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ENDOVASCULAR MANAGEMENT OF ACUTE TANDEM OCCLUSION STROKE

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Abstract:

Acute ischemic stroke caused by tandem occlusion is characterized by a blockage or severe narrowing of the cervical internal carotid artery along with an embolic obstruction in a major intracranial vessel, such as the terminal carotid or proximal middle cerebral arteries. This condition accounts for 10–20% of all ischemic strokes. Without treatment, studies estimate that 40–69% of affected patients either suffer severe disability or do not survive. Tandem occlusion is associated with poor prognosis in acute stroke management due to its high mortality and morbidity rates, as well as its low response to intravenous tissue plasminogen activator (tPA). However, endovascular treatment has demonstrated advantages in restoring blood flow in large vessel occlusions, preserving the cerebral penumbra, and improving clinical outcomes. Current treatment guidelines recommend an initial administration of intravenous thrombolytic therapy, followed by endovascular thrombectomy. Despite these recommendations, there is still no standardized endovascular approach specifically for managing this stroke subtype.

Keywords: Acute ischemic stroke, endovascular treatment, tandem occlusion, middle cerebral arteries.

Introduction

Tandem occlusion—cervical internal carotid artery stenosis and major intracranial vascular embolic occlusion—causes 10–20% of acute ischemic strokes. Studies suggest 40–69% of patients die or suffer severe impairment without therapy **[1].** High mortality, morbidity, and little responsiveness to intravenous tissue plasminogen activator make this condition prognostically bad **[2].**

Endovascular therapy can recanalize massive artery occlusions, preserve cerebral penumbra, and improve clinical results. Though there is no standard treatment for this subtype of stroke, current recommendations propose intravenous thrombolytic therapy followed by endovascular thrombectomy **[3].**

Cerebral Vascular Anatomy

The brain is supplied by two arterial pairs: the internal carotid arteries (ICA) for anterior circulation and vertebral arteries (VA) for posterior circulation. These circulations connect at the brain's base via the circle of Willis **[4].** The common carotid arteries (CCAs) bifurcate into the internal and

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external carotid arteries, with the ICA entering the skull and giving rise to the ophthalmic artery. The ICA then branches into the anterior cerebral artery (ACA) and middle cerebral artery (MCA), with the anterior choroidal and posterior communicating arteries branching before this bifurcation. The VA, originating from the subclavian arteries, join to form the basilar artery, which branches into the posterior cerebral arteries **[2]** (Fig. 1).

