

ATHENS CARDIOLOGY UPDATE 2010

Persistent Bradycardia in a Patient With Coronary Artery Disease and Concomitant Carotid Artery Disease

Adamantia Polydorou, MD,¹ Panagiotis Megalooikonomos, MD,² Athanasios Portinos, MD,³ Efi Prapa, MD,⁴ Panagiota Kara, MD,⁵ Alexia Tsigas, MD,⁵ Joseph Moutiris, MD,⁶ Victoria Polydorou, MD,¹ Ion Belenis, MD,³ Antonios Sideris, MD,⁴ Antonis S. Manolis, MD,² Antonios Polydorou, MD⁶

¹Cardiology Department, Internal Medicine Department 251 General Air Force Hospital of Athens

²First Department of Cardiology, "Evangelismos" General Hospital of Athens

³Department of Vascular Surgery, "Evangelismos" General Hospital of Athens

⁴Second Department of Cardiology, "Evangelismos" General Hospital of Athens

⁵Department of Surgical Pathology, "Evangelismos" General Hospital of Athens

⁶Department of Interventional Cardiology, "Evangelismos" General Hospital of Athens

KEY WORDS: *persistent bradycardia, carotid angioplasty, percutaneous transluminal coronary angioplasty, case report*

ABBREVIATIONS

ACS= acute coronary syndrome

CAS= carotid angioplasty & stenting

ECG= electrocardiography

INR= international normalized ratio

LAD= left descending coronary artery

LBBB= left bundle branch block

LICA= left internal carotid artery

PTCA= percutaneous transluminal coronary angioplasty

RICA= right internal carotid artery

Correspondence to:

Antonios Polydorou, MD

Director of Interventional Cardiology
Evangelismos Hospital, Athens, Greece

E-mail:

antonios.polydorou@ontelecoms.gr

ABSTRACT

We report a case of a 75-year-old male admitted to our hospital via the emergency room because of wide-QRS complex tachycardia. Non-ST elevation acute coronary syndrome (NSTE-ACS) was observed after termination of tachycardia. Coronary angiogram showed subtotal occlusion at the mid segment of the left anterior descending (LAD) coronary artery, which was treated by percutaneous transluminal coronary angioplasty (PTCA) and stenting. Because of persistent bradycardia (40-45 bpm) in the absence of β -blocker therapy, not responding to intravenous atropine, a temporary pacemaker was inserted. As the patient had bruits on auscultation of both carotid arteries, at the end of the coronary angioplasty procedure, carotid angiography was performed. This revealed severe bilateral disease which was treated with left internal carotid artery (LICA) angioplasty and stenting (CAS) performed at the same session, and right internal carotid artery (RICA) endarterectomy performed the day after. Interestingly, the bradycardia resolved a few hours post CAS. Before discharge, an electrophysiology study, undertaken to evaluate for the wide-QRS complex tachycardia and the persistent sinus bradycardia, yielded negative results. In conclusion, persistent bradycardia, in the absence of bradycardic agents, may be a clue to underlying carotid artery disease.

CASE REPORT

A 75-year-old patient was admitted to our hospital via the emergency room due to two episodes of wide-QRS complex tachycardia. Non-ST elevation acute coronary syndrome was diagnosed after termination of the tachycardia.

Patient's cardiovascular risk factors included hypertension, smoking, dyslipidemia and a positive family history of coronary artery disease. Seventeen years ago, the patient had his aortic valve replaced because of significant stenosis and was since that time on anticoagulation with warfarin. On admission he gave a history of constrictive retrosternal pain which lasted for 3 hours. He was found to be in wide-QRS complex tachycardia, with a heart rate of 187 bpm with the following ECG characteristics:

cycle length 320msec, QRS=120 ms, LBBB-like pattern, normal axis and no concordance pattern of the QRS complex in the precordial leads. The wide complex tachycardia was terminated by intravenous lidocaine. Initial blood pressure was 70/40 mmHg and temperature was normal. He had mild tachypnea, while his oxygen saturation was at 87%. Upon restoration of sinus rhythm, a 2-mm ST-segment depression in leads I, aVL, V₅, V₆, was observed, with the heart rate around 45 bpm. The wide-QRS tachycardia episode recurred once and sinus rhythm was restored following 300 joules DC shock. Physical examination, following cardioversion, was unremarkable, apart from heart sounds characteristic of a metallic aortic valve and of bilateral carotid bruits. The lungs were clear. Biochemistry showed elevated cardiac troponin I (0.6 ng/ml), creatine phosphokinase (419 IU/l) and creatine phosphokinase-myocardial band (52 IU/l). Serum electrolytes and thyroid hormones were normal. Following cardioversion, the ECG was in sinus bradycardia with 1.5 mm ST-segment depression in leads I, aVL, V₅-V₆. Prior to admission, the patient was only receiving warfarin (the INR upon admission was at 1.4).

