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CASE REPORT

Hyperacute Intracerebral Hemorrhage Complicating Carotid Stenting Should Be Distinguished from Hyperperfusion Syndrome

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SUMMARY: We describe a patient who experienced a fatal ipsilateral basal ganglia hemorrhage within an hour after carotid angioplasty and stent placement. In the few similar cases published there were no prodromata, but hyperacute onset of severe neurologic deterioration corresponding to intracerebral hemorrhage (ICH). Our findings suggest that besides the delayed ICH that is associated with hyperperfusion syndrome (HPS), a second type of hyperacute and usually fatal ICH exists that resembles hypertensive hemorrhage.

Carotid artery stent placement (CAS) is a treatment option in symptomatic internal carotid artery (ICA) stenosis. A rare complication of any of the cervical recanalization procedures, be it CAS or carotid endarterectomy (CEA), is the so-called hyperperfusion syndrome (HPS)[1] that usually develops between the 5th and 7th days after the procedure and presents with ipsilateral frontotemporal or retro-orbital headache, nausea, vomiting, and other neurologic signs of elevated brain pressure. Intracerebral hemorrhage (ICH) is not an obligate component of this complication but can also occur. Large clinical series about CEA have shown that the overall incidence of ICH complicating the procedure is on the order of 0.2%–0.7%.[2-5] In contrast, recent data on CAS suggest a relatively higher ICH rate of up to 5%.[6]

We report a case of fatal ICH subsequent to CAS after a time of 30 minutes. Diffusion-weighted cranial MR imaging a few days before the procedure showed acute hemodynamic lesions in the ipsilateral centrum semiovale. However, ICH occurred in ipsilateral basal ganglia and did thus not at all affect the region of acute infarction. This suggests that acutely infarcted brain parenchyma is not necessarily at risk of hyperacute ICH after CAS. There may be a second entity of HPS with a different pathophysiologic mechanism that leads to early ICH in typical locations and has to be differentiated from the HPS described by Sundt et al.[1] We aim to support this theory by reviewing the relevant literature concerning cases similar to ours.

Case Description

A 65-year-old woman was admitted after an episode of brachiofacial hemiparesis of the left side that remitted incompletely. She gave a history of both severe small and large vessel disease. She had been suffering from arterial occlusive disease for many years. In 2000, she was supplied with a femoral bypass on the right side; in 2002, amputation of the right lower limb was necessary. A 95% stenosis of the right internal carotid artery was diagnosed, and the contralateral side showed an 80% stenosis (both according to North American Symptomatic Carotid Endarterectomy Trial criteria[7]). The further medical history revealed a tachyarrhythmia and an older embolic insult of the right posterior cerebral artery. Furthermore, she suffered from chronic renal failure and subcortical arteriosclerotic encephalopathy. Vascular risk factors included arterial hypertension, high blood cholesterol, smoking (approximately 30 pack-years), many years of alcohol abuse, and a positive family history of vessel disease (Table). Coagulation parameters were normal, and blood pressure on admission was 150/80 mm Hg.

An unenhanced cranial CT performed at admission demonstrated old microvascular lesions in the brain stem and basal ganglia as well as a residual territorial infarct in the right posterior cerebral artery and older, well-demarcated hemodynamic lesions in the right hemisphere. The patient was treated with intravenous heparin. CT angiography confirmed the Doppler-sonographic finding of high-grade (95%) stenosis of the right ICA and high-grade stenosis (80%) of the contralateral vessel. The contralateral A1 segment and the anterior communicating artery were normal. Therefore both anterior cerebral arteries (ACA) were supplied from the contralateral side. There was no cross-flow through ACA to ipsilateral because of the ipsilateral A1 aplasia. The right posterior cerebral artery (PCA) was occluded, corresponding to an older embolic infarction. Both vertebral arteries had an ostial stenosis of at least 50%; the basilar artery was ectatic.

