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How Can Obstructive Sleep Apnea Syndrome Affect Right Ventricle: About an Important and Reversible Pulmonary Hypertension Case

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Abstract **Case Report**

Obstructive sleep apnea syndrome (OSAS) is a common, and often under-diagnosed, affecting 9% of men and 4% of women in the middle age. The prevalence of pulmonary hypertension in OSAS varies from 15 to 20%. It is generally moderate and due to intra-thoracic pressure variations and episodes of oxygen desaturations. We report the case of a patient in whom the obstructive sleep apnea syndrome, was resposible for a very important pre-capillary pulmonary hypertension and right ventricular systolic dysfunction (RV). We also report the marked improvement of this pulmonary hypertension (PH) and the recovery of a good function of the right ventricle under continuous positive pressure therapy. Rare are the authors who reported the occurrence of dysfunction of RV in the context of an OSAS, ethere rmains a need for serial evaluation of RV function in pulmonary hypertension during OSAS

Keywords: OSAS, pulmonary hypertension, right ventricle systolic dysfunction.

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a common, and often under-diagnosed, affecting 9% of men and 4% of women in the middle age [1].

It is defined by the repetitive onset, during sleep, of complete or partial obstructions of the upper airways responsible for apnea or hypopnea [2]. The prevalence of pulmonary hypertension in OSAS varies from 15 to 20%[3]. It is due to intra-thoracic pressure variations and episodes of oxygen desaturations. The pulmonary hypertension observed during OSAS is generally and moderate with possible exacerbation during exercise or sleep. It is often post-capillary related to cardiovascular complications of OSAS [4].

Objectives

The aim is to show through the observation of our patient that OSAS, in the absence of any cardiovascular nor pulmonary pathology, can cause a very important pre-capillary pulmonary hypertension and right ventricular dysfunction (RV). We also report the marked improvement of this pulmonary hypertension (PH) and the recovery of a good function of RV under continuous positive pressure therapy.

MEDICAL OBSERVATIONS

We report a 60-year-old woman without cardiovascular risk factors, hospitalized for right heart failure. On admission ; she was hemodynamicly stable with 100 pulses per minute and 110-60 mmhg on his blood pressure and had signs of right heart failure. Cardiopulmonary auscultation finds a systolic murmur of tricuspid insuffisancy, pulmonary B2 glow.

The Eelectrocardiogram and Chest X-ray showed indirect signs of pulmonary hypertension. Tansthoracic echocardiography showed a significant pulmonary hypertension with dilatation and à limiting function of the right ventricle. Left ventricle (LV) systolic function was preserved. No anomaly of the LV nor congenital pathology was found. The patient was put on diuretics and oxygen with partial improvement.

Cardiac catheterization showed a significant average lung pression at 40mmhg, capillary pulmonary pressure at 10mmhg, positive vasoreactivity to oxygen and NO (nitric oxide). A complete assessment for pulmonary pathology or systemic disease was carried out but did not find any cause.

Beside, medical staff at night reports significant snoring by the patient. In view of this, a ventilatory polygraph was performed showing moderate OSAS. The patient was ventilated by continuous

positive airway pressure with ball T-shirt. To our surprise, after 6months of treatement the RV has recovered a good systolic function and the PH has clearly regressed.

The diagnosis of severe pre-capillary pulmonary hypertension with alteration of the systolic RV function due to OSAS was retained.

