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Case Report

Severe hemodynamic depression after carotid artery stenting: The problem overcome with a transvenous temporary cardiac pacemaker

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ABSTRACT

Background: Carotid angioplasty stenting (CAS) may have adverse events including perioperative hemodynamic depression. A transvenous temporary cardiac pacemaker (TTCP) is an option for preventing devastating sequelae due to circulatory failure. An exploration of the predictors of hemodynamic depression following CAS is valuable for selecting candidates for preoperative TTCP implantation before CAS.

Case Description: An 84-year-old man underwent CAS for asymptomatic left carotid severe stenosis. He had no history of bradycardia arrhythmia. A TTCP was implanted in advance in view of the likelihood of perioperative hemodynamic depression. CAS was accomplished successfully, but severe hypotension and vanishing of self-heartbeat occurred about 90 min after the procedure. By activating the pre-implanted TTCP, spontaneous circulation was readily recovered with vasopressor administration. He was discharged with no additional neurological deficits. A literature review using a random effect model found that smoking (odds ratio [OR] 1.68, 95% confidence interval (CI) 1.13–2.52) and severely calcified plaque (OR 3.70, 95% CI 2.15–6.35) were significant predictors of perioperative hemodynamic depression following CAS.

Conclusion: TTCP can be recommended for a patient receiving CAS to prevent catastrophic consequences, particularly in cases with a history of smoking or severely calcified plaque.

Keywords: Bradycardia, Carotid artery stenting, Hypotension, Predictor, Temporary cardiac pacemaker

INTRODUCTION

Carotid artery stenosis is one of the causes of ischemic stroke, in which surgery is a treatment option for selected cases. Carotid endarterectomy (CEA) is the standard long-standing surgical procedure and was proved to be effective in preventing ischemic stroke in patients with moderate to severe carotid stenosis as long as three decades ago.[7,9] Because of recent advances in neuroendovascular surgery, carotid artery stenting (CAS) has become an alternative strategy for CEA. Its outcomes compared with CEA have not been inferior in prospective randomized trials in symptomatic or in asymptomatic patients.[2,3,20,21]

Apart from perioperative ischemic complications, CAS often involves hemodynamic instabilities such as hypotension and bradycardia, at least during the period of balloon inflation. A recent

study found that periprocedural hemodynamic instability predicted periprocedural stroke, myocardial infarction, death, and longer hospitalization.[1] It can, therefore, be supposed that prevention of hemodynamic instability would improve the outcome of CAS. Pharmacological agents such as atropine and vasopressors are routinely used. Some single-institute studies have reported the efficacy of a transvenous temporary cardiac pacemaker (TTCP).[4,10,14]

We report here a patient in whom severe circulatory depression after CAS was readily managed as a result of the previously implanted TTCP, and we discuss the necessity of TCP in CAS. We have also conducted a meta-analysis of the literature concerning predictors of periprocedural hemodynamic depression following CAS.

CASE REPORT AND INTERVENTION

An 84-year-old man, with a history of hypertension and chronic kidney disease, was referred to the emergency room with a complaint of dysphagia and hand clumsiness on his left side since the previous day. He had no history of smoking and alcohol drinking. The National Institutes of Health Stroke Scale score was 2. Magnetic resonance imaging (MRI) of the brain revealed an acute infarction in the right frontal insulo-opercular area [Figure 1a]. MR angiogram demonstrated stenosis of the bilateral cervical carotid artery. He had already been taking aspirin (81 mg/day), so clopidogrel (75 mg/day) was added, and argatroban was administered intravenously with no early neurological deterioration and recurrence.

An electrocardiogram (ECG) found a normal sinus rhythm. The man had no history of atrial fibrillation (AF) or bradycardia. ECG monitoring did not capture any episode of AF. His baseline serum low-density lipoprotein cholesterol level was 59 mg/dl, and his high-density lipoprotein cholesterol level was 121 mg/dl.

A computed tomography (CT) angiogram found bilateral carotid artery stenosis, which was more severe on the left side (right: 47%, left: more than 90%, on NASCET criteria[17]), with the distal nontapering portions of the internal carotid artery serving as the reference segment [Figure 1b and c]. CT of the neck demonstrated non-circumferential but severe calcification of bilateral carotids [Figure 1d]. MRI of the plaque was carried out to determine the consistency and vulnerability of the plaque. The plaque components in the T1- and T2-weighted images showed with iso- and hyper-intensity, respectively.

