

## Analysis of Reserve Capacity and Subsequent Stenting in a Case of Subacute Occlusion of the Internal Carotid Artery

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### Introduction

In stroke patients, an acute thrombotic occlusion of an internal carotid artery (ICA) is a poor predictor for clinical outcome [1]. It has occasionally been managed by carotid endarterectomy [2] (CEA) or percutaneous transluminal angioplasty (PTA) with [3] or without [4] stent protection (carotid angioplasty and stenting, CAS). On the other hand, a chronic thrombotic occlusion (CTO) of the ICA may be treated successfully by external–internal carotid artery (EC–

IC) bypass surgery [5, 6]. However, EC–IC together with best medical treatment (BMT) does not reduce the risk of an ipsilateral stroke compared with BMT alone [7]. In addition, there have also been recent reports on CAS in patients with symptomatic CTO [8, 9] and CAS has a serious role in patients with high operative risk.

We herein report on the diagnostic work-up and subsequent therapeutic management of a patient with symptomatic thrombotic occlusion of the left extracranial ICA and a pre-existing occlusion of the contralateral ICA [10].

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### Case Report

A 65-year-old male with multiple cardiovascular risk factors presented with dysarthria and central right-sided facial palsy following an episode of syncope. The patient was admitted to our *stroke unit* where he experienced blood pressure-dependent right-sided weakness and sensorimotor aphasia, initially controllable by conservative therapy.

### Pre-Interventional Assessment

Computed tomography (CT)-angiography (CTA; Aquilion, Toshiba Medical Systems Europa, Zoetermeer, Niederlande) showed bilateral ICA occlusion and the magnetic resonance (MR) scan on a 3Tesla MR-system (TIM TRIO®, Siemens Medical Solutions, Erlangen, Germany) demonstrated multiple chronic microvascular lesions bilaterally on T2-weighted images. Diffusion-weighted imaging (DWI) revealed acute infarctions in a watershed distribution with additional significant prolongation of both the mean transit time (MTT) and time to peak (TTP) in the left hemisphere. Conventional catheter angiography (digital subtraction angiography, DSA) finally confirmed the diagnosis of bila-



**Fig. 1** Catheter angiography (DSA) demonstrates complete occlusion of the left internal carotid artery

teral ICA occlusion (Fig. 1) and revealed collateralisations via the ophthalmic arteries as well as via vertebro-basilar pathways. Importantly, origin and subsequent course of the left external carotid artery (EC) were found to be entirely normal and an EC–IC bypass was considered accordingly.

Given the patient's recurrent symptoms, assumingly of hemodynamic origin, an urgent intervention was required. To further support such a decision, a *Blood Oxygenation Level Dependent* MR scan was obtained (Fig. 2) to evaluate the patient's cerebrovascular reserve capacity (CRV) [11, 12]: The experimental set-up had previously been approved by the local ethics committee and included a bottled carbon dioxide (CO<sub>2</sub>) source and a dedicated magnetic resonance imaging (MRI) patient-monitoring device (GE Medical Systems Information Technologies, Freiburg, Germany). The patient was prepared with a conventional facial non-rebreathing mask and a gas bag connected for a 3 min-CO<sub>2</sub> challenge to induce hypercapnia.

The analysis of BOLD signal intensity changes was performed using an in-house software package (Brainshow [13]). The BOLD response was diminished in the left

hemisphere, mainly in the middle cerebral artery (MCA) territory, thus indicating a marked reduction of reserve capacity (Fig. 2).

### Intervention

The patient was treated with 100 mg aspirin p. o. before admission. Then, he was given an additional 75 mg clopidogrel p. o. and because of the fluctuating symptoms the aspirin dosage was raised to 300 mg and also low-dose heparin was administered.

