Chronic innominate artery occlusion with hyperacute intracranial thromboembolism: Revascularization with simultaneous local thromboaspiration and mechanical thrombectomy

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ABSTRACT

Chronic innominate artery occlusion with acute right internal carotid terminus thromboembolism and successful revascularization using simultaneous local thromboaspiration and mechanical thrombectomy has not been previously described. A 51-year-old male presented with transient left hemiparesis. A CT angiogram of the head and neck demonstrated chronic occlusion of the right innominate artery with no intracranial thromboembolism. More profound symptoms recurred twelve hours after admission. A diagnostic catheter-based angiogram confirmed occlusion of the innominate artery and identified hyper-acute right carotid terminus thromboembolism. Angioplasty of the innominate artery was followed by simultaneous mechanical and aspiration thrombectomy of the right internal carotid artery terminus. Combination local thromboaspiration and mechanical thrombectomy was shown in this case to be effective in achieving a favorable clinical outcome.

CASE REPORT

A 51-year-old male presented to the emergency room with left-sided weakness and right facial droop. Admission NIHSS (National Institute of Health Stroke Scale) score was 13 and blood pressure taken from the left arm was 154/79 mmHg with a pulse rate of 67 beats per minute. Past medical history was significant for a 20 pack year history of smoking and was otherwise unremarkable. There was no prior history of right upper arm claudication, syncope, vertigo, or visual impairment. Symptoms occurred with no prior activity of his right upper extremity.

A non-contrast head computerized tomographic (CT) examination identified no intracranial hemorrhage or early changes of acute ischemic injury (Fig. 1A). A CT angiogram (CTA) of the head and neck after initial clinical evaluation demonstrated no carotid bifurcation stenosis or intracranial large vessel thromboembolic occlusion (Fig. 1 B,C). A brain MRI examination with evaluation of diffusion and apparent diffusion coefficient (ADC) sequences demonstrated no acute brain parenchymal infarction (Fig. 1 D,E,F). The CTA evaluation did however demonstrate occlusion of the proximal right innominate artery (IA) with opacification of a diminutive right cervical internal carotid artery (ICA) relative to the contralateral side. The non-dominant right cervical vertebral artery (VA) and the right subclavian artery (SA) were patent. Increased right hemispheric cortical branch vascularity relative to the left was identified. Diminished caliber of the right common carotid artery (CCA) and right ICA with increased
ipsilateral hemispheric cortical branch vessels suggested chronic IA artery occlusion with right hemispheric pial collaterals (Fig. 2 A, B and C). No CTA findings compatible with fibromuscular dysplasia (FMD) or vasculitis was noted. No cervical ribs were identified. A CT perfusion study was not performed as no intracranial thromboembolic occlusion was identified by CTA. The Electroencephalography (EEG) study and the serum coagulation profile were normal.

Neurologic symptoms resolved within five hours after onset, compatible with a transient ischemic attack. The patient re developed acute left upper and lower extremity weakness with mental status change twelve hours after his admission. Hydrochlorothiazide (12.5 milligrams) as well as a nicotine patch (21 mg per 24 hour) had been given by his primary care caregiver. Left arm blood pressure had transiently dropped to 92/64 mm Hg with a pulse rate of 60 beats per minute. An emergent catheter based angiogram was requested for by the attending vascular surgeon to definitively evaluate the aortic arch and IA prior to surgical intervention. Due to the patient's rapid clinical decline no repeat CTA, MRA or CT perfusion imaging was obtained. Angiographic evaluation of the arch demonstrated occlusion of the proximal IA with delayed retrograde filling of the right cervical VA and post stenotic IA. Via retrograde filling within the post stenotic distal IA, delayed ante grade flow within the right CCA with distal occlusion of the visualized right ICA was identified (Fig. 3 A and B). To evaluate the delayed filling and distal occlusion, a 5 French JB2 diagnostic catheter in tandem with a stiff Storq Standard 300 cm .035-inch (Cordis Corporation, Miami Lakes, FL) guidewire was advanced through the IA occlusion into the right CCA. Digital subtraction angiography (DSA) of the right CCA revealed carotid terminus (Carotid-T) occlusion with no right hemispheric intracranial flow (Fig. 3C). No catheter based angiographic findings compatible with FMD (Fibromuscular disease) or vasculitis was identified.

