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Case Report

Diagnostic and Management Approach of Obesity with Multiple Complications in a Child: A Case Report

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Abstract:

Background: Adults with obesity may already experience obesity during childhood or adolescence, highlighting the critical importance of early intervention. This is particularly concerning given that childhood obesity, a growing component of Indonesia's "triple burden of malnutrition," significantly increases the risk of developing severe non-communicable diseases and reducing life expectancy.

***Case*:** A 10-year-old male patient presented with a chief complaint of shortness of breath for the past two weeks, worsened when lying down and improved when sitting up. The patient also snored, often woke up due to difficulty breathing. The patient had experienced rapid weight gain since the age of 2 years. He ate in large portions, frequently snacked, and consumed sugary drinks daily. He had no regular physical activity and was mostly sedentary. He showed signs of obesity (BMI 35.8 kg/m^2), short stature, and physical abnormalities including a rounded face, double chin, acanthosis nigricans, and bowed legs.

***Discussion*:** Diagnosing obesity requires comprehensive history and physical examination to distinguish between primary and secondary causes. Our patient's early-onset obesity and hyperphagia prompted leptin level evaluation, although the result was within normal limit, leptin resistance or receptor imbalance was suspected. In this case, familial lifestyle factors appear to play a role, highlighting the importance of a family-centered approach. Management of obesity includes dietary modification, physical activity, sleep and behavioral regulation, and pharmacologic therapy when indicated.

Conclusion: An accurate diagnostic approach is crucial to guide optimal management strategies in complicated cases of obesity.

Keywords: children, diagnostic, management, obesity

Introduction

Obesity is one of Indonesia's "triple burden of malnutrition," falling under the category of overnutrition.[1] Globally, approximately one in five children are affected by obesity, a figure that has increased by 2.5% in the last two decades.[2] According to the 2018 Basic Health Research, the prevalence of obesity among children aged 5 to 12 years in Indonesia was 9.2%.[3] Obesity is a major risk factor for various non-communicable diseases (NCDs), which were estimated to contribute to 73% of all deaths in 2018.[4,5] Importantly, 15 – 30% of adults with obesity had already experienced obesity during childhood or adolescence.[6]

Children with obesity face a two-fold increased risk of developing cardiovascular diseases in adulthood.[7] This heightened risk comes from factors such as elevated blood pressure, increased cholesterol levels, and the accumulation of plaque in blood vessels. Other complications arising from childhood obesity include type 2 diabetes mellitus, hypertension, dyslipidemia, fatty liver disease, respiratory disorders like obstructive sleep apnea syndrome (OSAS), and other metabolic disturbances. Children with complicated obesity tend to experience chronic diseases earlier in adulthood, leading to a reduced life expectancy compared to those with uncomplicated obesity.[8-10]

Furthermore, approximately one in ten obese children experience depression due to social stigma and emotional distress, a higher rate than in overweight children.[11] Given the wide range of complications posed by obesity, the aim of this case report is to explore comprehensive diagnostic and management approaches. These approaches include dietary modifications, increased physical activity, and psychosocial support, all intended to mitigate the negative impacts of obesity and its complications, thereby improving the quality of life for affected children.

Case

A 10-year-old male patient presented to the Pediatric Nutrition and Metabolic Disease clinic at Dr. Cipto Mangunkusumo Hospital (CMH). He was referred from a regional hospital with a chief complaint of progressively worsened shortness of breath for two weeks prior to hospital admission. The patient's history revealed a rapid weight gain beginning at the age of two, reaching 50 kg by age five. He exhibited a large appetite, ate more than three times a day with each meal portion equivalent to twice as much as an adult. In addition to large meals, he frequently consumed snacks due to persistent hunger, even less than an hour following a meal. He reported no fruit consumption as he disliked it.

Two years prior to admission, the patient began experiencing shortness of breath during light activities and reported easy fatigability, which led to reluctance in participating in school sports. For the past year, he noted breathlessness even when walking short distances, such as to the bathroom.

