Periprocedural Hemodynamic Management for Transcarotid Artery Revascularization

Adequate blood pressure regulation is crucial for maintaining flow reversal and neuroprotection.

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The carotid baroreceptors regulate blood pressure (BP) and heart rate (HR) in response to the pressure on the arterial wall by altering sympathetic and parasympathetic activity.[1] It has been suggested that this baroreflex is dysfunctional in the setting of chronic illnesses such as hypertension, coronary artery disease, carotid artery disease, diabetes mellitus, as well as advanced age.[2] Throughout the literature, the general consensus on the definitions of hypotension, hypertension, and bradycardia in the perioperative period of a carotid revascularization is: < 100 mm Hg, > 160 mm Hg, and < 60 bpm, respectively.[1-8] However, the use of mean arterial pressure and its clinical correlations during carotid surgery is not well documented.

In the setting of carotid endarterectomy (CEA), the baroreceptor sensitivity is diminished with the surgical disruption and removal of the nerve endings, resulting in hemodynamic instability (hypotension or hypertension and bradycardia) in up to 55% of patients.[3-5] This hemodynamic instability may last hours to days.[6]

On the contrary, in the setting of carotid artery stenting (CAS), hypotension and bradycardia have been observed in up to 76% of patients, secondary to stimulation of the carotid body receptors from the angioplasty balloon and/or stent placement.[7] In CAS, hypotension and/or bradycardia may last 12 to 24 hours.[8] True hemodynamic instability was seen in 39.4% of CAS patients, and instability lasting > 1 hour was seen in 19.2% of cases.[9] These patients were at higher risk for postoperative cerebrovascular and cardiac ischemic events.[1-8]

Many studies have investigated the risk factors for hemodynamic instability after CAS including > 70% stenosis, severely calcified plaque, bilateral stenting, balloon dilation pressure > 8 atm, overlapping stents, symptomatic carotid disease, and intraprocedural hypotension.[7,10-12] One factor that has been shown to be protective against bradycardia and hypotension is previous CEA.[7]

Transcarotid artery revascularization (TCAR) offers a unique hybrid approach with direct access to the common carotid artery in the neck that avoids the navigation of the aortic arch as in transfemoral CAS, but also avoids surgical dissection of the carotid bifurcation, as in CEA. This carries the potential benefit of minimizing the number of events of labile BP or HR. However, appropriate BP control is essential to maintain robust flow reversal and neuroprotection.

INTRAPROCEDURE

Intraprocedural hemodynamic instability has been shown to be an important predictor of postprocedural hemodynamic complications. Most of the hemodynamic instability events during and after stenting are transient and self-limiting, with most patients experiencing transient bradycardia with or without asystole that resolves after balloon deflation and intravenous administration of glycopyrrolate or atropine. Administration of prophylactic atropine (0.5 mg intravenously) before balloon inflation during CAS decreases the incidence of intraoperative bradycardia and cardiac morbidity in primary CAS patients.[7] Periprocedural bradycardia, hypotension, and the need for vasopressors occur more frequently with primary CAS than with repeat CAS procedures. With a similar action on acetylcholine receptors, glycopyrrolate (0.4 mg intravenously) has a shorter duration of action and more predictable course than atropine. Furthermore, glycopyrrolate possesses a superior adverse effect profile with a markedly lower incidence of cardiac morbidity following its administration than observed following atropine.[13,14]

TCAR offers the advantage of neuroprotection with flow reversal before crossing the carotid atherosclerotic lesion. Flow reversal is based on the difference between the arterial BP in the common carotid artery and the common femoral vein. Keeping a constant systolic BP between 140 and 160 mm Hg is crucial to achieve

robust flow reversal for neuroprotection by recruiting oxygenated blood flow across the circle of Willis and other collateral pathways.

POSTPROCEDURE

BP management is a key component in the postoperative period with any carotid intervention. Strict monitoring with an indwelling arterial hemodynamic catheter is mandatory because hypertension or hypotension may lead to significant complications such as cerebral hyperperfusion syndrome (CHS) or watershed infarcts.

Hypertension

Maintaining systolic BP < 160 mm Hg or within 20% of the preprocedure value is recommended. It is mandatory to treat perioperative hypertension in a controlled and titrated manner using short-acting antihypertensive drugs. Data from literature comparing the efficacy among antihypertensive agents after carotid artery surgery are scarce. In addition, the wide variability of responses in patients makes it difficult to predict the most efficient drug. The efficacy of α- and β-blocking agents, such as labetalol and esmolol, have been shown to be suitable for the treatment of perioperative hypertension. Typical dosing for esmolol for rapid BP control includes an initial bolus of 1 mg/kg, followed by an infusion of 0.15–0.3 mg/kg/min titrated to the systolic BP. For gradual postprocedure control, an initial bolus of 0.5 mg/kg is followed by an infusion starting at 0.05 mg/kg/min that is titrated based on systolic BP. These agents have no cerebral vasodilatory effects and do not influence intracranial pressure.

Hypotension

Persistent hemodynamic instability (defined as hypotension; systolic BP ≤ 90 mm Hg) lasting for > 1 hour and requiring vasopressor therapy is present in 19.2% of patients undergoing CAS.[9] The implication of sustained or sudden hypotension is an increased risk for periprocedural major cardiac events and stroke.[1-8,14] To help prevent this occurrence, intravenous hydration, use of glycopyrrolate or atropine, and use of vasopressors is recommended. An algorithm for the management of periprocedural hypotension during CAS has been published.[14]

CEREBRAL HYPERPERFUSION SYNDROME

CHS constitutes an infrequent but devastating complication after CEA and CAS. First described in 1981 by Sundt et al, it is defined as a clinical triad that includes ipsilateral headache, transient focal seizures and intracranial hemorrhage (ICH).[15] The combination of hypoperfusion associated with a significant carotid stenosis, with impaired brain reserve due to inadequate collaterals leads to compensatory dilatation of the distal cerebral vasculature as part of the cerebral autoregulatory mechanism. Once the carotid stenosis is treated, there is loss of autoregulation with associated hyperperfusion in previously underperfused areas. The capillaries are then more prone to rupture, culminating in hemorrhagic infarct.

Data comparing postoperative CHS and ICH incidence between open and endovascular repair are limited. In a recent meta-analysis, CEA appeared to be associated with a higher risk for CHS compared to transfemoral CAS, although this difference was seen in the older studies.[16] It has also been suggested that there is an earlier onset of CHS after CAS, possibly due to the prolonged baroreceptor stimulation by the stent that may induce bradycardia, hypotension, and ischemic damage. Many factors have been attributed with the increased risk of CHS (eg, age, diabetes, poorly controlled preprocedure hypertension, recent contralateral CEA, contralateral carotid occlusion, exhausted cerebrovascular reserve), but postoperative hypertension and inadequate control of arterial BP are probably the most important and most preventable.

CONCLUSION

Hemodynamic instability after carotid intervention necessitates an offensive strategy of early management to prevent adverse sequelae. Maintaining systolic BP between 140 and 160 mm Hg during flow reversal and between 100 and 140 mm Hg postoperatively further compounds the safety and success of TCAR. ■

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