Cranial MR imaging revealed focally restricted diffusion in the right centrum semiovale typical of hemodynamic lesion in that no cortical structures were involved. The lesions were only beginning to demarcate in the fluid-attenuated inversion recovery (FLAIR) sequence (Fig 1). There were no other acute signs of infarction, especially not in the basal ganglia and the thalamus (Fig 1). Because of the patient's comorbidity, CAS of the right ICA was performed via femoral approach under local anesthesia. An intravenous bolus of 5000 IU of heparin was administered; thereafter, the activated clotting time was 278 seconds. A 7/40 carotid Wallstent (Boston Scientific, Natick, Mass) was positioned with the use of a Strategy guidewire (Cook, Bloomington, Ind) and a 7F Mach 1 Guide catheter (Boston Scientific). After placement of the stent, the residual stenosis was dilated with the use of a Submarine-Rapido 5/20 balloon catheter (INVAtec, Roncadelle, Italy). The morphologic result was excellent, with no residual stenosis after dilation (Fig 2). There were no procedural complications. Because of the known stenoses in all other brain-supplying vessels, blood pressure was not lowered under 140/70 mm Hg to prevent further hemodynamic impairment. On the other hand, blood pressure never rose above 160 mm Hg systolic before the ICH.

Approximately 30 minutes after the intervention the patient suddenly developed headache and vomiting and became unresponsive. Immediate unenhanced cranial CT showed a large right-sided ICH affecting the basal ganglia and the thalamus (Fig 3). In addition there

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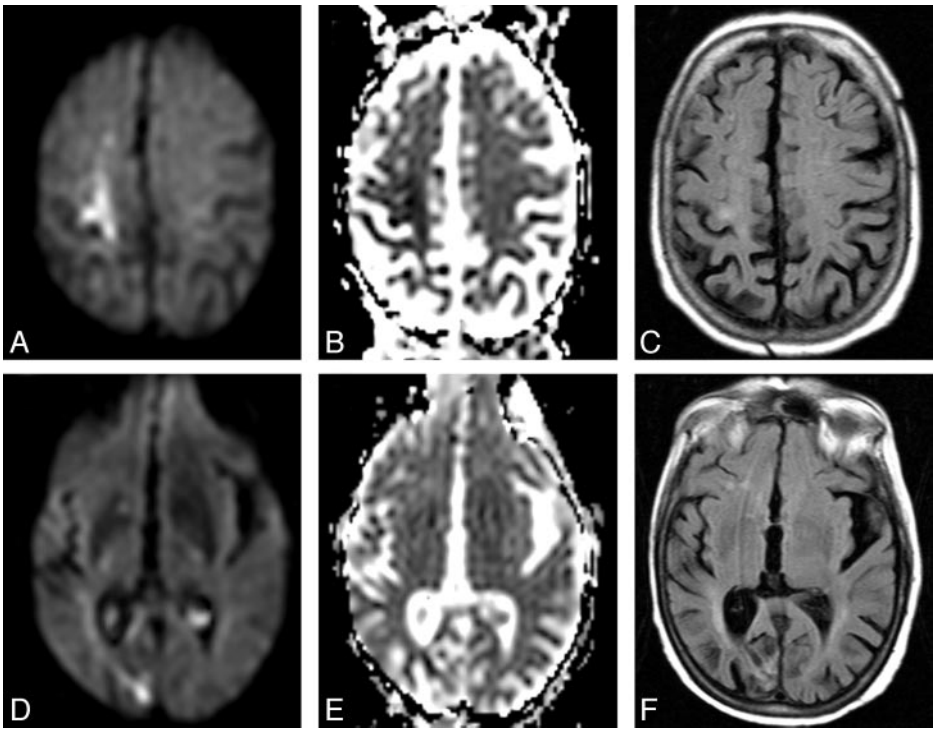

Fig 1. MR imaging before CAS.

A–C, Lesion of focally restricted diffusion in the right-sided centrum semiovale, characteristic of acute hemodynamic infarction (*A,* diffusion-weighted MR imaging [DWI][; *B*, apparent diffusion coefficient [ADC]). In the FLAIR sequence, an incomplete demarcation can be seen (*C*).

D–F, At the level of the basal ganglia there is no evidence of an acute ischemic lesion. The right occipital pole is part of the residual posterior territorial infarction. The FLAIR sequence demonstrates mild microangiopathic changes (*F*).

