

## POSTERIOR CEREBROVASCULAR ACCIDENTS AFTER PERCUTANEOUS CORONARY INTERVENTION

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Received 2012-05-31; Revised 2014-04-10; Accepted 2014-04-14

### ABSTRACT

Percutaneous Coronary Interventional procedures (PCIs) are recognised as gold standard treatment for high risks patients with diffuse coronary atherosclerosis. Neurologic complications are one of the most dreaded procedural outcomes, stroke after PCI affects mainly elderly and high-risk patients (age more than 80 years and use of intra-aortic balloon pump), with longer times of the procedure and angiographic complications, such as dissection, abrupt closure and no reflow. We report two consecutive cases of posterior CVAs after percutaneous coronary procedures involving the ponto-mesencephalon and the cerebellum. Especially when risk factors are present, careful attention should be devoted to the preparation of the patient and to minimize catheter manipulation and exchanges.

**Keywords:** Posterior Cerebrovascular Accidents, Percutaneous Coronary Interventions

### 1. INTRODUCTION

Percutaneous Coronary Interventional procedures (PCIs) are recognised as gold standard treatment for high risks patients with diffuse coronary atherosclerosis.

Neurologic complications are one of the most dreaded procedural outcomes, leading to an enormous impact on resource consumption, on patient's prognosis and quality of life, with a rate of reported stroke ranging from 0.11 to 0.4% (Fuchs *et al.*, 2002).

Stroke after PCI affects mainly elderly and high-risk patients (age more than 80 years and use of intra-aortic balloon pump), with longer times of the procedure and angiographic complications, such as dissection, abrupt closure and no reflow (Fuchs *et al.*, 2002).

Cerebrovascular Accidents (CVAs) complicating left heart catheterization procedures have been described most commonly in the fore part of the brain, as a

consequence of embolic involvement of carotid supplied territories (Fuchs *et al.*, 2002; Lund *et al.*, 2005).

We report two consecutive cases of posterior CVAs after percutaneous coronary procedures involving the ponto-mesencephalon and the cerebellum.

### 2. CASE REPORT

#### Case 1

A 57-year-old woman, dislipidemic, with a two-year history of sudden and severe 10-min pre-cordial pain irradiated to the neck, jaw and to both upper limbs, spontaneously resolved.

The patient received ECG which revealed a mild mitral incontinence.

A <sup>99</sup>Tc-Pyrophosphate myocardial scan demonstrated a reversible mild signal in the anterior descendent coronary artery territory.

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Admitted to the Cardiology Department she underwent diagnostic coronarography showing normal arteries.

