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Cardiogenic shock associated with subarachnoid hemorrhage

Choque cardiogênico associado à hemorragia subaracnóidea

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ABSTRACT

Systemic complications are frequent in subarachnoid hemorrhage patients. Among these complications, electrocardiographic abnormalities simulating ischemic cardiomyopathy may occur, possibly associated with myocardial dysfunction. This manuscript aims to report a case of subarachnoid hemorrhage associated with myocardial dysfunction and cardiogenic shock. A 45 years old woman was admitted with subarachnoid hemorrhage and coma, showing Glasgow scale = 7, Hunt-Hess = 5 and Fischer computed tomography classification = 3. On the second day, the patient underwent anterior cerebral communicant artery aneurysm embolization. The clinical evaluation revealed diffuse pulmonary infiltration, dyspnea and hypotension. Additional tests showed electrocardiographic lateral wall repolarization changes and elevated creatine kinase-MB fraction (36U/L). The cardiac index was 2.03 L/minute/m², Vascular systemic resistance was 3728 dynes.sec/cm². The non-responsiveness to volume demonstrated a cardiogenic shock pattern. The ventricular ejection fraction was 39%. The coronariography was normal, showing no obstructive lesions. Six days later the patient was removed from respiratory support and after eight days the dobutamine infusion was discontinued. The ejection fraction recovered up to 65%. Serial transcranial Doppler evaluations did not show vascular spasm. After ten days the patient was discharged form the intensive care unit. Patients with subarachnoid hemorrhage may be complicated with ventricular dysfunction and cardiogenic shock, increasing the cerebral ischemia risk. Diagnosis optimization and hemodynamic stabilization are essential to minimize the risk of cerebral vasospasm and ischemia.

Keywords: Subarachnoid hemorrhage/etiology; Shock, cardiogenic; Heart failure; Cardiomyopathies; Case reports

INTRODUCTION

Subarachnoid hemorrhage (SAH) is a challenging clinical condition, its incidence ranging according to the country (2 cases/100,000 inhabitants in China to 22.5 cases/100,000 inhabitants in Finland). The most affected age range is between 40 and 60 years-old, but this condition may affect from children to elderly adults. The hospital mortality rates range between 33% and 45%.[1-3]

SAH is frequently associated with systemic complications such as pulmonary edema, sodium disorders and deep venous thrombosis.[2,4] Among the cardiovascular changes, may be found electrocardiographic signs (50-90% of the cases), increased heart enzymes, and global or partial contractile

dysfunction mimicking ischemic heart disease which may result in cardiogenic shock. Herein we report on a SAH case with severe ventricular dysfunction and cardiogenic shock.[3,5-7]

CASE REPORT

A previously healthy 45 years-old woman lost consciousness suddenly while riding a bicycle. She was initially seen by paramedics and had Glasgow = 7, non-measurable blood pressure (BP), cold skin, heart rate (HR) = 126 beats per minute (bpm) and respiratory frequency (RF) = 10 movement per minute (mpm).

Upon arrival to the emergency room she had Glasgow grade 5, BP 98/66, HR 70 bpm, arterial oxygen saturation (SaO2) 85%. After orotracheal intubation, the arterial oxygen saturation (SaO2) was improved, however a large amount of pink and foamy secretion came out from the orotracheal tube, suggesting acute pulmonary edema. The chest radiography showed diffused pulmonary infiltrate (Figure 1) and the electrocardiogram had sinusal rhythm, prolonged QT and lateral wall repolarization changes (Figure 2). CK-MB was 36 U/L (normal range up to 16 U/L). Metabolic acidosis was identified, with lactate 2.1 mmol/L (normal range up to 1.6 mmol/L).

Head computed tomography showed diffusely cortical sulci stretching, hyperdense basal cisterns, Silvian sulcus and inter-hemispheric clouding (Fisher's tomography classification = 3) (Figure 3). The Hunt-Hess clinical classification was grade 5. In the second hospital day the patient underwent cerebral arteriography showing a bilobar saccular aneurysm in the anterior communicant artery measuring about 5x4 mm (Figure 4). During the test she had mechanic vasospasm, which improved following intra-arterial nimodipine. The aneurysm was embolized with 4, 3 and 2 mm platinum microcoils. Following the endovascular procedure and
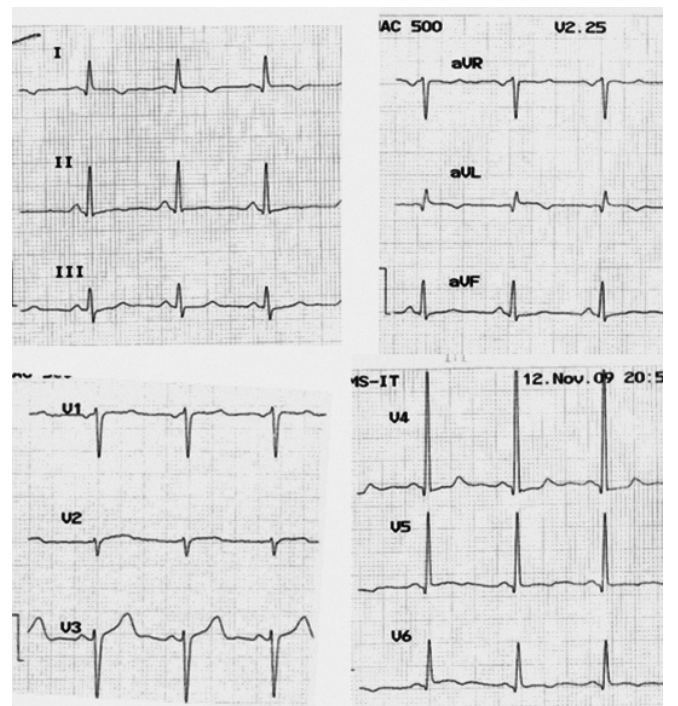
Figure 2 – Admission electrocardiogram. Sinusal rhythm, inverted T wave on DI and aVL leads, and enlarged QT interval.

