Hyperperfusion syndrome following endovascular cerebral revascularization

A review

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Endovascular cerebral revascularization is becoming a frequently used alternative to surgery for the treatment of atherosclerotic disease, especially in the intracranial circulation where options are limited. Recent literature regarding the equivalent efficacy of carotid artery stenting and carotid endarterectomy in certain patient populations, as well as the recognition of the significant risk for recurrent stroke posed by intracranial lesions, will only serve to amplify this trend. Hyperperfusion syndrome has been well documented in the setting of carotid endarterectomy; however, a paucity of literature exists regarding the incidence, pathophysiology, and management as it relates to percutaneous interventions. The purpose of this review is to outline the current state of knowledge, with particular attention to the distinct attributes of endovascular treatment that would be expected to modify the course of hyperperfusion syndrome. (DOI: 10.3171.2009.1.FOCUS08276)

KEY WORDS • angioplasty • carotid artery stenosis • hyperperfusion syndrome • intracranial atherosclerosis • stent

ENDOASCULAR cerebral revascularization is being increasingly used as an alternative to surgical intervention, especially in the management of intracranial atherosclerosis. Recent literature revealing a similar efficacy between CAS and CEA in certain populations, as well as the escalating recognition of intracranial lesions as a significant contributor to the incidence of ischemic stroke, has only served to augment this trend.^{5,12,41} Concerning intracranial lesions, treatment was previously limited to medical therapy, including anticoagulation treatment, and the modification of risk factors. However, since the pioneering work of Sundt et al.,³⁷ endovascular management has become an efficacious alternative.⁷ Furthermore, substantial impetus for persistent evolution exists, as despite the implementation of maximal medical management, stroke recurs with considerable frequency.^{5,14,15} Consequently, the use of endovascular cerebral revascularization has intensified. Unfortunately, as is the case with any procedure, this therapy is not entirely without adverse effects.

In 1978, Spetzler et al.³⁴ first described the “normal perfusion pressure breakthrough theory” to describe the precipitous decline observed in a 49-year-old woman following the resection of a large right parietal arterio-

venous malformation. Despite a technically successful surgery the patient died of cerebral edema and hemorrhage, a phenomenon hypothesized to occur as a result of impaired autoregulation in an area of chronic ischemia. Sundt and colleagues³⁶ later provided the first detailed description of HPS following CEA. In their analysis of 1145 patients undergoing CEA, they noted a constellation of delayed-onset symptoms including atypical migrainous phenomena, focal seizures, and ICHs in a minority of patients, with a predilection for those having > 100% increase in CBF from baseline. As recognition of HPS broadened,³ multiple clinical and laboratory investigations were conducted to better elucidate the incidence, treatment, and pathophysiology of reperfusion injury in this setting.^{27,28,32,35} Notwithstanding this, evidence regarding HPS following CAS remains sparse, in addition to the significant void that exists when considering intracranial revascularization. As such, the purpose of this review is to evaluate the current literature with special attention to the distinct attributes of endovascular therapy expected to modify the course of HPS that has previously been outlined for CEA.

Hyperperfusion Syndrome

Epidemiology and Clinical Presentation

Hyperperfusion injury is estimated to occur in 1.1–

Abbreviations used in this paper: CA = carotid artery; CAS = CA stenting; CBF = cerebral blood flow; CEA = carotid endarterectomy; HPS = hyperperfusion syndrome; ICH = intracerebral hemorrhage.

TABLE 1: Incidence of hyperperfusion syndrome following endovascular revascularization of the craniocervical arteries*

Year	Authors	Intervention	No. of Cases	Hyperperfusion	Associated Factors
2000	Meyers et al.	craniocervical PTA/stenting	140	5.0%	
2000	Morrish et al.	CAS	102	3.9% ICH	
2003	Coutts et al.	CAS	44	6.8%	
2004	Abou-Chebl et al.	CAS	450	1.1%	severe stenosis; contralateral stenosis; hypertension
2004	Kaku et al.	CAS	30	30%	age (>75 yrs); CVR < 20%
2006	Terada et al.	intracranial PTA/stenting	106	2%	age (>70);† severe stenosis; poor collaterals; low perfusion; & poor reactivity‡
2004	Zaidat et al.	early CAS	39	2%	
2007	Abou-Chebl et al.	CAS	836¶	BP-controlled group 0.5%; noncontrolled group 1.9%	hypertension
2007	Ogasawara et al.	CAS	2898	1%	none (for ICH)
2008	Miyamoto et al.	early CAS	10	0%	

* BP = blood pressure; CVR = cerebrovascular reactivity; PTA = percutaneous transluminal angioplasty.