Angioplasty of the IA stenosis was initially performed using a 9 mm x 40 mm over the wire EverCross angioplasty balloon (EV3 Inc., Plymouth, MN) (Fig. 4A). Mechanical and combined aspiration thrombectomy was preceded by intra- arterial administration of 10 mg tPA (tissue plasminogen activator). A 054 Reperfusion Catheter (Penumbra Inc., Alameda, CA) was navigated to the petrous segment of the right ICA (Fig. 4B). A 6 mm x 20 mm Solitaire FR stent retrieval system (EV 3, Plymouth, MN) was deployed within the distal right M1 MCA trunk (Fig. 4B). The stent retrieval system was proximally withdrawn with simultaneous aspiration through the 054 Reperfusion Catheter using the Penumbra aspiration pump. Retrieval of the stent system was proximally withdrawn with simultaneous thromboaspiration and mechanical thrombectomy was preceded by intra-arterial administration of 10 mg tPA (tissue plasminogen activator).

Imaging Findings
The right SA and right CCA arise from the IA in 99% of cases. With proximal IA occlusion, there is retrograde flow from the right VA into the right SA, referred to as "steal phenomenon". Flow then occurs retrograde through the distal IA and then ante grade into the right CCA. This alteration in blood flow, results in chronic reduced perfusion to the right cerebral hemisphere. A case report identifying this phenomenon has been shown with MR angiogram and perfusion imaging [5]. In our patient, CT angiogram findings of chronic IA stenosis was evidenced by the diminution in caliber of the right carotid system reflecting diminished wall shear stress from decreased flow and increased cortical branch vascularity of the right cerebral hemisphere, compatible with pial collaterals.

### DISCUSSION

Right MCA acute thromboembolic stroke from occlusion of the IA with unsuccessful revascularization and a poor patient outcome has been reported [1]. Right carotid-T thromboembolism (intracranial carotid bifurcation occlusion with involvement of A1 and M1 segments) from IA steno-occlusive disease with successful endovascular intervention and complete clinical recovery has not been reported. Carotid-T occlusions have poor clinical outcomes even after endovascular intervention [2]. Simultaneous thromboaspiration and mechanical thrombectomy may additionally improve recanalisation rates, particularly for carotid-T occlusions.

### Etiology & Clinical Findings
Extracranial carotid artery disease accounts for 15-20% of all ischemic strokes, the majority as a result of carotid bifurcation disease. The gender prediction is not known for innominate stenosis. There is a slight male preponderance for extra-cranial carotid bifurcation disease that is often identified with innominate stenosis. In a study of 94 patients with innominate artery stenosis, the mean age was 62 [3].

In an extensive angiographic study, IA steno-occlusive disease was identified in only 4% of patients [4]. This may or may not present with posterior circulation and upper extremity ischemic symptoms. Dizziness, visual impairment, vertigo and syncope are the most common neurologic symptoms. Diminished or absent upper extremity pulses ipsilateral to the stenosis and asymmetric blood pressures are the most consistent clinical findings in symptomatic patients. Anterior circulation ischemic symptom in subclavian artery (SA) stenosis is more likely consequent to extracranial carotid bifurcation disease [5]. With IA stenosis, right hemispheric ischemic symptoms may occur.
IA steno-occlusive disease is distinct from subclavian steal as it affects the anterior and posterior circulations [5]. Patient habitus and anatomy significantly effects imaging assessment of the vertebral and subclavian arteries. Sonographic evaluation of the IA is limited by overlying ribs and tracheal shadowing. In subclavian steal or significant IA stenosis, altered vertebral artery waveforms are seen only when the degree of stenosis is greater than 60%. Permanent flow reversal can be seen when a SA or IA stenosis is greater than 75% [5]. Ultrasound can be helpful in determining MCA occlusion; however this is not frequently performed.