Two weeks prior to admission, the patient was hospitalized at a regional hospital due to shortness of breath, high blood pressure, and swelling extending from his abdomen to his genitalia. He was diagnosed with cardiomegaly and received diuretics and antihypertensive medication captopril 25 mg twice daily. At that timehis shortness of breath was most pronounced when lying flat and improved in a sitting position. He denied chronic cough, drastic weight loss, or prolonged fever, but he frequently experienced recurrent cough and cold, attributed to allergies. The patient found it more comfortable to sleep with two pillows or in a sitting position. His symptoms were also accompanied by snoring and frequent awakenings due to breathlessness during sleep.

A dietary intake analysis at home revealed the patient typically consumed 3–4 meals per day, with each meal consisting of 2–2.5 portion of carbohydrate (rice), 1.5–2 portion of animal protein, 0.25–0.5 portion plant-based protein, and 2–3 tablespoons of vegetables. His vegetable intake was limited to a few types, such as water spinach and long beans. Fruit intake was rare, with bananas being the only preferred fruit. According to the mother, the child often purchased snacks without parental supervision, such as one portion of meatballs, chicken noodle, cakes, sweet bread, and commercially sweetened tea beverages up to 4–5 times a day. The patient did not engage in regular physical activity. He also frequently felt drowsy and was unable to walk long distances due to feeling heavy-breathed. At home, he mostly lay down, watched television, or slept. There was no history of steroid use.

The patient was the first of two siblings, born full-term via normal spontaneous vaginal delivery at a hospital. His birth weight was 2800 grams; birth length and head circumference data were unavailable. The patient's younger brother was 4 years old, healthy, and was not obese like him.

The patient's father had been obese since childhood, frequently consumed snacks, was an active smoker, and had a low level of physical activity. The patient's mother began gaining weight after marrying the patient's father. Upon encounter, the father weighed 97 kg with a height of 170 cm (BMI 33.5 kg/m^2), while the mother weighed 73 kg with a height of 153 cm (BMI 31.2 kg/m^2). There was no family history of similar complaints, nor any history of heart disease, hypertension, or diabetes mellitus.

On examination, the patient's blood pressure was 131/73 mmHg (P95+12 to P95+30), consistent with hypertension. All other vital signs were within normal limits. The patient weighed 56 kg and was 125 cm tall, with a body mass index (BMI) of 35.8 kg/m^2. His head circumference (HC) was 53 cm. Based on the Centers for Disease Control (CDC) growth curves, the patient's weight-for-age was 179% (56/32), height-for-age was less than the 3rd percentile (125/138), weight-for-height was 233% (56/24), and BMI-for-age was greater than the 95th percentile (35.8/16.7). The head

circumference-for-age was at the median (53/53). These measurements indicated that the patient's nutritional status was obese with short stature.

The patient presented with a rounded face, chubby cheeks, double chin, and a short neck with acanthosis nigricans. The physical examination showed enlarged tonsils (T3/T3) without visible crypts or detritus, enlarged breasts, a distended abdomen with folded walls, a buried penis with intrascrotal testes (3ml bilaterally, Tanner stage G1P1 and normal stretched penile length. His digits appeared short, with no edema, and there were bowing of the lower extremities, with normal range of motion in all extremities.The patient underwent routine blood tests (hemoglobin 12.4 g/dL, hematocrit 41.3%, leucocyte 11,480/uL, thrombocyte 321,000/uL), lipid profile (high-density lipoprotein (HDL) 35 mg/dL, low-density lipoprotein (LDL) 123 mg/dL, triglyceride 67 mg/dL), uric acid (3 mg/dL), liver function (SGOT 29 U/L, SGPT 30 U/L), renal function (ureum 10.7 mg/dL, creatinine 0.3 mg/dL, glomerulus filtration rate 171 ml/minute/1,73m^2), electrolytes (sodium 138 mEq/L, potassium 4.1 mEq/L, chloride 104.6 mEq/L), and blood glucose parameters (HbA1c 5.2% and fasting blood glucose 65 mg/dL), all of which were within normal limits. The patient also underwent a leptin laboratory test with a result of 44 ng/mL, and whole exome sequencing was planned due to suspicion of a genetic defect.

