

Tako-tsubo Cardiomyopathy Probably Associated With Administration of Sulprostone Following Cesarean Delivery

Síndrome de tako-tsubo probablemente asociado con la administración de sulprostone tras una cesárea

To the Editor,

Postpartum hemorrhage is a cause of maternal deaths and morbid events in western countries, due to uterine atony in the majority of cases. In addition to monitoring, resuscitation, and transfusion procedures, the management of post-partum hemorrhage generally follows a stepwise protocol in international guidelines. The first step consists of the administration of oxytocin or ergometrin. If bleeding persists, then prostaglandins (sulprostone, carboprost and misoprostol) are usually recommended.

A 50-year-old pregnant woman was admitted to our hospital at 28 weeks gestation for an elective cesarean delivery. Her medical history included only obesity. During the fifth month of pregnancy, she had a screening transthoracic echocardiography (TTE) which showed preserved systolic and diastolic functions without valvular disease. Cesarean delivery was performed following the administration of standard spinal anesthesia without hemodynamic instability. After delivery, 30 UI of oxytocin were administered to treat uterine atony associated with postpartum hemorrhage. A few minutes later, she experienced chest pain and palpitations. Electrocardiography (ECG) showed a junctional tachycardia with narrow QRS complex, retrograde P waves, and significant diffuse ST-segment depression (Fig. 1). The supraventricular tachycardia spontaneously resolved without occurrence of heart failure. Four hours later, as uterine atony and bleeding continued, she received 1.5 mg sulprostone over 3 h with a stable infusion rate of 0.5 mg/h. Thirty minutes before the end of infusion, she became hypertensive (220/110 mmHg) and tachycardic (130 bpm). She also complained of sudden chest pain and shortness of breath. Her oxygen saturation fell to 85% percent and lung crackles appeared, leading to the diagnosis of hypertensive pulmonary edema. She was rapidly treated with reservoir face-mask oxygen, intravenous nitrates, and diuretics. ECG showed only a sinus tachycardia without T-wave or ST-segment abnormalities. Chest radiography confirmed pulmonary edema. In addition, she had elevated cardiac

enzyme 6 h later (troponin I 3.5 $\mu\text{g/L}$, normal range < 0.2 $\mu\text{g/L}$). She was admitted to the cardiology intensive care unit. The initial TTE revealed a severe left ventricular (LV) systolic dysfunction (left ventricular ejection fraction [LVEF] estimated at 35%) due to an extensive apical and midventricular akinesis contrasting with hypercontraction of the basal segments. Moreover, a small pericardial effusion was noted. Eighteen hours after the onset of heart failure symptoms, coronary angiography ruled out obstructive coronary disease and there was no angiographic evidence of acute plaque rupture. Ventriculography showed the same LV wall motion abnormalities as TTE (Fig. 2). The diagnosis of tako-tsubo cardiomyopathy (TTC) was considered and acute heart failure treatment continued with angiotensin-converting enzyme inhibitors and beta-blockers. She made good clinical progress and was discharged from the intensive care unit at day 4. The follow-up TTE, 5 days after discharge, showed a total recovery of LVEF and the ECG indicated sinus rhythm without T-wave or ST-segment abnormalities.

Diagnosis criteria of TTC are: a) transient hypokinesis, akinesis, or dyskinesis in the LV mid-segments with or without apical involvement¹ and frequently, but not always, a stressful trigger event²; b) the absence of obstructive coronary artery disease or angiographic evidence of acute plaque rupture; c) new ECG abnormalities or modest elevation in cardiac troponin, and d) the absence of pheochromocytoma and myocarditis.³ Hormonal disturbances secondary to excessively elevated serum catecholamine levels have been suggested to be the cornerstone of the transient LV apical dysfunction. Other possible mechanisms like coronary vasospasm, coronary microcirculation alterations, or generation of a transient intraventricular dynamic gradient have also been proposed. A recent literature review found 6 published cases of TTC after cesarean delivery.⁴ In 4 cases, the onset of symptoms was preceded by administration of vasoconstrictive substances, including adrenergic stimulants (adrenaline, phenylephrine, ergonovine, and ephedrine) and anticholinergics (atropine). Our case report presented a similar pattern, as sulprostone is also a vasoconstrictive substance that elevates plasma levels of noradrenaline.⁵ To our knowledge, we described the first case of TTC after intravenous administration of sulprostone. However, we found 4 cases of cardiac arrest and one of pulmonary edema associated with sulprostone administration.⁶ All of them could



Figure 1. Twelve-lead electrocardiogram demonstrating junctional tachycardia with retrograde P waves (arrows) and diffuse ST-segment depression.