MR Angiography with 3D gadolinium enhanced technique requires a shorter acquisition time, limiting motion artifact and is useful for determining IA stenosis. 2D TOF (Time of flight) technique provides information about the direction of flow in the vertebral arteries. If enhancement is present in a VA on gadolinium-enhanced MRA, but no corresponding flow-related enhancement is seen on TOF MRA, then flow in that vessel must be reversed. CTA can identify stenosis of the IA but does not evaluate VA flow reversal. Chronic IA occlusion can be determined by extra-cranial carotid artery caliber and right hemispheric pial collaterals. CT and MR brain perfusion with evaluation of cerebral blood flow, cerebral blood volume and mean transit time maps can identify hypoperfusion within the right cerebral hemisphere with significant IA stenosis. These perfusion parameters can also be used to determine a viable ischemic penumbra in the instance of carotid-T and or MCA occlusion. Digital Subtraction angiography (DSA) remains the reference standard for diagnosing and characterizing IA stenosis and steal phenomena as well as intracranial thromboembolic occlusion. This modality also provides opportunity for endovascular intervention [5].

**Differential Diagnosis**

Anterior circulation strokes from IA thrombus formation due to non-atherosclerotic disease have been reported in a few case reports [6,7,8, 9]. The diagnostic differential of IA disease with embolic phenomenon includes cervical ribs, dissection, nephrotic syndrome hypercoagulability, thoracic mural thrombus syndrome (TMT), FMD and vasculitis. Prolonged IA compression at the thoracic outlet due to a cervical rib results in fibrotic and inflammatory change causing platelet aggregation, mural thrombus formation and macro embolism [6]. Ischemic stroke from supra-aortic artery dissection accounts for 10-25% of patients under the age of fifty and is spontaneous in 60% of cases. Isolated dissection of the IA is extremely rare and is more frequently associated with residual dissection following repair of acute type A aortic dissections [7]. If IA dissection is present urgent CTA and trans-esophageal echocardiography should be performed to exclude aortic dissection. In hypercoagulable states arterial thrombosis is rare and assessment of hypoalbuminemia in nephrotic syndrome may be useful in identifying at risk patients that may benefit from early anticoagulation treatment [8]. TMT syndrome occurs in healthy young people with risk factors that include smoking and a family history of atherosclerotic disease and is characterized by thrombus formation in the aortic arch and branch vessels [9]. FMD is a non-atherosclerotic and non-inflammatory vasculopathy that most frequently affects renal and carotid arteries and can involve any artery [10]. IA stenosis is also recognized in other entities such as radiation and Takayasu's arteritis with ancillary findings [11]. The natural history of radiation arteritis of larger vessels spans many decades, with development of thrombosis within the first five years, fibrotic lesions in the first decade, and lesions resembling atherosclerotic disease after the second decade [12]. Takayasu's arteritis is characterized by idiopathic inflammation of the aortic arch, pulmonary, coronary, and cerebral vessels. This presents typically in females in the second to fourth decades with stroke, transient ischemic attack, myocardial infarction, and upper extremity claudication [13].

**Treatment & Prognosis**

Percutaneous transluminal angioplasty of the brachiocephalic vessels with or without stenting is a viable alternative to surgery with lower peri-procedural complications [14]. Angioplasty alone has a high failure rate. With stent placement clinical outcomes are equivalent to surgery [15]. In the acute stroke setting, IA angioplasty without stent placement as performed in the case presented is favored, as it would mitigate the requirement of immediate post procedural anti-platelet medications. With an established infarction anti-platelet agents would increase the risk for hemorrhagic transformation. Avoiding stent placement also facilitates delivery of mechanical thrombectomy devices into the right carotid system without hindrance. Stenting can be performed electively after clinical recovery.