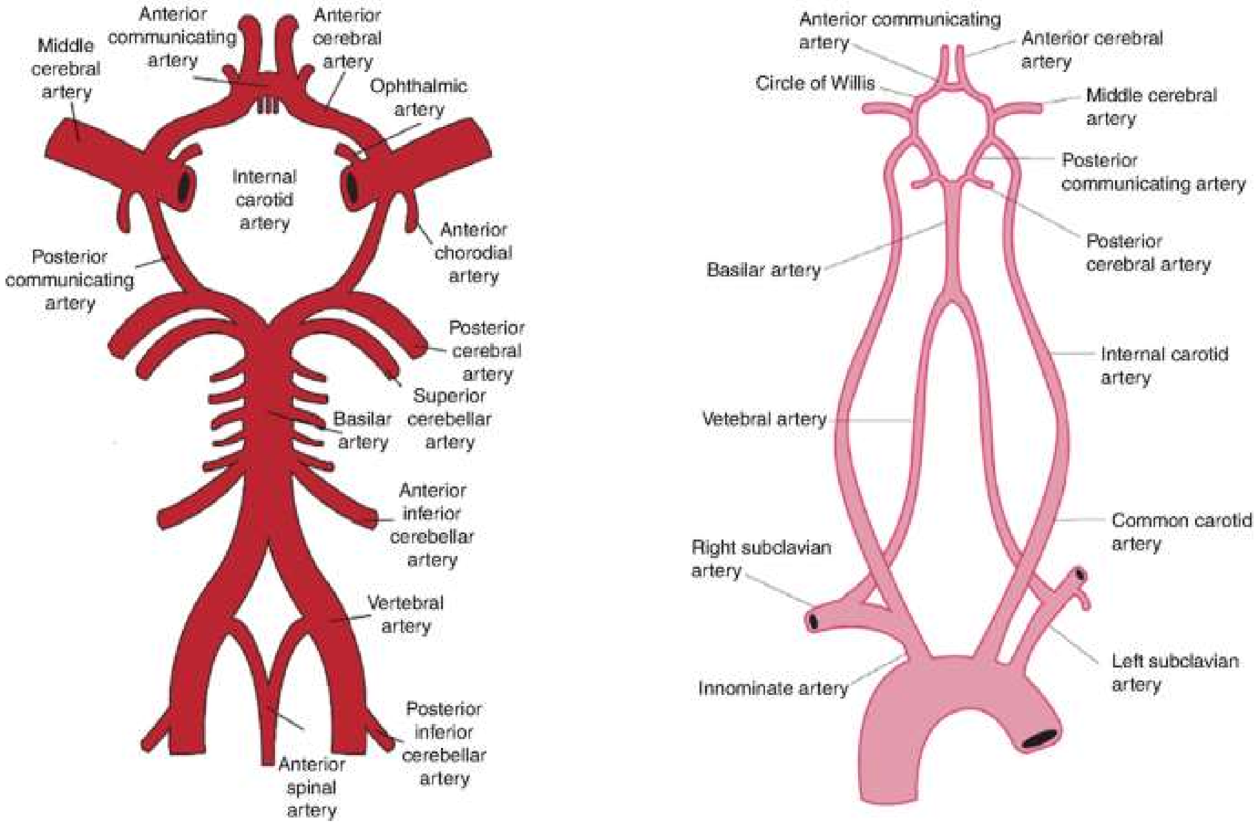

Fig (1): Intracranial arteries and their origination from the heart. Note the anastomosis of blood between the anterior and posterior intracranial circulations by way of the circle of Willis (picture on the left) and the origination of the internal carotid arteries and vertebral arteries from the aortic arch (picture on the right) **[4].**

ICA Segments: The ICA is divided into several segments:

- **Cervical (C1)**: From CCA bifurcation to the petrous bone.
- **Petrous (C2)**: Through the carotid canal.
- **Lacerum (C3)**: Between petrous and cavernous segments.
- **Cavernous (C4)**: Forms the carotid siphon.
- **Clinoid (C5)**: Continuation of the cavernous segment.
- **Ophthalmic (C6)**: Where the ophthalmic artery branches.
- **Communicating (C7)**: From the posterior communicating artery to the MCA and ACA bifurcation **[5, 6].**

Middle Cerebral Artery (MCA): The MCA, the ICA's largest terminal branch, is divided into four segments (M1 to M4). The M1 segment is insonated in TCD, with blood flow directed towards the transducer **[4].**

Anterior Cerebral Artery (ACA): The ACA, part of the circle of Willis, supplies the medial frontal and superior parietal lobes. It has five segments, but only the first two are relevant for TCD studies:

- **A1**: From the ICA to the anterior communicating artery.
- **A2**: From the anterior communicating artery to the corpus callosum, terminating at the callosomarginal artery or corpus callosum genu **[7].**

Internal Carotid Artery Occlusion (ICAO) in Acute Ischemic Stroke (AIS)

ICA occlusion is a common and significant large vessel occlusion (LVO) site in AIS, with varied pathophysiology depending on occlusion type. It is categorized into true cervical ICA (cICA) occlusion, pseudo-occlusion of cICA, and distal ICA (dICA) occlusion (Figure 5) **[8].** ICAO affects 6–15% of acute ischemic events, with 40–69% of patients experiencing AIS and a 16–55% mortality rate **[9].**

