Letter by Cao et al Regarding Article, “Impact of Target Arterial Residual Stenosis on Outcome After Endovascular Revascularization”

To the Editor:

We have read the article by Hwang et al1 about the impact of residual stenosis on outcome after endovascular revascularization in patients with acute anterior circulation stroke and middle cerebral artery (MCA) occlusion. In their study, 21 patients had instant reocclusion (IR) during endovascular procedure and 9 patients had delayed reocclusion (DR) on follow-up angiographic imaging acquired 5 to 7 days after endovascular therapy. In situ thrombosis (IST) secondary to atherosclerotic lesion was determined to be the hidden pathogenesis of MCA occlusion in 8 of 9 patients with DR and 16 of 21 patients with IR.1

In 8 patients with IST and DR, excellent pretreatment collateral flow was demonstrated to be associated with DR.1 Considering the multiple beneficial effects of good collaterals in acute stroke,2 this finding is somewhat novel. The authors attributed it to the following 2 speculated reasons: (1) the preexisting excellent collaterals may preclude maintenance of reestablished antegrade perfusion and (2) competing between antegrade and collateral flow may result in increased thrombogenicity at the site of a stenosis because of slower flow.3

In patients with MCA occlusion, collateral flow, if exists, is from pial arteries and extracranial to intracranial anastomoses. These secondary collaterals usually require time to develop. Diminished perfusion pressure in vessels distal to stenosis or occlusion is considered a key variable to stimulate collateral growth.4 Decline of perfusion pressure in vessels distal to an embolic occlusion or a mild to moderate MCA atherosclerotic stenosis of MCA may be either too rapid or too slight to induce collaterals growth. Accordingly, excellent pretreatment collateral flow most likely indicates a severe chronic atherosclerotic stenosis that has critically impaired perfusion pressure in distal vessels for a long period of time before acute IST. From our perspective, the intrinsic atherosclerotic pathology and the severity of target vessel lesion rather than collateral flow itself are pivotal factors associated with both IR and DR. Our speculation was first supported by the high portion of IST as the pathogenesis of MCA occlusion and incomplete recanalization status (grade 2 on Arterial Occlusive Lesion Scale) after endovascular therapy in patients with IR or DR.1 Culprit plaques underlying IST usually tether to the vessel wall tightly and are relatively difficult to be removed by endovascular procedure. When the thrombus resulting from IST is removed or lysed by endovascular procedure, the underlying culprit plaque with or without additional procedure-related damage is exposed to blood again. The process of IST might repeat at the top of original plaque and result in reocclusion. Procedure-related damage to the plaque, if occurred, would further increase the risk of reocclusion. Patients with severe stenosis may be more liable to procedure-related damage than those with only mild to moderate stenosis before IST. Considering that the severity of stenosis before IST was unavailable, it was a reasonable finding that excellent pretreatment collateral flow turned out to be associated with DR independently.1 According to our speculation, IR might be also associated with excellent pretreatment collateral flow. However, the pretreatment collateral status in patients with IR was not described. Detailed data about the 20 patients with IR and IST from the online-only Data Supplement1 showed that 5 of 8 patients with DR and IST had experienced IR, which suggested shared pathogenesis of IR and DR.

Findings of Hwang et al1 might be instructive for antiplatelet strategy in endovascular recanalization of MCA. More intensified antiplatelet therapy during and after endovascular procedure might be reasonable in patients with excellent pretreatment collateral flow as it indicates a severe atherosclerotic stenosis before IST and high risk of reocclusion.

Disclosures

None.

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