THE RELATIONSHIP BETWEEN MALLAMPATI SCORE AND OBESITY WITH THE RISK OF OSAHS IN DOWN SYNDROME CHILDREN

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ABSTRACT

Obstructive Sleep Apnea/Hypopnea Syndrome (OSAHS) has commonly occured in Down syndrome children caused by the abnormalities in upper respiratory tract anatomy and obesity. This study aims to identify the correlation between Mallampati score and obesity and the risk of OSAHS in Down syndrome children. This was an analytical descriptive cross-sectional study. This study was conducted in children with Down syndrome from September until November 2017. All parents filled the Indonesian Pediatric Sleep Questionnaire (PSQ), then all of the children were examined for Mallampati score and Body Mass Index (BMI). The correlation was analyzed by the Pearson Chi-Square model. Thirty-six subjects were included in this study. The number of male subjects were slightly more (61.11%), the mean age of the subjects was 8.42±4.45 years, with 52.78% (19 subjects) having OSAHS. There were 23 subjects (63.89%) who had Mallampati scores of 3 and 4, with 13 subjects (36.11%) were obese. This study concluded a statistically significant correlation between Mallampati score and obesity and the risk of OSAHS in Down syndrome children (p-value 0.001 and 0.029). Mallampati score and obesity had a significant correlation with the risk of OSAHS in Down syndrome children.

Keywords: Down syndrome; Mallampati; Obesity; OSAHS

INTRODUCTION

With an incidence of 1 in 1000 live births, Down syndrome (trisomy chromosome 21) is the most prevalent chromosomal condition.[1] People with Down syndrome often experience congenital complications, including mental retardation and disorders, including sleep disorders. Down syndrome children experience sleep disturbances more often than other normal children.[2] Churchill et al. reported the prevalence of Obstructive Sleep Apnea/Hypopnea Syndrome (OSAHS) in Down syndrome children between 24-59%.[3] While Brockmann et al. reported a prevalence of OSAHS as 61%.[4] Sleep-disordered breathing (SDB), namely OSAHS, is characterized by the presence of upper airway obstruction during sleep, which causes episodes of apnea (complete airflow obstruction) and hypopnea (reduced airflow), hypoventilation, hypercarbia, and hypoxemia.[5] Children with Down syndrome have phenotypes that predispose them to OSAHS, including adenotonsillar hyperplasia, midfacial and mandibular hypoplasia, hypotonia, small upper airways, macroglossia, and choanal atresia.[6, 7] These anatomical structural abnormalities of the upper airway may increase the risk of OSAHS in Down syndrome children. The Mallampati score is used to assess the density of the oral cavity.[8,9] Currently, the Mallampati score has been used as a simple method that is quite good for predicting the incidence of OSAHS in children. The higher the Mallampati score, the more difficult it is to intubate.[10] A study by Kumar et al. in Chicago found a significant correlation between Mallampati scores, tonsil size, and AHI (Apnea-Hypopnea Index) in

children with OSAHS. In this study, it was demonstrated that every increase in Mallampati's score would six times increase the odds ratio of the risk of OSAHS.[11] Su M-S et al.'s study in China found an association between Mallampati scores and the risk of OSAHS in preschool and school-age children.[12]

The prevalence of overweight and obesity in Down syndrome children is 23-70%.[13] Research by Samarkandy et al. in 2012 and Wee et al. in 2015 found a higher Body Mass Index (BMI) in children with Down syndrome than those without Down syndrome.[14, 15] Basil et al. reported that 74% of Down syndrome children had OSAHS, and children with obesity were more likely to have moderate to severe OSAHS.[16] Maris et al., by polysomnography, found that 66.4% of Down syndrome children had OSAHS, and there was a significant relationship between BMI and the severity of OSAHS.[6] Obesity, particularly central obesity, will cause the accumulation of fat around the airways and abdomen, which cause mechanical loads on the chest wall, reduce lung expansion (volume), reduce airway retraction caudally, and cause pharyngeal collapse.[17]

Research on OSAHS sleep disorders, especially in Down syndrome children in Indonesia, has not yet been published. Based on the above background, his study aimed to determine the relationship between Mallampati scores and obesity with the risk of OSAHS in Down syndrome children. Thus, early medical interventions for OSAHS sleep disorders can be carried out earlier to improve the quality of life in Down syndrome children.

