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Cerebral Angiography Can Demonstrate Changes in Collateral Flow During Induced Hypertension

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A 52-year-old woman with a large left-hemispheric stroke was transferred to our hospital for possible endovascular treatment. The patient underwent a cerebral angiogram at 7 hours after symptom onset with intent to treat and was found to have occlusion of the proximal M1-segment of the left middle cerebral artery (MCA). At that time it was felt that this was a high-risk patient for mechanical clot retrieval and it was decided to treat her with induced hypertension. The diagnostic catheter was left in place in the left internal carotid artery (ICA) and hypertension was induced in the angiography suite by means of an infusion of neosynephrine. Ten minutes after the goal blood pressure levels had been reached, a repeat left ICA injection was performed, which demonstrated more extensive collateralization of the MCA territory from anterior cerebral artery branches. Mean transit times (MTT) for the left ICA circulation improved from 9.5 seconds prior to induced hypertension to 6.0 seconds. The neosynephrine infusion was continued for a total of 24 hours and the patient showed neurological improvement. We suggest that induced hypertension led to the improved collateralization to the left MCA as evidenced by the improved MTT and augmentation of leptomeningeal collaterals, which in turn led to the patient's clinical improvement.

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Abbreviations: CT, computed tomography; ICA, internal carotid artery; MCA, middle cerebral artery; MTT, Mean transit times; NIHSS, National Institutes of Health Stroke Scale; SBP, systolic blood pressure;

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Introduction

Thrombolysis, pharmacological and/or mechanical, is the treatment of choice for patients presenting with acute ischemic stroke. However, a large proportion of patients either do not meet criteria, or show only partial or no response to thrombolysis. Inducing hypertension is one of the few alternatives for intervention that are available for such patients. The concept of induced hypertension is based on the assumption that systemic hypertension will preferentially augment blood flow to the area of stroke because of ischemia-induced impairment of autoregulation. Although no randomized trials have been conducted to demonstrate the efficacy of this treatment, there are studies that have reported that induced hypertension is relatively safe and can lead to improved outcome in selected patients (1-3). We report that cerebral angiography can demon-

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strate improved collateral flow after induced hypertension and might therefore be used for patient selection.

Introduction

A 52-year-old woman who presented with right-sided hemiplegia and aphasia while hospitalized at an outside medical center was transferred to our hospital for possible endovascular treatment. Her estimated National Institutes of Health Stroke Scale (NIHSS) score on admission was 20. The patient was not eligible for intravenous thrombolysis because of severe thrombocytopenia with recent history of bleeding. The patient's transfer was delayed and she arrived at our institution at 7 hours after stroke onset. She had been paralyzed and intubated after an episode of decreased responsiveness, which was attributed to seizure activity.

Cerebral angiography was performed with the intent of possibly treating the patient with a clot retrieval device. Cerebral angiography showed occlusion of the left proximal middle cerebral artery (Fig. 1).



Figure 1. 52-year-old woman with right hemiplegia and aphasia. Left internal carotid artery injection with blood pressure at baseline, early arterial phase, shows that the left middle cerebral artery is occluded (white arrow).

Following this study, intervention was not performed as it was felt to be too risky. Induced hypertension was felt to offer a safer alternative for this patient. Following cerebral angiography, the patient's systolic blood pressure (SBP) was 100 mmHg. A phenylephrine (our drug of choice for inducing hypertension in patients who are not significantly bradycardic) drip was initiated and the patient's SBP was raised to 160 mmHg. Ten minutes later, a repeat injection showed increased filling of the left MCA territory (Fig. 2).

The mean transit time (MTT, i.e. the time interval between opacification of the petrous segment of the internal carotid artery and opacification of the transverse sinus on the same side) for the left ICA circulation had improved from 9.5 to 6.0 seconds (decrease of 37 percent). All images were acquired at 7 frames per second after contrast was injected through an automated system at a rate of 6 ml/second for a total of 8 ml. The catheter was not moved between injections.

The patient was sedated and paralyzed and therefore neurological examination was limited. However, based on the angiographic results, the decision was made to continue induced hypertension for 24 hours aiming at SBP values in the vicinity of 160 mmHg. The following day the patient remained intubated but was off sedation. Repeat assessment showed good anti-gravity strength in the upper and lower extremities and therefore an improvement of 4 points on the NIHSS to a score of 16. The patient subsequently deteriorated due to a large contralateral MCA stroke and expired after care was withdrawn. The etiology of the patient's strokes was thought to be cardioembolic though no definite embolic source could be established.

Discussion

Induced hypertension is often the only possible intervention in patients with acute ischemic stroke. Patient selection is based sometimes solely on safety criteria (3) and in other cases on the presence or absence of clinical improvement after a trial of hypertension (2). The problem with the first approach is that some patients may be exposed to unnecessary risk, whereas with the second approach, patients are excluded if their neurological function can not be adequately assessed, as was the case with our patient.

Assessing the effect of induced hypertension on collateral circulation by neuroimaging provides for an alternative method of patient selection. It has been recently shown that magnetic resonance angiography and MRI perfusion studies can be used to that end (4). CT perfusion and tran-

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scranial Doppler are other non-invasive studies that could also be applied.

Our report shows that induced hypertension can increase collateral flow and improve MTT on conventional cerebral angiography. In our patient, we believe that the MTT measured represents mainly the ACA circulation, but since the ACA provides collateral flow to the MCA, a shorter MTT for the ACA should result in improved MCA perfusion as well. These findings are likely to be the underlying mechanism for the clinical improvement that the patient exhibited.

In patients who are undergoing cerebral angiography as part of their acute work-up or treatment, our method might thus be used to select the subset most likely to respond to induced hypertension. Patient selection should also take multiple elements of the patient's medical history into consideration, so as to minimize the risk of acute cardiovascular complications. Although hypertension is related to repeat ischemic events, we feel that it is unlikely that the acute hypertension that we induced over a brief period of time could be the cause of the patient's second stroke. Her second stroke was thought to be cardioembolic in origin, although no definite embolic source could be established.

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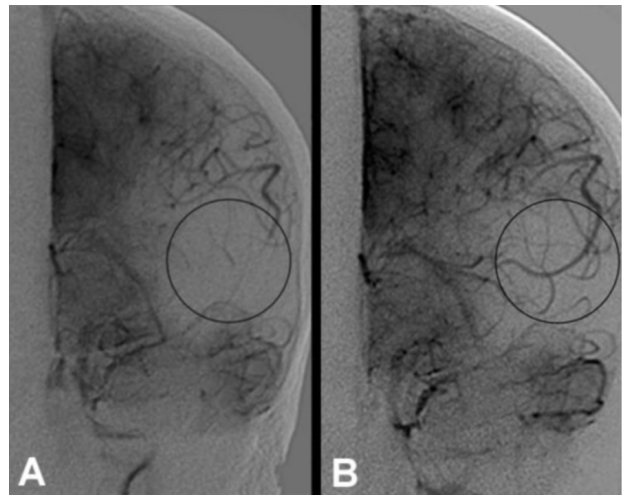


Figure 2A-B. A. Left internal carotid artery injection with blood pressure at baseline, late arterial phase. The black circle highlights a part of the left MCA territory with poor collateral flow. B. Repeat injection after 10 minutes of induced hypertension. Late arterial phase. There is significant improvement in the perfusion of the MCA territory that is highlighted in the black circle.