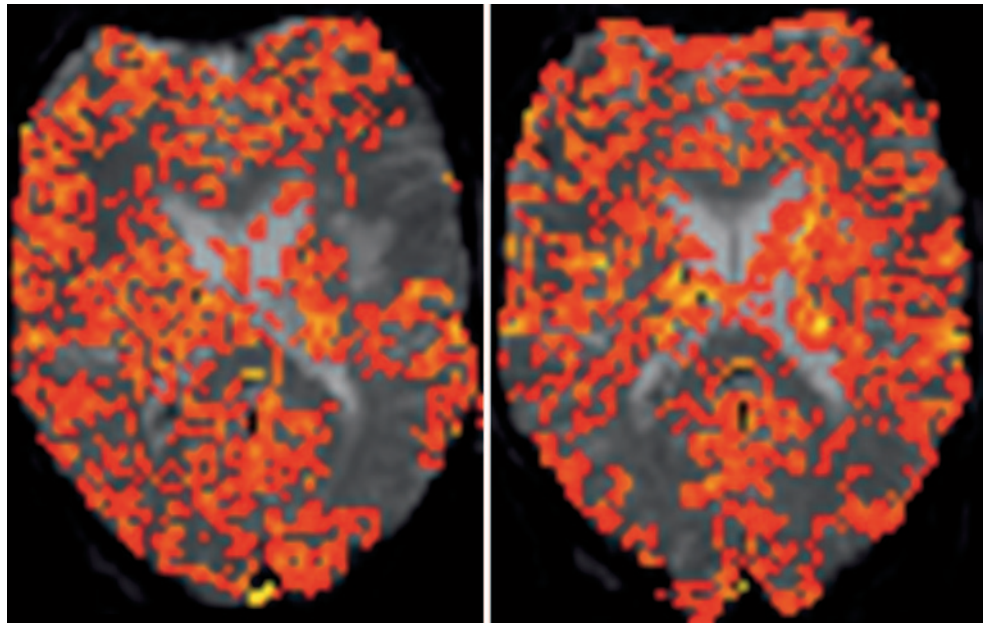
Eight days following the initial event, the patient's symptoms suddenly deteriorated irrespective of maximum conservative therapy. Since emergency EC–IC bypass surgery was not possible logistically, the patient agreed to attempt a neuroendovascular revascularisation. Written informed consent was obtained and stent-protected CAS performed. In addition to the established anticoagulation, further heparin was given via intravenous infusion during the procedure (activated clotting time, ACT=250–300 s).

Selective left common carotid artery catheterisation was performed according to standard aseptic technique using a guide-wire (Terumo, Tokyo, Japan) and a 7F guide catheter (Mach 1™, Boston Scientific, Natick/MA, USA). A microcatheter (FastTracker 18™, Boston Scientific, Natick/MA, USA) with a 0.014-in guide-wire (Synchro 14; Boston Scientific, Natick/MA, USA) was utilised to carefully negotiate the lumen of the occluded vessel under biplanar fluoroscopic control to a level beyond the occlusion; intraluminal position confirmed by microcatheter series. A 7/40 Carotid Wallstent™ Monorail™-System (Boston Scientific, Natick/MA, USA) was then deployed, followed by balloon angioplasty using a 5/20 Sterling™ balloon catheter (Boston Scientific, Natick/MA, USA) at an inflation pressure of 8 bar.