Since the symptomatic right side demonstrated stenosis <50%, we proposed to manage the right IC stenosis conservatively. For the asymptomatic side, showing severe stenosis, carotid angioplasty was planned to prevent a further ischemic stroke.

After obtaining written informed consent from the patient and his family, we performed CAS for the left high-grade carotid stenosis, because of preexisting dysphagia.

TTCP

Before CAS, the transvenous cardiac pacemaker was placed through the left femoral vein, because the serious

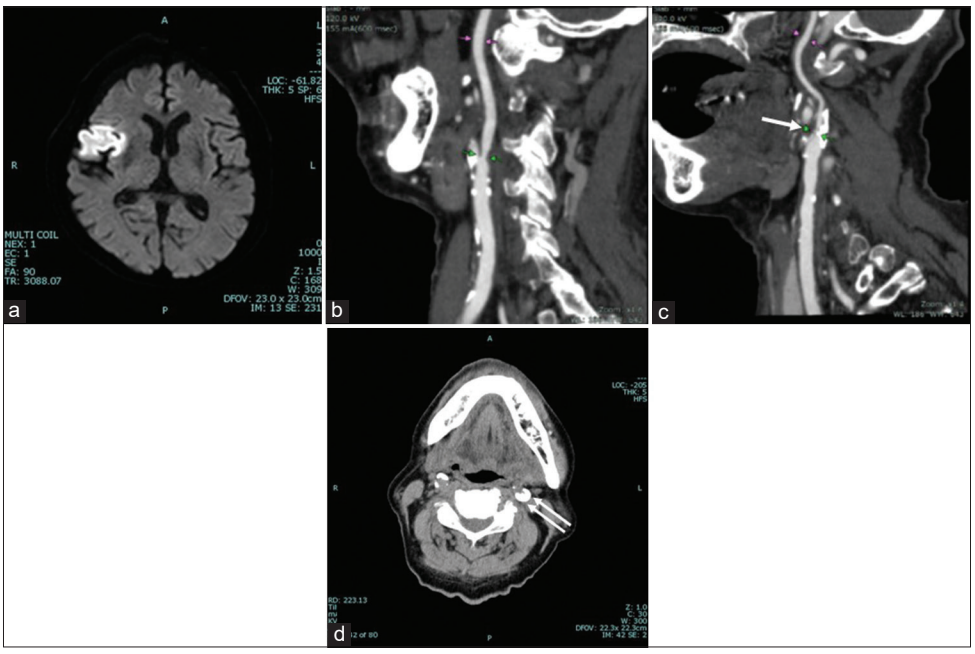

Figure 1: (a) Diffusion-weighted image at initial presentation showing the right frontal insulo-opercular infarction. Sagittal image of CT angiogram of (b) the right and (c) the left carotid artery, showing mild stenosis on the right and severe stenosis on the left side (arrow). (d) CT without contrast medium of the neck, showing severe calcified carotid plaque on the left side (double arrows).

hemodynamic depression was expected due to severe calcified plaque [Figure 1d]. With fluoroscopic guidance, a temporary pacer (Edwards Life Sciences, Irvine, CA, USA) was introduced into an appropriate position through flow direction, and the tip was seated in the right ventricle. Pacemaker function was confirmed by setting the rate above the natural heart rate (HR), and then the demand pacer (VVI mode, output intensity: 5 mA) was set to capture a HR below 55 beats/min, based on the preoperative ECG, with a sensitivity of 2 mA.

Surgical procedure

Under intravenous sedation with propofol, and local infiltration anesthesia with lidocaine, a 9-Fr occlusion balloon-guiding catheter (OPTIMO; Tokai Medical Products, Aichi, Japan) was inserted through the right common femoral artery, and was placed at the left common carotid artery. Unfractionated heparin (100 units/kg of body weight) was given intravenously to keep the activated clotting time above 300 s. The selective digital subtraction angiogram showed a severe stenosis of the left carotid artery [Figure 2a].

Because of the difficulty in navigating a filter-based embolic protection device, we carried out the procedure with proximal balloon protection [Figure 2b].

After the intravenous injection of atropine (0.5 mg), a balloon catheter of small diameter (UNRYU 2 mm, Kaneka, Japan) was induced to the stenotic region with a micro-guidewire (Chikai 0.014, ASAHI INTECC, Japan) and was inflated at 6 atm for 30 s. Predilation was then performed using a 4 mm balloon catheter (RX-Genity, Kaneka, Japan). A self-expandable open-cell stent (4 × 40 mm, PRECISE Pro RX, Cordis Japan) was deployed successfully, with no post dilation [Figure 2c]. The proximal balloon protection was released; the total proximal occlusion time was 13 min. A final angiogram confirmed that there was neither distal embolism nor acute occlusion.

