SÃO PAULO MEDICAL JOURNAL

CASE REPORT

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Neurological Complications of Coronary Arteriography

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The authors report a 46 year-old female who was submitted to a right femoral artery coronary arteriography. During the procedure she presented an upward, downward and convergence gaze paresis; an upbeating nystagmus. A NMRI showed a change in T-2 weighted signal of the left paramedian mesencephalic region. A diagnosis of stroke was made and dextran 40 was prescribed. Vertical gaze paresis progressively disappeared. Convergence paresis and nystagmus improved partially only. The possible pathophysiological basis underlying this clinical picture are: catheter-induced dislodgement of an atheromatous plaque; small thrombi formation and release from the catheter tip; catheter-induced vascular lesion with subsequent thromboembolism or dissection of the intima; cardiac arrhythmia; catheter-induced or contrast medium-induced arterial spasm; a combination of two or more of these.

UNITERMS: coronary arteriography, neurological complications, vertebro-basilar circulation, NMRI.

INTRODUCTION

Coronary arteriography (CA) is a commonly performed procedure in the diagnosis of various cardiac diseases. Its overall complications rate is 1,8% and its neurological complications stands as much as 0.1% (2,3,6). The most common risk factors for emergence of neurological complications are: hypertension; atherosclerosis; valve disease; previous neurological deficit (2,3,6). The most frequent neurological manifestations are: gaze or motor deficits; pupillary, visual field, sensorium changes; ataxia and migraine attacks (2,3,4,5,6). The vertebro-basilar system is the most affected circulation in CA (2,3,4,6) .

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CASE REPORT

A 46 year-old female was submitted to a CA because of a cathecolamine-dependent ventricular tachycardia. Personal history disclosed recurrent headaches. Family history revealed sudden death of a brother and a son. Her father died after a cerebral stroke and had hypertension. Physical exam and blood tests were unremarkable. A negative Machado-Guerreiro reaction was obtained. A 24-hour Holter demonstrated 4 episodes of atrial tachycardia with abnormal conduction. Heart rate ranged from 31 to 101bpm. Her CA disclosed normal coronary arteries; a mild diffuse left ventricle hypokinesia; mild left ventricle enlargement; a mild diffuse right ventricle hypokinesia.. Cardiac muscle biopsy showed mild cell hypertrophy. Soon after the procedure, the patient referred blurred vision and diplopia. Physical exam at that time was unremarkable. The neurological exam diclosed a clear sensorium, and an upward, downward, convergence gaze paresis, upbeating nystagmus with no pupillary changes. A normal CAT was obtained. A NMRI showed a change in T-2 weighted signal on the left paramedian

mesencephalic region. A diagnosis of stroke was made and 500 ml of dextran 40 TID was prescribed. Vertical gaze paresis progressively disappeared and it was absent at discharge. However, convergence deficit and nystagmus beating to the right were still present.

DISCUSSION

Vertical and convergence gaze paresis is usually associated with mesencephalic lesions (1,7). Some authors claim lesions causing vertical and convergence paresis should be bilateral whereas others believe that a paramedian lesion is sufficient to produce paresis. Additionally, the vertebro-basilar circulation (VBC) is intimately involved with the blood supply of the structures controlling overall eye movements (2, 3,4). The presence of gaze paresis herein, due to a mesencephalic lesion as demonstrated in the NMRI is consistent with involvement of the VBC (2,3,4,6). The literature concurs on the more frequent involvement of the VBC over the carotid circulation (CC) in the genesis of the neurological complications of CA (2,3,4,6). The possible mechanisms for the vascular nature of the clinical manifestations and the other non-clinical findings are: catheter-induced dislodgement of an atheromatous plaque; small thrombi formation and release from the catheter tip; catheter-induced vascular lesion with subsequent thromboembolism or dissection of the intima; cardiac arrhythmias; catheter-induced or contrast medium-induced arterial spasm; a combination of two or more of these factors (2,3,5,6). However, no clear explanation has been found yet for the more common involvement of VBC over the CC in the neurological complications after a CA. Patients submitted to CA with femoral artery catheter insertion and catheter progression through aorta develop a similar rate of VBC complication as individuals who are submitted to CA with brachial artery catheter insertion and progression through the subclavian and inominnate arteries (3,6). This finding, that is, the similar complication rate for both CA catheter traject, raises some doubts about a catheter-induced dislodgement of an atheromatous plaque as a possible mechanism in the genesis of neurologic complications. In this report, the patient was submitted to a femoral CA with catheter progression through the aorta. Neither had the patient any angiographic signs suggestive of atheromatosis as shown in the femoral CA, nor coronary arteriography through femoral artery necessarily leads to preferrentially posterior circulation thrombi lodgement (3,5). The vascular anatomy of the aortic trunk suggests that anterior circulation should be more likely affected by a catheter-induced dislodged thrombus. These two elements taken together, that is, the normal femoral CA and aortic trunk anatomy speak against the possibility of catheter-induced plaque dislodgement in this case report. Therefore, catheter-induced dislodgement of a plaque is not a likely mechanism of vascular lesion in this case. Formation and release of small thrombi from the catheter tip into the systemic circulation may also play an important role in the determination of symptoms. These small thrombi may cause no clear symptoms when launched in the carotid circulation. Thrombi size and the physiology of the CC may explain the lack of clear-cut or long-standing clinical manifestations and findings (3,6). The anterior circulation may simply be less vunerable to an insulting agent. However, when thrombi lodging occurs in the VBC, manifestations and findings occur more readly and more proeminently (5,6). In keeping with the lesser vunerability of the anterior circulation theory, is the patient's NMRI showing no hemispheric lesions whereas the mesencephalic lesion was inequivocally apparent (Fig 1). Accordingly, cardiac arrhythmias, hemorrhagic shock, dislodgement of a cardiac thrombus should in general lead preferentially to hemispheric involvement over brain stem manifestations as usually reported in the literature (3,5,6). It is the authors' impression that both circulation systems are at least equally attacked by the same insulting agent during a complicated CA. However, the intrinsic mechanisms determining the findings and symptoms are different for each system. The posterior circulation with a different vascular anatomy irrigates a rather different anatomy of the CNS, that is, the the brain stem and occipital cortex. The brain stem may be more vulnerable and has a lower threshold for the emergence of symptoms under the same insult whereas the anterior circulation may be more resistant. In summary, this case reported here demonstrates that involvement of the vertebro-basilar circulation may occur despite a major preexisting factor for the emergence of neurological complication during a CA. The exact causes for this more frequent clinical involvement of the posterior circulation is yet to be determined.

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RESUMO

Este artigo relata um caso de uma mulher de 46 anos submetida a cateterismo cardíaco femoral direito, que desenvolveu paresia do olhar conjugado para cima, para baixo, déficit de convergência e nistagmo para cima logo após o procedimento. Ressonância nuclear magnética mostrou hipersinal de T-2 paramediana mesencefálica esquerda. Dextran 40 proporcionou progressiva regressão do déficit do olhar vertical. Nistagmo e convergência regrediram parcialmente. Possíveis mecanismos lesionais: lesão vascular pelo cateter e tromboembolismo ou dissecção da intima; arritmia cardíaca; espasmo arterial pelo cateter ou contraste; combinação de dois ou mais destes fatores são discutidos.