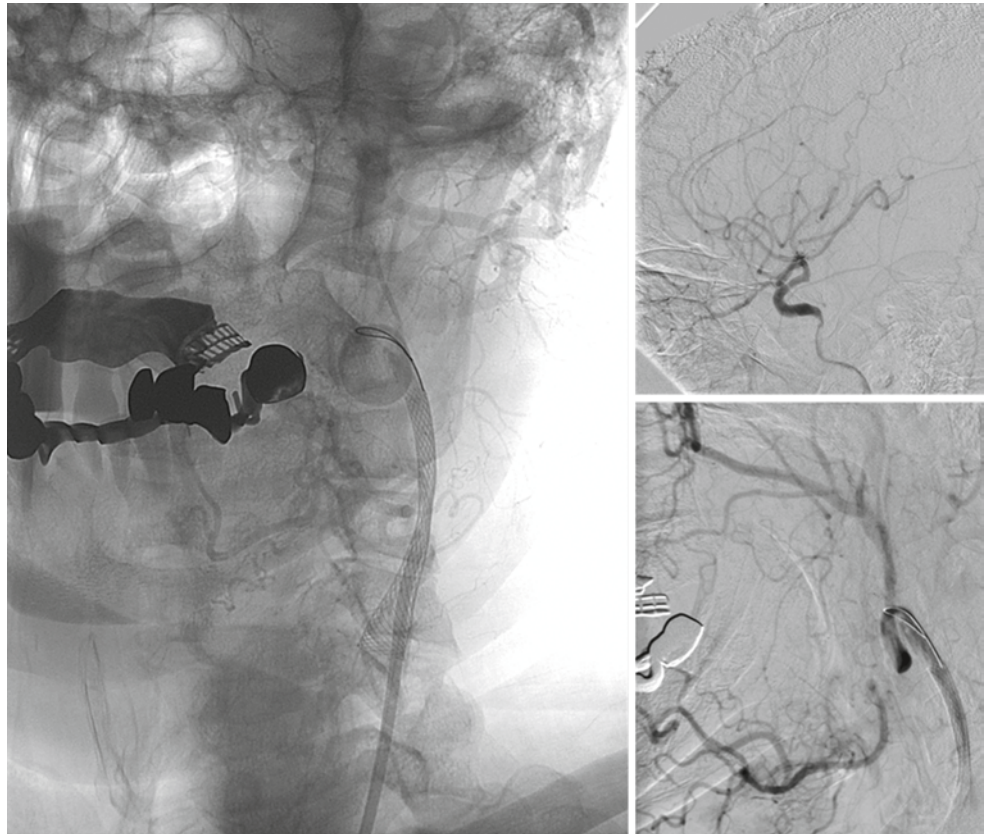
The final angiogram series demonstrated a small intimal flap distal to the stent indicating a minor dissection which was considered to be adequately treated by the present anticoagulation regimen. However, we attributed the very small post-interventional diameter of the extracranial ICA primarily to the previous collapse (Figs. 3 and 4). The vessel diameter was monitored by duplex-ultrasound and normalised within 48 h. The control CT-angiography after 1 week confirmed a patent vessel diameter and the very small intimal flap which finally healed without any additional interventional treatment.

The patient's subsequent course was uneventful. His pre-interventional neurological symptoms subsided. Follow-up MRI showed no new infarct. Cerebral perfusion as well as CRV (Fig. 2) improved markedly. A follow-up CTA examination of the carotid arteries at 3 months revealed persistence of excellent luminal reconstruction without recurrent stenosis.

**Fig. 2** Representative supra-tentorial slices prior to (*left*) and after (*right*) carotid artery stenting show a significant difference in hypercapnia-induced BOLD signal intensity changes between the hemispheres: an initial restriction (*left*) and the subsequent improvement (*right*) of cerebrovascular reactivity can be clearly seen in the left MCA territory



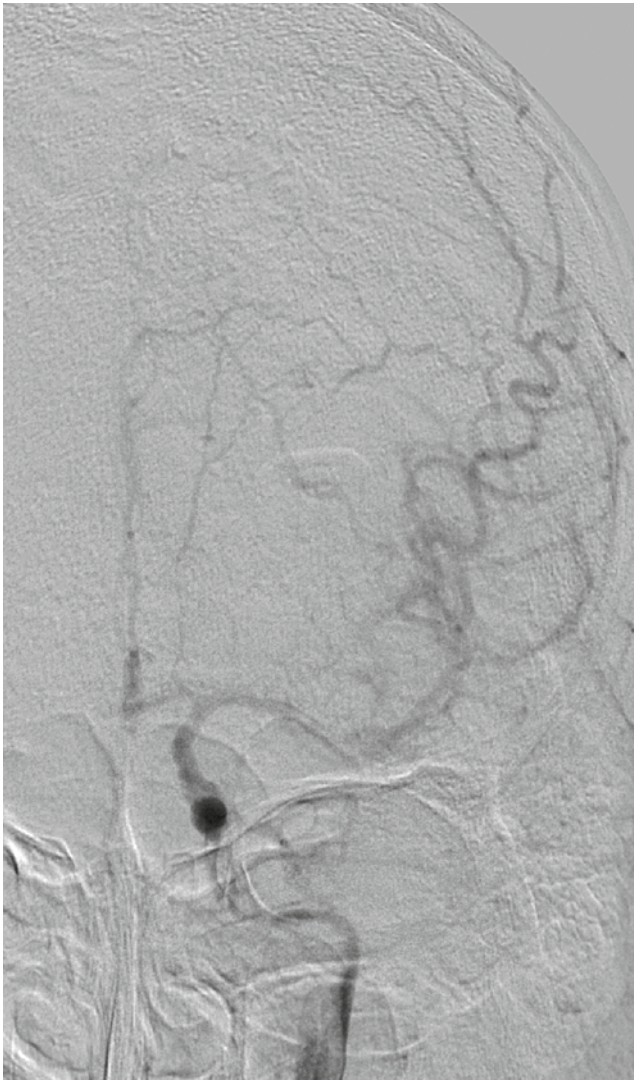
**Fig. 3** Catheter angiography images showing successful left internal carotid artery (LCA) revascularisation with antegrade filling of MCA branches after stenting and stent position (un-subtracted image)