Figure 1 – Chest X-ray evidencing diffuse pulmonary infiltration.
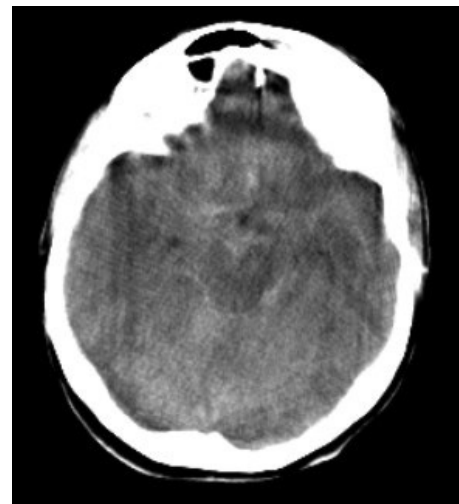
Figure 3 – Axial computed tomography image, evidencing subarachnoid hemorrhage – Fisher III.

Figure 4 – Digital angiography evidencing saccular aneurysm in the anterior communicant artery.

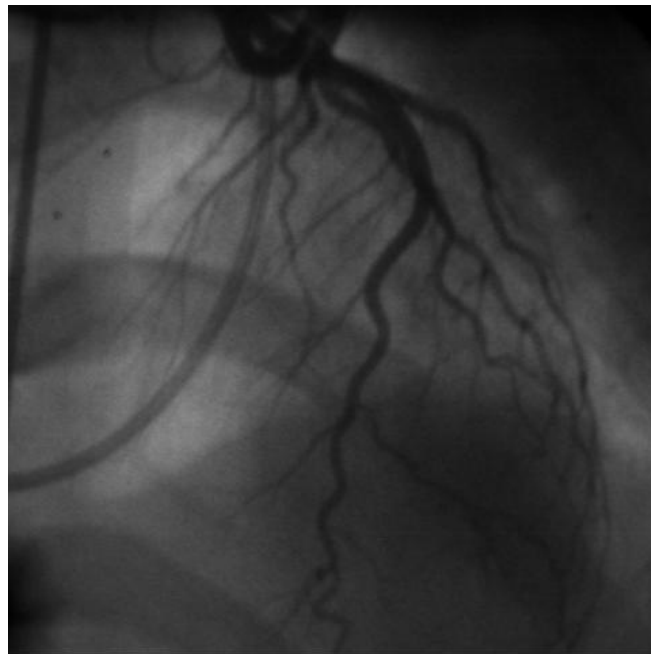**Figure 5 – Left coronariography without obstructive lesions.**

consciousness improvement, the patient was extubated and referred to the intensive care unit (ICU). Dyspneic, she required non-invasive ventilatory support (NIV). The electrocardiographic (ECG) changes were maintained, and the transthoracic echocardiogram showed lateral-dorsal wall akinesia and hypokinesia.

In the third day she had Glasgow 15, with no focal deficits. The ventilatory pattern worsened, and orotracheal plus controlled mechanic ventilation was required. She involved with volume-refractory hypotension (80/40). Intravenous norepinephrine infusion was started. Daily transcranial Doppler showed no vasospasm or significant effects on cerebral flow, even during hypotension.

The respiratory arterial pulse changes (ΔPp = 7%) showed the patient to be volume unresponsive. The low cardiac index (2.03 L/min/m^2) and increased systemic vascular resistance (3728 dynes.sec/cm^5/m^2) evidenced a hemodynamic pattern of cardiogenic shock. A new transthoracic echocardiogram showed lateral-dorsal akinesia, severe left ventricular function impairment, ejection fraction (EF) 39% and venous congestion signs. Evident improvement of hemodynamic parameters was seen after dobutamine start and gradual norepinephrine weaning.

Heart catheterization on the fifth hospital day showed no coronary arteries obstructive lesions, left ventricle with moderate to severe hypokinesia, and unchanged thoracic aorta (Figure 5).

The patient was extubated on the sixth hospital day. She had then Glasgow 15 and transcranial Doppler showed no vasospasm. On the eighth hospital day, a new echocardiogram showed EF 65%, and mild to moderate diffused hypokinesia. Dobutamine was then weaned off.

