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Case Report

Fatal delayed hemodynamic depression after carotid artery stenting

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ABSTRACT

Refractory and fatal hemodynamic depression remarkably occurred eight hours after left carotid artery stenting in a 62-year-old male who had no hemodynamic instability till then; possible contributory factors were pre-existing moderate left ventricular systolic dysfunction and new-onset complete heart block caused by vasopressor-induced sympathetic stimulation in the presence of covert distal conduction system disease.

1. Introduction

Hemodynamic depression (HD) is the most common adverse event following carotid artery stenting (CAS); it occurs due to the stretching of carotid sinus baroreceptors resulting in stimulation of vagal fibers.[1] Hypotension and bradycardia, the most frequent manifestations of HD, occur mostly at the time of balloon inflation post-stent deployment, and usually settle in the immediate post-procedural period; HD does not lead to excess mortality, stroke or myocardial infarction since it is usually a transient phenomenon.[2] HD occurring beyond six hours of CAS is most often a recurrent episode in patients who had HD earlier; occurrence of the first episode of HD beyond six hours of CAS is rare.[1] We report a case of severe refractory HD occurring eight hours after CAS, despite absence of HD earlier, that was associated with new-onset complete heart block (CHB) and fatal myocardial depression.

2. Case report

A 62-year-old man presented with bilateral lower limb claudication. He had sustained a cerebrovascular accident with right hemiparesis in the past that had recovered fully. There was also a past history of inferior wall myocardial infarction, but there were no cardiac symptoms currently. He had diabetes mellitus, systemic hypertension and a 30-pack-year history of smoking. Physical examination revealed left carotid bruit and feeble lower limb pulses. His renal function was normal. The electrocardiogram (Fig.1A) showed normal sinus rhythm (95/m), non-specific intra-ventricular conduction defect (QRS duration 127 ms) with QRS axis minus 59° and normal PR interval (169 ms). The echocardiogram revealed moderate left ventricular systolic dysfunction (ejection fraction 43%) with inferior and posterior wall hypokinesia.

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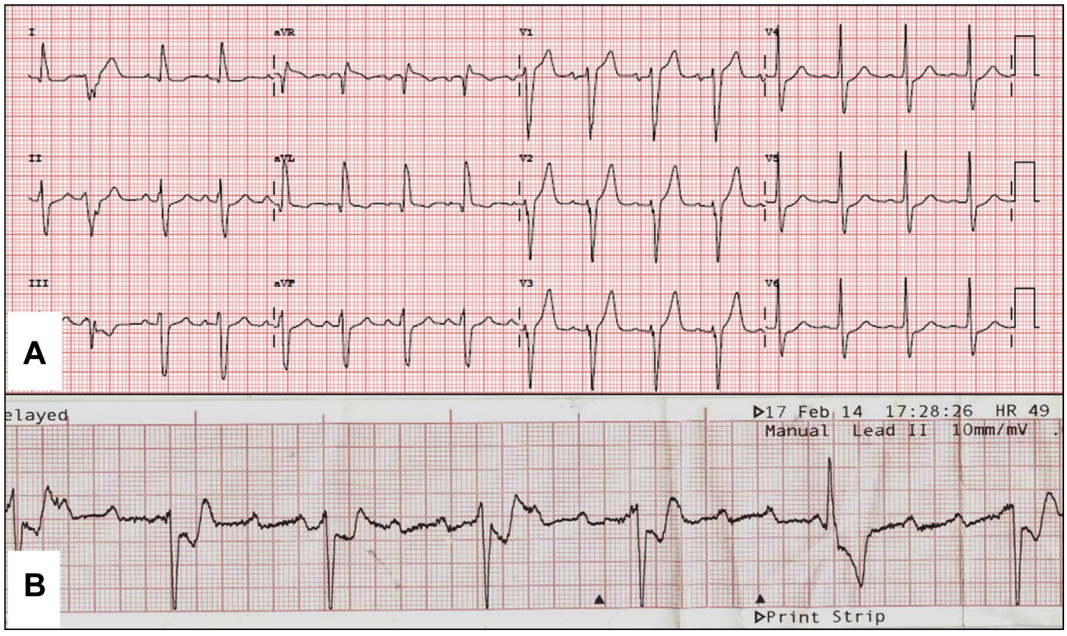