After the neurointerventional procedure the anticoagulation regimen was maintained for 1 week and then reduced to 100 mg aspirin and 75 mg clopidogrel for 3 months. The patient is on 100 mg aspirin since.

## Discussion

In the setting of acute or chronic carotid artery occlusion, cerebral ischemia may principally result from either embolic occlusion with resulting territorial ischemia or from reduced



**Fig. 4** The post-interventional DSA image in frontal view shows the filling of the ipsilateral MCA (image quality is considerably diminished as the patient was only minimally sedated for the procedure and became progressively agitated)

regional perfusion with consequent hemodynamically distributed infarcts. While the value of thrombolysis and fibrinolysis in the acute setting has been shown to be beneficial [14], the mechanism underlying the acute deterioration in our case was rather due to hypoperfusion rather than thromboembolism. EC–IC bypass has been reported in a selected group of patients with poor reserve capacity when CEA was not possible [6]. In our case, EC–IC bypass appeared appropriate but was not applicable because of the acuteness of the patient's deterioration. Paty et al. [15] reported on success rates of CEA of the order of 30 % when performed within 14 days after the onset of symptoms. Furthermore, successful CAS [16] and stent-assisted revascularisation of intracranial vessels [17] in a subacute stage had been reported.

In patients with co-existing conditions leading to a higher operative risk, CAS is not inferior to CEA [10]. However, at this particular time, there is no scientific evidence for the sufficient safety of endovascular revascularisation of totally occluded ICAs.

Measurement of CRV by the means of Xenon-CT has been suggested as a surrogate parameter when selecting patients for cerebrovascular therapy [18]. Alternatively, the applicability of CO<sub>2</sub> exposure-induced hypercapnia in combination with BOLD-contrast functional MR has been demonstrated [11, 19, 20]. Considering our patient's symptoms and his restricted cerebrovascular reserve, an attempt of revascularisation was indicated.

Taking the risk of distal embolisation into account, a blood flow manipulation with proximal balloon occlusion of the common carotid artery was discussed before the intervention but not taken into account because it would not have added any benefit as long as external artery branches were still open [21]. Furthermore, it would have led to a flow reversal in the ophthalmic artery with the threat of a steal phenomenon and unpredictable consequences. The only double-balloon device available in our department has a very small lumen which seemed inappropriate in the present setting. In addition, the pre-operative work-up for the EC–IC bypass demonstrated a substantial contribution from the external carotid artery to the brain perfusion. Any further reduction of arterial input should be avoided under those precarious circumstances. The contrast injection through the microcatheter showed no relevant thrombus load in the ICA distal to the occlusion—in this case the procedure would have had to be adjusted, e.g. applying vacuum thrombectomy [22, 23].

In the past, several reports of reperfusion injury following recanalisation by either CEA [24–26] or CAS [27, 28] have led to a very careful selection of patients, especially in cases with prolonged ischemia. For this reason, initial therapy in our patient comprised medical therapy with anticoagulation as well as blood-pressure elevation only. However, this approach did not yield a satisfactory result: symptoms were fluctuating and aggravated over time. Following further deterioration with sudden onset of aphasia and hemiparesis, an ultima ratio decision was therefore made to attempt neuroendovascular revascularisation.

CAS in subacute ICA occlusion is feasible and may lead to a favourable outcome but should be restricted to carefully selected cases. The non-invasive assessment of cerebrovascular CO<sub>2</sub> reactivity using BOLD-contrast MRI might provide valuable information in this setting.

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**Conflict of Interest** The authors declare that there is no actual or potential conflict of interest in relation to this article.

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