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Case Report

The Pickwickian child: a case report of paediatric obesity hypoventilation syndrome

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ABSTRACT

Childhood obesity is on the rise and with it are also some unusual complications like Obesity hypoventilation syndrome, the triad of which includes obesity, hypoventilation and sleep disordered breathing. The absence of a mechanical, neuromuscular or metabolic explanation is essential before making this diagnosis. We report the case of a 10-year-old male child who was brought to us for the first time with acute onset breathlessness on exertion and generalised oedema, with progressive weight gain in the last 3 years. He was diagnosed with OHS with severe Obstructive sleep apnoea leading to cardiac failure and eventually improved with antifailure treatment, sildenafil, lifestyle modifications and BiPAP. Prompt recognition and early initiation of treatment measures including stringent lifestyle modifications are essential in preventing complications of OHS like cardiac failure.

Keywords: Pediatric obesity, Obesity hypoventilation syndrome, Pediatric sleep

INTRODUCTION

With an epidemiological transition taking place worldwide, India has seen its fair share of rise in lifestyle related disorders in children, especially obesity. The incidence has been steadily increasing with the rampant consumption of junk food, decline in physical activity and increased use of devices. Recent times have seen a significant increase in the number of obese children. Obesity, which is defined in children using age- and sex-specific criteria, typically as a Body mass index (BMI) at or above the 95th percentile, is not only a risk factor for cardiometabolic disease but also an important contributor to sleep-related breathing disorders, including Obstructive sleep apnea (OSA) and less commonly, Obesity hypoventilation syndrome (OHS).[1] While OSA is relatively well described in the pediatric population, OHS is often overlooked and remains underdiagnosed, particularly in resource-limited settings. OHS is classically defined in adults by the triad of obesity, sleep-disordered breathing and chronic daytime hypercapnia ($PaCO_2 \geq 45$ mm Hg at sea level), in the absence of alternative causes of hypoventilation.[2]

OHS is extensively studied in adults, but its presentation in children can be subtle and is frequently masked by overlapping features of OSA. Delayed recognition may lead to significant morbidity, including pulmonary hypertension, right heart failure and neurocognitive impairment.[1]

Children with sleep-disordered breathing often present with nonspecific symptoms, which may be misattributed to behavioral or lifestyle factors. Weight gain further exacerbates ventilatory compromise, creating a vicious cycle that can culminate in acute decompensation. In such cases, the child may present with respiratory failure requiring intensive care support.[3] The diagnosis and management of OHS in children in low-resource environments are particularly challenging due to limited access to advanced diagnostic tools and also to long-term ventilatory support. Hence, there is a need for early clinical

suspicion and pragmatic treatment approaches tailored to individual circumstances.

We report the case of an obese 10-year-old boy who presented with respiratory failure and was subsequently diagnosed with OHS with severe obstructive sleep apnea, leading to cardiac failure. This case highlights the importance of early recognition of sleep-disordered breathing in children with obesity and illustrates the potential consequences of delayed diagnosis, especially in settings with limited healthcare access.

CASE REPORT

A 10-year-old boy presented to the emergency room with breathlessness on exertion and generalized edema for 3 days. His oxygen saturation on room air was 70%, hence he was started on oxygen by high flow nasal cannula but he collapsed soon after and was ventilated and kept on mechanical ventilation. The first blood gas showed profound respiratory acidosis with PCO_2 of 120 mm Hg and a pH of 7.020 and bicarbonate level of 30 mmol/l pointing towards an acute on chronic respiratory acidosis. The oxygen saturation gradually improved thereafter to 86%. The sensorium too improved gradually.

