Tako-Tsubo cardiomyopathy presenting with cardiogenic shock successfully treated with milrinone: A case report

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A B S T R A C T

We report the case of a middle age patient presenting with Tako-Tsubo cardiomyopathy (TTC) complicated by cardiogenic shock that was successfully handled with milrinone. A 64-year old man presented with cardiogenic shock after benzodiazepine and alcohol intoxication. A slight elevation of troponin and typical left ventricular ballooning without coronary lesions suggested TTC. Within a few hours milrinone infusion normalized the cardiac index. TTC is responsible for severe transient left ventricular dysfunction occurring after physical or psychological stress. The major pathophysiological mechanism involved is disproportionate catecholamine secretion, which may stun the myocardium. We considered if treatment of this unique physiopathology with catecholamines could be dangerous in these patients and if alternative inotropes such as milrinone should be preferred.

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Introduction

Tako-Tsubo cardiomyopathy (TTC) is a recently identified entity that is increasingly diagnosed to be responsible for severe transient left ventricular dysfunction, usually occurring after psychological or physical stress. TTC concerns preferentially post-menopausal women but can also affects men. The prevalence of TTC is up to 7% of all suspected acute coronary syndromes in women and 1% when both sexes are considered. This syndrome is characterized by regional wall motion abnormalities, which mimic on ventriculography a Tako-Tsubo, i.e. a crock used in Japan to capture octopuses. TTC presentation features signs of acute coronary syndrome but is not the consequence of obstructive coronary disease. This condition is considered to be related to disproportionate catecholamine secretion, which may stun the myocardium. As a result, catecholamine infusion can worsen the phenomenon and therefore TTC presenting with cardiogenic shock is difficult to handle. Cardiogenic shock complicates approximately 6.5% of TTC. Only a few data are available regarding the management of this unique condition. Here we describe the successful treatment of a patient presenting with Tako-Tsubo-related cardiogenic shock using milrinone, a non-catecholamine inotrope.

Case report

A 64-year old man was admitted for coma induced by benzodiazepine and alcohol intoxication. Endotracheal intubation was performed to protect the upper airways. On admission, clinical examination revealed skin mottling with hypotension. Low-dose norepinephrine infusion (0.2 μg/kg/min) and fluid expansion were started. Transthoracic echocardiography showed severe left ventricular dysfunction (left ejection fraction of 20%), while basal segments were hyperkinetic (Fig. 1A and B; Video 1). The cardiac index as estimated by echocardiography was low (1.25 L/min/m²). The electrocardiogram showed inverted T waves in the precordial leads. Analysis of blood samples revealed high lactate levels (6.5 mmol/L) and a mild increase in troponin I (3.68 ng/mL). Emergency coronary angiography was normal. The typical ventriculographic presentation suggested TTC. Because the lactate levels continued to increase despite norepinephrine infusion, and based on the TTC
pathophysiology, milrinone was introduced as inotropic agent (bolus of 50 μg/kg over 10 min then continuous infusion of 0.375 μg/kg/min), rather than dobutamine, which could theoretically worsen the cardiac catecholamine toxicity. Four hours later, the lactate level normalized and echocardiography revealed an improved left ventricular ejection fraction (Fig. 1C and D; Video 2). Norepinephrine was maintained at the same dosage at admission (0.2 μg/kg/min) and stopped a few hours after milrinone initiation. Milrinone was administered during 48 h. No arrhythmias were recorded. After milrinone discontinuation, echocardiography showed complete left ventricular function recovery and the patient was successfully weaned from mechanical ventilation.

Discussion

To our knowledge, this is the first report of a TTC complicated by cardiogenic shock that was successfully managed with milrinone. The major pathophysiological phenomenon involved in TTC is considered as disproportionate catecholamine discharge in response to stress. This “catecholamine rush” is thought to saturate beta receptors and to literally stun the myocardium. At supraphysiological concentrations, catecholamines could result in beta-receptor paradoxical negative inotropic effects. Cardiogenic shock is mostly treated with dobutamine or epinephrine. But in TTC patients, these catecholamines could, given their beta agonist action, worsen myocardial stunning. Milrinone is a well known non-catecholamine inotrope that is used less than catecholamines. By inhibiting type III phosphodiesterase, milrinone increases the calcium influx and improves myocardial contraction without any beta agonist action. This drug also decreases the systemic vascular resistance and pulmonary capillary wedge pressure. Furthermore there is a lower increase in the heart rate and myocardial oxygen consumption with milrinone compared with catecholamines. However, the induced peripheral vasodilatation may promote hypotension.

Because the lactate levels and cardiac index improved only after starting milrinone infusion, we supposed that milrinone stabilized the hemodynamic condition. Anyway, low-dose norepinephrine was not sufficient to explain the improvement in the cardiac index. However, initial norepinephrine administration could have avoided hypotension induced by milrinone. Mebazza et al suggested norepinephrine infusion in the case of hypotension caused by milrinone.

Padayachee et al proposed the use of levosimendan, a calcium sensitizer, to treat TTC presenting with cardiogenic shock. They handled successfully two patients with this drug. Levosimendan binds to the N-terminal lobe of cardiac troponin C and stabilizes the calcium-bound conformation, resulting in sustained interaction between actin and myosin filaments in systole and improves the myocardial systolic function. However, levosimendan administration is also associated with vasoplegia and hypotension.

Conclusion

TTC presenting with cardiogenic shock represents a difficult challenge in intensive care units. Considering the unique physiopathology of TTC, the use of catecholamines could be dangerous and alternative inotropes such as milrinone or levosimendan should be preferred.

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Supplementary data

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