Risk of procedural thromboembolism into the VA and ICA for extra-cranial vascular stent angioplasty has been addressed with placement of distal protection devices [16]. This would be a moot point for the already occluded right CCA in the acute setting. The value of embolic protection within the vertebral artery is doubtful especially in the presence of flow reversal, as was present in our patient. There is a significant delay in establishment of antegrade flow after stent angioplasty that ranges from 20 seconds up to a few minutes [17], diminishing the risk of thromboembolism to the posterior circulation.

The self-expanding retrievable and reusable Solitaire stent retriever device mechanically engages the thrombus within its stent struts. The device with the thrombus is then retracted with or without flow arrest from a balloon tip guide catheter. A recent systematic review of the effectiveness of the Solitaire stent retriever in acute intracranial stroke revealed an 89.7% recanalization rate with a procedure induced complication rate of 3.4% [18]. Concurrent use of the stent retrieval device before and after adjunctive measures that include pharmacologic intra-arterial thrombolysis, aspiration thrombectomy with Penumbra Reperfusion catheters and stent deployment or angioplasty was attempted in 21% of cases in the Solitaire systematic review [18]. The Penumbra Pivotal Stroke Trial demonstrated an 81.6% intracranial vessel recanalization rate and a cerebral hemorrhage rate of 11.2% using only Penumbra Reperfusion catheters [19].

Carotid-T occlusions have a large clot burden with a dismal clinical outcome [2]. Even after endovascular
recanalization, only 21.4% achieve functional independence [20]. Direct large caliber catheter aspiration of the internal carotid artery was independently used in one study [21], however for successful recanalization more than one device was used in 50% of patients in another [20]. Simultaneous use of the stentretriever and aspiration catheter has been referred to as the “Solumbra” technique and has no published data to attest to its effectiveness. Clinical outcomes have been shown to improve after angiographic recanalization [22]. Combination endovascular therapy for intracranial thromboembolic stroke as compared to standard intravenous tPA alone, showed no difference with no increased benefit in clinical outcomes in a randomized controlled study. This study was not inclusive of newer stentretriever technology in the endovascular therapy arm. In the post hoc analysis of this study carotid T occlusions with M1 MCA occlusions showed greater recanalization and better outcomes with use of combined IV tPA and endovascular therapy [23].

**TEACHING POINT**

Innominate artery occlusive stenosis with acute carotid-T thromboembolic occlusion requires early catheter based angiographic evaluation for endovascular intervention. Revascularization using aspiration and mechanical thrombectomy at the carotid terminus simultaneously preceded by innominate artery angioplasty is effective in establishing a favorable clinical outcome.

**REFERENCES**


18. Koh JS, Lee SJ, Ryu CW, Kim HS. Safety and Efficacy of Mechanical Thrombectomy with Solitaire Stent Retrieval for
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Figure 1: A 51-year-old male presented with acute transient left sided weakness. Chronic innominate artery steno-occlusive disease was identified by initial CT angiography without intracranial thromboembolic occlusion. Early recurrence and worsening of left sided weakness prompted a catheter based angiographic study, identifying hyperacute thromboembolic occlusion at the right carotid terminus. This was retrieved using combined local thromboaspiration and mechanical thrombectomy preceded by innominate artery angioplasty.

Findings: A: Select axial non contrast head CT image slice shows no attenuation change compatible with right hemispheric infarction. No dense right middle cerebral artery sign compatible with thrombus is identified (black arrow). B: Maximum intensity projection CTA shows patent right carotid terminus (thick black arrow) and right proximal middle cerebral artery (thin black arrow). C: Curved planar sagittal reformat CTA demonstrates a normal caliber and contour of the right distal common carotid artery, carotid bifurcation and internal carotid artery. D, E and F: Select axial FLAIR (D), axial diffusion (E) and axial apparent diffusion coefficient (ADC) mapped sequence (F) slices show no signal change compatible with right hemispheric infarction.

