Emergent Carotid Stenting After Thrombectomy in Patients With Tandem Lesions

Daniel Behme, MD; Carlos A. Molina, MD; Magdy H. Selim, MD; Marc Ribo, MD

The Case
A 68-year-old diabetic woman presents to the emergency department 1 hour after right-sided hemiplegia and global aphasia. National Institutes of Health Stroke Scale score is 21; Alberta Stroke Program Early CT score (ASEPCTS) on computed tomographic scan is 8; and computed tomographic angiography shows severe left internal carotid artery stenosis and proximal left M1 middle cerebral artery occlusion (tandem lesion). She is treated with intravenous tPA (tissue-type plasminogen activator) and is being considered for endovascular intervention.

The Questions
Would you consider a left internal carotid artery stenting immediately after successful thrombectomy (thrombolysis in cerebral infarction [TICI]-3)? If so, when do you start dual-antiplatelet therapy?

The Controversy
Emergent carotid stenting after thrombectomy in patients with tandem lesions.

Yes, Carotid Artery Stenting Should Be Considered Immediately After Successful Thrombectomy

Daniel Behme

I would always go for stenting of the internal carotid artery (ICA) stenosis because the clot in the middle cerebral artery was likely caused by the stenosis. Therefore, acute stenting in combination with thrombectomy allows for treatment of the symptomatic middle cerebral artery occlusion and the suspected cause of the stroke, the severe ICA stenosis, simultaneously. More importantly, I would never refuse to provide endovascular therapy (EVT) because of an underlying tandem lesion. The data of the MR CLEAN (Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in The Netherlands) have shown that patients having tandem occlusions benefit from EVT. This was also true for the other randomized trials, which are summarized in the HERMES meta-analysis (Highly Effective Reperfusion Evaluated in Multiple Endovascular Stroke trials).

Immediate stenting is thereby justified for 2 reasons. First, the ICA stenosis is obviously symptomatic and therefore should be considered for stenting or surgical treatment according to the current recommendations anyway. Second, it is much easier to perform intracranial thrombectomy if one can place a large guiding catheter or a femoral long sheath distally in the ICA or at least if one can pass the cervical ICA with a large bore aspiration catheter, which might not be possible in the presence of severe ICA stenosis. Although balloon angioplasty without stenting might be an option, this technique is associated with increased risks of ICA dissection and recurrent stenosis. In addition, balloon angioplasty without stenting is not the standard of care in symptomatic ICA stenosis. If you favor of carotid endarterectomy, you probably should ask a vascular surgeon what he thinks about administering intravenous tPA right before surgery. If you only worry about the time lost because of stenting before thrombectomy, you can simply do it the other way around; pass the stenosis with an intermediate catheter, if necessary, after angioplasty of the ICA and perform thrombectomy first. One can save ≈30 minutes by applying this technique. About antiplatelet therapy, I would start it right away before stent placement. Ideally, after a flat detector computed tomographic scan is performed after intracranial thrombectomy and intracranial hemorrhage (ICH) is ruled out before stent placement. I would leave the patient on acetylic salicylic acid monotherapy until tPA is eliminated completely, that is, for 24 hours. To my knowledge, existing guidelines do not recommend starting dual-antiplatelet therapy within 1 hour of intravenous tPA administration. However, one should not be afraid to use dual-antiplatelet therapy with acetylic salicylic acid and clopidogrel based on the guidelines for ICA stenting. Available data suggest that there is no higher risk of symptomatic ICH with dual-antiplatelet therapy in combination with intravenous
tPA when ICA stenting is performed in tandem lesions compared with intracranial thrombectomy alone. This seems also to be true for intravenous antiplatelet inhibitors like Tirofiban (Aggrastat) or Eptifibatide (Integrilin). Especially in our particular case with a high ASPECTS and a short time from symptom onset to recanalization, the expected infarcted tissue should have a small volume. Therefore, a major bleeding complication seems very unlikely to me.

No, Carotid Artery Stenting Should Not Be Considered Immediately After Successful Thrombectomy

Marc Ribo

Endovascular treatment of tandem lesions in patients with acute stroke is controversial and open to debate because most recent large randomized clinical trial had these lesions listed in their exclusion criteria. The main questions are which of the 2 lesions should be treated first and should the extracranial carotid lesion be stented or not. Immediate stenting of the carotid lesion aims to secure permeability of the ICA, for which we have to pay the price of loading the patient with antiplatelet therapy to reduce the risk of stent thrombosis. A loading dose of dual antiplatelet in the acute phase of a large stroke is known to significantly increase the risk of ICH, especially in this case of particularly large stroke (National Institutes of Health Stroke Scale 21) with baseline parenchymal ischemic damage (ASPECTS 8). Not to mention that the patient just received intravenous tPA, meaning that even single-antiplatelet therapy is contraindicated in the first 24 hours. For these reasons, the rate of ICH after acute stenting in patients with stroke presenting with tandem occlusions has been reported to be as high as 20%.