Clinical Presentation and Prognosis

- **Acute Presentation**: Symptoms depend on occlusion location and collateral circulation. Common signs include contralateral hemiparesis and sensory loss, aphasia, and homonymous hemianopia **[10].** Malignant brain edema and herniation may occur with MCA and ACA infarctions.
- **Chronic Presentation**: Often asymptomatic, detected incidentally on imaging. Chronic ICAO may present with premonitory symptoms such as amaurosis fugax and transient aphasia **[11].**

Pathophysiological Mechanisms

- **Embolic Phenomenon**: Micro and macro emboli from atherosclerotic plaques or thrombus can lead to downstream ischemia, causing transient ischemic attacks (TIA) and strokes **[12].**
- **Hemodynamic Alterations**: Conditions like low cardiac output or hypotension increase stroke risk, particularly with severe ICA stenosis **[13].**
- **Collateral Failure**: Reduced collateral circulation can exacerbate ischemia, especially in patients with anatomical variants like fetal PCoM **[14].**

Etiologies of ICAO

- **Atherothrombosis**: Accounts for 20% of strokes, with carotid atherosclerosis contributing to 7% of all strokes. Severe stenosis (70–99%) often leads to transient neurological symptoms **[11].**
- **Cardioembolism**: Atrial fibrillation is the leading cause, contributing to one-quarter of AIS cases. Cardioembolic strokes usually occur in supraclinoid ICA segments, leading to severe symptoms and poor outcomes without revascularization **[15].**
- **Cervical ICA Dissection**: Accounts for 1–2% of strokes but is more common in younger patients (25–45 years). It typically results from trauma or connective tissue disorders **[16].**

Other Etiologies: Include post-stent atherothrombosis, neck irradiation **[17],** and vasculitis **[18].**

Acute Tandem Occlusion Stroke

Pathophysiology and Diagnosis of Tandem Lesions

Tandem lesions occur in 15% of patients undergoing endovascular treatment, primarily due to atherosclerotic carotid artery stenosis **[19].** These lesions are more common in men, with varying prevalence across racial groups—Native Americans and Caucasians have the highest rates, while

African Americans and Asians have the lowest **[19].** The most common causes of tandem lesions are atherosclerotic plaques (60-70%), followed by internal carotid artery (ICA) dissection (20-30%), and rarely, carotid web (<5%) **[20].** Carotid web is associated with recurrent strokes and is most often found in the carotid bulb **[20].**

ICA Dissection

Arterial dissection is more common in younger patients and presents with fewer vascular risk factors, leading to a lower stroke recurrence compared to atherosclerotic **[21].** Diagnosis of ICA dissection is highly accurate with non-invasive imaging (angio-MR and angio-CT), which can reveal characteristics such as a "flame-shaped" lumen and mural thrombus **[19].**

Imaging and Diagnosis

CT and MRI are useful for diagnosing tandem occlusions. Non-contrast CT can detect ischemia and arterial wall hematomas, while angio-CT shows features like a narrowed, eccentric lumen **[19].** MRI sequences, including T1 FS and T2, help identify high-signal intramural hematomas **[22].**

Case Example

A patient with severe right hemiplegia and aphasia exhibited a DWI-ASPECTS of 6 after 4.5 hours, showing left ICA and middle cerebral artery occlusions. Angiography revealed left ICA dissection, followed by successful thrombectomy with restoration of flow through the circle of Willis **[23]** (Fig. 6).

