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# Cardiogenic pulmonary edema following β2 agonist infusion for acute bronchospasm

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#### Abstract

We report the case of severe acute pulmonary oedema (APO) secondary to the administration of salbut-amol to a postpartum patient treated for acute bronchospasm. The diagnosis of acute pulmonary edema was suspected on the clinical examination, chest radiography, biological and echocardiographic findings. Rapid improvement under dobutamine and mechanical ventilation argue in favour of cardiogenic pulmo-nary edema. The young age of our patient, the absence of history of cardiovascular disease and the chro-nology of this complication onset regarded to salbutamol infusion could suggest  $\beta^2$  agonist involvement in this event. The improvement of cardiac function on echocardiography.

Keywords: Salbutamol; Acute Pulmonary Edema; Acute Bronchospasm.

# 1. Introduction

Acute pulmonary oedema (APO) is one of the complications of the following treatments beta2-mimetic, which has only been reported in pregnant women. There appears to be a genuine beta2-stimulant cardiomyopathy, which rapidly improves when adequate treatment is initiated. The incidence is between 0.3 and 9% and the mortality rate 3%, depending on the publication. We report a case of PAO due to peripartum cardiomyopathy secondary to the administration of beta2-mimetics.

# 2. Observation

A 34 year old woman, with a history of a tonsillectomy at the age of 5 had had a pregnancy, correctly monitored, she gave birth on 27/12/2018 vaginally to a viable Female Newborn who weighed 3600g in a hospital centre. An hour later the patient presented with a delivery haemorrhage with shock. The patient was transferred to the operating theatre, where a deep tear in the cervix was sutured. The patient was transfused with 04 units of packed red blood cells and 04 units of FFP. On the second day, the patient developed dyspnoea with bronchospasm and sibilant rales on pulmonary auscultation, and was put on intravenous salbutamol at a dose of 3 µg/min for 48 h. The worsening respiratory condition required ventilatory assistance.

The patient's progress was rapidly favourable, enabling mechanical ventilation to be weaned after 24 hours.

On the fourth day of hospitalisation, the patient again developed acute respiratory failure.

Pulmonary auscultation revealed diffuse crackles in both lung fields. Cardiac auscultation revealed only regular tachycardia. The chest X-ray showed a bilateral alveolar syndrome.

The ECG trace was that of a sinus tachycardia, the echocardiography showed non-dilated right cavities, EF = 58%, FR = 26%, no signs of PAH, global hypokinesis of the LV in favour of cardiomyopathy. There was no valvular disease. Arterial blood gas showed PCO2 at 32 mmHg and PO2 at 55 mmHg in room air. Biological tests showed no abnormal renal function or DIC.

Thoracic angioscan: bilateral alveolar syndrome, no signs of pulmonary embolism.

The diagnosis was beta2-mimetic-induced APO. The patient received intravenous furosemide.

Progress was rapidly favourable, with disappearance of dyspnoea and oedema of the lower limbs.

A follow-up cardiac ultrasound performed on the tenth day of hospitalisation showed echographic normalisation of left ventricular function.

# 3. Discussion

Many observations of Acute pulmonary oedema (APO) during pregnancy have been reported in the literature. These mainly concern patients treated with beta2-mimetics for tocolysis. In these cases, the incidence of APO is high, with 1 in 400 patients treated [1], sometimes resulting in death [2]. The patient in our case presented with acute dyspnoea in the post-partum period, and several diagnoses need



to be considered [1]. Diagnostic arguments in favour of beta2-mimetic-induced APO are: recent discontinuation of beta2-mimetic therapy, and rapid favourable evolution when adequate treatment is initiated. If there is no significant clinical improvement after 12 to 24 hours of treatment, the diagnosis of beta2-mimetic-induced APO should be reconsidered.

The mechanism of beta2-mimetic-induced APO is not fully understood and appears to be multifactorial [3]. Multiple hypotheses have been put forward:

Cardiogenic APO, hydrosodium inflation, or non-cardiogenic APO with increased capillary permeability. There appears to be a genuine beta2-stimulant cardiomyopathy. In a series of 15 cases of peripartum cardiomyopathy, four patients had received prolonged treatment with beta2-mimetics; the course of their cardiopathy was favourable, with complete recovery of cardiac function in a few weeks or months. An isolated case of tocolytic-induced PAO with evidence of true cardiomyopathy has been reported [4]. Various mechanisms have been suggested to explain this myocardial damage: desensitisation of beta-adrenergic receptors as in catecholamine-reversible cardiopathies [5], [6], small foci of myocardial necrosis as in pheochromocytoma cardiopathies [7]. The patient in our study had no systolo-diastolic dysfunction on ultrasound. In addition, the patient benefited from many investigations, particularly haemodynamic investigations, as part of the diagnostic and therapeutic approach. There are currently many publications comparing the benefits of tocolytic treatment in terms of lengthening gestation, the incidence of maternal side-effects, and the causes and rate of perinatal mortality, with conflicting results [2-4], [6]. If tocolytic treatment with beta2 stimulants is decided upon, the dose and duration of treatment should be limited as much as possible. In order to limit hydrosodic intake, beta2-mimetics should be used as soon as possible. In order to limit fluid intake, beta2-mimetics should preferably be administered in concentrated form by electric syringe. During treatment, clinical monitoring should include water intake and output, daily weighing, pulmonary auscultation and, if necessary, daily measurement of saturation using a pulse oximeter.

#### 4. Conclusion

We report a case of severe PAO induced by beta2-mimetics, requiring a stay in intensive care. Parturients in whom beta2-mimetics are used for tocolysis, severe acute asthma or bronchospasm for more than 48 hours should have their cardiac function assessed. Characterised by a rapidly favourable outcome, this entity should be reconsidered in the absence of significant clinical improvement after 12 to 24 hours of treatment. [1]

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