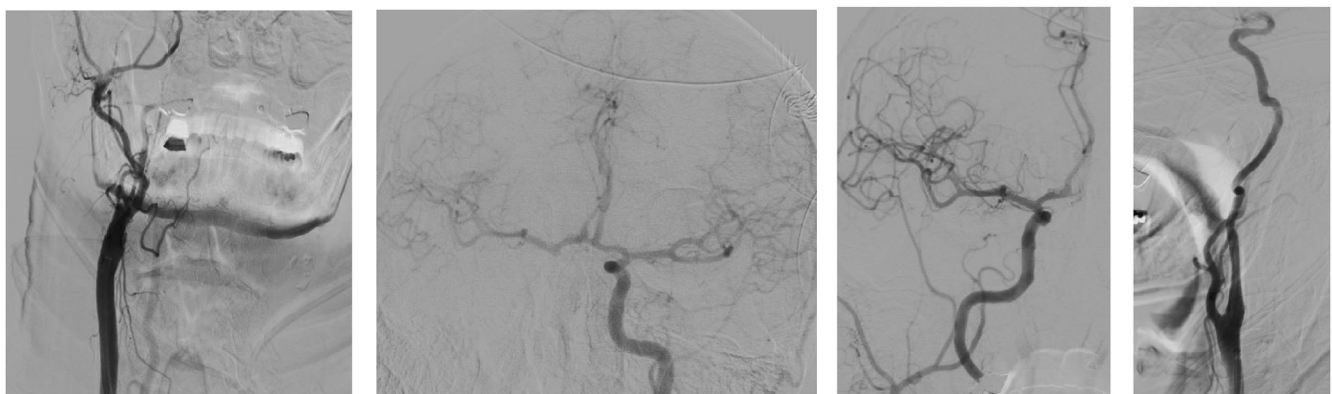


Fig. 5. Emergent revascularization. (A) Right ICA with no antegrade flow; (B) Left ICA with cross flow; (C) Right ICA recanalization; (D) Right cICA stent.

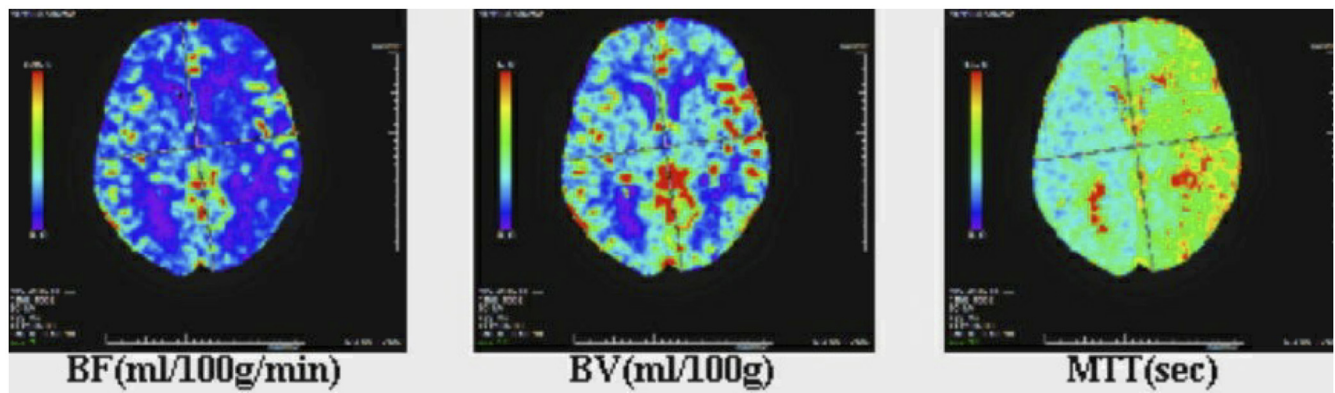


Fig. 6. CTP imaging with delayed perfusion metrics.

expansion of the infarct core and shrinking penumbra. One theory is that increased oxygen extraction by penumbral tissue leads to decreased oxygen delivery to distal leptomeningeal collaterals and further metabolic deterioration [8]. Ischemia also impairs cerebral autoregulatory mechanisms (vascular resistance, collateral flow) resulting in capillary dysfunction. Microvascular endothelium and perimicrovascular glia swell in response to ischemia causing the microvascular lumen to significantly narrow increasing the vascular resistance that the perfusion pressure must overcome [9]. Edema secondary to cerebral ischemia may increase ICP, which further increases resistance to collateral flow. Taking into account the decreased perfusion of tissue distal to the site of occlusion and several factors contributing to cerebrovascular resistance, it seems intuitive that collaterally perfused penumbra is highly susceptible to hypotension and factors affecting cardiac output. In other words, cerebral perfusion in patients with ICA occlusion has increased dependence on blood pressure due to impaired autoregulatory mechanisms [10]. Permissive hypertension has been shown to increase leptomeningeal collateral flow improving penumbra oxygen metabolism and resulting in smaller infarct volumes.

Collateral flow plays a significant role in determining final infarct volume and functional outcome. Poor baseline collateral flow is associated with accelerated infarct growth or infarcts that have already progressed into the majority of the occluded vessel's territory, while patients with robust baseline collaterals have reduced initial infarct volume, lower NIHSS at presentation, and less infarct growth within ischemic penumbra [7,11,12]. Robust collaterals may decrease the rate at which ischemic penumbra metabolically deteriorates, but collateral flow is dynamic and can fail over time [2,12]. A study published in 2013 found that collateral deterioration was strongly related to increased absolute ( $P = 0.02$ ) and relative infarct growth ( $P < 0.0001$ ). Although robust collaterals at baseline enable the survival of ischemic penumbra, robust collaterals combined with large perfusion mismatch indicate the potential for significant infarct growth [12].