MATERIAL AND METHODS

This research is analytical descriptive, cross-sectional study design. The participants were Down syndrome children whose parents are members of the Bandung POTADS (Persatuan Orang Tua Anak dengan Down Syndrome) community. Inclusion criteria included children aged 3-18 years, having parents/caregivers living with the children, and both parents/caregivers and children willing to participate in the study. Exclusion criteria include children suffering from lung diseases such as bronchopneumonia or pulmonary TB diagnosed by a pediatrician, children who were taking muscle relaxants drugs, children with epilepsy and cerebral palsy, and children with behavioral disorders. The research was conducted through interviews, filling out the OSAHS sleep disorder screening questionnaire in children using the Indonesian version of the Pediatric Sleep Questionnaire (PSQ), which has been validated. Then, the participant underwent physical examination, including weight, height, and Mallampati by a Sleep Medicine expert. This research was conducted from September to November 2017 and has been approved by the Universitas Padjadjaran Research Ethics Committee through the ethical clearance No. 929/UN6.C.10 /PN/ 2017.

The sampling technique used in this research is non-probability sampling with a consecutive sampling method. The sample size in this study was determined using the formula to determine the sample size for correlation study. Based on the minimum sample calculation formula for the relationship parameters studied, a minimum sample size of 32 subjects was obtained. Data collected were then processed using 24.00 SPSS for Windows version. To analyze the relationship between the Mallampati score and the risk of OSAHS and the relationship between obesity and the risk of OSAHS, the Pearson Chi-Square was used. The relationship was considered significant if the p-value was 0.05 or less.

RESULT

This study included thirty-six children with characteristics as listed in Table 1.

Table 1. Characteristics of Research Subjects

Characteristics	Average (SD)	Median	*Range*	n(%)
Age (years)	8.42 (4.45)	7.50	13 (3 – 16)	
Gender				
- Male				22 (61.11)
- Female				

				14 (38.89)
PSQ Score	7.42 (3.70)	8.00	15 (0 – 15)	
PSQ Classification - OSAHS (8) - Non-OSAHS (< 8)				19 (52.78) 17 (47.22)
BMI Status - Obesity - Non-Obesity				13 (36.11) 23 (63.89)
Mallampati Score - 1 - 2 - 3 - 4				- 13 (36.11) 15 (41.67) 8 (22.22)
Mallampati Score Risk - Low Risk (1-2) - High Risk (3-4)				13 (36.11) 23 (63.89)

Note: categorical data are presented in frequency and percentage, while numerical data is presented in mean, standard deviation (SD), median, and range.

Table 1 shows that subjects were 36 children with an average of 8.42 ± 4.45 years. The sex distribution is slightly more male (61.11%). Based on the classification of PSQ scores, it was found that most of the subjects (52.78%) suffered from OSAHS. Based on BMI status, 13 (36.11%) subjects were obese. According to Mallampati score, most of the subjects had a Mallampati score of 3 (41.67%). Based on the risk classification of the Mallampati score, 23 (63.89%) subjects were at high risk of suffering from OSAHS.

Table 2. Analysis of the Relationship Between Mallampati Score and OSAHS Risk in Down Syndrome Children

Variable	OSAHS (n=19)		Non-OSAHS (n=17)		*p-value*
	N	%	N	%	
Mallampati Score Risk - Low Risk (1-2) - High Risk (3-4)	2 17	15,40 73.90	11 6	84.60 26.10	0.001**

Description: Pearson Chi-Square Correlation; the significance value of $p < 0.05$.
The sign ** indicates a statistically significant result.