Echocardiography revealed mild concentric left ventricu-

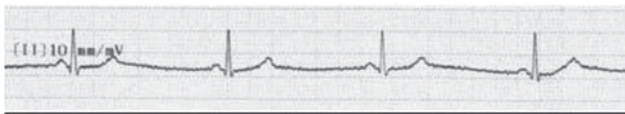


FIGURE 1. Single lead strip (II) showing sinus bradycardia at 43 beats/minute.

lar hypertrophy (intraventricular septum diameter in diastole ~12 mm), normal left ventricular systolic function (ejection fraction ~60%), mildly dilated left atrium and ascending aorta, well-functioning prosthetic aortic valve and minimal mitral and tricuspid valve regurgitation. He was admitted to the coronary care unit for continuous monitoring and was placed on oral aspirin 100 mg qd, oral clopidogrel 75 mg qd, oral rabeprazole 20 mg bid and subcutaneous enoxaparin 60 mg bid.

The day after admission the patient underwent coronary angiography, with a temporary pacemaker inserted prophylactically because of sinus bradycardia. The angiogram demonstrated subtotal occlusion of the mid segment of the left anterior descending (LAD) coronary artery, 50% stenosis in the mid left circumflex coronary artery (LCx) which was an ectatic vessel and a hypoplastic right coronary artery (RCA). Carotid angiography was performed during the same session, and showed subtotal calcified stenosis of RICA and 85% stenosis of LICA. Adhoc coronary angioplasty and stenting of LAD was performed with use of a 2×12 mm Sprinter Legend balloon and successful implantation of a zotarolimus –drug eluting stent. He was then transferred back to the ward in stable condition without any symptoms, but with persistent bradycardia (~40-45 bpm).

Angioplasty and stenting of LICA (CAS) was performed a few days later, with use of a 7×10×40 mm Acculink conical stent; pre- and post-dilation was achieved with use of a Clear-stream-Lite PAC 5×20 mm balloon. The procedure was performed with use of the Abbott Vascular brain protection device. Patient's condition improved and bradycardia resolved