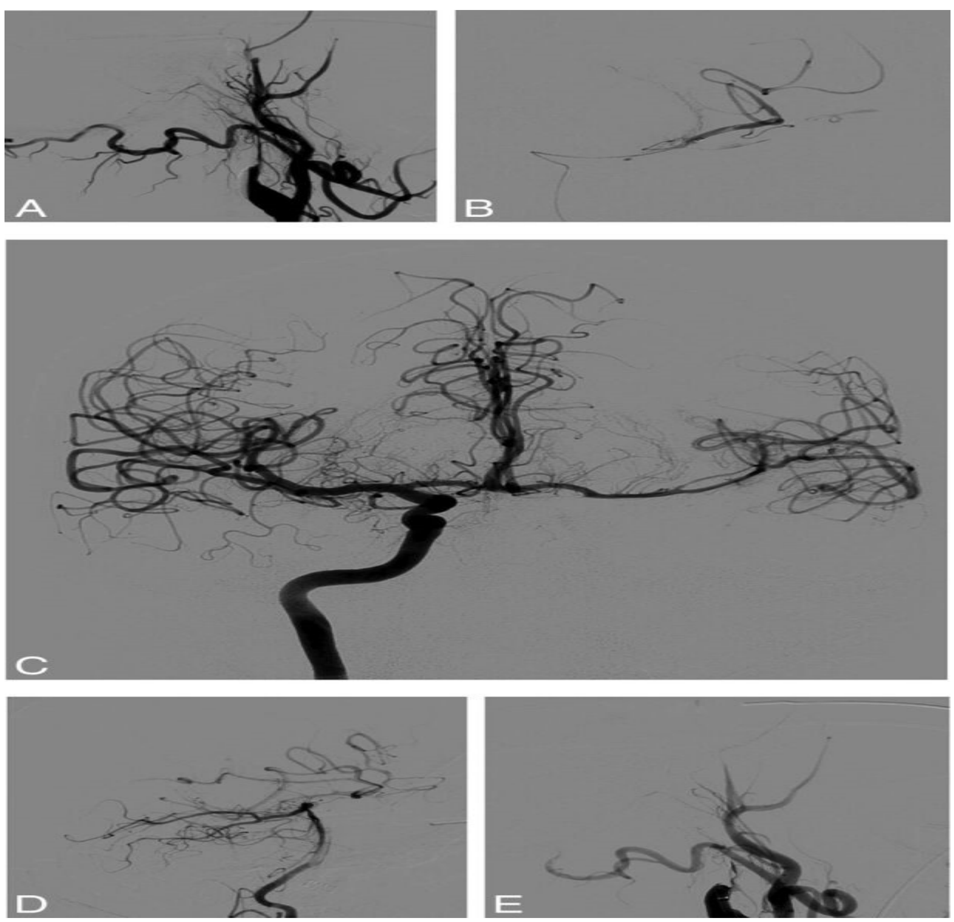

Fig (6): Shows patient presented with severe right hemiplegia and aphasia. Initial MR imaging revealed a DWI-ASPECTS = 6 after 4.5 hours since symptom onset, associated with left tandem ICA and middle cerebral artery occlusions. The initial angiogram (*A*) demonstrates left internal carotid occlusion related to cervical dissection. the microcatheter is navigated through the dissected ICA to the intracranial occlusion (*B*). Thrombectomy performed after contralateral femoral puncture and right ICA run shows a functional circle of Willis and no residual left M1 occlusion (*C*). The posterior communicating artery is also permeable as seen on the left vertebral artery run (*D*). Consequently, artery was left in its initial condition (*E*) **[23].**

Atherosclerosis vs. Dissection

Atherosclerotic lesions are common in older males and typically involve vulnerable plaques at the carotid bifurcation, often with adjacent calcified plaques. Unlike dissection, which develops acutely, atherosclerosis evolves slowly, allowing for collateral circulation development **[24].**

Differential Diagnosis: Pseudo-Occlusion

Tandem lesions can be confused with pseudo-occlusion, where apparent ICA occlusion results from stagnant blood, complicating diagnosis and treatment. Advanced imaging techniques, such as multiphase CTA, improve diagnostic accuracy **[25].**

Treatment Strategies for Tandem Occlusion (TL)