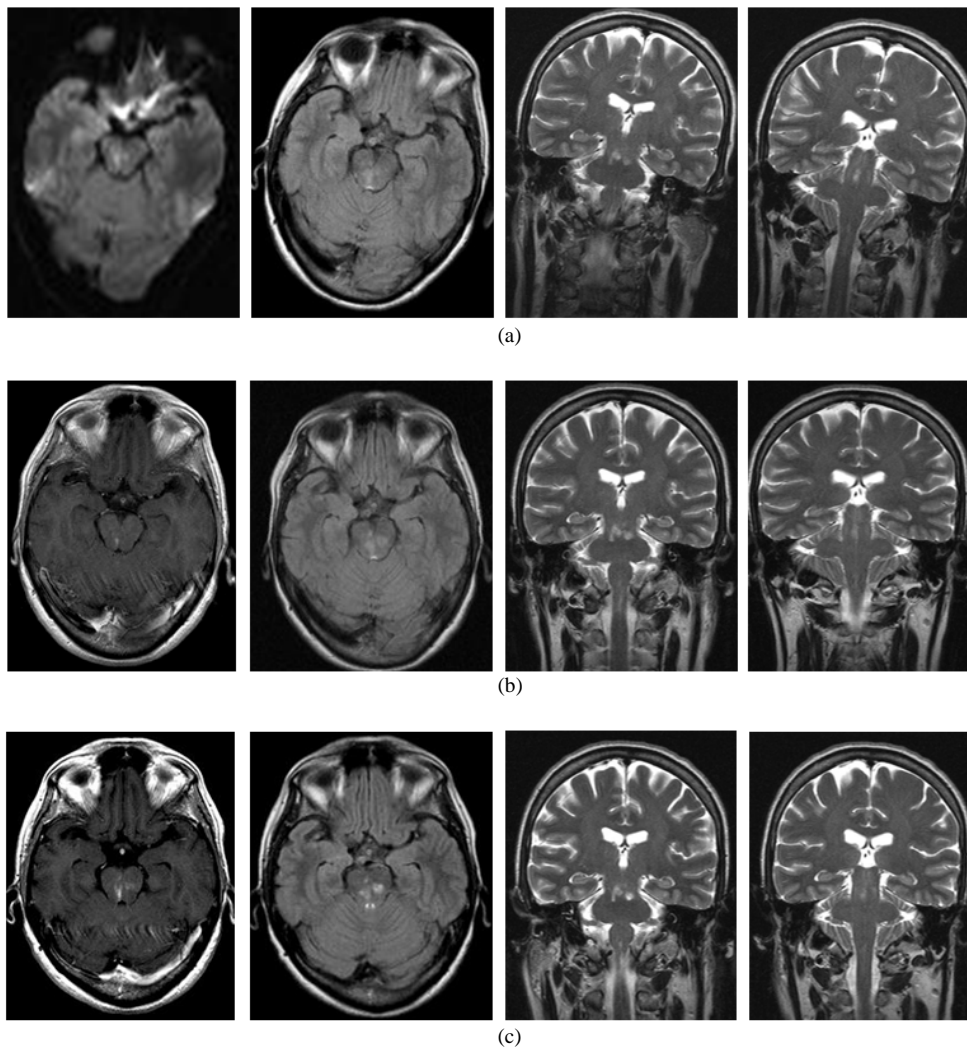
The day after she suddenly developed left labial paresthesia, nausea, vomit and ocular disorders. She was oriented and alert. Neurological examination showed internuclear ophthalmoplegia, positive bilateral index-nose test, more evident on the right and mild postural tremor of the upper limbs.

MRI imaging of the brain at 48h revealed small areas of high signal on diffusion weighted images in the right inferior quadrigeminal tubercle and in bilateral median and para-median portion of quadrigeminal lamina, a

small left cerebellum infarct and small areas of high signal on T2-weighted and FLAIR images in the right subcortical and left fronto-parietal areas (**Fig. 1a**).

Ecocardiography and supra-aortic trunk ecocolor Doppler showed no significant findings. The patient was managed conservatively.

At the 7 and 15 days follow-up MRI, blood-brain barrier disruption due to hemorrhagic reperfusion was observed as high signal intensity on T1-weighted images at the level of the lesions, in the right inferior quadrigeminal tubercle and in quadrigeminal lamina (**Fig. 1b and c**).



**Fig. 1.** MRI imaging of the brain at 48h, 7 days and 15 days after diagnostic coronarography (explanation in text): (a) Diffusion weighted image, axial FLAIR image and coronal T2-weighted images, (b) Axial T1-weighted image, axial FLAIR image and coronal T2-weighted images, (c) Axial T1-weighted image, axial FLAIR image and coronal T2-weighted images

## Case 2

A 57-year-old man, dyslipidemic, obese, with hypertension, ex-smoker, had positive history for recurrent anterior myocardial infarction treated with thrombolysis and with anterior descending artery stenting.

He developed unstable angina for which he was admitted to the Cardiology Department, after being asymptomatic for a period of 6 years.

Angiography showed total occlusion of the beginning of the right coronary artery, collateral circles and proliferation of intimal cells layers inside the stent along the anterior descending artery.

After the procedure, the patient complained dyspnea, nausea, vomit with a blood pressure of 140/70 mmHg, normal rate and blood oxygenation. He immediately started O<sub>2</sub> therapy and Lasix 20 mg ev.

Echocardiography showed left ventricle hypertrophy, aortosclerosis, moderate mitralic and tricuspid incontinence and moderate left atrium enlargement. Ventriculography showed normal size of the left ventricle but moderate anterolateral and postero-basal hypo kinesis and low ejection fraction (50%).