The patient was diagnosed with obesity and short stature, suspected with primary obesity with a differential diagnosis of secondary obesity due to possible leptin hormone deficiency, complicated by congestive heart failure (New York Heart Association Class (NYHA) class III) secondary to hypertension and severe obstructive sleep apnea syndrome (OSAS), and tonsillar hypertrophy. Admission was planned for nutritional management (1,920 kcal/day divided in three main course meals, with fruit as snacks, and water as the only permitted beverage), antihypertensive therapy, and multidisciplinary consultations.

Over 4 days of hospitalization, the patient continued to sleep with 2–3 pillows and exhibited persistent snoring. The patient experienced nocturnal desaturation episodes during sleep, requiring supplemental oxygen via nasal cannula. Blood pressure remained between the P95 and P95+30 range.

On day 5 of hospitalization, the patient underwent echocardiography, which revealed tricuspid and mild aortic valve regurgitation with an ejection fraction of 60%. Captopril (12.5 mg orally every 8 hours) was continued, with follow-up echocardiography scheduled in 6 months. The respirology team recommended nightly continuous positive airway pressure (CPAP). The nephrology team advised continuation of antihypertensive therapy and a low-sodium diet. The rhinolaryngscopic evaluation by ear, nose, and throat (ENT) team showed adenoids occupying 60% of the choanae, nasopharyngeal reflux, laterolateral oropharyngeal

narrowing, and grade 1 lingual tonsil hypertrophy. Subsequently, fluticasone proprionate nasal spray was prescribed twice daily.

The persistent loud snoring with awakenings and oxygen desaturation (down to 93%) during sleep was still observed on the 6th day of hospitalization. The patient was only able to walk 50 meters without experiencing difficulty breathing. His chest X-ray revealed cardiomegaly and early pulmonary edema, prompting the addition of furosemide (10 mg orally twice daily). The physical medicine and rehabilitation team initiated a moderate-intensity aerobic exercise program (3–5 times per week) to improve the patient's physical tolerance.

The patient underwent a 24-hour polysomnography, which confirmed severe OSAS with desaturation phases down to 50%. Based on these results, CPAP therapy was initiated at a positive end-expiratory pressure of 5 mmHg. Following CPAP initiation, snoring and sleep apnea improved, and dyspnea decreased. With CPAP, the lowest recorded nocturnal oxygen saturation was 89%, with levels reaching up to 98%.

On day-13, drug-induced sleep endoscopy (DISE) and bilateral tonsilloadenoidectomy were performed. Following the procedures, snoring continued to improve, and CPAP therapy was maintained. The total duration of CPAP use was 7 days.

Two days following the surgery, the patient reported no complaints. The patient was discharged with routine medications (captopril 12.5 mg every 12 hours orally, furosemide 10 mg every 12 hours) and dietary education (3 large low-sodium meals with more vegetables and 2 fruit snacks per day).

During the hospital stay, the patient's weight decreased by 4000 grams (267 grams/day). The final diagnoses were severe OSAS, obesity with short stature (with differential diagnoses of suspected leptin receptor resistance and other secondary obesity types), NYHA Class III congestive heart failure secondary to hypertension and severe OSAS, grade 1 hypertension, and tonsillar hypertrophy post-tonsilloadenoidectomy. The patient did not meet the criteria for any metabolic syndrome. The patient was then scheduled for outpatient follow-up with Nutrition-Pediatric Metabolism and Respirology clinics.