Fig. 1 – A. Baseline ECG showing normal sinus rhythm and intra-ventricular conduction defect with left axis deviation. B. ECG rhythm strip soon after onset of hemodynamic depression and resuscitation. Complete heart block, rapid *p* rate and broad QRS complexes are seen.

Coronary angiography showed triple-vessel coronary artery disease with discrete 80% stenosis in the proximal left anterior descending coronary artery and diffuse narrowing in the right and circumflex coronary arteries. Peripheral angiography revealed long-segment 90% stenosis in the left internal carotid artery just after its origin (Fig.2A), and significant obstructive disease in both external iliac arteries as well as in the right superficial femoral and anterior tibial arteries. In view of the left ventricular systolic dysfunction, diffuse vascular disease and multiple medical co-morbidities, he was considered a poor candidate for anesthesia and surgery. After written informed consent, percutaneous coronary, bilateral external iliac and left internal carotid artery revascularization was planned in a staged manner with a view to limit the radiographic contrast load in each sitting. The indication to treat the left internal carotid artery lesion was the history of recovered right hemiparesis and high-grade of stenosis. In the first sitting, angioplasty and stenting of the left anterior descending coronary and bilateral external iliac arteries was successfully performed, with no subsequent deterioration of renal function. Carotid intervention was performed two days later. Anti-hypertensive medications, including a beta-blocker, were administered as usual on the morning of the procedure.

Using percutaneous femoral arterial access and an 8F guiding catheter, the left internal carotid artery lesion was crossed with a 0.014″ Cougar wire (Medtronic) and a 6 mm SpiderFx embolic protection filter (eV3/Covidien) was deployed distal to the lesion. The lesion was dilated with a 3 mm coronary balloon and stented with a 7 × 40 mm Acculink carotid stent (Abbott Vascular); post-deployment, the stent was dilated with a 5 mm balloon at 12 atm. Atropine 0.6 mg was administered intravenously just before stent deployment. Temporary insertion of a right ventricular pacing lead was not done. Neither bradycardia nor hypotension was encountered during the procedure. Final angiogram (Fig.2B) showed minimal residual stenosis, no dissection and normal flow across the stented segment. Completion cerebral angiograms showed no evidence of distal embolization; there was another lesion in the intracranial segment of the left internal carotid artery causing 70% stenosis (Fig. 2C) which was left alone; the left middle cerebral artery was supplied by the left internal carotid artery, whereas the left anterior cerebral artery was supplied by the right internal carotid artery via the anterior communicating artery (Fig. 2D). Femoral sheaths were removed immediately after the procedure and hemostasis was obtained by manual compression. There were no peri-procedural complications and patient remained hemodynamically stable for the next several hours, during which time his blood pressure ranged from 100/60 to 120/70 mm Hg and the pulse rate varied between 72 and 84/m without inotropic support.

Eight hours after the procedure the patient suddenly became unresponsive with no premonitory symptoms; peripheral arterial pulses could not be felt and blood pressure was not recordable. Cardiopulmonary resuscitation was promptly initiated, which included administration of adrenaline and atropine. ECG done soon after this showed a rapid atrial rate, CHB and slow ventricular rate (Fig.1B). External cardiac pacing was immediately initiated, after which transvenous right ventricular pacing was begun. Despite this he continued to be hypotensive. Severe global left ventricular hypokinesia was seen on echocardiography. Resuscitatory efforts were unsuccessful and he expired an hour later.

3. Discussion

The most frequent complication of CAS is HD in the form of hypotension and bradycardia. The incidence of HD has varied