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Case Report

Management of catecholamine-induced stunned myocardium—a case report

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Abstract Hypertensive, hypervolumic, and hemodilution therapy (triple-H therapy) is administered to patients with symptomatic cerebral vasospasm after intracranial aneurysm clipping. This therapy can sometimes result in cardiac dysfunction because of pharmacologically induced hyperadrenergic state. The diagnosis may be missed if blood pressure alone is monitored to guide triple-H therapy. In this report, we describe one such patient who developed cardiac failure after triple-H therapy. This was diagnosed by using a bioreactance noninvasive cardiac output monitoring. Continuous cardiac output monitoring by this technique facilitated treatment of cardiac failure with milrinone and dobutamine. At discharge, the patient had no neurologic deficits.

1. Introduction

Acute stress can result in transient cardiac dysfunction caused by excess sympathetic activity [1–3]. Similar hyperadrenergic state may occur during induced hypertensive, hemodilution, and hypervolumia as a part of triple-H therapy, used to treat symptomatic cerebral vasospasm in postoperative patients after intracranial aneurysm clipping, and cause cardiac failure and pulmonary vascular congestion. Titrating triple-H therapy using only blood pressure monitoring may overlook the cardiac dysfunction associated with this therapy. Here, we report one such patient where the triple-H therapy caused cardiac dysfunction which was diagnosed and treated successfully by using a bioreactance noninvasive cardiac output monitor.

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2. Case report

Approval from the institutional review board (National Institute of Mental Health and NeuroSciences ethics committee) was obtained for the publication of this case report. A 45-year-old woman was admitted with a grade III (Fisher scale) subarachnoid hemorrhage (SAH). Cerebral angiography revealed a right posterior communicating artery aneurysm (Fig. 1). Preoperative biochemical and hematological investigations and electrocardiogram (ECG) were within normal limits. The patient underwent clipping of the aneurysm, and the postoperative course was uneventful for 5 days. On the sixth postoperative day (POD), the patient became drowsy. Cerebral angiography revealed right A1 and M1 segment vasospasm (Fig. 2). Triple-H therapy was initiated using dopamine, noradrenaline, and intravenous fluids administered through a central venous line. Doses of dopamine and noradrenaline were increased up to 23 and 0.2 $\mu g\,kg^{-1}\,min^{-1}$, respectively, to achieve a mean arterial pressure (MAP; noninvasive) of 110 to 120 mm Hg from an MAP of 90 mm Hg. Her consciousness

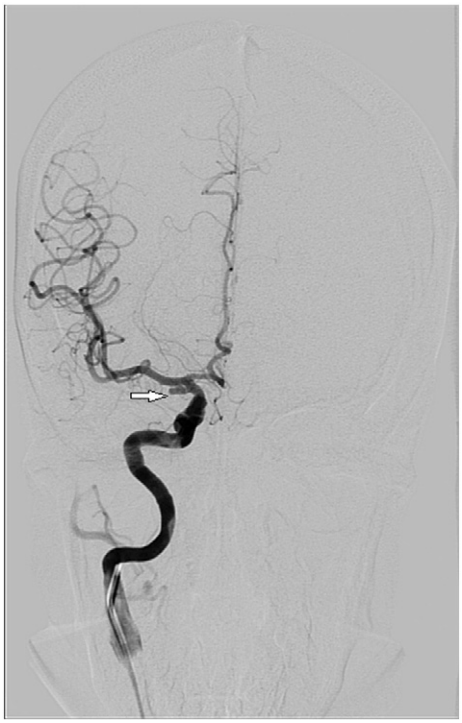

Fig. 1 Four-vessel digital subtraction cerebral angiography at hospital admission. White arrow: right posterior communicating artery aneurysm.

improved. However, on the 11th POD, she developed dyspnea and productive cough. A Doppler study of the lower limbs did not show any evidence of deep venous thrombosis. Her trachea was intubated and mechanical ventilation initiated, with a