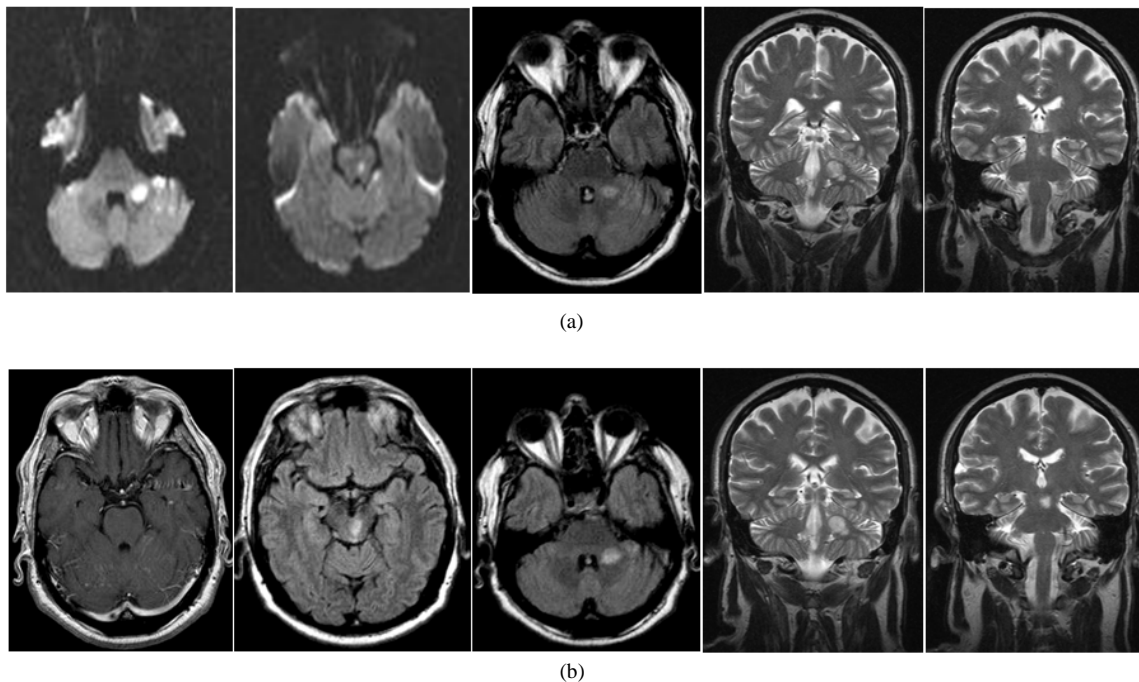
Before discharging the patient, two events of angina were resolved with sublingual nitrates. Discharge therapy was: Cardioaspirin 100 mg, epinitril 10 mg, enalapril 20 mg.

Then he was admitted to our hospital because of a 36-h loss of balance and co-ordination and 24-h lateral dyplopia and dysarthria appeared. The symptoms had been preceded by nausea and vomit 5 days earlier.

Neurological examination revealed horizontal lateral nystagmus (fast phase to the right), left adiadococinesia and impaired walking, suggesting internuclear ophthalmoplegia. Positive left index-nose test. Tone, strength and muscular trophism were preserved.

MRI imaging of the brain showed multiple areas of high signal on T2-weighted and FLAIR images involving the left paramedian ponto-mesencephalic region, the left cerebellar hemisphere and the right parieto-mesial region (**Fig. 2a**).

The high signal intensity on diffusion weighted images suggested subacute CVA in the vertebro-basilar territory. On the following MRI, 7 days later, the lesions were moderately enlarged and showed high signal intensity on T1-weighted images as for reperfusion blood-brain barrier damage (**Fig. 2b**).



**Fig. 2.** MRI imaging of the brain at 48h and 7 days after diagnostic coronarography (explanation in text): (a) Diffusion weighted images, axial FLAIR image and coronal T2-weighted images, (b) Post-contrast T1-weighted image, axial FLAIR images and coronal T2-weighted images

### 3. DISCUSSION

Ischemic strokes most often involve the middle cerebral artery territory (up to 50%) (Fuchs *et al.*, 2002). Radiologic and postmortem studies indicate that perioperative strokes are predominantly ischemic and embolic. Ischemic stroke most often involve the major cerebral-arterial territories (middle cerebral artery, 47.6%; posterior cerebral artery, 23.8%; and anterior cerebral artery, 9.6%).

The timing of embolic strokes after surgery has a bimodal distribution: 45% of perioperative strokes are identified within the first day after surgery, 55% occur after recovery from anesthesia from the second postoperative day on. Early embolism results especially from manipulations of the heart and aorta. Lund *et al.* (2005; Dukkipati *et al.*, 2005) found that brain lesions were associated with a significantly higher number of solid micro-emboli during transradial than during transfemoral catheterization.