According to Table 2, seventeen subjects had a high-risk Mallampati score (3 and 4) and OSAHS. Analysis of the relationship between Mallampati scores and the risk of OSAHS in Down syndrome children was significant, with a p-value of 0.001.

Table 3. Analysis of the Relationship Between Obesity and the Risk of OSAHS in Down Syndrome Children

Variable	OSAHS (n=19)		Non-OSAHS (n=17)		*p-value*
	N	%	N	%	
BMI Status - Obese - Not obese	10 9	76.90 39.10	3 14	23.10 60.90	0.029**

Description: Pearson Chi Square Correlation; the significance value of $p < 0.05$.
The ** sign indicates a statistically significant result.

Table 3 demonstrates that ten subjects were obese and had OSAHS. Analysis of the relationship between obesity and the risk of OSAHS in Down syndrome children was significant, with a p-value of 0.029.

DISCUSSION

In this study, the prevalence of OSAHS in Down syndrome children with an average age of 8.42 ± 4.45 years was 52.78%. These results complement the data on the prevalence of OSAHS in Down syndrome children in Indonesia, which has not yet been published. A similar study in the United States conducted by Hoffmire et al. in 2014, using the PSQ questionnaire, found that 46% of Down syndrome children aged 7 to 17 years indicated to have OSAHS.[18] While Bassell et al. in 2015 in Atlanta, United States, using the Childen's Sleep Habit Questionnaire (CSHQ) questionnaire, 51% of Down syndrome children aged 1.5 to 13.4 years indicated having OSAHS.[18, 19] Research in Hong Kong by Ng et al. in 2006 found the prevalence of OSAHS in Down syndrome children was 59%, with an average age of 10.8 ± 5.93 years by polysomnography examination.[7] In Belgium, Maris et al. found a prevalence of OSAHS of 66.4% of Down syndrome

children aged 2.8 to 10.5 years with polysomnography.[6] This depicts that the prevalence of OSAHS in Down syndrome children in Indonesia is slightly higher than the prevalence in New York, United States of America using the PSQ questionnaire.

This study found that Down syndrome children had Mallampati scores of 2, 3, and 4. About 23 subjects (63.89%) had Mallampati scores of 3 and 4 who were at high risk for OSAHS, of which seventeen subjects were indicated to have OSAHS. Based on statistical analysis, there was a significant relationship between Mallampati scores and the risk of OSAHS in Down syndrome children. The study of Skotko et al. in 2017 used the Mallampati score as a significant physical examination to predict the incidence of OSAHS in Down syndrome children, in addition to using polysomnography and PSQ and Children's Sleep Habit Questionnaire (CSHQ) questionnaires.[20] Allareddy et al.'s research in 2017 found that 76.92% of Down syndrome children examined had high Mallampati scores (scores 2, 3, and 4).[21] The study of Kumar et al. in Chicago, United States, in 2014 also found that the Mallampati score was an independent predictor for OSAHS in children. There was a significant relationship between Mallampati score, tonsil size, and AHI in this study. An increase in one Mallampati score led to a six-fold increase in the risk of OSAHS in children.[11]

Overweight and obesity are typical in Down syndrome children. The prevalence is about 23 to 70%.[13] Research by Samarkandy et al. in 2012 and Wee et al. in 2015 found that children with Down syndrome had a higher BMI than children without Down syndrome.[14, 15] Obesity is classified into two types: the primary type (visceral or abdominal) and the peripheral type. In the primary type, fat accumulates in the abdominal region and the upper body, such as the upper chest, neck, and shoulders, while in the peripheral type, fat accumulates in the pelvis and thighs.[22] Obesity, especially central obesity, causes fat accumulation around the airways and abdomen, which can cause mechanical loads on the chest wall, reduce lung expansion (volume), reduce airway retraction caudally, and cause pharyngeal collapse.[23]