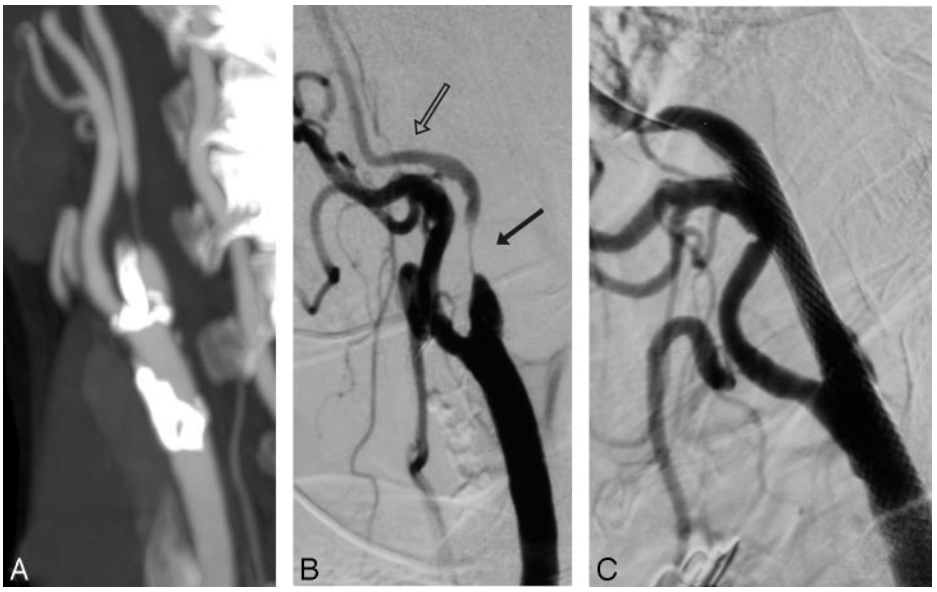

Fig 2. A maximum intensity projection (MIP) image reconstructed from CT-angiographic data shows the high-grade stenosis of the right ICA as well as associated calcified atherosclerotic plaques (*A*). Digital subtraction angiography of the right carotid bifurcation before and after the intervention shows a tight, short-segment, hemodynamically significant stenosis (*B, closed arrow*)—note the low attenuation of contrast medium in ICA distal to the stenosis (*B, open arrow*)—and a good hemodynamic result after stent-placement and dilation (*C*).

was subarachnoid extension. There was no bleeding in the regions where diffusion-weighted imaging had shown acute ischemic lesions.

An external ventricular drain was placed under general anesthesia, further complicated by myocardial infarction. Neurologic examination showed an unresponsive patient with dilated pupils and no reaction to painful stimuli. Follow-up CT revealed bleeding progression with subfalcine herniation. Therapy was then minimized to high-dose morphine. The patient died 2 days after the intervention.

Review of the Literature

Few cases with fatal hyperacute ICH after CAS have been published. All of these cases had in common a short time course,

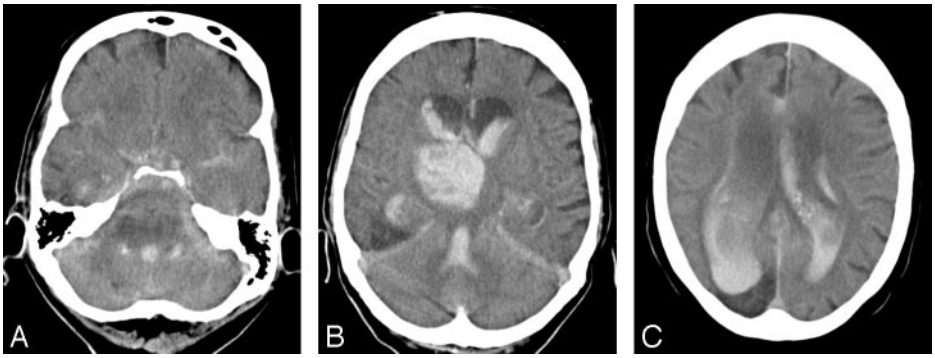

Fig 3. Unenhanced cranial CT from approximately 1 hour after the intervention demonstrates a large right basal ganglial hemorrhage with extension to intra-axial and extra-axial CSF spaces. The hematoma is ipsilateral to the treated stenosis. The uppermost section shows the residual lesion after previous infarction in the right PCA territory.

hours at most, between the end of the procedure and the acute onset of neurologic deterioration, presumably corresponding to onset of ICH. ICH predominantly occurred in basal ganglia, and all cases presented with a high-grade stenosis. The Table gives an overview about all relevant cases we found in the literature.