† None of the variables listed demonstrated statistical significance.

‡ As assessed by SPECT after an acetazolamide challenge.

¶ Includes a portion of the patient population in the previously presented data by the same first author.

6.8% of patients following endovascular cerebral revascularization, with mortality rates ranging from 3 to 26% when ICH is present (Table 1).^{2,6,20,27,39} Although this occurs with similar frequency to that seen following CEA,²⁷ several investigators have recognized that for endovascular treatment there often exists a differing postprocedural time course.⁶ Consistent with the description by Sundt and colleagues,²⁷ the syndrome usually occurs 5 to 7 days following endarterectomy,^{27,36} whereas with CAS it is more likely to occur within the first 24 hours.^{2,6,10,17,18,21,23,25,27,29,30,33,38,43} Concurrent with these observations, Coutts et al.⁶ have proposed that HPS be divided into 3 categories based on presentation: acute hemorrhage, acute focal edema, and delayed-classic presentation. In one of the largest published series containing 4494 patients, Ogasawara and colleagues²⁷ found that onset occurred 5.8 days following CEA in contrast to 1.5 days after CAS ($p < 0.0001$). The authors hypothesized that this variation occurred as a result of 2 factors: 1) a greater incidence of distal emboli and 2) more profound cerebral ischemia with a subsequent increase in the production of reactive oxygen species following stent placement. Concerning distal emboli, this would represent hemorrhagic transformation and not hyperperfusion, but this too remains questionable, as their definition of HPS appropriately excluded patients in whom there was evidence of restricted diffusion on postoperative MR imaging. Regarding increased ischemia, the authors proposed that this occurred in conjunction with transient bradycardia and hypotension due to damage to the carotid baroreceptor,¹⁹ yet no evidence exists that this is significantly different from that seen with cross-clamping during surgery. Overall, these

explanations are insufficient and serve to underscore an underlying variation in the pathophysiology based on the modality of treatment.

Pathophysiology

Together carbon dioxide and cerebral autoregulation serve to maintain the consistency of cerebral perfusion over a wide range of systemic blood pressures (60–160 mm Hg).^{16,40} While carbon dioxide significantly influences the smaller arteries, it has little effect on the CAs or the circle of Willis, areas where myo- and neurogenic autoregulation dominate. Increases in blood pressure cause stretching of the vascular smooth muscle, resulting in depolarization and subsequent contraction that limits perfusion. Once the pressure exceeds the ability of this mechanism, further vasoconstriction is dependent on the sympathetic innervations of the vascular adventitia. Notably, the vertebrobasilar circulation has a lower density of this adventitial innervation, making it potentially more susceptible to extreme changes in systemic blood pressure.^{16,40}

Ischemic conditions have variable effects on the vasculature, which are dependent on the duration of exposure. Acutely, this leads to endothelial changes that initiate leukocyte and platelet adhesion and subsequent activation. This process can lead to irreversible vascular injury within hours, noted by a breakdown of the blood-brain barrier beginning in the venules. Chronically, ischemia causes maximal dilation of the resistance arteries and arterioles, with a consequent decrease in their responsiveness and tonicity.¹⁶ In addition, the endothelial damage to the microvasculature produced by diabetes and hypertension,

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TABLE 2: Risk factors for the development of HPS*

patient age >75 yrs
long-standing hypertension
history of prior stroke
recent stroke (see Discussion)
poor cerebrovascular reserve
severe stenosis ($\geq 90\%$)
contralateral stenosis
poor collateralization
postprocedural hypertension

*Derived from the works of Abou-Chebl and colleagues (2004), Kaku and associates, and Terada and coworkers.

common comorbidities in this patient population, have also been shown to impair myogenic regulation.⁴⁰ Ensuing reperfusion of the affected territory, even under normotension, is marked by a substantial increase in blood flow. In conjunction with capillaries and venules being exposed to unregulated arterial pressure, the generation of reactive oxygen species occurs, further potentiating endothelial damage.^{35,40} The hydrostatic forces lead to the development of edema, and if sufficient vascular injury has occurred, or if the blood pressure is uncontrolled, intracranial hemorrhage ensues.