The incidence of perioperative stroke depends on the type and complexity of the surgical procedure. The risk of stroke after general, noncardiac procedure is very low, whereas cardiac and vascular surgeries are associated with higher risk.

Fuchs *et al.* (2002) reported an incidence of stroke complicating PCI of 0.38% in 9,662 patients collected for 10 years. Recently Dukkipati *et al.* (2004) (Lund *et al.*, 2005) published the largest single study, involving 20,679 patients collected for 9 years, with an event rate of 0.3%.

The timing of procedure, total amount of contrast material or the type of contrast material are considered important factors to predict the risk.

Büsing *et al.* (2005) found that the rate of asymptomatic cerebral infarction after cardiac catheterization seems to be about 10-fold higher than it is for clinically apparent cerebral lesions.

Other groups have recently documented that silent acute brain injury can also be associated with percutaneous cardiac interventions, with possible cognitive impairment for patients in whom new lesions are identified (Büsing *et al.*, 2005; Dukkipati *et al.*, 2004).

Even though the carotid system is probably more often involved, it is arguable that structures supplied by the vertebro-basilar system are more frequently symptomatic than those of the carotid system, because of low compensatory function in damaged cerebellum and brainstem and because of the absence of collateral circulation to the end-arteries supplying especially the brainstem.

Diffusion-Weighted Magnetic Resonance Imaging (DW-MRI), which provides image contrast based on random translational motion of water molecules, substantially altered by acute cerebral ischemia, is the most sensitive technique for early detection of cerebral infarction (Fiebach *et al.*, 2001; Burdette *et al.*, 1998).

The addition of DW-MRI to conventional sequences makes it possible to detect very small and hyperacute infarction at almost any anatomic location within the brain hemispheres, the brainstem and the cerebellum, as shown in our cases. And structural damage to the brain has been detected by DW-MRI also in asymptomatic patients, caused by silent embolism during cerebral and coronary angiography (Hähnel *et al.*, 2001; Bendszus *et al.*, 1999; Omran *et al.*, 2003), as well as during surgical and endovascular revascularization procedures in the carotid artery (Jaeger *et al.*, 2001; Wityk *et al.*, 2001).

Independent predictors for major periprocedural stroke have been shown to be: Age, sex, diabetes, history of hypertension, unstable angina, body weight, congestive heart failure, peripheral vascular disease, chronic renal dysfunction, history of transient ischemic attacks, vein graft intervention, use of abciximab. Fuchs *et al.* (2002) showed that stroke after PCI affects mainly elderly patient with risk factors for atherosclerosis. Cerebral hemodynamic impairment in these patients would increase the risk of ischemic disease secondary to reduced flow (Weiller *et al.*, 1991; Miyazawa *et al.*, 2001), which may be additionally compromised by systemic hypoperfusion.

Aortic atherosclerosis is an independent predictor of the risk of perioperative stroke: The use of ecocardiography-guided aortic cannulation and intra-aortic filtration can reduce the risk of stroke (Omran *et al.*, 2003; Jackson *et al.*, 2000).

Systolic dysfunction increases the risk of perioperative stroke, particularly among patient with atrial fibrillation, therefore preoperative echocardiography may help to stratify the risk of stroke. Atrial fibrillation occurs in 30 to 50% of patients after cardiac surgery with a peak incidence between the second and fourth postoperative days and it is a major cause of many perioperative strokes.

The incidence of postoperative atrial fibrillation and stroke may be reduced by the prophylactic administration of amiodarone and beta-blockers, beginning 5 days before cardiac surgery.

The discontinuation of warfarin or antiplatelet agents in anticipation of surgery exposes patients to an increased risk of perioperative stroke. The risk is particularly high among patients with coronary artery



disease. Urgent or emergent catheterization confer an additional risk for CVA.

Possible explanations include the greater propensity for hemodynamic compromise in these patients, which may increase the risk of ischemic stroke and the less meticulous care in the advancement of catheters through the aorta during urgent or emergent PCI, which increases the risk of embolization to the brain. Keely and Grines (1998), in fact, showed that scraping of aortic plaque occurs in more than 50% percutaneous revascularization procedures.

#### 4. CONCLUSION

This report shows that occurrence of periprocedural CVAs related to cardiac catheterization can be observed and that, especially when risk factors are present, careful attention should be devoted to the preparation of the patient and to minimize catheter manipulation and exchanges.

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