There are 2 options if we decide not to stent the carotid lesion. The first option is to perform an angioplasty of the carotid lesion to minimize the degree of residual stenosis. Angioplasty is often sufficient to ensure ICA permeability during the following days or weeks; however, in some cases, a reocclusion may occur, usually progressively in the following minutes after angioplasty and can be observed on an angiogram performed a few minutes later. The second option is to perform the intracranial thrombectomy to reestablish intracranial flow with minimal interaction with the carotid plaque (to minimize plaque activation) leaving a residual stenosis that sometimes may even reocclude during the procedure. In any case, if an acute ICA reocclusion is observed, an angiogram from the contralateral ICA confirming a robust cross-filling flow through the anterior communicating artery may indicate that no further action is needed. Stenting should be performed only in those rare cases with ICA reocclusion and an insufficient circle of Willis. Avoiding acute stenting and therefore acute antiplatelet loading will not increase the bleeding risk. Subacute carotid stenting according to the patient outcome and final parenchymal lesion can be safely performed in the following days to weeks.

In the decision-taking process, we should balance the following: lower risk of ICA reocclusion with increased risk of ICH versus higher risk of ICA reocclusion with no increased

risk of ICH, that is, reocclusion versus bleeding. Hemorrhagic transformation in a patient with acute stroke on dual antiplatelet (who just received intravenous tPA) often represents an irreversible fatal outcome. On the contrary, a reocclusion of an extracranial ICA lesion may still be perfectly compensated by contralateral cross-filling. In cases of immediate clinical recovery, a reocclusion can be clinically monitored; however, it is not unusual that these reocclusions may be even clinically unnoticed and represent a radiological finding on follow-up imaging. In cases without immediate clinical improvement, a bedside monitoring technique such as repeated ultrasound during the first hours can identify a reocclusion. In the event that a reocclusion is detected, there might still be a second endovascular therapeutic option for the patient.
Comments by Drs Molina and Selim

EVT has been demonstrated to improve long-term outcome in severe stroke patients with proximal intracranial occlusions, including those patients with underlying tandem lesions. Intuitively, emergent carotid stenting—before or right after successful thrombectomy—would remove the big carotid truck from the circulation avoiding further traffic jams because of early rethrombosis and embolization. However, clearing the road by carotid stenting requires dual-antiplatelet loading that may potentially increase the risk of ICH in a freshly reperfused ischemic tissue, specifically when tPA is on board.

Our opponents defend 2 different strategies for carotid stenting after thrombectomy. Dr Behme, advocate for a retrieve and stent position. He argues that emergent carotid stenting is mandatory right after successful thrombectomy, to clear the pathway for imminent reocclusion or recurrent embolization, regardless of the magnitude of initial ischemic damage and that the risk of ICH in these patients on dual-antiplatelet therapy is low (9%) and comparable to patients with nontandem lesions. When tPA is on board, he proposes to use of aspirin monotherapy <24 hours to prevent stent thrombosis. In contrast, Dr Ribo defends a retrieve and wait approach. He is concerned about the explosive cocktail of dual antiplatelets and tPA. He considers that the risk of ICH in our particular case exceeds the risk of recurrent embolism and proposes a halfway approach of emergent balloon angioplasty and delayed definitive stenting few days after thrombectomy.

Both timing of carotid stenting and optimal antithrombotic strategy in this setting remain controversial. Emergent carotid stenting increases the risk of ICH—because of double antiplatelet regimen—especially in severe strokes, with low ASPECTS scores and tPA on board. Retrospective studies suggest that the risk of symptomatic ICH ranges from 9% to 20% in the retrieve and stent approach. On the contrary, the retrieve and wait may lead to an unacceptable high risk of recurrent disabling stroke. In fact, the recurrent risk in patients with persistent severe carotid stenosis may be as high as 16% at 24 hours. In patients with tandem lesions undergoing emergent balloon angioplasty after thrombectomy, the 7-day stroke risk is ≈10%. Several factors may shift the balance between recurrent stroke and ICH risk including the extent of baseline infarction, pretreatment with intravenous tPA, degree of clinical improvement and residual ischemic lesion after thrombectomy, collateral flow status, availability of flat detector computed tomographic scan to discriminate bleeding from contrast extravasation, type of stent, and antithrombotic regimen chosen. Taken together, the current evidence is limited and mainly based on relatively small retrospective studies with a wide range of clinical and imaging characteristics and different antithrombotic protocols.

A pragmatic algorithm for identifying patients for emergent or delayed stenting is crucial. A randomized trial of early versus delayed carotid stenting after thrombectomy is needed. Such a trial should also evaluate the effects of different antithrombotic strategies to balance the risk between stent rethrombosis and ICH. Until then, the best modality and timing to treat a carotid stenosis after successful thrombectomy should be based on a case-by-case basis.

Disclosures

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References

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