Discussion

A 10-year-old male patient, who had been obese since the age of 2, presented with clinical manifestations of shortness of breath during sleep and while walking, indicating obesity-related complications. The initial approach to his obesity should involve distinguishing between primary and secondary causes through history-taking and physical examination, as outlined in **Table 1**. History-taking and physical

examination are essential to assess complications, evaluate all potentially affected target organs, identify possible comorbidities, and guide lifestyle modifications in patients with obesity.[12]

Table 1. Physical examination in obese children[12]

Organ system	Physical examination findings	Patient's physical examination
Integumen/ subcutaneous tissue	Acanthosis nigricans, skin tags, hirsutism, striae, pseudo gynecomastia (male), intertrigo, xanthelasma (hypercholesterolemia)	Acanthosis nigricans, pseudo gynecomastia
Neurology	Papilledema and/or decreased venous pulsation on fundoscopic examination	None
Head and neck	Tonsillar hypertrophy, airway obstruction	Tonsillar hypertrophy, laterolateral oropharyngeal narrowing
Cardiovascular	Hypertension, heart rate	Hypertension
Respiratory	Physical activity intolerance, asthma	Physical activity intolerance
Gastrointestinal	Hepatomegaly and hepatic tenderness (non-alcoholic fatty liver disease), abdominal tenderness (secondary to gastroesophageal reflux or gallbladder stone)	None
Musculoskeletal	Pes planus, groin pain, waddling gait, tibia vara (Blount disease), arthralgia in the lower extremities, and restricted joint movement.	Short digits and bowing of the lower extremities
Endocrine	Goiter, extensive striae, hypertension, dorsocervical fat pad, pubertal stage, and decreased growth velocity	Dorsocervical fat pad, hypertension
Psychosocial	Flat affect and low mood, low self-esteem, and social withdrawal	Frequently falls asleep during class

Others	Short stature, disproportion, dysmorphic features, developmental delay	Short stature

The patient was initially suspected to have primary obesity due to excessive caloric intake unbalanced by inadequate physical activity. This was supported by a family history of obesity in both parents and a permissive parenting style, allowing the patient to have unrestricted access to food and no regular physical activity at home. However, the possibility of secondary obesity was considered upon finding that the patient's height was classified as short stature, falling below the range of his genetic height potential (159.5 – 176.5 cm). Such abnormalities necessitate further differentiation based on the age of onset, which is important for differentiating the underlying causes of obesity in children, as outlined in **Figure 1.**

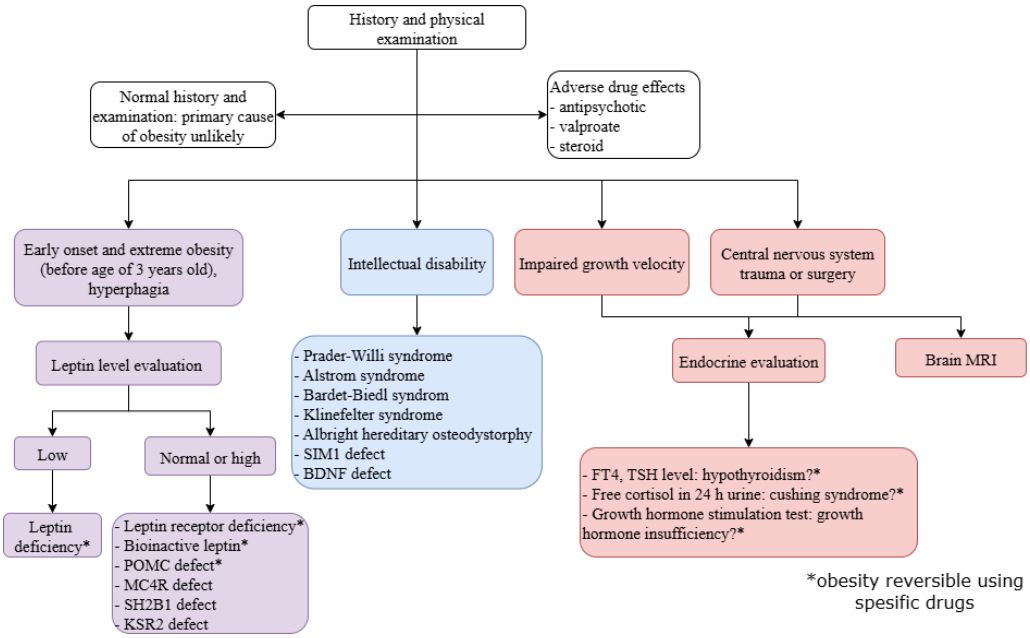

Figure 1. Algorithm for differentiating obesity etiologies.[13]