There was no bradycardia during the surgical procedure. Blood pressure was decreased depending on the propofol infusion, which was managed by means of intermittent administration of 1 mg etilefrine [Figure 3a]. The patient did not exhibit any novel neurological deficits.

Postoperative course

Blood pressure gradually decreased, necessitating a reduction in the infusion of Propofol. Approximately 45 min after transfer to the intensive care unit (about 90 min after the procedure), blood pressure suddenly fell to 37/18, and his heart beat depended completely on TTCP [Figure 3b]. The patient became comatose and tetraparetic. The pre-implanted TTCP allowed the immediate recovery of self-circulation by administering a single bolus of noradrenaline (2 mg) together with continuous dopamine infusion. The man's blood pressure rose immediately to 128/47 and his neurological condition returned to the baseline. A continuous infusion of dopamine was maintained for 12 h so as to maintain the systolic blood pressure above 100 mmHg. TTCP was required for the next 3 days, because he exhibited intermittent bradycardia, necessitating pacing.

Postoperative MRI did not show any high-intensity signal on a diffusion weighted image. Aspiration pneumonia associated with acute heart failure was observed on postoperative day (POD) 5; this was managed with antibiotics, diuretics and high flow nasal cannula oxygen therapy. SPECT on POD4 revealed hyperperfusion in the left middle cerebral artery area without any corresponding symptoms, which subsequently subsided POD11.

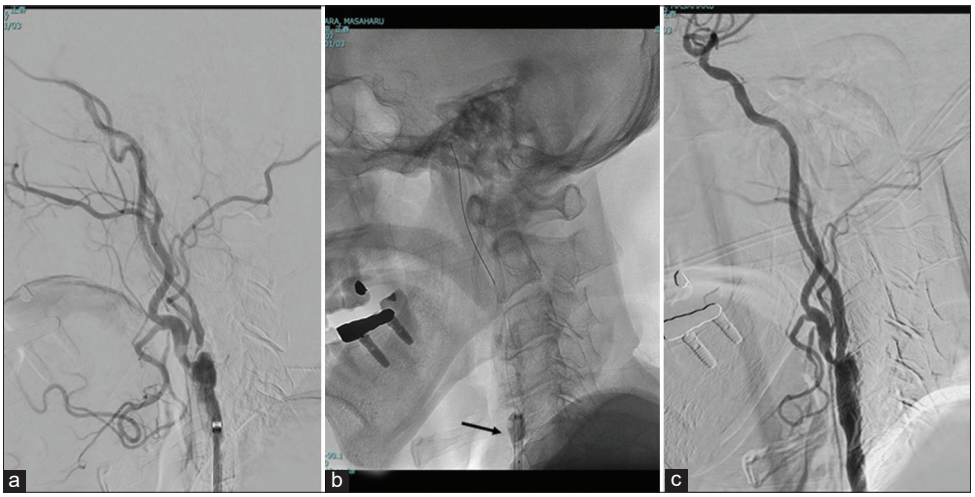

Figure 2: Lateral projection of the left carotid angiogram during CAS procedure. (a) Control angiogram before operation showing severe stenosis of the left internal carotid artery. (b) Proximal balloon protection (arrow) before stenting. (c) Final angiogram after stent deployment.