The first report on ICH after CAS was published by Schoser et al[8] and is the first report on HPS after CAS. They describe a 59-year-old woman with mild right-sided sensomotor hemiparesis and CT diagnosis of a left-sided borderzone infarction. No MR imaging is documented in this report. However, the patient experienced a fully developed HPS with ipsilateral putaminal hemorrhage that was diagnosed on the 3rd day after CAS of a high-grade stenosis of the left ICA. No hemorrhage occurred in any region affected by hemodynamic lesions. Outcome in this case was not fatal; the patient recovered with a mild upper limb paresis.

McCabe et al[9] were the first to report the occurrence of fatal ICH soon after CAS of a high-grade stenosis in a symptomatic patient. In their case, neurologic deterioration followed CAS after a few hours and occurred without any prodromata postulated by Sundt et al[1] to be an obligate component of the hyperperfusion syndrome (ie, clinical signs of elevated brain pressure: severe headache, nausea, and seizures [Table]). Mori et al[10] describe a quite similar case. A symptomatic patient was treated with CAS in a high-grade stenosis and few hours afterward suddenly manifested signs of acutely elevated brain pressure (Table). In both cases, ICH was extensive and occurred in the ipsilateral basal ganglia with ventricular and subarachnoid extension. Both patients had morphologic signs of cerebral microangiopathy. The authors describe a small site of paraventricular infarction as a possible origin of ICH, but the lesion may have been an old lacunar infarction.

In a letter referring to the case described by McCabe et al,[10] Chamorro et al[11] report a further case of fatal hyperacute ICH in basal ganglia after CAS. Theirs is the only case in which ICH occurred on the contralateral side, but—as in our case—ICH did not affect an area that had been considered as acutely infarcted before the intervention. The patient they describe presented with acute onset of neurologic deterioration accompanied by increase of blood pressure (Table). The authors do not explain why the ICH could have occurred in the contralateral thalamus, so this remains unclear. Thus, it may as well have been a coincidental hypertensive ICH or an effect of the anticoagulation regimen that included urokinase in addition to heparin, aspirin, and clopidogrel. Corresponding to the cases mentioned above, the case described by Chamorro et al[11] showed radiologic signs of small vessel disease, which is claimed by the authors to be a possible risk factor to develop ICH after CAS.

Morrish et al[6] report a retrospective study of 104 CAS procedures in 90 patients; 4 patients developed ICH after the procedure, which is a quite high rate (4.4% of the patients). Two of them experienced hyperacute fatal ICH. Those 2 had previously presented with symptomatic high-grade stenoses; in both, ICH occurred in the basal ganglia without any prodromata before neurologic deterioration (Table).

Qureshi et al[12] have published a series of 7 patients with fatal ICH after a neurointerventional procedure under a special anticoagulation regime that included abciximab in addition to clopidogrel, aspirin, and heparin. In 4 patients, the extracranial ICA was treated (Table). They do not give the total number of patients treated, so the percentage of occurrence of the complication remains unclear. However, all of the patients who experienced fatal ICH had symptomatic high-grade stenoses before CAS. Fatal ICH followed the intervention with a short delay ranging from 10 minutes to 8 hours. Most of the ICH occurred in basal ganglia; the authors did not show radiographic data for each patient.

The case described by Friedman et al[13] about hyperacute fatal ICH in the ipsilateral thalamus is interpreted by the authors as ICH in the "wrong territory," in that the posterior communicating artery was hypoplastic and ICH occurred in a territory not likely to be part of the ICA territory. The CT images shown in the article do not completely convince that the origin of ICH is the posterior thalamus and therefore part of the posterior circulation. It could as well be interpreted as a primary basal ganglia hemorrhage in the territory of the anterior perforating arteries. However, the symptomatic patient had high-grade stenoses in both ICAs and cranial MR imaging (radiographic data not shown in the report) revealed a diffusion-restricted area in the corona radiata as sign of acute hemodynamic infarction.