Stenting vs. Angioplasty Alone

Two primary approaches for TL management are stenting during thrombectomy (Fig. 7) and balloon angioplasty followed by delayed stenting (Fig. 8) **[26]**. Stenting offers immediate recanalization, reduces stroke recurrence risk, and supports clot lysis but increases thrombotic risk, necessitating acute antithrombotic therapy **[27].** Angioplasty alone avoids acute therapy and shows lower bleeding risk but may lead to reocclusion and recurrence **[19].** A meta-analysis on stenting vs angioplasty found no mortality benefit but reduced revascularization risk with drug-eluting stents. Stenting combined with thrombectomy showed better outcomes but a higher risk of hemorrhage **[25].**

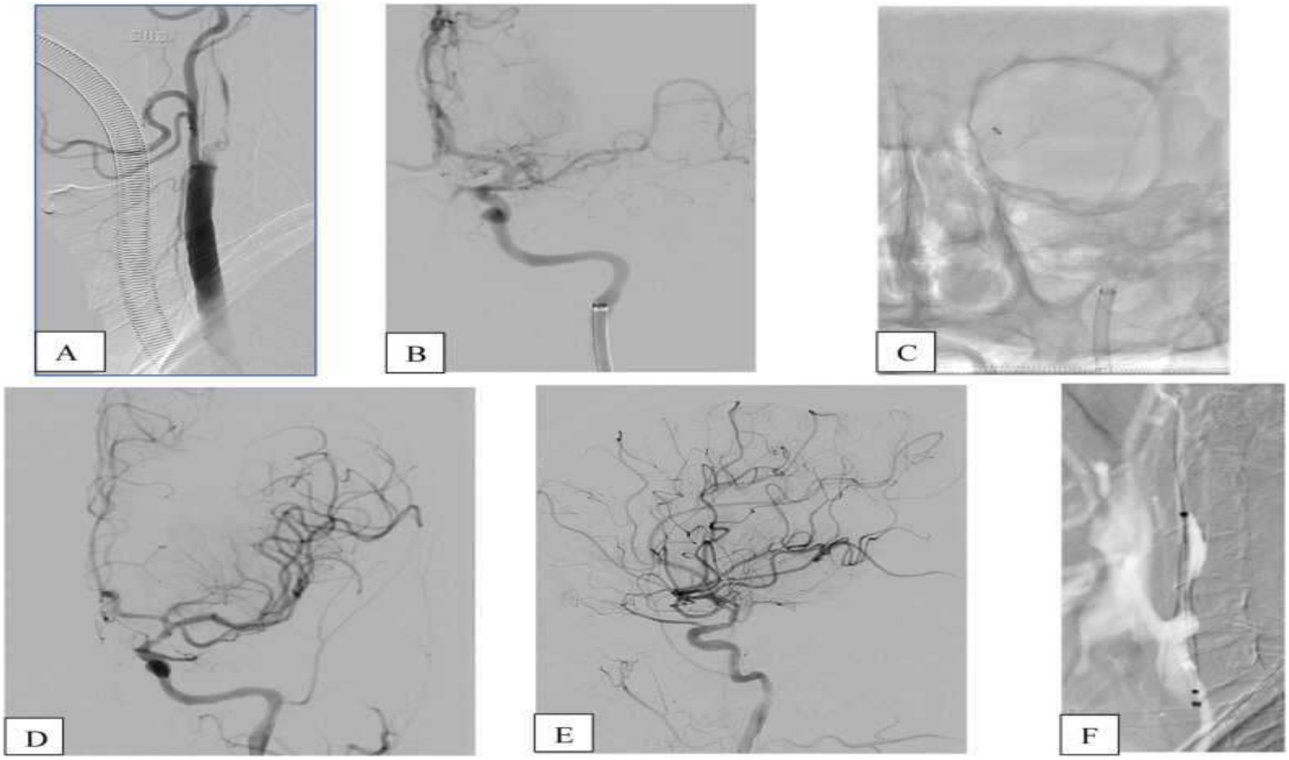

Fig (7): Angiographic data(A-B) showed tandem occlusion of left proximal internal carotid artery+occlusion of M1 tract of MCA. After crossing extracranial lesion, a first attempt of thromboaspiration was performed(C), with a complete recanalization, TICI 3, of the intracranial circulation (D-E). Carotid stent was then deployed with dilatation of angioplastic Balloon(F) **[19].**