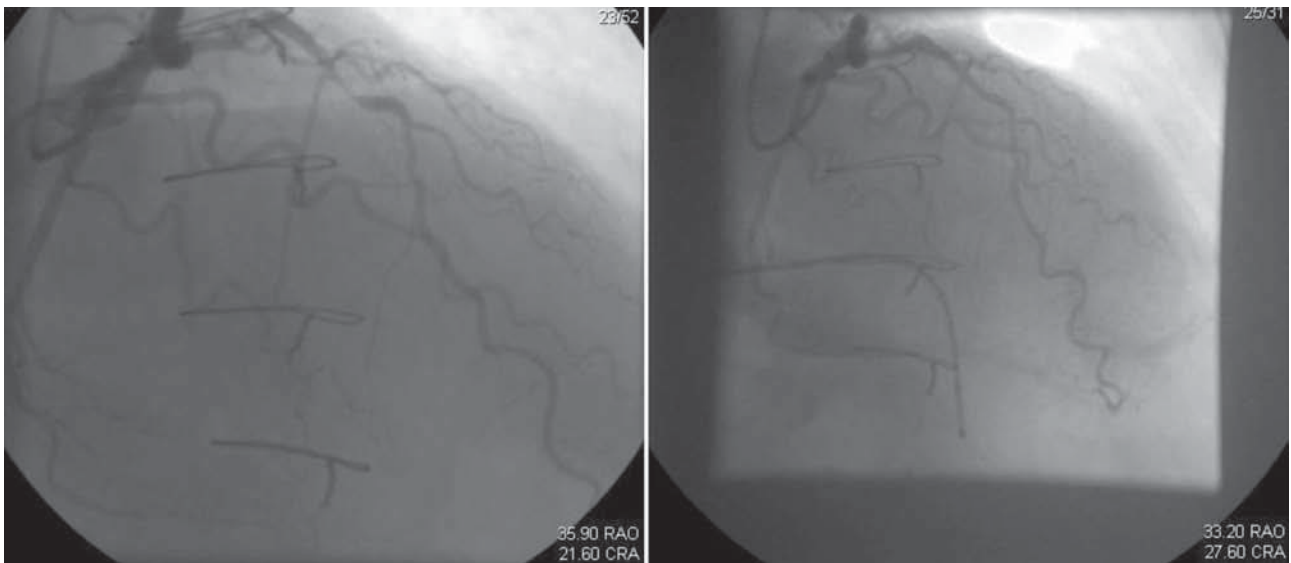


FIGURE 2. LAD pre and post PTCA & stenting. LAD = left anterior descending (coronary artery); PTCA = percutaneous transluminal coronary angioplasty.

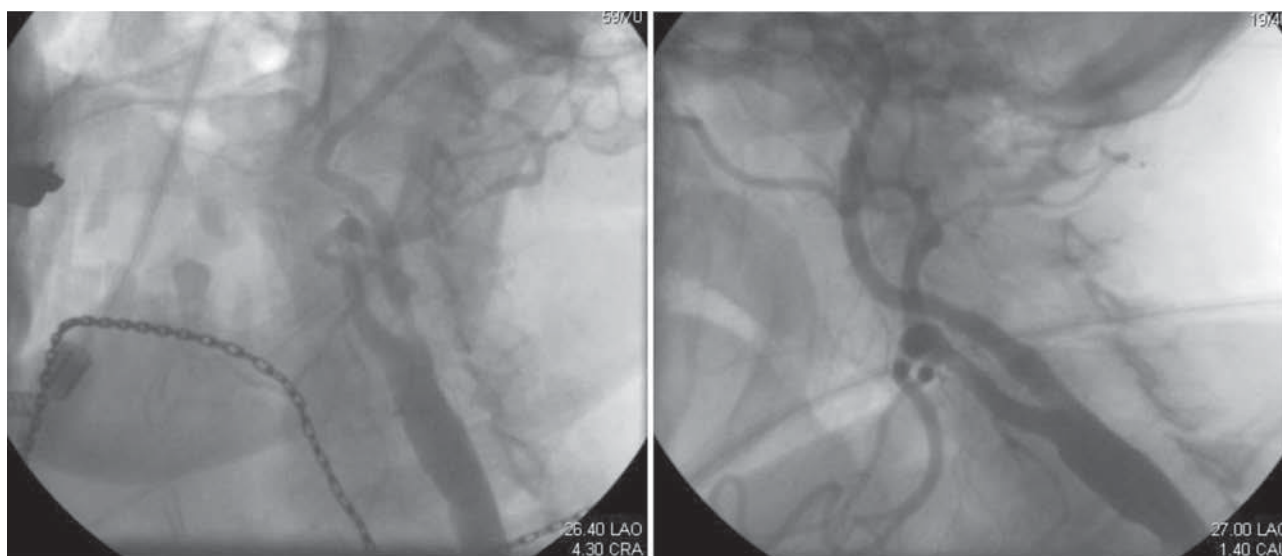


FIGURE 3. LICA pre and post CAS. CAS = carotid artery stenting; LICA = left internal carotid artery.

within 6 hour following CAS. The following day, the patient underwent successful endarterectomy (CEA) of the right internal carotid artery (RICA), under double antiplatelet therapy (clopidogrel, aspirin). Histological examination of a specimen of the atheromatous plaque from the RICA demonstrated extensive calcification of the media tunica, lipid-rich foamy histiocytes, cholesterol clefts, multinucleated giant cells, fresh hemorrhage (red blood cells, fibrin) and a mild chronic reactive inflammatory cellular infiltrate.

Finally, an electrophysiology study was performed which showed normal AH, HV, RR, PR, QRS, QT intervals, normal sinus node recovery time and corrected sinus node recovery time, Wenckebach at 420 ms without induction of any sustained arrhythmia with use of programmed ventricular stimulation with 1 up to 3 extrastimuli. In the ensuing days, the patient had a sustained clinical and hemodynamic improvement and was discharged home free of any symptoms.

