Sinus Bradycardia Persisting for 9 Days after Carotid Angioplasty and Stenting

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Introduction

Carotid angioplasty and stenting (CAS) has gained popularity in recent years as an option for treating extracranial carotid occlusive disease. Despite having the clear advantages of less invasiveness and postprocedural discomfort, angioplasty is associated with several complications, mainly hemodynamic depression and embolic events[1].

Stenting procedures are commonly considered for patients at high risk for endarterectomy because these procedures arguably decrease the risk of stroke, death, and myocardial infarction in this specific group[2,3]. However, precise selection criteria have not been determined for this approach, and the surgeon's experience or preference is usually the determining factor.

The present case is important because it shows that postprocedural bradycardia may present as a prolonged condition rather than as a short-lived complication easily controlled in the operating room[4].

Case Report

A 76-year-old previously asymptomatic male presented at hospital admission with recurrent transient ischemic attacks. The symptoms reflected involvement of right medial cerebral artery territory and were attributed to ipsilateral carotid obstruction, which was confirmed by imaging studies. The ischemic attacks lasted 10–30 minutes and manifested as aphasia and left hemiparesis.

The patient had a clinical history of stable coronary artery disease, hypertension, hypercholesterolemia, and chronic (non-dialytic) renal insufficiency with baseline creatinine clearance of 20 mL/min. His surgical history included cardiac revascularization at the age of 45 years, and left internal carotid angioplasty and stenting after a stroke episode in 2004, which left no persistent deficits. Recent myocardial perfusion scintigraphy showed no signs of myocardial ischemia except for fibrosis in the inferior wall; gated SPECT revealed mild left ventricular dysfunction (EF = 48%). In addition, the patient had an asymptomatic abdominal aortic transrenal aneurism (7 cm), for which surgical intervention had been postponed following the recent neurological events.

Computed tomographic angiography of the neck was not performed because of the patient's renal dysfunction, but duplex ultrasound revealed stenosis greater than 70% with an unstable and calcified plaque in the internal right carotid artery, and a patent stent (< 50% stenosis) in the internal left carotid artery. Due to the severity of the stenosis and the increasing frequency of attacks (refractory to clinical treatment despite full anticoagulation and antiplatelet therapy), the patient was considered for intervention at 6 days after the initial event.

A less invasive approach was chosen because of the overall high risk of the patient's clinical conditions, and CAS was performed following the technique used in most standardized protocols. The procedure was performed in an operating room and the patient was monitored by an anesthesiologist; anxiolytic medication was administered for comfort. The right common carotid artery was easily cannulated and a selective angiogram of the carotid bifurcation showed the exact location of the target lesion. The stenosis was easily crossed and a distal cerebral protection device was positioned in the ICA. A self-expanding stent (Wall-Stent, 7 x 30 mm) was deployed in the internal carotid artery followed by dilatation with a 5 x 20 mm balloon for no more than 2 seconds. A control angiogram of the cervical carotid circulation showed adequate flow through the previously stenotic lesion.

The surgical procedure was flawless and no significant hemodynamic complications were observed during the intra-operative period, except for transient sinus bradycardia (50 bpm) at the time of balloon inflation, effectively normalized with atropine (1 mg) administration. A minor stroke was noticed after the patient had completely regained consciousness, with left hemiparesis that persisted for 24 hours after the surgical event.

At 12 hours after intervention, the patient already showed significant improvement of the neurologic deficit, but persistent asymptomatic sinus bradycardia (mean heart rate = 35 bpm) was observed, with no accompanying hypotension or ischemic electrographic alterations. Bradycardia showed only temporary chronotropic response to bolus infusion of atropine (0.5 mg), raising the heart rate to 85 bpm. The patient had been prescribed long-term carvedilol (12.5 mg/day) and his heart rate before the procedure was 55–60 bpm. He was taking no other medication with a chronotropic effect.

Considering the severe level of bradycardia in the acute phase of the stroke, beta-blocker therapy was suspended

Keywords

Bradycardia; arrhythmias, cardiac; angioplasty; stents; carotid arteries.

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and continuous infusion of dopamine was initiated; the dose was titrated up to 10 mcg/kg/min, to maintain a heart rate of 45–50 bpm. Several attempts were made to minimize the infusion rate of medication, all of which were followed by reestablishment of the persistent bradycardia. Blood pressure levels remained stable throughout hospital admission (around 140 x 90) and did not fluctuate during the attempts to remove intravenous medication. After 9 days of dependence on vasopressor treatment, the patient regained adequate control of cardiac frequency, enabling the full withdrawal of medication. Twenty-four-hour ECG-monitoring performed after the suspension of dopamine revealed an average heart rate of 52 bpm with an appropriate variability, similar to the pre-operative pattern.

Discussion

Postprocedural hemodynamic depression is a well-described complication related to carotid angioplasty. It is caused by the direct mechanical dilation of the carotid artery and bulb, which leads to an increase in parasympathetic discharge[5,6]. This condition is generally self-limiting, being triggered after balloon dilation during the transoperative period, and with an incidence rate varying from 19% to 68%[1,4,7]. Sometimes, however, such as in the present case, hemodynamic depression may manifest several hours after the surgical manipulation, justifying the need for monitoring for a minimum of 12 hours[8].

Studies evaluating predictors for hemodynamic depression after CAS have suggested various risk factors associated with the development of this complication, all of which were observed in the present patient. High-grade stenosis (>70%) as well as unstable and calcified plaque morphology lead to more intense parasympathetic stimulation after stent implantation; this results in the stent exerting a greater force on a less compliant arterial wall[7]. Lesions close to the carotid bifurcation, where the greatest concentrations of baroreceptors are located, also predict the presence of post-operative hemodynamic depression.

Advanced age, low cardiac ejection fraction, and coronary disease are other previously documented risk factors. Older patients with impaired cardiac function are more vulnerable to hemodynamic instability due to a lack of myocardial reserve and age-related poor neuronal response mechanisms.

Diabetes mellitus, a history of smoking, and previous endarterectomy are conditions that reduce the risk of hemodynamic instability after CAS[1,7]. Diabetes mellitus interferes with cardiovascular autonomic response by reducing parasympathetic nerve function, which may attenuate the carotid baroreceptor stimulation triggered by carotid manipulation. Similarly, long-term smoking alters baroreceptor response and augments sympathetic tone, thereby raising heart rate and blood pressure. The reduced number of baroreceptors in the carotid bulb after endarterectomy also acts as a protective factor.

Chronic beta-blocker use could be a confounding variable, but the appropriate heart rate observed before CAS, the degree of bradycardia after the procedure, and its long persistence after medication withdrawal suggests minor or no beta-blocker effect as a causal factor or even as a maintaining factor of the described complication. A retrospective analysis of 500 CAS procedures showed that beta-blocker therapy is in fact associated with a reduced risk of developing persistent hemodynamic depression[7].

In terms of technical approach, the use of balloon angioplasty increases the risk of hemodynamic instability because of the greater radial force exerted on stretch receptors of the carotid sinus. The deployment of self-expandable stents only, without angioplasty, is a protective factor[6]. However, such practice is uncommon because without proper post-deployment stent dilation, significant residual stenosis generally persists.

Sinus bradycardia is an autonomic response of variable duration: the longest reported period over which vasoactive drugs were required is 96 hours[7]. To the best of our knowledge, the present case of 9 days of persistent bradycardia is the longest documented in the literature. This case reinforces the importance of cardiac monitoring after CAS, and the potential for bradycardia to be reversed after a long period, without pacemaker therapy.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

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