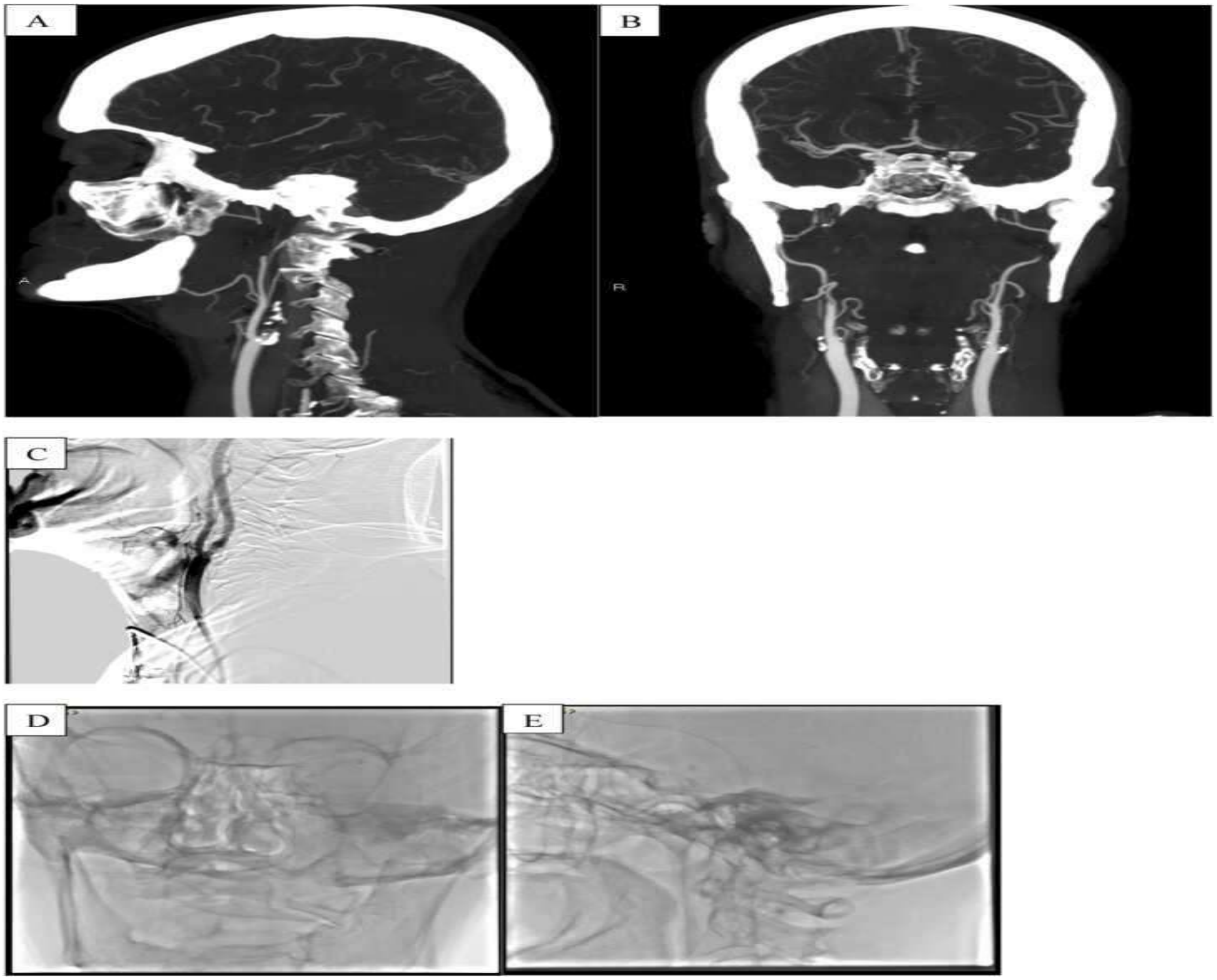

Fig (8): Angio-CT (A-B) showed tandem occlusion of left proximal internal carotid artery+occlusion of M1 tract of MCA. Dilatation with angioplastic balloon, without deployement of carotid stent, was performed (C), in order to cross extracranial lesion.After two attempts of thromboaspiration, complete recanalization, TICI 3, of the intracranial circulation was obtained (D-E) **[19].**

Endovascular Treatment Studies

Endovascular treatment of TL is under-researched, with studies like ESCAPE and ESCAPE-NA1 showing varying results on stenting timing **[28]** and functional outcomes **[29].** Notably, in the TITAN registry, 79.4% achieved successful reperfusion, and 53.4% had good outcomes **[19].** The STRATIS registry found stenting during thrombectomy resulted in better outcomes, though observational bias may exist **[30].** The EASI-TOC trial **[31]** and TITAN trial **[32]** are ongoing to define the best strategy.

Anterograde vs. Retrograde Approach

The anterograde approach, which treats cervical lesions first, offers faster distal recanalization but risks delaying intracranial treatment **[33].** In contrast, the retrograde approach, treating intracranial lesions first, reduces ischemic time but may increase distal embolization risk **[34].** Both approaches have similar safety profiles, with no significant differences in hemorrhage, mortality, or 90-day independence **[35, 36].** Embolic protection devices, though beneficial in non-acute stenting, are rarely used in acute settings **[37].**

Deferred ICA Intervention

Deferred ICA intervention involves a staged approach for carotid recanalization, typically with CAS or endarterectomy after intracranial EVT. During the acute phase, balloon angioplasty is used to access intracranial vessels. In the ESCAPE trial, 57% of intervention-arm subjects received emergency endovascular treatment for extracranial disease; only 4 out of 13 required staged revascularizations **[3].