The development of OSAHS is influenced by both anatomic and pharyngeal collapsibility variables. Sleep-related collapse of the upper airway at the pharyngeal level is the major pathophysiological manifestation.[24] Even in healthy persons, sleep is associated with a constriction of the pharynx and an increase in inspiratory resistance due to a decrease in upper airway muscular tone and pharyngeal protective reflexes against collapse. Apnea is caused by a total blockage. The effects of partial collapse include snoring, hypopnea, and persistent obstructive hypoventilation. Sleep fragmentation caused by recurrent awakenings is the primary cause of hypersomnolence as a result of decreased sleep efficiency and total sleep duration. Moreover, some authors propose that the severity of OSAHS, nocturnal hypoxemia, and sleep fragmentation is an independent predictor of hypersomnolence in sleep disorders.[25] Soft tissue mass is often disproportionate to the space made accessible by pharyngeal bone structures, and soft tissues in excess and/or tiny bone structure predominate in the majority of OSAHS patients.[24]

Upper airway narrowing tends to also cause OSAHS.[26] Obese people with OSAHS have augmented pharyngeal wall fat pads.[27] However, airways are not always constricted by these fatty deposits, indicating that there may be other processes that cause constriction.[28] Oedema may be produced by distension and/or increased neck vein pressure, vascular congestion, inflammation owing to tissue vibration during snoring, or pulmonary hypertension (PH) due to arterial vasoconstriction due to repeated hypoxia.[29] Obesity-related inflammation may also cause soft tissue inflammation and oedema.[30]

The hormones generated by adipose tissue provide a second potential route linking obesity to OSAHS.[31] Leptin is a hormone generated by adipose tissue that plays a crucial function in body-weight management by stimulating hypothalamic satiety

pathways.[32] Leptin levels thus connect with BMI and insulin levels. Obesity is often accompanied with elevated leptin levels, indicating the presence of leptin resistance.[33] In addition to its effects on bodyweight regulation, leptin also has effects on respiratory centre regulation. Some authors propose that OSAHS may also contribute to body-weight increase due to hyperleptinemia, insulin resistance, and inflammatory activity, which, as in a vicious loop, exacerbates the condition.[34] There may be further functions played by genetic variables.[35]

Basil et al. found that children with Down syndrome are at risk for obesity and OSAHS. In this study, 47.8% were obese, and 74% had OSAHS with polysomnography examination. Analysis of the relationship between obesity and OSAHS found that the risk of OSAHS increased significantly in obese children.[16] Shires et al. stated that BMI has a significant relationship with the onset of OSAHS in Down syndrome children.[36] Hill et al. stated that the increase in BMI was in line with the increase in AHI and the decrease in blood oxygen saturation levels in Down syndrome children.[37] Canapari et al. found that visceral body fat distribution was associated with obesity and a strong predictor of OSAHS in children.[17] Chen et al.'s study found that Down syndrome children with severe OSAHS symptoms had a higher BMI than children with mild OSAHS symptoms.[38] In this study, ten subjects from thirteen subjects who were obese also suffered from OSAHS. In line with other existing studies, it can be concluded that obesity contributes as a risk factor for OSAHS in Down syndrome children.

CONCLUSION

Mallampati score had a significant relationship with the risk of OSAHS in Down syndrome children (p-value 0.001). Obesity has a significant relationship with the risk of OSAHS in Down syndrome children (p-value 0.029).

CONFLICT OF INTEREST

The authors declared that there is no conflict of interest.