Information regarding collateral status and cerebral blood flow (CBF) may be obtained via various angiographic and perfusion imaging techniques [2,11,13]. Computed tomographic angiography (CTA) may be used to identify the location of vascular occlusion and to accurately visualize collateral patency, although it only provides a snapshot in time [7,12]. Robust collaterals could be observed on CTA early post-occlusion, but progressively fail to sustain perfusion over time [2]. CTA also does not accurately visualize salvageable tissue at risk of infarction. Only measurements of tissue perfusion can assess the effectiveness of collateral flow [7]. Computed tomographic perfusion (CTP) imaging creates cerebral perfusion maps displaying variables such as mean transit time (MTT), cerebral blood volume (CBV), and CBF, which may be used to predict tissue outcome [14]. CTP is able to distinguish critically hypoperfused parenchyma that will inevitably progress to infarction (ischemic core) from sub-critically hypoperfused, functionally suppressed parenchyma that is potentially salvageable (penumbra) from non-critically reduced flow of metabolically stable regions (benign oligemia) [2,7]. Previous studies have found that MTT and  $T_{max}$  (delay time without delay correction) may accurately identify at risk ischemic penumbra, while infarct core threshold may be obtained via CBF or CBV measurement [14].

CTP imaging identifying penumbra and delayed MTT indicate potential collateral failure to sustain effective parenchymal perfusion. The standardization of perfusion imaging selection criteria may help identify patients at risk for stroke due to collateral failure with potential to benefit from intervention. For example, patients with MTT of 200% or greater may be at increased risk of hemispheric stroke [14]. A 2015 study on acute stroke patients with ICA occlusion used CTP imaging to select patients for emergent intervention due to risk of hemisphere stroke. It found that ideal patients should have infarct core size less than 50 cc, total hypoperfusion area (defined as  $T_{max}$  greater than 10 s delay) of 100 cc or less, ischemic penumbra (mismatch volume) of 15 mL or greater, and mismatch ratio (total hypoperfusion:core) greater



Fig. 7. (A) Left cICA occlusion pre-operatively; (B) Left ICA balloon inflated with simultaneous suction via ACE60 catheter penumbra; (C) Left ICA recanalization.

than 1.8 on imaging. However, this study was conducted on patients presenting with severe neurological deficits and has a very limited sample size [15].

Current evidence suggests that initial stroke management should be to determine tissue viability based on collateral status via non-invasive imaging and treatment tailored accordingly [7]. Collateral flow is independently associated with and has more influence on final infarct volume than time from symptom onset to treatment [11]. A study published in 2016 found that patients had improved outcomes with higher collateral grades despite longer times from last known well to intervention [16]. Perfusion mismatch, the difference between penumbra (MTT) and infarct core (CBV), has also been shown to be an independent prognostic factor [17]. Even in the presence of robust collateral flow, high grade reperfusion is associated with improved outcomes after EVT [2,16]. Information obtained via CTP imaging may be used to identify patients at risk for stroke and significantly extend the time window for recanalization to salvage ischemic penumbra [7,18].

Whether MTT or  $T_{max}$  maps display the optimal penumbra threshold is uncertain. A study published in 2016 concluded that whole brain CT perfusion via multidetector row CT scanners is just as accurate in measuring ischemic penumbra and core as MR imaging when using the following thresholds:  $T_{max}$  greater than 6 s or MTT greater than 200%, and CBF less than 25% or CBV less than 55%. It also found that  $T_{max}$  greater than 6 s is the optimal penumbra threshold predicting infarction and neurologic deterioration in the absence of reperfusion suggesting that intervention would be beneficial in this patient subgroup [14]. Infarction risk increases with increasing degrees of  $T_{max}$  delay, which has been hypothesized to indicate tissue further along the collateral flow pathway with increased vulnerability to reductions in collateral flow [12,14]. Repeated perfusion imaging may provide further insight into infarct evolution, mechanisms regulating collateral flow, and additional indicators of collateral failure [12].

CT perfusion can be a valuable tool to determine patients at risk for stroke and prevent large stroke in patients with ICA occlusion and normal neurological exam. In patients at risk for stroke and requiring intervention, a major concern is distal embolization to previously unaffected territories further reducing collateral flow to ischemic penumbra and worsening clinical outcome. Aspiration with temporary flow arrest has been shown to increase suction efficacy and to decrease the probability of shower emboli [19].

The current study is limited by the small number of patients. The study was intended to demonstrate the potential role of CT perfusion imaging and EVT in the management of patients with acute cICA occlusion and normal neurological exam after experiencing neurologic deficits in addition to discussing a revascularization technique to decrease the risk of distal embolization in patients requiring emergent revascularization.

## 5. Conclusion

Patients with acute ICA occlusion experiencing neurological deficits that promptly return to baseline, but have not yet experienced stroke, can progress to suffer large strokes due to flow failure or clot propagation. We suggest obtaining CTA/CT perfusion studies in all patients to determine collateral status in order to tailor treatment on a case by case basis. These patients should be monitored in the ICU for neurologic deterioration and given the option of intervention if mismatch is noted on CT perfusion imaging. It seems that  $MTT \geq 200\%$  or  $T_{max} > 6$  s is the optimal penumbra threshold predicting infarction and neurologic deterioration. In patients undergoing intervention, we suggest a technique using proximal flow arrest to minimize risk of shower emboli. Further studies are needed to more accurately assess the role of CTP and the efficacy and safety of EVT in this patient population.

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**Data sharing statement:** The relevant anonymized patient level data are available on reasonable request from the authors.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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