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Transient cortical blindness during coronary and aortocoronary bypass graft angiography

Cegueira cortical transitória durante coronariografia e estudo de enxertos aortocoronarianos

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ABSTRACT – The prevalence of cerebrovascular complications after cardiac catheterization is low. These include stroke, transient ischemic attack, and amaurosis fugax. Cortical blindness is a rare, bilateral clinical condition of largely ischemic etiology, characterized by damage to the cerebral cortex, which manifests with acute reduction of visual acuity. Usually, neuro-ophthalmic complications of cardiac catheterization are correlated with embolic phenomena or migraine. We report a case of transient cortical blindness during coronary and aortocoronary bypass graft angiography.

Keywords: Angiography; Cortical blindness; Coronary angiography

RESUMO – A prevalência de complicações cerebrovasculares após cateterismo cardíaco é baixa. Tais complicações incluem o acidente vascular cerebral, o ataque isquêmico transitório e a amaurose fugaz. A cegueira cortical é uma condição clínica rara e bilateral, de etiologia, em grande parte, isquêmica, caracterizada por lesão no córtex cerebral, que se manifesta como redução aguda da acuidade visual. Geralmente, complicações neuro-oftalmológicas do cateterismo cardíaco correlacionam-se com fenômenos embólicos ou enxaqueca. Relatamos um caso de cegueira cortical transitória durante realização de coronariografia e estudo de enxertos aortocoronarianos.

Descritores: Angiografia; Cegueira cortical; Angiografia coronária

INTRODUCTION

The prevalence of cerebrovascular complications after cardiac catheterization is low.¹ The National Institutes of Health (NIH) reports a rate of 0.03%,² and the report of the British Cardiovascular Society (BCS), which evaluated 34,041 coronary angiographies, shows a prevalence of 0.06%.² Cortical blindness is a rare, bilateral clinical condition, of mostly ischemic etiology, characterized by damage to the cerebral cortex (bilateral retrogeniculate pathways), which presents as acute poor visual acuity.³ It was first correlated with coronary angiography in 1970.⁴ Since then, less than two dozen cases have been reported, some of which include coronary and aortocoronary bypass graft angiography.^{5,6}

The literature indicates only 17 cases of transient cortical blindness after coronary angiography.^{7,8} Thirteen of these events occurred in men and only four in women, but this reflects the larger number of men who undergo investigation for coronary disease. The mean age of patients was 58 years, and more than half (ten patients) had a diagnosis of hypertension (HTN). Eleven (65%) patients had left internal thoracic artery (LITA) graft angiography. The contrast volume used ranged from 80mL to 400mL. There was complete and spontaneous reversal of the deficit in almost 95% of cases, with time required to regain normal vision ranging from 15 minutes to 3 weeks, and a mean of 3 days. Selective spinal angiography adds a greater risk of neurological complications. Since the internal thoracic artery used for coronary grafting is adjacent to the origin of the vertebral artery, notably on the left side, it is likely that an inadvertent injection into the vertebral artery may occur.^{8,9}

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We report a case of transient cortical blindness during coronary and aortocoronary bypass graft angiography. This study was approved by the patient, who signed an Informed Consent Form.

CASE REPORT

A 64-year-old female patient, presenting dyslipidemia with good pharmacological control, former smoker for 10 years, affected by non-ST-segment elevation acute coronary syndrome (NSTEMI-ACS) in December 2011, when she underwent invasive risk stratification, having severe and single stenosis in the proximal third of the left anterior descending artery (LAD). She was submitted to percutaneous coronary intervention (PCI) with implantation of a bare metal stent, and met success criteria. She remained clinically asymptomatic for 6 months, when presented progressive angina, culminating with a new coronary angiography, in June 2012.

In this new exam, diffuse intra-stent restenosis was described with an estimated obstruction of 70%. Considering the impossibility of using a drug-eluting stent, coronary artery bypass grafting surgery was indicated, with a LITA graft for LAD. The patient evolved asymptomatic over 9 years, when she again presented with symptoms of progressive angina and dyspnea on moderate exertion.

Considering the patient's history, a new invasive stratification was indicated in August 2020, and the procedure was performed using right femoral access. During coronary angiography, parietal irregularities were found in the right coronary artery (RCA) and circumflex artery territory. As to LAD territory, the stent had mild neointimal hyperplasia, a finding different from that described in the procedure performed in 2012 (Figure 1).