The patient also experienced hyperphagia, without any psychomotor or intellectual impairment, prompted consideration of a leptin hormone disorder affecting appetite regulation. While his leptin was elevated (cut-off value: 13.4 ng/mL) making leptin hormone deficiency unlikely, leptin receptor resistance remains a possibility.[14] Other genetic differential diagnoses were considered and ruled out: Proopiomelanocortin (POMC) gene defects were unlikely, as the patient did not present with red hair and pale skin; Melanocortin 4 Receptor (MC4R) gene defects were with the patient's short stature, as these typically cause increased linear growth; Src Homology 2 B Adaptor Protein 1(SH2B1) gene defects were unlikely given the lack of behavioral problems;

and Kinase Suppressor of Ras 2 (KSR2) gene defects were not supported, as the patient did not exhibit bradycardia.

Leptin, primarily produced by adipocytes and, to a lesser extent, by muscle cells, is crucial for regulating body weight, energy balance, and appetite. It signals the hypothalamus regarding the status of the body's energy reserve, particularly fat stores. This signaling subsequently influences feeding behavior.[15, 16] This patient had excessive fat storage, which theoretically should lead to increased leptin production, signaling the body to suppress appetite and enhances energy consumption. However, leptin resistance or an imbalance in leptin receptors are commonly observed in obesity. An increase in fat mass without a corresponding rise in energy consumption may contribute to this resistance.[16, 17]

The severity and duration of obesity significantly influence its complications, which can affect nearly every organ system, including the nervous, respiratory, cardiovascular, metabolic, gastrointestinal, renal, urinary, endocrine, musculoskeletal, dermatological, and psychosocial systems. In this patient, a respiratory complication, OSAS was confirmed by polysomnography (PSG). The severity of OSAS directly correlates with the degree of obesity. Studies indicate that a Body Mass Index (BMI) over 28 kg/m^2 increases the risk of OSAS by 4 – 5 times in children aged 2 to 18 years. Notably, severe obesity can lead to alveolar hypoventilation, resulting in oxygen desaturation.[9, 18, 19]

Hypertension is another common complication of obesity. Obesity can induce changes in cardiac morphology, myocardial dysfunction, and remodeling, ultimately leading to heart failure. Hypertension is also a known complication of OSAS. To confirm hypertension in children, ambulatory blood pressure monitoring (ABPM) is recommended. In cases like this patient's, the use of antihypertensive medications, specifically angiotensin-converting enzyme (ACE) inhibitors, can significantly improve cardiac function in obese children experiencing heart failure.[7]

Dyslipidemia should also be evaluated in obese children, as it increases their risk by about 1.7 times.[7] Although the patient did not present with hyperlipidemia, routine lipid screening performed between ages 9 – 11 years should be repeated between ages of 17 – 21 years old.[19-21] Another metabolic complication that may arise in children with obesity is type 2 diabetes mellitus. Insulin resistance is the key underlying mechanism of metabolic dysfunction in these patients. In this case, the presence of acanthosis nigricans served as a clinical marker associated with insulin resistance, indicating a future risk despite currently normal fasting blood glucose (65 mg/dL) and HbA1c levels (5.2%).