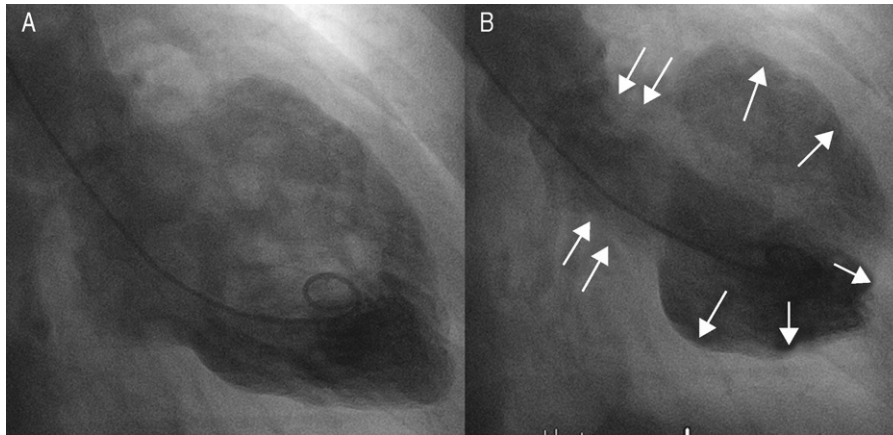


Figure 2. Left ventriculography (right anterior oblique 30°) showing apical ballooning. A, diastole; B, systole.

have also suffered from a TTC, because there were no detailed data concerning cardiac function in these reports. In our report, two other trigger factors also could have promoted the TTC: the cesarean delivery itself or the oxytocin infusion, which induced a junctional tachycardia with myocardial ischemia. However, the delay between the stressful trigger and the first symptom is usually less than 2 h in the published cases of TTC after cesarean delivery.¹ Moreover, our patient became transiently asymptomatic when the junctional tachycardia ended. These factors argue in favor of a probable relationship between sulprostone and the TTC.

The lesson gleaned from the described cases is worthwhile: cardiac symptoms, LV systolic dysfunction, or a slight troponin increase after sulprostone administration should lead to a suspicion of TTC. Moreover during cesarean delivery, the differential diagnosis could be an acute coronary syndrome caused by spontaneous coronary artery dissection. These two diseases can be distinguished by TTE and coronary angiography. Clinicians should be aware of this potential adverse effect when monitoring patients receiving sulprostone.

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Staphylococcus Aureus Endocarditis on Transcatheter Aortic Valves

Endocarditis por Staphylococcus aureus sobre válvula aórtica percutánea

To the Editor,

Transcatheter aortic valves are becoming an alternative for patients with symptomatic severe aortic stenosis when comorbidity makes conventional valve replacement surgery an unaffordable risk. We describe one of the first cases of infective endocarditis on the CoreValve[®] model (Medtronic, Minneapolis, Minnesota) prosthetic aortic valve.

An 81-year-old male with dyspnea secondary to severe degenerative aortic stenosis. Comorbidity consisted of diabetes

mellitus, chronic kidney failure, and a severely depressed left ventricular ejection fraction with pulmonary hypertension >60 mmHg. The coronary arteries showed no significant lesions. The surgical risk according to EuroSCORE was 29%, so it was decided to implant a CoreValve[®] transcatheter prosthesis, which took place after prophylaxis with ampicillin, with no major complications. According to the aortography, there was moderate, grade II/IV residual aortic regurgitation and the patient was discharged after a week.

During the following months, an improvement of 50% was seen in the left ventricular systolic function, with a decrease of pulmonary pressure to 45 mmHg. The patient remained asymptomatic, except for an admission 4 months after the implant for self-limiting gastrointestinal bleeding leading to anemia. A colonoscopy was performed without prior antibiotic prophylaxis and diverticulosis was diagnosed.