CASE REPORT

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Pickwickian syndrome with severe pulmonary hypertension: Deciphering the early connection

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ABSTRACT

Pulmonary Hypertension (PH) is commonly found in obesity hypoventilation syndrome but it has been assessed in very few studies. In patients referred to sleep clinics pulmonary hypertension is common among with OHS and must therefore be included in their regular assessment. This case report highlights a young male (20 year old) presenting with severe pulmonary hypertension who was having morbid obesity diagnosed as pickwickian syndrome.

Keywords: obesity; pulmonary hypertension; pickwickian syndrome.

1. INTRODUCTION

Pickwickian syndrome or obesity hypoventilation syndrome is a condition seen in those with a body mass index of > 30 kg/m2 with daytime PaCO2 >45 mmHg after other causes for alveolar hypoventilation have been ruled out (Mokhlesi, 2010; Kessler et al., 2001; Olson et al., 2005). It's associated with cardiovascular and metabolic derangements (Piper et al., 2011; Borel et al., 2012; Salih et al. 2020). Pulmonary hypertension and right heart failure are commonly seen and are associated with poor outcome (Kessler et al., 2001; Overvad et al., 2008). Pulmonary Hypertension is a condition in which the mean pulmonary artery pressure is >20mmHg. On literature search from PUBMED, google search scholar, MEDLAR, SCOPUS, no article was found regarding pickwickian syndrome in young presenting with severe pulmonary hypertension. In this case we have reported a young male, 20year old presenting with severe pulmonary hypertension with morbid obesity diagnosed as pickwickian syndrome.

2. PRESENTATION

A 20 year old obese male from rural India presented to the OPD with the complaints of breathlessness on exertion (and later at rest) associated with orthopnea and paroxysmal nocturnal dyspnea since 10 days with no complaints of palpitations, chest pain, syncope, anxiety, cough or fever. He also gave history of bilateral pedal edema, distention of abdomen and difficulty in passage of stools since then. Symptoms like pain in abdomen, nausea, vomiting and burning micturition were absent. No comorbidities or addictions were noted with the exception of occasional alcohol consumption. Being hefty since the past 2 years with increasing severity of breathlessness and the number of PND episodes as he added pounds was evident while procuring his details in the OPD. On admission, He was 178cm tall with a weight of 116 kg and a BMI of 36.6kg/m^{2}. He was hemodynamically stable with mild tachycardia and 100% O_2 saturation. On auscultation he had normal heart sounds and diffuses crepitations over both lung fields. His abdomen was distended and pitting edema could be demonstrated bilaterally.

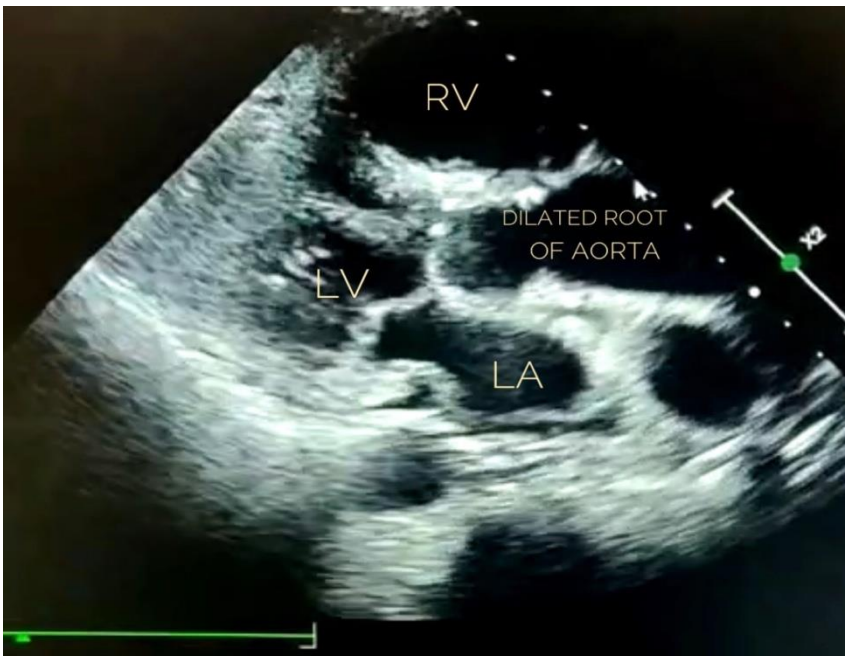

Figure 1 RV: Right Ventricle, LV: Left Ventricle, LA: Left Atrium; 2D Echo image showing dilated root of aorta with dilated right ventricle and left ventricular hypertrophy

