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Unexplained refractory prolonged hypotension after carotid endarterectomy

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Abstract
Acute hemodymanic instability after CEA is a well – recognized phenomenon. Both hypotension and hypertension can occur and may result in prolonged hospital admission or more seriously, may be associated with neurological complications. In almost all cases hypo/ hypertension is transient and rarely persist beyond 24 hrs. Herein we report an unusual case of unexplained, refractory prolonged hypotension after carotid endarterectomy.

Keywords: hypotension, unexplained, rarely, carotid

Introduction
CEA has become an important approach to the treatment of carotid stenosis and occlusion by removing atheroma from the carotid bifurcation and internal carotid artery. The surgery may decrease the subsequent risk of fatal or disabling stroke in patients with significant carotid stenosis. However, hemodynamic instability, especially postoperative hypertension, is very common after CEA], which can directly or indirectly influence morbidity and mortality after the surgery. It is also associated with increasing use of critical care facilities and longer hospitalization. A possible explanation for this phenomenon is that CEA would damage the carotid sinus and then impair BP homeostasis. In addition, other important factors also affect the hemodynamic instability, such as baroreceptor function, surgical factors and anesthetic factors.
The hemodynamic instability caused by CEA has a close relationship with the baroreceptor function. Baroreflex plays an important role in regulating arterial BP and is altered in different disease states, such as chronic hypertension, diabetes, recent transient ischemic attack or stroke, contralateral carotid stenosis and so on. Research has shown that patients with contralateral carotid stenosis are more likely to present baroreflex dysfunction and hemodynamic instability as well as a hypertensive shift after CEA. These comorbidities are prevalent in patients presenting for CEA. Surgical removal of the carotid plaque causes immediate partial disruption of baroreceptor activity, leading to hypertension and increased arterial pressure instability.
There is no significant difference in neurologic mortality when using either general (GA) or regional anesthesia (RA). However, the choice of the anesthetic method for CEA also affects the postoperative hemodynamic profile. CEA performed with RA leads to significantly less postoperative hemodynamic instability than CEA performed with GA, and these patients receive less vasoactive medications during surgery and have a shorter duration of hospitalization. However, the specific mechanism is unclear. Maybe the change of baroreceptor function caused by the manipulation is abolished by infiltration of the local anesthetic into the periadventitial tissue around the carotid sinus. Different GA drugs may have different effects on the cardiovascular function as well as baroreflex activity, resulting in differences in arterial pressure when using either RA or GA. This may give guidance for the patients with substantial cardiac risks who would poorly tolerate significant BP and heart rate shifts.

Case Presentation
A 55 year old male presented with h/o difficulty in speech and right sided Transient Ischemic attack – 2 weeks ago. He was normotensive (Blod Pressure: 130 /80 mm Hg) and diabetic for last 9 years. He had undergone PTCA 5 yrs ago.
On clinical evaluation, and investigations viz Color Doppler ultrasound & CT angio > than 90% stenosis of Carotid bulb and proximal ICA was observed. His Echocardiography showed good cardiac function i.e. LVEF was 40% and there were no regional wall motion abnormality.
He underwent standard, longitudinal left carotid endarterectomy with patch repair under general anaesthesia. On surgical exploration Carotid sinus nerve was blocked with 0.25% of lidocaine at carotid bifurcation. On arteriotomy, fibrocalcified plaque with ulceration causing severe stenosis was found. Intraluminal Shunt (Pruit – Inhara) was used. After endarterctomy, The arteriotomy was closed with Dacron patch 8 mm x 45 mm. During the operation, his blood pressure was well controlled.
During post-operative period in ICU, he required Ionotropes. Despite Dopamine infusion for 24 hours his remained hypotensive (SBP: 80 mm Hg) hence Dobutamine & Noradrenaline infusions were given for 7 days. On 3rd post-operative day, S. Creatinine started rising from 1.2 mg/ dl to 4.5 mg/ dl over successive 5 days. His Troponin – I was within normal limits. His central venous pressure was also within normal limits. We also gave Intravenous Methyl Prednisolone for 3 days which had only transient effect on elevation of blood pressure. There was no evidence of blood loss. His oxygen saturation as well as blood gas (ABG) was normal without oxygen support. His hypotension persisted for almost one week. After a week, gradually all iono tropes

were withdrawn and his blood pressure remained within normal range.

Discussion

The arterial baroreceptor mechanism is responsible for acute modulation of arterial pressure and it is altered in different disease states e.g. chronic poorly controlled hypertension and bilateral carotid artery disease which are usually prevalent in patients presenting for CEA.