At the time of left subclavian artery catheterization and the initial tests for LITA cannulation, the patient reported acute visual alteration (visual blurring), followed

by blindness (the LITA graft was chronically occluded at its origin). During the entire examination, there was no hemodynamic instability, and a low osmolality nonionic contrast agent (iohexol) was used, with a total volume of 80mL. Left ventriculography was not performed. A brief neurological examination performed in the cath lab did not show any focal motor deficit. Capillary blood glucose was 108mg/dL.

The patient was kept under hemodynamic monitoring, followed by vigorous hydration, keeping the mean arterial pressure at approximately 68mmHg. The neurological examination was systematically repeated after 15 minutes, and remained unchanged in relation to the first examination. Ophthalmoscopy also showed no signs suggestive of retinal ischemia or even papillary edema. The patient was transferred to the intensive care unit and evaluated by the neurology team. After approximately 4 hours, with no clinical improvement, magnetic resonance imaging (MRI) of the brain was performed (Figure 2), where no areas of restricted diffusion were observed in the echo-planar sequence, ruling out the possibility of an acute/subacute ischemic process.

The ophthalmology team was called in, performed ophthalmoscopy again, and no modifications suggestive of retinal ischemia were detected. Tonometry was normal. No specific measures were taken. Twelve hours after the event, the patient fully recovered her vision, with no change in visual acuity. The patient was discharged from hospital 24 hours after cardiac catheterization. She was submitted to a new ophthalmologic evaluation 5 days after the event, with retinography, which showed no alterations (Figure 3).

Based on the absence of ischemia on diffusion MRI, the clinical course with reversal of deficit without specific therapy, and ruling out other possible causes of acute visual disturbances, the diagnosis of cortical blindness became more likely, related to neurotoxic changes associated with iodinated contrast medium.

LITA: left internal thoracic artery.

Figure 1. (A and B) Coronary angiography in right anterior oblique and left anterior oblique projections, showing previously implanted stent in the proximal third of the left anterior descending artery. (C) Occlusion of left internal thoracic artery.

Figure 2. Brain magnetic resonance imaging. (A) T2 sequence with no sign of ischemia. (B) Diffusion with no sign of restriction.

Figure 3. Retinography of the right (A) and left eyes (B). Papillae with clear edges, normal color, physiological excavation. Arteriovenous ratio, arteriolar brightness, and crossing within normal range. Choroid and retina with no hemorrhages and/or exudates.

DISCUSSION

The mechanism of brain damage in transient cortical blindness remains uncertain, but the posterior cerebral circulation is known to be more susceptible to these lesions. According to Rapaport and Levitan,¹⁰ prolonged contact of the contrast with the blood-brain barrier may lead to neurotoxicity in two ways: lipid solubility allows the contrast to

cross it, and the high osmolarity of the contrast may open the endothelial junctions, and release its entry into the neural tissue. Added to these two factors is the fact that the visual cortex probably has an incomplete blood-brain barrier, which would explain the selectivity of cortical amaurosis after catheterization.

Other possible causes would be embolism and ischemia related to prolonged hypotension, which should always

be ruled out by tomography or MRI. When compared to CT, MRI is more accurate in diagnosing ischemic stroke. T2-weighted sequences are able to demonstrate infarcted tissue, but fail to assess acute regional ischemia. On a diffusion-weighted imaging (DWI) sequence, however, areas of high water proton restricted diffusion, corresponding to the ischemic territory affected by cytotoxic and vasogenic edema, are differentiated as zones of hypersignal, with sensitivity and specificity of 99%.^{11,12}

Because of this scenario, several authors believe that the higher the volume of contrast used, the greater the possibility of developing this complication. Such a severe complication is devastating for the patient, family, and operator. Fortunately, the condition is exceedingly rare and, usually, transient. We reported this case with the purpose of warning for this situation, which is little known and has peculiar etiological aspects.

CONFLICTS OF INTEREST

The authors declare there are no conflicts of interest.

CONTRIBUTION OF AUTHORS

Conception and design of the study: AVB and RS; data collection: DOB; data interpretation: CNV; text writing: PAV and AML; approval of the final version to be published: AVB.

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