Abou-Chebl et al[14] report a retrospective single-center study on 450 patients who had been treated with CAS. Three patients (0.67%) are reported to have developed ICH after the intervention. The authors do not give much specific information about the individual patients, but 1 of the 3 is reported to have experienced hyperacute fatal ICH that occurred approximately 1 hour after the intervention. ICH in this case was

Characteristic features of cases reported with hyperacute ICH following CAS

Author	Age (y)/ Sex	Vessel Treated (NASCET)	Other Territories	Vascular Risk Factors	Time from Last Symptom	Radiographic Pathologies before Treatment			Blood Pressure (mmHg)			Anticoagulation (Procedural)		Time between End of Procedure and Setup of Neurologic Event	Localization of ICH	HPS Suspect Prodro-mata	Clinical Out-come
						Embolic	Hemodynamic	Microangio-pathic	During Procedure	After Procedure	During Neurologic Event	Heparin	Others				
McCabe et al[9]	68/M	Left ICA (95%)	No data	Smoking, alcohol	5 mo, incomplete remission	Cortical	None	Moderate periventricular	160/90–175/ 105	140–160/95	150/85	5000 IU	ASA, dipyridamole	7 h	Ipsilateral basal ganglia	None	Fatal
Mori et al[10]	71/M	Left ICA (99%)	No data	Hypertension	None, recurrent TIAs	None	None	Yes	≤142 systolic	No data	170–190 systolic	10 000 IU	Warfarin, ticlopidin	4 h	Ipsilateral basal ganglia	None	Fatal
Morrish et al[6]	87/M	Left ICA (95%)	No significant stenosis in contralateral ICA	No data	4 mo	None	None	Subcortical	No data	110/60	125/80	7000 IU	ASA, ticlopidin	10 h	No data	None	Fatal
Morrish et al[6]	62/F	Right ICA (95%)	No significant stenosis in contralateral ICA	No data	6 wk	Large cortical	None	None	No data	173/64	173/64	9000 IU	ASA, ticlopidin	15 min	Ipsilateral basal ganglia	None	Fatal
Chamorro et al[11]	43/M	Right ICA (95%)	No stenosis	Hypertension	7 d	Acute embolic	None	Lacunar	135/75–180/ 80	100/60–125/ 60	180/75	5000 IU	ASA, clopidogrel, urokinase	~6 h	Contralateral basal ganglia	None	Fatal
Qureshi et al[12]	66/F	ICA (99%)	No data	Hypertension, smoking, hyperlipid-emia	2 d after ischemic stroke	None	None	Lacunar	No data	Max. 165 systolic	No data	ACT: 356 s	ASA, clopidogrel, abciximab	1 h	Basal ganglia	None	Fatal
Qureshi et al[12]	56/M	ICA (90%)	No data	Hypertension, coronary and peripheral artery disease	4 d after TIA	None	None	None	No data	Max. 199 systolic	No data	ACT: 180 s	ASA, clopidogrel, abciximab	8 h	Basal ganglia	None	Fatal
Qureshi et al[12]	61/M	ICA (90%)	No data	Coronary and peripheral artery disease	4 wk after ischemic stroke	Multiple small infarcts ipsilateral to stenosis	None	None	No data	Max. 187 systolic	No data	ACT: 320 s	ASA, clopidogrel, abciximab	10 min	Lobar	None	Fatal
Qureshi et al[12]	46/F	ICA (80%)	No data	Hypertension, smoking	1 d after TIA	None	None	None	No data	Max. 260 systolic	No data	ACT: 273 s	ASA, clopidogrel, abciximab	1 h	Lobar	None	Fatal
Friedman et al[13]	82/M	Right ICA (90%)	Left ICA (60%), no collateraliza-tion from anterior or posterior communi-cating arteries	Positive family history	Few days	None	Yes, restricted diffusion	None	~140 systolic	~140 systolic	No data	5000 IU	ASA, clopidogrel	45 min	Ipsilateral basal ganglia	None	Fatal
Abou-Chebl et al[14]	Not appli-cable	Left ICA (99%)	Right ICA (80%)	Hypertension	Not applicable							~5000 IU (ACT > 250 s)	ASA, clopidogrel	1 h	Ipsilateral basal ganglia	None	Fatal
Case reported	65/F	Right ICA (95%)	Left ICA (80%), proximal VA stenosis bilateral	Hypertension, smoking, alcohol, positive family history	Maximum 14 d, incomplete remission	Cortical	Yes, restricted diffusion	Mild	140/70	120/70	Max. 160 systolic	5000 IU (ACT: 278 s)	ASA, clopidogrel	30 min	Ipsilateral basal ganglia	None	Fatal