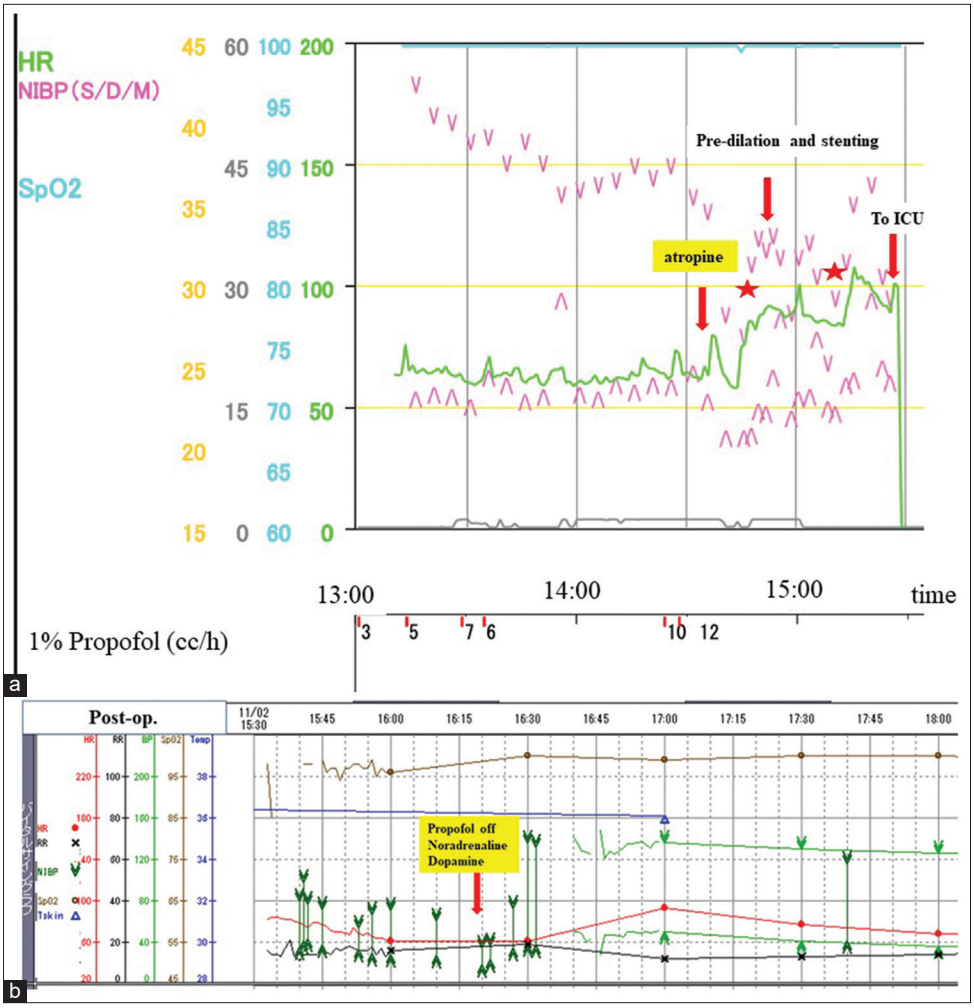

Figure 3: (a) Intraoperative and (b) postoperative cardiopulmonary monitoring chart. (a) For sedation, propofol was administered continuously, causing a gradual decrease in systolic blood pressure (sBP). Atropine was given intravenously, and increases in sBP and heart rate (HR) were confirmed. Balloon angioplasty with stent deployment was then conducted successfully. (b) About 45 min after ICU admission, severe circulatory depression developed, but recovery from it was accomplished immediately on cessation of propofol and administration of noradrenaline and dopamine. HR: Heart rate, NIBP: Non-invasive blood pressure, S: Systolic, D: Diastolic, M: Mean, SpO2: Percutaneous oxygen saturation, ICU: Intensive care unit.

After confirmation of absence of re-stenosis or plaque protrusion, using USG, the patient was discharged home on POD 26 without any neurological sequelae on POD 26.

Predictors of periprocedural hemodynamic depression after CAS by literature review

Two authors (M.F. and T.S.) independently searched the PubMed database up to the last day of 2020 with the following keywords: ("bradycardia" OR "hypotension" OR "hemodynamic instability") AND ("carotid" AND ("artery" OR "angioplasty") AND ("stent" OR "stenting") OR ("carotid" AND "stenosis") AND ("predictor" AND "risk factor"). This initial literature search yielded 23 articles.

Studies eligible for inclusion had the following features: (1) case series observational studies, whether retrospective or prospective, in which at least 100 CAS procedures were conducted and the predictors of periprocedural hemodynamic depression were investigated (hypotension and /or bradycardia); (2) the stent used was explicitly described as self-expandable; (3) raw data of predictors of interest could be extracted adequately and (4) age, mean and standard deviation were presented; and (5) the articles were peer-reviewed and written in English. Studies in which the specific CAS technique was investigated in relation to periprocedural hemodynamic instability were excluded from analysis.