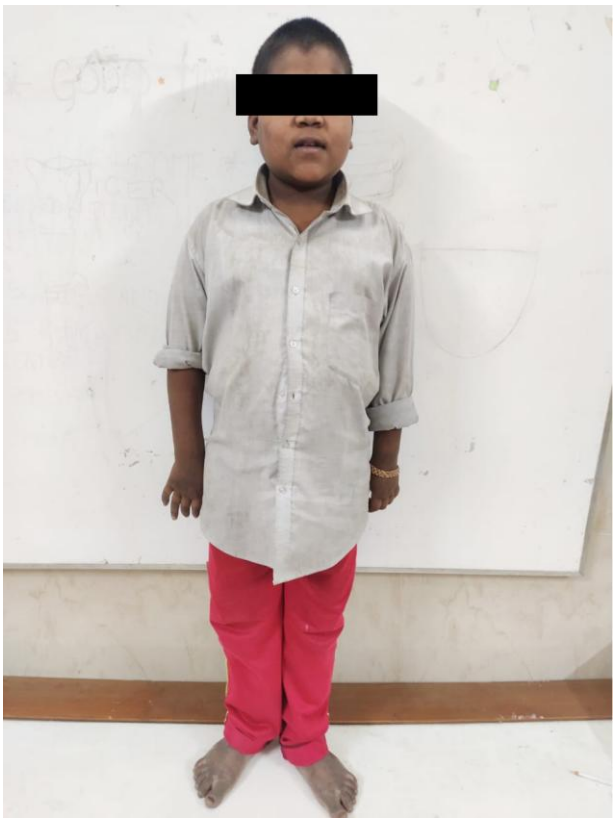

Figure 1: Clinical photograph of the child.

The child was obese with a weight of 58 kg and height of 136 cm and a BMI of 31.3kg/m^2. He had a short neck with acanthosis nigricans and most of the body fat was abdominal in distribution. There were no dysmorphic features and the genitalia were normal. The child had an enlarged liver with facial oedema and pitting oedema in both lower limbs. The blood pressure was at the 99th centile for age and height. He had bilateral crepitations in the lung bases with an enlarged and tender liver of around 7-8 cm below costal margin. He responded well to positive pressure ventilation, inotropes and diuretics. The CO_2 retention gradually improved to 58 mm Hg. A portable Chest X-Ray revealed pulmonary plethora with straightening of the left heart border. There was no polycythaemia on the complete blood count but leucocytosis with polymorphic predominance and elevated CRP.

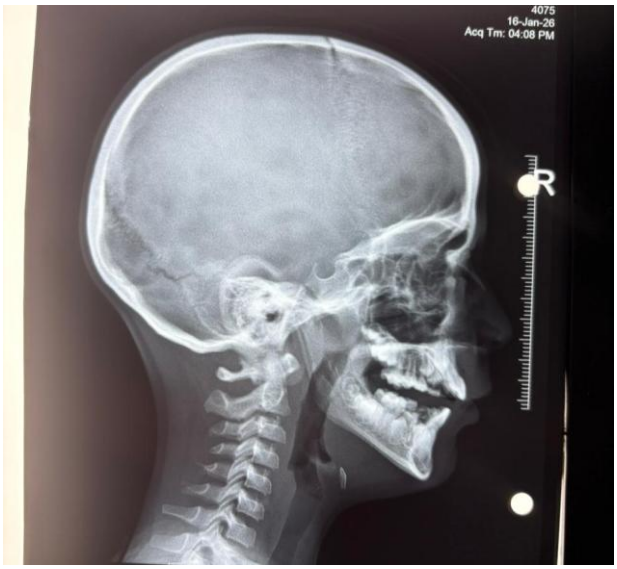

Figure 2: X-ray nasopharynx of the child showing adenoid hypertrophy.

He was eventually weaned from the respiratory support and 2D ECHO was performed on stabilization which revealed an unpleasant surprise in the form of severe pulmonary hypertension with right sided heart failure. He was started on oral sildenafil for pulmonary hypertension. The blood pressure fell to below the 90th centile on its own as the effect of Sildenafil started to take effect.

On enquiry the mother admitted that the child has gained a large amount of weight in the last 3 years. She also reported that the child frequently snored and breathed from his mouth through the night. This resulted in a disturbed sleep and subsequent daytime sleepiness which led to a gradually declining scholastic performance. An X-Ray of the nasopharynx revealed enlarged adenoids and the child was started on fluticasone nasal spray for the same.

A sleep study was performed and revealed severe obstructive sleep apnoea with 23 documented apnoeic episodes and an apnoea hypopnoea index of 15. A decision to initiate BiPAP therapy was considered, however, the child lived in a tribal region with no access to a reliable supply of electricity. As the parents were very reluctant for BiPAP therapy, a compromise was reached and tennis ball

technique was considered. The child tolerated the same for 3 nights and was discharged on oral sildenafil with a plan of tapering once the pulmonary pressures reduced. Regrettably, the parents never brought the child for a follow-up visit and attempts to contact them were unsuccessful.