Note:—NASCET indicates North American Symptomatic Carotid Endarterectomy Trial; ICA, internal carotid artery; VA, vertebral artery; TIA, transient ischemic attack; ASA, acetyl salicylic acid; ACT, activated clotting time.

extensive and occurred in the ipsilateral basal ganglia with extension to the subarachnoid space; again, the patient showed no prodromata.

Further reports on results and complications after CAS have been published. Some cases appear similar to those mentioned above. Masuo et al[15] report 2 cases of intracranial hemorrhage after CAS. One of the patients experienced an ipsilateral basal ganglia ICH that occurred the day after CAS without any focal prodromata but headache since the procedure. ICA-stenosis had been high grade, but ICH was not fatal in this case. The series of 161 patients reported by Koch et al[16] contains 1 case of fatal ICH after the intervention in under 10 hours. In this case, the carotid stenosis had been high-grade and symptomatic as well. They do not give information about the location of the ICH. Coutts et al[17] report a series of 129 cases of CEA and 44 cases of CAS in which the existence of 3 different entities of hyperperfusion syndrome is postulated: acute focal edema, acute hemorrhage, and the delayed classic presentation as described above.[1] In 3 cases (1 after CEA, 2 after CAS), hyperacute ICH occurred after the procedure. Two of the patients died of the complication, and all of them had had high-grade stenoses. At least 1 of the fatal cases symptomatic on presentation had a comorbidity that was not well described. This patient was treated with CAS. No postinterventional hypertension and no prodromata were documented in this particular case until sudden deterioration occurred 3 hours after the intervention.

Discussion

At least 2 different types of hyperperfusion injury in brain parenchyma may have to be discriminated. Sundt et al[1] described the clinical presentation of a HPS that typically developed 5 to 7 days after CEA. Patients with this complication typically present with neurologic signs of elevated brain pressure, such as severe headache, nausea, and seizures. ICH is not an obligatory component of "classic" HPS but may occur in some cases. Outcome may vary from complete restitution to fatal. The "classic" HPS described by Sundt et al[1] has been reported after carotid angioplasty as well. Schoser et al[8] were the first to describe cases in which the patient presented with that delayed complication. Recent studies report quite different rates of HPS after endovascular treatment of the carotid arteries that range from 1.1%[14] to 5%.[18]

The second type of brain injury complication after treatment of an ICA stenosis occurs in a different and more dramatic way. Hyperacute fatal ICH after CEA has been described and considered to be due to hyperperfusion injury.[4,19,20] Coutts et al[17] were the first to distinguish between early and late types of presentation of hyperperfusion injury after CEA as well as CAS. In a retrospective review of their own cases (129 of CEA and 44 of CAS), they found that 3 entities of HPS could be differentiated, 2 early types and the "classic" delayed type of presentation. The 2 early categories include acute focal edema (2 patients) with favorable outcome on the one hand and acute ICH (3 patients) with often fatal outcome on the other. From a pathophysiologic point of view, one could differ between a primary ICH as 1 type of complication (often hyperacute and fatal) and a primary brain edema (which does not always occur hyperacutely and is not necessarily associated with ICH) as the second type. Outcome is better by far in this second type.

In this report, we concentrate on CAS as the underlying procedure. Concerning CAS, the first case of hyperacute fatal ICH was published by McCabe et al.[9] All well-documented cases we found in the literature are detailed in Table 1.