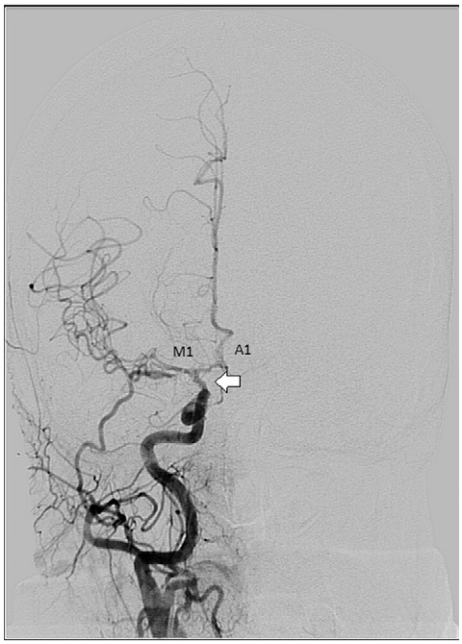

Fig. 2 Four-vessel digital subtraction cerebral angiography done on sixth POD showing proximal vessel narrowing (white arrow). A1, anterior cerebral artery; M1, middle cerebral artery.

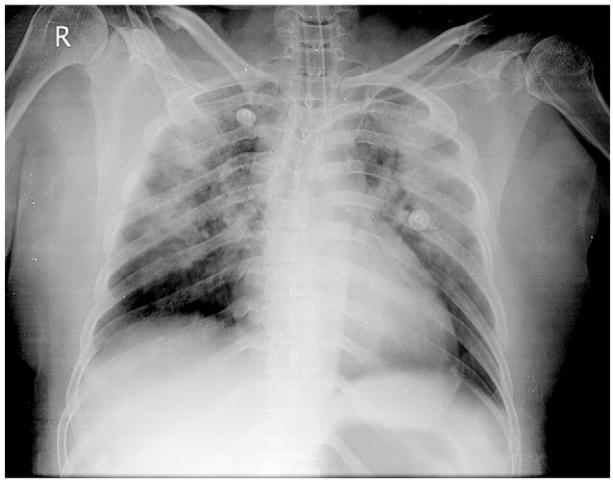

Fig. 3 Bedside x-ray chest at ICU admission. X-ray reveals bilateral diffuse infiltrates.

fraction of inspired oxygen of 0. 7 (partial pressure of oxygen, arterial/fraction of inspired oxygen = 150) and 10 cm H_2O positive end-expiratory pressure. Central venous pressure at this time was 20 cm H_2O. The patient did not have electrocardiographic changes suggestive of myocardial ischemia, and troponin levels were not assessed. Hemodynamic parameters were continuously monitored by using an invasive femoral arterial line and bioreactance-based noninvasive cardiac output monitor (NICOM; Cheetah Medical, Tel-Aviv, Israel).

The cardiac index (CI) was 1.9 L min^{-1} m^{-2}, stroke volume (SV) was 26 mL, and systemic vascular resistance was 3751 dyne · s cm^{-5} m^{-2}. Low SV, bilateral extensive chest infiltrates (Fig. 3), and the need for high doses of ionotropes suggested cardiac failure with a low cardiac output state which was initially treated with milrinone infusion (0.25 μg kg^{-1} min^{-1}). This was later changed to dobutamine infusion (due to nonavailability of milrinone) at 24 hours at doses up to 20 μg kg^{-1} min^{-1}. Transthoracic echocardiography a few hours after her hemodynamic deterioration showed left ventricular regional wall motion abnormalities in the form of apical and mid-distal anterior hypokinetic segments with an ejection fraction of 44%. As these findings were in favor of stress-induced myocardial dysfunction (precipitated by high doses of catecholamines) rather than neurogenic pulmonary complication, dopamine and noradrenaline were tapered, whereas the MAP was maintained stable at 100 to 110 mm Hg. Over a period of 7 days, dopamine and noradrenaline were tapered and stopped. The CI and SV improved to 4.4 L min^{-1} m^{-2} and 66 mL, respectively (Fig. 4), whereas the systemic vascular resistance decreased to 1979 dyne · s cm^{-5} m^{-2}. Furosemide was also administered daily keeping in mind the cardiac cause of respiratory distress and a cumulative positive fluid balance of around 5 L caused by the previous triple-H therapy. A negative fluid balance of around 6 L was achieved over the next 5 days. Bacteriologic cultures of tracheobronchial secretions, urine, and blood at 48 hours of