While the aforementioned may help explain HPS in general, it does little to elucidate a physiological disparity between the 2 procedures that would explain the variant time course. Investigators have evaluated the hemodynamic changes that occur following CAS and CEA,^{19,24,32} but the only significant difference was more frequent and prolonged hypotension over the first 24 hours after CAS.¹⁹ Other theories include the potential propensity of endovascular therapy to result in a larger ischemic burden as a result of distal emboli, yet this is unlikely as in nearly all of the reported cases authors have described sudden onset without prodromal symptomatology.^{4,6} Nevertheless, a greater incidence of clinically silent ischemia cannot entirely be ruled out. Another potential contributor, at least for the development of ICH, is the routine use of dual antiplatelet therapy following angioplasty and stent therapy.¹ Consistent with this is the slightly higher rate of ICH that has been consistently demonstrated following percutaneous intervention.^{6,23,27}

Management of HPS

Risk Factors. Appropriate management begins with preprocedural evaluation to identify those patients at greatest risk (Table 2), as the prevention of HPS is the primary objective given that treatment is largely symptomatic. Risk factors include patient age (> 75 years), history of stroke, long-standing hypertension, severe stenosis, contralateral stenosis, and poor collateralization.^{2,13,40} The latter 3 factors are indicative of poor cerebrovascular reserve, a parameter that may be evaluated using advanced imaging such as CT perfusion scanning (Figs. 1 and 2), SPECT, perfusion-weighted MR imaging, and dynamic

susceptibility contrast MR imaging.^{9,13,40} Kaku et al.¹³ retrospectively analyzed the results obtained in 30 patients who underwent CA angioplasty and stent placement. All patients underwent preoperative assessment of cerebrovascular reactivity by way of an acetazolamide challenge using SPECT. Cerebrovascular reactivity was calculated as the CBF during the acetazolamide challenge minus the resting CBF divided by the resting CBF. Those with a cerebrovascular reactivity of < 20% were more likely to develop hyperperfusion following the procedure. This strategy may allow the identification of individuals in whom incremental revascularization would be preferred. Such patients may benefit from serial procedures with initial underinflation to permit the vascular territory sufficient opportunity to regain the ability to autoregulate, the same idea advocated by Spetzler et al.³⁴ in their early description.

Prophylaxis. As an alternative to these advanced imaging techniques, some writers have advocated the uniform initiation of strict blood pressure control in the early postprocedure period. Abou-Chebl and colleagues¹ aggressively managed the blood pressure in 570 patients following CAS, and compared data acquired in these 570 patients with those in 266 patients treated prior to the institution of the protocol. Patients who were hypertensive at baseline had their home medications restarted, with those needing additional control receiving oral metoprolol and intravenous nitroglycerin as necessary. Additionally, patients were stratified based on their risk factors, with those having hypertension, $\geq 90\%$ stenosis, and/or $\geq 80\%$ contralateral CA stenosis, being held to < 120/80 mm Hg as opposed to 140/90 mm Hg for all others. This resulted in a nonsignificant decrease in the incidence of all HPS from 1.9 to 0.5%, and a significant decrease in the rate of ICH from 1.1 to 0% ($p = 0.032$). More remarkable results were obtained in the high-risk population, with the rate of HPS decreasing from 29.4 to 4.2% ($p = 0.006$). While

Fig. 1. Preprocedural CT perfusion scans (reference slice [A], cerebral blood volume [B], CBF [C], and mean transit time [D]) demonstrating an increase in mean transit time in the posterior aspect of the left middle cerebral artery (MCA) territory coupled with a decrease in CBF.