Possible risk factors for hyperacute ICH after CAS have been discussed. McCabe et al[9] and Chamorro et al[11] claim that the presence of microvascular changes indicates a higher risk of developing a hyperperfusion injury. Insufficient intracranial collateralization and signs of cerebral microangiopathy are frequently present, if documented.[6,9-12] Our patient showed these attributes as well. The small infarction declared by Mori et al[10] as the origin of the hemorrhage seems more likely to have been a lacunar residuum that had already become isointense to CSF, thus corresponding to microangiopathy. It is well known that hypertensive encephalopathy does not only consist of periventricular demyelinization but possibly also small areas of perivascular hemorrhage that are associated with higher risk of hypertensive ICH.[21,22] Thus, the hypodense lesions on cranial CT could be interpreted as a morphologic indicator for risk but not as origin of hemorrhage.

Nearly all reports on ICH soon after carotid recanalizations in general and CAS in particular have in common patients who had high-grade stenoses in the treated vessel (Table 1). Few reports on ICH in patients with possibly hemodynamically symptomatic carotid stenosis are supported by radiographically documented hemodynamic lesions.[8,13] Thus, it is often unclear whether stenoses were symptomatic of hemodynamic or embolic causes. Our case is the first in which diffusion-weighted MR imaging demonstrated ipsilateral hemodynamic lesions before the intervention followed by a "mismatch" between the parenchymal lesions and the region of ICH. With our data, we can prove that the stenosis treated was definitely hemodynamically symptomatic. Only Friedman et al[13] report a somewhat similar case. However, they focus on an ICH having occurred in the territory of the posterior circulation and therefore not due to hyperperfusion injury. They describe their patient as having a small diffusion-restricted area in the ipsilateral corona radiata but show no MR imaging data. So, it remains unclear whether this infarction could be claimed as hemodynamic or microangiopathic.

Lovblad et al[23] have demonstrated that interventional treatment of the carotid artery stenosis can lead to silent or symptomatic infarction. In our case, infarction seen before the procedure was not in the area of hemorrhage. Because no MR imaging could be performed between the procedure and the fatal bleeding event, we cannot exclude the possibility of hemorrhage into an acute infarction. However, this seems very unlikely, because our patient did not develop new neurologic signs before the dramatic clinical deterioration. In addition, in all reports of similar cases, there were no focal neurologic signs before deterioration (Table). In contrast, early stent placement in patients with diffusion-restricted lesions is considered as a feasible treatment option. Zaidat et al[24] report that patients with smaller infarction volume and mild neurologic deficits can be relatively safe to treat in acute stroke. The recent case report by Geisler et al[25] about CAS in acute hemodynamic stroke supports these findings.

All well documented cases with early ICH after CAS[6,9-14] report extensive and subsequently fatal ICH in the basal gan-

glia occurring without any prodromata, as described above. Thus, the most striking similarities among all these cases may be not only the devastating outcome but also the time course and the localization of ICH. Excessive basal ganglia hemorrhage is a complication well known in patients with arterial hypertension.[26,27] We believe that the pathophysiologic mechanism of this early variant of HPS must be different from that of the "classic" HPS. Although the delayed HPS is believed to be due to impaired cerebral autoregulation, the early variant—like typical hypertensive ICH—might be due to rupture of small perforating arteries in the basal ganglia that are acutely exposed to suddenly normalized perfusion pressure after angioplasty of a high grade stenosis. This theory is additionally supported by the Doppler-sonographic findings of Niesen et al[28] who measured hemodynamic parameters before and in the first hours after CAS of high-grade stenoses and did not depict signs of hyperperfusion but normalization of previously impaired cerebral perfusion.

Along with Coutts et al,[17] we propose a more differentiated view on the term hyperperfusion syndrome. The mechanism of parenchymal injury in all cases of hyperacute ICH we found seems to be not hyperperfusion but rather blood pressure normalization. Therefore, it might be justifiable to not call that complication HPS and to treat it in a different way. Not only monitoring but also, if possible, moderate lowering of blood pressure during and especially after the procedure might help to prevent this often fatal complication in patients with severe vessel disease.

Conclusions

Our findings support the existence of a second pathophysiologic mechanism leading to ICH as a complication after CAS that is most probably different from that of the "classic" HPS. A possible explanation—as in hypertensive ICH—is the rupture of small perforating arteries in the basal ganglia that are acutely exposed to suddenly normalized perfusion pressure. In high-risk patients with severe vessel disease and high-grade stenosis, an antihypertensive treatment during and after the intervention might therefore be helpful in preventing that severe complication.

Acknowledgments

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