From each article, we extracted data for the following parameters: age, sex (male), hypertension, diabetes mellitus, dyslipidemia, coronary artery disease, peripheral artery disease, chronic obstructive pulmonary disease, smoking,

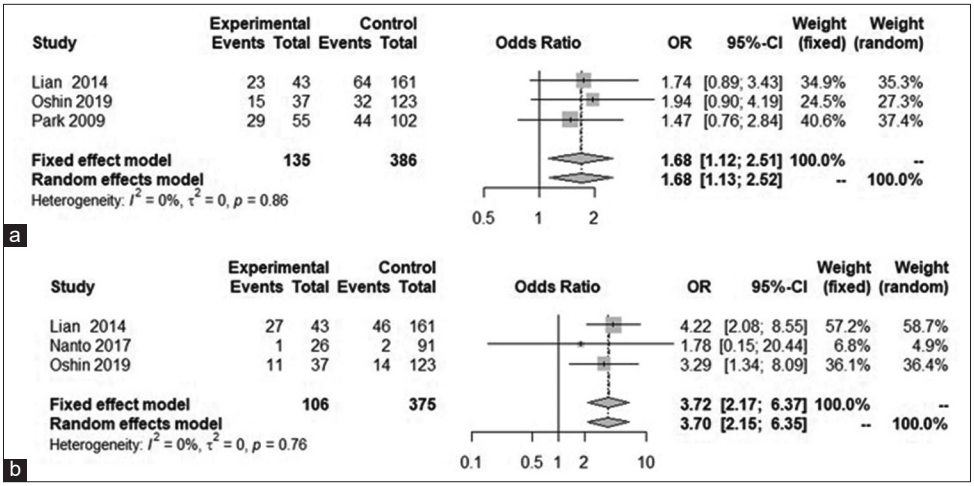

Figure 4: Forest plots of predictors of periprocedural hemodynamic depression with statistical significance: (a) smoking, (b) calcification.

stenosis with severe calcification, lesion near the carotid bulb (within 10 mm), side of the lesion (right), and symptomatic lesion. Any discordant results were resolved by discussion. The odds ratio (OR) and 95% confidence intervals (CIs) for periprocedural hemodynamic depression were then computed through the Mantel-Haenszel meta-analytic method. For age, the mean difference and 95% CIs were calculated using the inverse variance method. Statistical analysis was performed using R version 4.0.3 (R Core Team) and free software EZR.[12] $P < 0.05$ was taken as statistically significant.

The inclusion and exclusion criteria specified above gave us four articles,[13,16,18,19] which were analyzed further to study the predictors of periprocedural hemodynamic depression.

Based on our random effects meta-analysis model for all variables, significant predictors of periprocedural hemodynamic depression were found to be "smoking" (OR 1.68, 95% CI 1.13–2.52, $P = 0.01$) and "calcification" (OR 3.70, 95% CI 2.15–6.35, $P < 0.0001$); [Figure 4].

DISCUSSION

In the present case, severe circulatory depression following CAS was managed relatively easily due to the preexisting TTCP. If this TTCP had not been implanted, the measures for acute circulatory deterioration would have been more complicated, possibly resulting in a fatal outcome.

Circulatory instability is frequently seen after CAS occurring immediately (1 h) or within 20 h at latest;[8,15] this is attributed to the mechanical dilation and stimulation of the carotid sinus. Mechanical stretching of the carotid sinus causes an increase in parasympathetic activity, leading to hypotension and bradycardia.

The value of preoperative TTCP for CAS has been reported previously,[4,10,14] and in 22-57% of procedures TTCP was activated and helped to prevent the severe circulatory depression leading to neurological sequelae without complications. Bradycardia is usually treated with anticholinergic atropine, plus vasopressor agents. There is a time lag before they become effective, however. A shorter low-flow time is well known to be associated with favorable outcome in patients with in-hospital cardiac arrest.[6] TTCP assisted in the immediate recovery from hemodynamic depression in the present case. Alternatives to TTCP include transcutaneous temporary pacing.[11] This requires a higher current, however, causing pain and discomfort to the patient. None of their cases in this report underwent post-CAS pacing in the intensive care unit, and the duration of pacing was limited to the CAS procedure. In the present case, TTCP would have been better to avoid thermal burn due to prolonged use of transcutaneous pacemaker[5] since the present patient needed vasopressor administration for 1 day and intermittent pacing for the following 3 days.

Based on our meta-analysis of the literature, "smoking" and "severe calcification" were significant predictors of preprocedural hemodynamic depression. We, therefore, recommend, particularly in patients with severely calcified plaque and a history of smoking, that TTCP be implemented before CAS, given informed consent.

CONCLUSION

TTCP should be considered in performing CAS to prevent and promptly treat the resulting hemodynamic depression, particularly in patients with severely calcified plaque and a history of smoking.

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Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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