** Studies show balloon angioplasty-assisted thrombectomy can lead to successful recanalization and good outcomes **[38, 39]. However**, the evidence is limited, with retrospective studies showing no definitive conclusions **[31].**

Medical Treatment of Tandem Occlusion

Thrombolytics like tissue plasminogen activator (tPA) are commonly used, but their efficacy in tandem occlusions (TOs) is debated. IVT offers limited benefit, with only 20% of patients showing good outcomes. A combination of IVT and EVT improves outcomes **[40].** In acute stent placement, antithrombotic therapy is necessary to prevent thrombosis (2% incidence), but it may increase the risk of symptomatic intracranial hemorrhage (sICH) **[41].** Studies show IVT+EVT enhances outcomes without significantly increasing hemorrhagic risks **[42].** DAPT is commonly used post-stenting with no significant increase in sICH risk **[43].**

Antithrombotic Treatment Options

For acute carotid stent placement, several treatments have been explored. DAPT (aspirin and clopidogrel) is widely used, though it offers marginal benefits for functional outcomes. High-dose aspirin and clopidogrel with or without IVT have shown positive results **[44, 45].** Use of abciximab may increase sICH risk, while eptifibatide appears safe with low sICH rates **[46].** Emerging agents like ticagrelor are being explored, but evidence remains scarce **[19].**

Coil Occlusion and Other Treatments

Coil occlusion of the cervical ICA is used to prevent embolic recurrence in patients with good collateral flow. Labeyrie et al. reported coil occlusion in 12 out of 64 patients with tandem lesions, showing lower rates of intracranial hemorrhage and similar favorable outcomes at 90 days **[47].** However, due to small sample sizes, recommendations remain cautious.

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