Fig. 2. Posttreatment CT perfusion images obtained after revascularization of the left internal carotid artery and left MCA, demonstrating a significant increase in the cerebral blood volume and CBF, consistent with hyperperfusion. Additionally, there is resolution of the previously noted alteration in mean transit time.

these results are suggestive, they have not been consistently demonstrated, as evidenced by the review of Ogasawara et al.²⁷ in which poor postoperative blood pressure control was not significantly associated with a decrease in the incidence of hyperperfusion following CAS. Nevertheless, most clinicians believe that the benefits substantially outweigh the risks, and therefore this strategy is routinely practiced at our institution.

Other interventions aimed at decreasing the incidence of HPS include the use of free radical scavengers such as edaravone. Edaravone inhibits lipid peroxidation, thereby preventing endothelial injury,⁴⁰ and recent evaluation of its use in the setting of acute stroke has demonstrated a significant improvement in functional outcomes as assessed by the modified Rankin Scale.⁸ Ogasawara and colleagues²⁶ pretreated patients undergoing CEA with the compound and observed a significant reduction in the incidence of HPS (2 vs 16%, $p = 0.0310$); however, its evaluation in the setting of endovascular intervention has been limited.

Therapy. Following symptomatic presentation, therapeutic measures must focus on preventing further progression and on imaging performed to evaluate for cerebral edema or hemorrhage. Primary interventions include aggressive titration of blood pressure to a systolic value of < 120–140 mm Hg² and initiation of an anticonvulsant regimen in the setting of seizures (prophylactic use is not indicated).⁴⁰ Substantial evidence does not exist regarding the agent of choice for blood pressure control; however, drugs such as nitroprusside, whose mechanism of action is vasodilation, should be avoided so as not to increase CBF. Should cerebral edema progress such that significant increases in intracranial pressure occur, one should institute control in an orderly fashion using sedation, osmotic agents (mannitol and hypertonic saline), and paralysis if necessary. Nevertheless, it cannot be overemphasized that

prevention remains the primary objective, as the presence of severe HPS heralds mortality and long-term morbidity rates of up to 50 and 30%, respectively.⁴⁰

Timing. The question of the ideal timing of intervention following an acute event is another considerable concern with regard to the development of HPS. Classically, operative intervention has been considered to increase the risk if it is performed within 3–4 weeks following an ischemic event.³ However, a recent analysis of the data from the European Carotid Surgery Trial and the North American Symptomatic Carotid Endarterectomy Trial revealed that the greatest benefit was achieved in patients randomized within 2 weeks of their last ischemic event.³¹ Other authors have more specifically addressed endovascular intervention with similar conclusions.^{11,22,42} Zaidat et al.⁴² reviewed the outcomes in 38 patients undergoing CA angioplasty and stenting within a mean of 55 hours following acute ischemic stroke. Three patients (7.7%) developed worsening neurological deficits, with 2 suffering from further minor stroke and 1 from abciximab-related ICH. Hyperperfusion syndrome did not occur in any patient. The authors concluded that in those with small infarct volumes (≤ 10 – 12 cm³) and mild to moderate neurological deficits (National Institutes of Health Stroke Scale score ≤ 8), early revascularization was safe if deemed clinically necessary.

Conclusions

The use of endovascular cerebral revascularization will continue to increase, especially in cases of intracranial lesions for which there exist limited therapeutic options. Although HPS has been characterized following CEA, there is sparse evidence regarding its occurrence after percutaneous intervention. Clearly there is some underlying variation in the pathophysiology, as the time course and incidence of HPS are not identical between the 2 groups. To date, aggressive blood pressure management is the most commonly instituted prophylactic measure, but even this does not have consistent support. New treatments such as edaravone hold promise for the prevention of HPS, with further evidentiary support pending. Future research must focus on evaluating the hemodynamic variations following angioplasty and stent placement, especially those that involve smaller vascular territories as seen in middle cerebral artery and posterior circulation revascularization. Precise characterization of the specific risk factors, including timing of intervention, and how they relate to the individual procedures, will allow clinicians to more appropriately guide patient care.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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