Technique: A: Axial CT mA330, 140 kV, Slice thickness 2.5mm. B: Axial CT, 480mAs, 140Kv, Slice thickness 10mm, 75 ml Isovue 370 injection at 4ml/second. C: Axial CT, 480mAs, 140Kv, Slice thickness 0.63, 75 ml Isovue 370 injection at 4ml/second. D: Axial FLAIR TR 8000 TE 128.90, Slice thickness 5mm. E: Axial Diffusion, TR8600 TE80.10, Slice thickness 5mm. F: Axial ADC Axial Diffusion, TR8600 TE80.10, Slice thickness 5mm.
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Figure 2: A 51-year-old male presented with acute transient left sided weakness. Chronic innominate artery steno-occlusive disease was identified by initial CT angiography without intracranial thromboembolic occlusion. Early recurrence and worsening of left sided weakness prompted a catheter based angiographic study, identifying hyperacute thromboembolic occlusion at the right carotid terminus. This was retrieved using combined local thromboaspiration and mechanical thrombectomy preceded by innominate artery angioplasty.

Findings: Axial contrast enhanced CTA of the head and neck; A: At the level of the supra-aortic branch vessels, occlusion of the innominate artery (large solid arrow). Origin of the left vertebral artery from the aortic arch (dotted arrow). B: At the level of the C1 vertebral body, marked diminution in caliber of the right internal carotid artery (solid arrow), relative to the left (dotted arrow). C: At the level of the centrum semi ovale, increased cortical branch vessels on the right (solid arrow), relative to the left (dotted arrow) compatible with pial collaterals.

Technique: Axial CT, 480mAs, 140kV, Slice thickness 0.63mm, 75 ml Isovue 370 injection at 4ml/second.

Figure 3: A 51-year-old male presented with acute transient left sided weakness. Chronic innominate artery steno-occlusive disease was identified by initial CT angiography without intracranial thromboembolic occlusion. Early recurrence and worsening of left sided weakness prompted a catheter based angiographic study, identifying hyperacute thromboembolic occlusion at the right carotid terminus. This was retrieved using combined local thromboaspiration and mechanical thrombectomy preceded by innominate artery angioplasty.

Findings: A: Aortic arch angiography shows occlusion of the innominate artery (long arrow) and retrograde flow within the right vertebral artery (short arrow). B: Aortic arch angiography demonstrates delayed retrograde distal innominate artery flow (dotted arrow) and subsequent anti-grade flow into the right common carotid artery and internal carotid artery (long arrow) with distal occlusion (double striped arrow). C: Angiography of the right common carotid artery (short arrow) after traversing the innominate artery stenosis with a stiff guide wire shows absent flow within the intracranial segment of the right internal carotid artery compatible with carotid-T thromboembolic occlusion (long arrow).

Technique: A, B: Diagnostic 5French catheter injection of 30ml of Isovue 300 at 25cc/sec. C: Diagnostic 5F catheter injection of 6 of Isovue 300 at 4cc/sec.
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Figure 4: A 51-year-old male presented with acute transient left sided weakness. Chronic innominate artery stenotic-occlusive disease was identified by initial CT angiography without intracranial thromboembolic occlusion. Early recurrence and worsening of left sided weakness prompted a catheter based angiographic study, identifying hyperacute thromboembolic occlusion at the right carotid terminus. This was retrieved using combined local thromboaspiration and mechanical thrombectomy preceded by innominate artery angioplasty.

Findings: A: Fluoroscopic spot view shows an angioplasty balloon across the focal innominate artery stenosis (white arrow), performed prior to mechanical and aspiration thrombectomy of the carotid terminus and right middle cerebra artery. B: Road map image over the right frontal calvarium show the Solitaire stent retriever within the right middle cerebral artery (thin arrow) and the position of the 054 Penumbra Reperfusion Catheter within the right internal carotid artery (thick arrow). C: DSA of the right internal carotid artery with complete resolution of the carotid-T and right middle cerebral artery occlusion (arrows). D: Aortic arch angiography in the left anterior oblique projection demonstrates residual stenosis at the innominate artery (thick arrow) and revascularization of the right carotid and right anterior circulation with symmetric flow relative to the contra-lateral side (thin arrow).