On the eleventh day, the consciousness level was reduced (Glasgow 13). The head computed tomography (CT) showed mild hydrocephaly, no bleeding, and no medium line deviation. No vasospasm on transcranial Doppler was found, and conservative measures were adopted.

With complete neurological recovery, the patient was discharged from the ICU on the fourteenth day.

DISCUSSION

Heart changes overall affect 39% to 63% of SAH patients. Many of them have most severe presentations, remaining the heart lesion pathophysiology unclear.

Autopsies in SAH subjects have shown no correlation between SAH-associated electrocardiographic changes and relevant either coronary or myocardium disease. Additionally, other studies have concluded that these ventricular function changes are generally revertible, thus suggesting a non-ischemic "hibernating myocardium".[7] Some hypothesis were proposed to justify the SAH-associated heart dysfunction, as coronary

thrombosis, coronary spasm, secondary to hypertension myocardial oxygen offer and consumption unbalance, and tachycardia. Also it is believed that a post-SAH myocardial injury comes from a combination of neural and hormonal mechanisms. The cathecolamines release intensity is strongly correlated with SAH patients' heart complications. Also, cardiac lesions histopathology doesn't have a ischemic pattern, and is associated to sympathetic nerve terminations in the heart. Excessive norepinephrine release from these nerve terminations result in vascular constriction followed by subendocardial necrosis.[6]

SAH-associated cardiovascular dysfunction diagnosis is based on hemodynamic changes, electrocardiography, echocardiography and cardiac biomarkers testing.

SAH-associated electrocardiographic changes include heart arrhythmias, ST segment elevation or depression, T wave inversion, pathologic Q waves, and QT interval changes. These changes have no defined prognostic value, and are found in most of the patients.[6,7]

Cardiac biomarkers such as CK-MB and troponin I elevation have been seen in about 40% of SAH patients. These changes are often related to echocardiographic changes, increased cardiovascular risk, late cerebral ischemia and poorer prognosis.

Abnormal myocardial function is frequently (10% to 31%) seen in SAH patients echocardiography. These contraction changes may be either global or segmental, without confirmed concomitant coronary disease either by necropsy or coronariography.

Our patient had severe ventricular dysfunction and cardiogenic shock associated with the previously described electrocardiographic, enzymatic, echocardiographic and angiographic features. The SAH severity, characterized by the clinical (Hunt-Hess = 5) and tomographic (Fisher = 3) signs was itself a high risk condition for cerebral vasospasm and ischemia.[1] The association of low cardiac output has severely worsened the cerebral hypoflow and ischemia risk.[1,8] The hemodynamic support therapy associated with cerebral blood flow monitoring with transcranial Doppler likely contributed to cerebral ischemia prevention.

Finally, in SAH patients a ventricular dysfunction may be present, plus severe overall blood flow and cerebral blood flow impairment, and increased cerebral ischemia risk. Thus, early attention should be given to this diagnosis possibility, and to the hemodynamic optimization requirements, aiming to minimize vasospasm and ischemia risks.

RESUMO

Complicações sistêmicas são vistas frequentemente em indivíduos acometidos por hemorragia subaracnóidea. Dentre estas alterações podem ocorrer anormalidades eletrocardiográficas que simulam miocardiopatia isquêmica que podem ou não estar associadas com disfunção miocárdica. O objetivo deste é relatar um caso de associação de hemorragia subaracnóidea com disfunção miocárdica e choque cardiogênico. Mulher de 45 anos foi internada com quadro de coma secundário a hemorragia subaracnóidea. À admissão apresentava Glasgow = 7, Hunt-Hess = 5 e classificação tomográfica de Fisher = 3. O aneurisma cerebral de artéria comunicante anterior evidenciado pela arteriografia cerebral foi embolizado com sucesso no segundo dia de internação. Evoluiu com dispnéia e infiltrado pulmonar difuso. Havia alteração da repolarização ventricular em parede lateral, aumento da CK-MB (36 U/L) e hipotensão. O índice cardíaco de 2,03 L/min/m^2, a resistência vascular sistêmica 3728 dynes. seg/cm^5/m^2, e a irresponsividade a volume evidenciavam o padrão hemodinâmico de choque cardiogênico. A fração de ejeção do ventrículo esquerdo era de 39%. A cineangiocoronariografia não apresentava lesões coronarianas obstrutivas. Após 6 dias a paciente foi extubada e ao oitavo dia foi possível a retirada completa da dobutamina. A fração de ejeção passou a 65%. Sucessivos exames de Doppler transcraniano não apresentaram vasoespasmo. A paciente recebeu alta da unidade de terapia intensiva no décimo quarto dia. Pacientes com hemorragia subaracnóidea podem apresentar disfunção ventricular e choque cardiogênico, aumentando o risco de isquemia cerebral. O diagnóstico e a otimização hemodinâmica são essenciais para minimizar os riscos de vasoespasmo e isquemia cerebral.

Descritores: Hemorragia subaracnóidea/etiologia; Choque cardiogênico; Insuficiência cardíaca; Miocardiopatias; Relatos de casos

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