Haemodynamic instability associated with CEA is likely related to the carotid baroreceptors. Carotid baroreceptors denervation causes increased arterial pressure variability, because of decreased vagal and sympathetic baroreflex sensitivity, although this does not lead to prolonged hypertension as other baroreceptors are able to maintain normal chronic arterial pressure [1]. Baroreceptor function is inevitably disturbed in patients undergoing CEA due to dissection of the carotid bifurcation leading to either temporary or permanent dysfunction of carotid sinus nerve [2].

Due to the effects of carotid atheroma on arterial baroreflex sensitivity, perioperative immediate partial disruption of baroreceptor activity leading to increased arterial pressure instability. This may last for several hours or days after surgery and may be caused by stripping of sensory nerve endings from the arterial lumen. It may explain why carotid sinus nerve block during surgery has variable effects on perioperative arterial pressure control [3]. Patients with significant contralateral carotid atheroma suffer more intraoperative and postoperative hypertensive episodes than those with normal contralateral arteries because of bilateral baroreceptors dysfunction and reduced baro reflex reserve [4]. Equally, recovering baroreceptor function after CEA may account for the improved arterial pressure control sometimes observed in the late postoperative period [5].

Preoperative risk factors are like short interval between symptomatic neurological event and the time of surgery, recent TIA or stroke, Bilateral carotid stenosis, Fibrous plaque in morphology , Previous contralateral CEA, Poorly controlled preoperative hypertension, recent alteration in antihypertensive medications after stroke, effects of age, diabetic status etc. Intraoperative risk factors are like eversion endarectomy, local anaesthetic carotid sinus nerve block, distance (< 10 mm) between carotid bifurcation and maximum stenosis.

Transaction or local anaesthetic block of the carotid sinus nerve during Eversion endarterectomy is associated with increased hemodynamic instability [5] Carotid sinus nerve block is no longer recommended as a routine procedure[3]. Hypotension is also associated with standard longitudinal CEA with the usage of patch due to widening of reconstructed artery. This restores the compliance of the arterial wall leading to hyperactivity of the carotid baroreceptors. The hyperactive baroreceptors lead to bradycardia and hypotension.

Cross – clamping of the carotid artery leads to a compensatory increase in arterial pressure mediated by baroreceptor reflexes and an increase in sympathetic nervous activity. This is reversed on restoration of blood flow either by application of a shunt or by unclamping at the end of surgery and a short period of mild hypotension may follow. The magnitude of these changes depends on the degree of ipsilateral stenosis, the integrity of the collateral flow, the duration of cerebral ischemia, and surgical and anaesthetic factors. Changes may be less noticeable in patients undergoing CEA under deep general anaesthesia (GA) as both baroreceptor function and cerebral auto regulation may be attenuated by high concentrations of volatile anaesthetic agents. The duration of cross- clamping may also affect haemodynamic stability and, while hypertension is common, postoperative hypotension has also been reported possibly related to very short carotid cross clamp times of 10 – 15 min.

Although GA provides better surgical conditions & cerebral protection and RA provides awake neurological monitoring, recent systematic reviews [6, 7, 8, 9] and RCTs have shown no differences in outcome.

IV & inhalation anaesthetic drugs and Opoids, both affect cardiovascular function in a dose dependent manner by reducing central sympathetic tone, attenuating baroreflex activity, and by direct effects on the heart and peripheral vasculature [10].

To conclude, Mild hypotension is very frequent and rather nonspecific during carotid procedures but severe and persistent hypotension correlates to severe neurological events. Hence aggressive treatment of hypotension during surgery should be attempted to avoid neurological complications. Surprisingly our patient did not develop any major or minor neurological event. We believe persistent and refractory hypotension in our case was due to carotid sinus nerve blockade with local anaesthetic and /or compensatory response to increase in arterial diameter due to use of patch.

Conclusion

Hemodynamic changes are closely related to BRS variation. Severe hypertension occurs more frequently after CEA, while hemodynamic depression including perioperative hypotension or bradycardia occurs more frequently after CAS. Since hemodynamic instability is related to the patients' postoperative recovery, it is necessary to prevent or treat these conditions early. There are controversial opinions on the changes of BRS caused by CEA and CAS. However, there is no evident difference in short-term and long-term outcomes of the two main interventional ways to treat carotid artery stenosis. CAS is an alternative method to CEA, especially for patients at a high surgical risk and those younger than 70 years. Here we discussed the hemodynamic instability and the variation of BRS. The results may provide useful information for the choice between the two interventional methods. However, these conclusions still need more experiments to confirm the effects. Most importantly, we should pay more attention to the physiological index of BRS in the future.

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