Technique: C: 054 Reperfusion Catheter (Penumbra Inc., Alameda, CA) catheter injection of 4ml of Isovue 300 at 4cc/sec for right internal carotid artery digital subtraction angiography. D: Diagnostic 5 French catheter injection of 30ml of Isovue 300 at 25cc/sec for aortic arch digital subtraction angiography.
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<table>
<thead>
<tr>
<th>Etiology</th>
<th>Atherosclerotic stenoocclusive disease of the IA is most common. Other causes of IA artery stenosis includes traumatic injury, dissection, Takayasu arteritis, Thoracic mural thrombus syndrome (TMT), Fibromuscular dysplasia, radiation induced arteriopathy, vasculitis and external compression from a cervical rib.</th>
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<tr>
<td>Incidence</td>
<td>IA atheromatous steno-occlusive disease has a reported incidence of 4%. A few case reports have described thromboembolism to the carotid terminus and right middle cerebral artery. Extracranial carotid artery disease accounts for 15-20% of all ischemic strokes, the majority as a result of carotid bifurcation disease.</td>
</tr>
<tr>
<td>Gender Ratio</td>
<td>Not known for IA stenosis. Slight male preponderance for extracranial carotid bifurcation disease that is often identified with innominate stenosis.</td>
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<td>Age predilection</td>
<td>Mean age 62.</td>
</tr>
<tr>
<td>Risk factors</td>
<td>Risk factors for atherosclerotic disease that includes hypertension, diabetes, smoking, genetic predisposition and sedentary life style.</td>
</tr>
<tr>
<td>Treatment</td>
<td>Percutaneous transluminal angioplasty of the brachiocephalic vessels with or without stenting is a viable alternative to surgery with lower peri-procedural complications. IV/IA tPA and endovascular intervention for intracranial thromboembolism.</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Carotid-T occlusions have a large clot burden with a dismal clinical outcome. Even after endovascular recanalization, only 21.4% achieve functional independence.</td>
</tr>
<tr>
<td><strong>Imaging Findings</strong></td>
<td></td>
</tr>
<tr>
<td>Ultrasound:</td>
<td>Patient habitus and anatomy significantly effects assessment of the vertebral and subclavian arteries. User dependent sensitivity and specificity. In subclavian steal or significant IA stenosis, altered VA waveforms are seen only when the degree of stenosis is greater than 60%; permanent flow reversal can be seen when a SA or IA stenosis is greater than 75%. Ultrasound can be helpful in determining MCA occlusion; however this is not frequently performed.</td>
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<td>MR Angiography:</td>
<td>3D gadolinium enhanced technique require shorter acquisition time, limiting motion artifact and is useful for determining IA stenosis. 2D TOF technique provides information about the direction of flow in the VA. If enhancement is present in a VA on gadolinium-enhanced MRA, but no corresponding flow-related enhancement is seen on TOF MRA, then flow in that vessel must be reversed. Occlusion of the ICA and or MCA can be easily determined.</td>
</tr>
<tr>
<td>CT Angiography:</td>
<td>CTA can show stenosis in the IA but does not evaluate VA flow reversal. Occlusion of the ICA and or MCA can be easily determined. Chronic IA occlusion can be determined by extracranial carotid artery caliber and right hemispheric pial collaterals.</td>
</tr>
<tr>
<td>CT and MR perfusion:</td>
<td>Cerebral blood flow, cerebral blood volume and mean transit time maps can identify hypoperfusion to the right cerebral hemisphere with significant IA stenosis. These parameters can also be used to determine a viable penumbra in the instance of carotid-T and or MCA occlusion.</td>
</tr>
<tr>
<td>Digital Subtraction angiography:</td>
<td>Reference standard for diagnosing and characterizing IA stenosis and steal phenomena. Provides opportunity for endovascular intervention.</td>
</tr>
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Table 1: Summary table for Innominate artery stenosis with thromboembolic Carotid-T occlusion.