DISCUSSION

Bradycardia, in the absence of a drug effect, can be caused by extrinsic stimulation of the parasympathetic system or intrinsic dysfunction of the sinus node or the atrioventricular node. The blood supply to both nodes comes primarily from the right coronary artery (RCA) and secondarily from the left circumflex (LCx).⁶ On the inferior wall of the left ventricle, most cardiac receptors with vagal afferents are located. Acute coronary syndromes mainly those affecting the inferior wall, are accompanied by stimulation of these receptors with concomitant reflex hypotension and sinus bradycardia.⁴ It is also well known that occlusion and subsequent reperfusion of

the RCA may result in abrupt bradycardia and hypotension, attributed to Bezold-Jarisch cardio-inhibitory reflexes arising from the ischemic left ventricle.⁵ In our case, patient's ischemia resulted from a significant lesion in the LAD coronary artery. It is obvious that this event could not explain the persistent bradycardia, since this continued and after correction of the LAD stenosis.

With regard to the differential diagnosis between ventricular (VT) and supraventricular (SVT) tachycardia, the age of the patient, the present history of acute coronary syndrome, the hemodynamical instability and the ECG criteria (notch on the descending part of the QRS complex at lead V₁, monophasic R morphology at lead V₆ and QS morphology at lead V₁, the duration of the R wave >30 ms at lead V₁ and the distance from the beginning of R to S nadir at lead V₁ >70 ms) favour the diagnosis of VT. Ventricular tachycardia was probably triggered by ischemia, since the electrophysiology study undertaken post-LAD angioplasty, failed to induce it.

Since PTCA to the LAD was unable to correct the persistent bradycardia, the issue of permanent cardiac pacing was raised. Following, however, a thorough discussion, it was decided to look into the possibility of carotid origin of the bradycardia, since this sign has also been observed in other patients with severe carotid artery disease in our institution. So, we proceeded to LICA angioplasty. The LICA lesion was severe, as was demonstrated by carotid angiography and the atheromatous plaque was type II, as was demonstrated by the duplex scan.⁷ Right internal carotid artery (RICA) stenosis was a long severe stenosis (95%) with ruptured plaque which indicated that this should be treated by endarterectomy. This was confirmed by duplex scan pre-procedure and atheromatous plaque biopsy post RICA endarterectomy.

It is well studied that hemodynamic instability, including bradycardia, is frequent during CAS.¹ Also, it is also well known that the phenomenon of bradycardia could persist post-CAS up to 24 hours, in a small percentage of patients, mainly with calcified plaques in the carotid sinus.² Administration of atropine, preventively, before balloon inflation, decreases the incidence of intraoperative bradycardia and morbidity in CAS patients.³

In conclusion, we strongly believe that the persistent bradycardia in this patient with severe myocardial ischemia caused by LAD stenosis, was a result of significant internal carotid artery disease and was not related to ischemia or to some kind of electrophysiological disorder. Correction of bradycardia a few hours post-CAS is a remarkable phenomenon which needs however further investigation.

REFERENCES

1. Lin PH, Zhou W, Koungias P, El Sayed HF, Barshes NR, Huynh TT. Factors associated with hypotension and bradycardia after carotid angioplasty and stenting. *J Vasc Surg* 2007; 46:846-53; discussion 853-854.
2. Gupta R, Abou-Chebl A, Bajzer CT, Schumacher HC, Yadav JS. Rate, predictors, and consequences of hemodynamic depression after carotid artery stenting. *J Am Coll Cardiol* 2006; 47:1538-1543.
3. Cayne NS, Faries PL, Trocciola SM, et al. Carotid angioplasty and stent-induced bradycardia and hypotension: Impact of prophylactic atropine administration and prior carotid endarterectomy. *J Vasc Surg* 2005; 41:956-961.
4. Wei JY, Markis JE, Malagold M, Braunwald E. Cardiovascular reflexes stimulated by reperfusion of ischemic myocardium in acute myocardial infarction. *Circulation* 1983; 67:796-801.
5. Goldstein JA, Lee DT, Pica MC, Dixon SR, O'Neill WW. Patterns of coronary compromise leading to bradyarrhythmias and hypotension in inferior myocardial infarction. *Coron Artery Dis* 2005; 16:265-274.
6. Esente P, Giambartolomei A, Gensini GG, Dator C. Coronary reperfusion and Bezold-Jarisch reflex (bradycardia and hypotension). *Am J Cardiol* 1983; 52:221-224.
7. Biasi GM, Froio A, Diethrich EB, et al. Carotid plaque echolucency increases the risk of stroke in carotid stenting: the Imaging in Carotid Angioplasty and Risk of Stroke (ICAROS) study. *Circulation* 2004; 110:756-762.