His investigations revealed a serum creatinine of 1.5, SGOT of 50, SGPT of 72 and a total bilirubin of 1.2 mg%. ABG showed a pH of 7.2, pCO2 of 45 mmHg and pO2 of 93 mmHg. Subsequently hypoxia and hypercapnea were observed with a fall in saturation to 82%. He was put on Biphasic Positive airway pressure 12/8 with FiO2 at 60%.Following this his saturation improved and ABG showed a Ph 7.3, pCO_2 of 50, po2 of 89.5 and a saturation of 96% on the ABG. Diffuse haziness in both lung fields was observed on chest X-ray. He was then started on treatment with loop diuretics (furesamide 40mg OD), bosentan 125 mg OD and other supportive treatment. T inversion in lead V3-V4 was observed on ECG. An ultrasound of the abdomen and pelvis showed hepatomegaly with grade II fatty liver with moderate splenomegaly and bilateral pleural effusion. 2 D echo was suggestive of Dilated Right atrium and right ventricle, dilated root of aorta, moderate tricuspid regurgitation (51mm), moderate to severe pulmonary arterial hypertension (PASP= 70mmHg) and normal biventricular function (figure 1).

Following initiation of diuretic therapy weight reduction was apparent with improvement in respiratory distress. On the 4^{th} day he weighed 110 kg with a further reduction of 2kg by the time he was discharged. On follow up patient was doing with marked weight reduction and reduced breathlessness.

3. DISCUSSION

Pickwickian syndrome and pulmonary hypertension are commonly observed together but this inclination has been reviewed upon in a limited few studies. One mechanism doesn't serve the purpose of sifting through the multiplicity of routes that explain this propensity. Cumulative adiposity particularly in the chest and abdomen serve to impair physiological pathways and cause functional de-compensation, which may revert following weight loss (Borel et al., 2012; Overvad et al., 2008). Fat dispersal is of light in comparison to the total body fat or BMI (Ladosky et al., 2001). Of particular importance is the decrease in the engaging capacity of the chest wall and respiratory muscles causing diminished total lung capacity and functional residual capacity with loss of expiratory reserve volume (Jones, 2006). Ventilation/perfusion mismatch occurs as an outcome of pulmonary hypoventilation due to basal atelectasis, shunting and overall reduction in lung volume, which eventually presents as hypoxemia (Jones, 2006; Koenig, 2001). Progressive deterioration is come upon more often than not in these cases. PCO2 retention, hypoxia and acidosis in addition to restricted lung pathologies are of significance in the mechanism of PH in OSA (Koenig, 2001; Friedman et al., 2012). Increased venous return and right ventricular filling will be noticed as a consequence of negative intra--thoracic pressure secondary to upper airway obstruction subsequently manifesting as decreased LV filling and reduced stroke volume. Assessed parameters rarely divulge paths with the presence PH in these patients. Amongst the cases reviewed in multiple studies an older age presets the likelihood of PH amongst those with OSA. Genetic adaptation to chronic hypoxia is prevalent in certain cases with recent data to suggest the same (Friedman et al., 2012). Being a reversible entity life style modification in addition to drug therapy remains mainstay in the overall management.

4. CONCLUSION

Though there is no evidence of severe PH in the long- term prognosis of pickwickian syndrome, it should be considered in the regular clinical assessment of all such patients.

Informed consent

Informed consent was taken from the participant included in the study.

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Conflict of interest

The authors declare that they have no conflict of interest.

Data and materials availability

All data associated with this study are present in the paper.

Peer-review

External peer-review was done through double-blind method.

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