<table>
<thead>
<tr>
<th></th>
<th>Ultrasound</th>
<th>CT/CTA</th>
<th>MR/MRA</th>
<th>Other pertinent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>External compression</strong>&lt;br&gt;(Cervical rib)</td>
<td>• Useful in detecting a cervical rib without ionizing radiation.</td>
<td>• Postural narrowing of the costoclavicular space with neurovascular compression at the thoracic outlet and identification of intraluminal thrombus.</td>
<td>• Postural narrowing of the costoclavicular space with neurovascular compression at the thoracic outlet and identification of intraluminal thrombus.</td>
<td>• Radiographs of both the cervical spine and chest acquired to identify bone abnormalities that may contribute neurovascular compression at the thoracic outlet. (e.g. cervical ribs, elongated C7 transverse process)</td>
</tr>
<tr>
<td><strong>Innominate Artery Dissection</strong></td>
<td>• Transthoracic echocardiography (TEE) for identification of a dissection of the innominate artery limited by overlying ribs and tracheal shadowing.</td>
<td>• Identification of a dissection flap, mural thrombus, intraluminal thrombus at the innominate artery and embolism within intracranial vasculature.</td>
<td>• Identification of a dissection flap, mural thrombus, intraluminal thrombus at the innominate artery and embolism within intracranial vasculature.</td>
<td>• Catheter angiography is the gold standard for diagnosis and provides access for endovascular therapy.</td>
</tr>
<tr>
<td><strong>Vasculitis</strong>&lt;br&gt;Takayasu’ arteritis and Radiation arteritis</td>
<td>• Homogenous circumferential thickening of affected vessels.</td>
<td>• Vessel wall thickening and enhancement in the acute phase.</td>
<td>• Vessel wall thickening with increased T2 signal and enhancement in the acute phase.</td>
<td>• Increased uptake in thickened vessel wall identified by PET (Positron Emission Tomography) in the acute phase.</td>
</tr>
<tr>
<td><strong>Fibromuscular dysplasia</strong>&lt;br&gt;(FMD)</td>
<td>• Segmental string-of-beads pattern with alternating regions of lumen narrowing and vessel dilatation distal to a completely normal segment of the vessel.</td>
<td>• Segmental string-of-beads pattern with alternating regions of lumen narrowing and vessel dilatation distal to a completely normal segment of the vessel.</td>
<td>• Segmental string-of-beads pattern with alternating regions of lumen narrowing and vessel dilatation distal to a completely normal segment of the vessel.</td>
<td>• Catheter angiography is gold standard for diagnosis.</td>
</tr>
<tr>
<td><strong>Thoracic mural thrombi syndrome</strong>&lt;br&gt;(TMT)</td>
<td>• Transthoracic echocardiography (TEE) for identification of thrombus within the innominate artery limited by overlying ribs and tracheal shadowing.</td>
<td>• Aortic and or supra-aortic focal thrombosis without underlying atherosclerotic disease.</td>
<td>• Aortic and or supra-aortic focal thrombosis without underlying atherosclerotic disease.</td>
<td>• Increased risk of thrombus embolization with catheter angiography</td>
</tr>
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</table>

**Table 2:** Differential diagnosis table for Innominate artery occlusion and Carotid-T thromboembolism.
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ABBREVIATIONS
CT = Computerized tomography
CTA = Computerized tomographic angiography
CAROTID-T = Carotid terminus
CCA = Common carotid artery
DSA = Digital subtraction angiography
EEG = Electroencephalography
FMD = Fibromuscular dysplasia
IA = Innominate artery
ICA = Internal carotid artery
IV = Intravenous
MCA = Middle cerebral artery
MRA = Magnetic resonance angiography
NIHSS = National Institute of Health Stroke Scale
SA = Subclavian artery
TEE = Transesophageal echocardiography
TOF = Time of flight
tPA = tissue plasminogen activator
VA = Vertebral artery

KEYWORDS
Carotid-T thromboembolism; Innominate artery occlusion; Middle cerebral artery thromboembolism; Thromboaspiration; Mechanical thrombectomy

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