DISCUSSION

With the incidence of obesity in the pediatric population showing a steady increase worldwide, it is crucial to understand it better. Our case highlights that pediatric OHS may have a life-threatening clinical presentation that is poorly understood especially in the pediatric population. It arises from a complex interaction of mechanical and hormonal factors, with impaired ventilatory response to hypoxia and hypercapnia, leptin resistance and obstructive sleep apnoea.[4]

Our patient had all the hallmarks of OHS namely obesity with a BMI of 31.3kg/m^2, chronic hypoventilation as suggested by his blood gas analysis and a prominent history of sleep disordered breathing with adenoid hypertrophy. The episodes of desaturation and arousals led to a very fragmented sleep with prominent daytime somnolence. The child's restless nights eventually led to a restless mind with attention deficits, impaired concentration and declining scholastic performance. He also complained of early morning headaches occasionally with a drop-in physical activity and endurance which may be early indicators of cardiac dysfunction.

The path to a clinical diagnosis of OHS is littered with obstacles in the form conditions that closely mimic OHS namely OSA, especially when daytime hypercapnia is not looked for. The nighttime symptoms may sometimes be attributed to bronchial asthma which may have concomitant allergic rhinitis and adenoid hypertrophy.[1] The neurocognitive disturbances may be attributed to a neurological or psychiatric disorder in the absence of a detailed sleep history. This overlap with so many conditions often results in a delayed diagnosis, many a times directly in the emergency room with respiratory or cardiac decompensation as with our patient.

It is likely that had this child been diagnosed earlier, the untoward complication of pulmonary hypertension could have been avoided. While the paucity of research in pediatric OHS curtails our understanding of the same, the evidence at hand indicates that compensatory mechanisms may not be as well-developed in children and adolescents as adults, resulting in a faster progression to chronic hypercapnia.

Fortunately, this progression may be halted by a holistic treatment approach with non-invasive ventilatory support, and comprehensive weight management.[5] Compliance to both with long-term follow up and psychosocial support is paramount to ensure a successful outcome. Medical management in the form of Montelukast and inhaled corticosteroids are helpful for children with concomitant adenoid hypertrophy and asthma.[6] Treating complications like right sided heart failure in our patient is important, so is preventing other complications like polycythaemia. Neurocognitive impairment should not be neglected and should be addressed and monitored with each follow up.

Unfortunately, a severe lack of pediatric literature on OHS limits our comprehension of this clinical entity. Moreover, most of the diagnostic criteria are extrapolated from adult data and trials without any age-specific cut offs for the parameters. Most of the data reported is from complicated cases in tertiary centers like our patient and is retrospective in nature, making it difficult to study the disease progression in mild to moderate cases and how timely intervention may help these children.

Although pediatric-specific prevalence data remain limited, available cohort studies suggest that OHS is uncommon but likely underdiagnosed. In a French retrospective cohort, Gachelin et al reported that OHS was identified in 3.9% of 102 obese children undergoing sleep evaluation, with significant overlap with obstructive sleep apnea.[7]

According to Isaac et al increasing BMI remains the most consistent and fundamental risk factor for OHS, demonstrating a dose–response relationship; however, in an Indian cohort, variables such as female sex (OR 4.1) and orthopnea (OR 4.8) showed stronger associations, likely reflecting disease severity rather than primary causation.[8]

The clinical significance of pediatric OHS must be interpreted in the context of the growing obesity epidemic. Current estimates suggest that 12.7% of children aged 2-5 years and 20.7% of those aged 6-11 years are obese, placing a large population at risk for obesity-related respiratory complications.[1]

CONCLUSION

This case highlights obesity hypoventilation syndrome as an important yet underrecognized complication of paediatric obesity, with the potential to present as acute respiratory failure and progress to significant cardiopulmonary morbidity if not identified in time. It underscores the need for a high index of suspicion in obese children presenting with symptoms of sleep-disordered breathing, daytime somnolence or unexplained hypercapnia. Early identification and prompt implementation of suitable therapies, such as systematic weight control and non-invasive ventilation, can dramatically change the course of the disease and avert long-term consequences such as right sided heart failure and pulmonary hypertension.

This example also highlights the practical difficulties in managing long-term conditions like OHS in settings with minimal resources, where access to long-term ventilatory

support and diagnostic instruments may be limited. Individualized and practical therapy approaches become crucial in these situations. To improve outcomes and lower the burden of this potentially preventable illness in the paediatric population, early screening, a multidisciplinary approach and increased clinician awareness are essential.

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