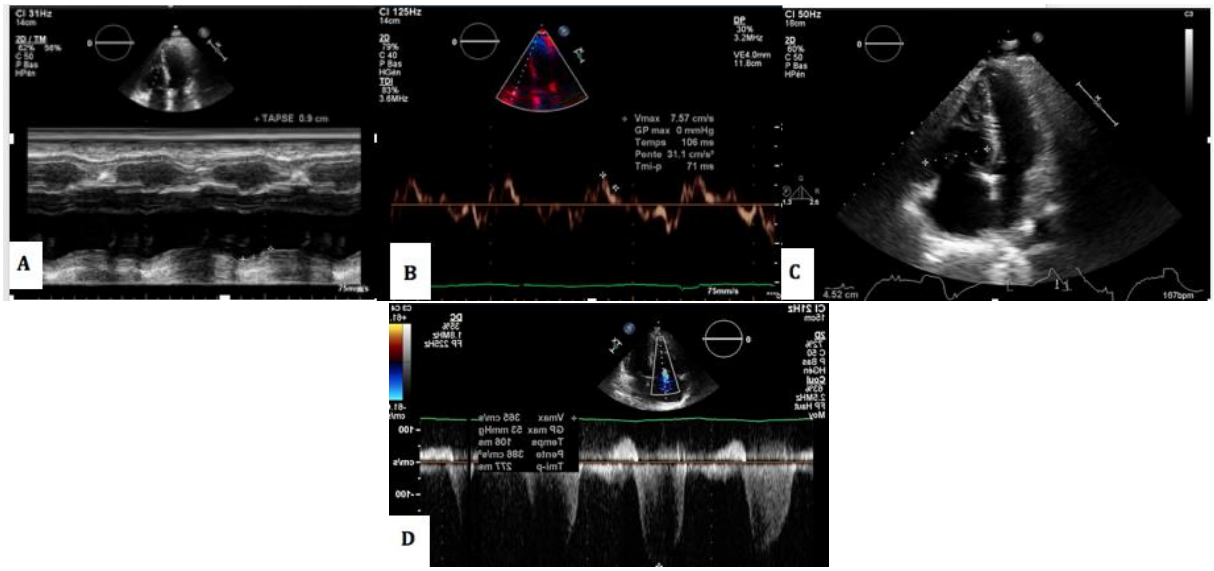

Fig-1: Transthoracic echocardiography showing a RV systolic dysfunction : TAPSE to 9mm (A), Tricuspid S wave at 7,5cm/s (B), RV dilatation, basal diameter at 41mm (C), and tricuspid insuffisancy giving a PAPS at 73mmhg (D)

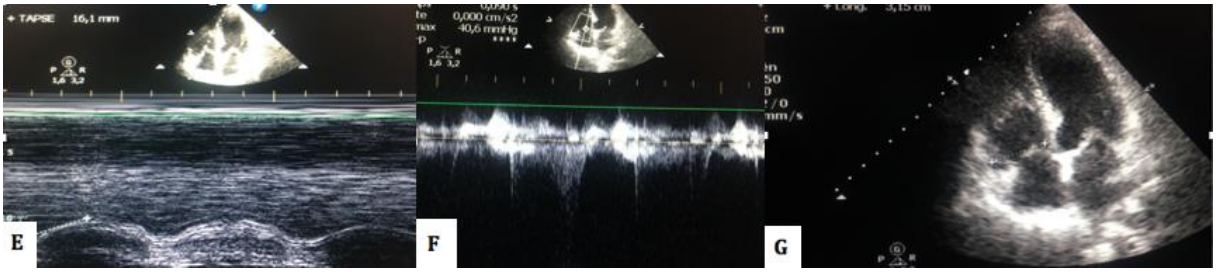

Fig-2: Shows the recovery of a normal size (G) and good systolic RV function (E), regression of the tricuspid insuffisancy (F)

DISCUSSION

Obstructive sleep apnea syndrome is a common but largely under-diagnosed condition [5, 6]. The objective value characterizing OSAS is the hypopnea apnea index (IAH) measured by polygraphy or polysomnography [7]. Heart failure, systemic and pulmonary hypertension, coronary artery disease, diastolic LV dysfonction are widely reported in OSAS, however the RV dysfunction is not very known during OSAS [8].

The pulmonary hypertension (PH) that shown in OSAS is generally moderate, related to changes in pulmonary arterial pressure (PAP) during sleep, and punctuated by respiratory events and intermittent desaturations secondary to apnea [9]. Indeed acute hypoxemia induced by nocturnal apneas leads to pulmonary arterial vasoconstriction responsible for repeated peaks of PH. Pulmonary arterial pressures increase during an apnea episode, often reaching their maximum at the end of apnea and decreasing when ventilation is restored [10]. This PH is pre-capillary with moderate PAPm and normal pulmonary capillary pressure [11]. Several studies have shown that increased PH is frequently observed when SAS is associated with daytime hypoxemia secondary to either significant obesity or chronic obstructive pulmonary disease (BPCO) [12].

In the case of our patient, the explorations showed a severe PH in the absence of obesity or obstructive ventilatory disorders, an idiopathic PH can also be evoked, but in front of the favorable evolution and the net decrease of this PH under ventilation by continuous positive airways pressure(CPAP), this suggests that SAS alone may be responsible for severe and permanent PH in the absence of any other cardiovascular risk factors or progressive pulmonary disease.

Subsequent pulmonary vasoconstriction to intermittent nocturnal hypoxia, and the exaggerated

intra-thoracic negative pressure leads to an increase post-load of right ventricle as well as a remodeling both the pulmonary vascular bed and the ventricular myocardium, and in the long run, RV systolic dysfonction (6).

RV dysfunction in OSAS is therefore plausible [13], and our patient's case supports it. The recovery of a good function after treatment confirms it more. However rare are the authors who reported the occurrence of dysfunction of RV in the context of an OSAS[14]. Therefore, there remains a need for serial evaluation of RV function in pulmonary hypertension during OSAS to confirm this.

CPAP allows to treat OSAS and decrease PH (13), there is no indication for specific treatments for pulmonary hypertension in this population.

CONCLUSION

Sleep apnea syndrome is a pathology that is underestimated. Our case shows that pulmonary hypertension due to nocturnal apnea episodes can be severe and responsible for a significant impact on the right ventricle, however this result needs to be further research to assess RV function during OSAS.

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