According to the International Society for Pediatric and Adolescent Diabetes (ISPAD) consensus, screening for glucose metabolism disorders is recommended for obese children aged 10 years or at puberty onset. This involves laboratory assessments like fasting blood glucose, HbA1c, and a 2-hour oral glucose tolerance test (OGTT). Furthermore, adiponectin, a hormone produced by adipose tissue that enhances insulin sensitivity, is typically reduced in obese individuals, contributing to insulin resistance.[19, 22, 23]

While the patient in this case did nott present with any neurological complication, a common issue observed was excessive daytime sleepiness, leading to students often falling asleep in class. This condition significantly reduces quality of life and can have a detrimental psychological impact on patients.[24]

Researches consistently show that childhood obesity can be effectively managed through weight loss, with better outcomes when initiated before puberty.[25] Family-based intervention approaches are particularly effective, demonstrating greater weight loss and an estimated increase in life expectancy of approximately 6–8 years. Even a modest weight loss of 5–10% can significantly improve cardiovascular function.[10, 13] A meta-analysis further supports this, reporting that a reduction in BMI of 1.25–1.3 kg/m^2 through lifestyle modification is associated with improved cardiometabolic outcomes in pediatric obesity.[26] In this case, the patient's parents exhibited a sedentary lifestyle, highlighting the need for a family-centered intervention approach.

Regulating nutritional intake is essential in the management of obesity, with strategies needing to be age-adjusted. For children aged 5 years and older, dietary recommendations include three main meals per day with 1 – 2 snacks. The daily food composition should consist of three portions of protein, 1–2 portions of milk, and 4–5 portions of non-starchy vegetables. Children should avoid sugary sweeteners and fast food. Portion sizes must be adjusted to the child's age, and positive reinforcement should be given whenever the child tries new food varieties.[27]

Gradual weight loss is crucial, with a safe reduction considered to be 2–4.5 kg or 0.5–2 kg per month.[28] In this case, the patient's estimated daily caloric intake was 2500 kcal, which was 131% of the Recommended Dietary Allowance (RDA) and significantly exceeded their daily requirement of 1900 kcal. Protein intake was also high at 75 grams (3 g/kg body weight).

To address this, a gradual dietary modification was implemented. The new plan adjusts caloric intake to meet daily requirements, focusing on staple foods and appropriate protein sources, and incorporating fruit-based snacks. This revised dietary plan includes three main meals and two snacks, providing a total of 1900 kcal (100% RDA) and 57 grams of protein (2.3 g/kg body weight).

For children over the age of 5, physical exercises and active play are essential, should be enjoyable, performed daily. Children need consistent encouragement and motivation to engage in physical activity as frequently as their healthy peers. Studies recommend at least 60 minutes of physical activity per day. To further increase motivation and adherence, older children can monitor their physical activity and food intake using mobile applications or other technological tools. These tools allow them to share their progress with peer groups, which can significantly boost their engagement.[27]

For children with heart failure, moderate-intensity activities such as walking or cycling, up to 60 minutes daily, are recommended. Daily home-based activities like sweeping and mopping are also encouraged. In this specific case, physical activity was initiated gradually, targeting 30 minutes of walking while maintaining a heart rate between 128 to 152 beats per minute. The patient successfully completed this activity without experiencing the previously reported shortness of breath. During outpatient follow-up, the patient was advised to continue walking, gradually increasing the duration to 60 minutes, 3 to 5 times per week.

Children with obesity have a higher risk of mortality due to complications that can affect nearly all organ systems. The prognosis for pediatric patients with obesity and multiple complications may vary, depending on the severity of the complications, the age at which obesity developed, and the effectiveness of the interventions implemented.[25]

Conclusion

Childhood obesity is a condition that demands serious attention due to its potential short- and long-term complications. Identifying the underlying cause of obesity is crucial to ensure appropriate and targeted management. Obesity management must be comprehensive, including adherence to dietary and physical activity recommendations, as well as pharmacologic treatment when indicated. Parental involvement is essential to maintain treatment consistency at home and to ensure regular follow-up for evaluating the effectiveness of interventions and monitoring the child's progress.

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Conflict of Interest

None declared

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