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REFERENCES

1. Mai CT, Kucik JE, Isenburg J, Feldkamp ML, Marengo LK, Bugenske EM, et al. Selected birth defects data from population-based birth defects surveillance programs in the United States, 2006 to 2010: featuring trisomy conditions. Birth defects research Part A, Clinical and molecular teratology. 2013;97(11):709.
2. Stores G, Stores R. Sleep disorders and their clinical significance in children with Down syndrome. Developmental Medicine & Child Neurology. 2013;55(2):126-30.
3. Churchill SS, Kieckhefer GM, Landis CA, Ward TM. Sleep measurement and monitoring in children with Down syndrome: a review of the literature, 1960–2010. Sleep medicine reviews. 2012;16(5):477-88.
4. Brockmann PE, Damiani F, Nunez F, Moya A, Pincheira E, Paul MA, et al. Sleep-disordered breathing in children with Down syndrome: Usefulness of home polysomnography. International journal of pediatric otorhinolaryngology. 2016;83:47-50.
5. Sinha D, Guilleminault C. Sleep disordered breathing in children. Indian Journal of Medical Research. 2010;131(2):311.
6. Maris M, Verhulst S, Wojciechowski M, Van de Heyning P, Boudewyns A. Prevalence of obstructive sleep apnea in children with Down syndrome. Sleep. 2016;39(3):699-704.
7. Hill EA. Obstructive sleep apnoea/hypopnoea syndrome in adults with Down syndrome. Breathe (Sheff).

2016;12(4):e91-e96. doi:10.1183/20734735.012116
8. Marshall S. Respiratory Care Anatomy and Physiology: Foundations for Clinical Practice. 3rd ed: Elsevier; 2012. 253-271
9. Friedman M, Hamilton C, Samuelson CG, Lundgren ME, Pott T. Diagnostic value of the Friedman tongue position and Mallampati classification for obstructive sleep apnea: a meta-analysis. Otolaryngology--Head and Neck Surgery. 2013;148(4):540-7.
10. Iyer MS, Pitetti RD, Vitale M. Higher Mallampati Scores Are Not Associated with More Adverse Events During Pediatric Procedural Sedation and Analgesia. West J Emerg Med. 2018;19(2):430-436.
11. Kumar HVM, Schroeder Jr JW, Gang Z, Sheldon SH. Mallampati score and pediatric obstructive sleep apnea. Journal of Clinical Sleep Medicine. 2014;10(9):985-90.
12. Su M-S, Zhang H-L, Cai X-H, Lin Y, Liu P-N, Zhang Y-B, et al. Obesity in children with different risk factors for obstructive sleep apnea: a community-based study. European journal of pediatrics. 2016;175(2):211-20.
13. Bertapelli F, Pitetti K, Agiovlasitis S, Guerra-Junior G. Overweight and obesity in children and adolescents with Down syndrome—prevalence, determinants, consequences, and interventions: A literature review. Research in developmental disabilities. 2016;57:181-92.
14. Samarkandy MM, Mohamed BA, Al-Hamdan AA. Nutritional assessment and obesity in Down syndrome children and their siblings in Saudi Arabia. Saudi Med J. 2012;33(11):1216-21.
15. Wee SO, Pitetti KH, Goulopoulou S, Collier SR, Guerra M, Baynard T. Impact of obesity and Down syndrome on peak heart rate and aerobic capacity in youth and adults. Research in developmental disabilities. 2015;36:198-206.
16. Basil JS, Santoro SL, Martin LJ, Healy KW, Chini BA, Saal HM. Retrospective study of obesity in children with Down syndrome. The Journal of pediatrics. 2016;173:143-8.
17. Canapari CA, Hoppin AG, Kinane TB, Thomas BJ, Torriani M, Katz ES. Relationship between Sleep Apnea, Fat Distribution, and Insulin Resistance in Obese Children. Journal of Clinical Sleep Medicine. 2011;07(03):268-73.
18. Hoffmire CA, Magyar CI, Connolly HV, Fernandez ID, van Wijngaarden E. High prevalence of sleep disorders and associated comorbidities in a community sample of children with Down syndrome. Journal of Clinical Sleep Medicine. 2014;10(4):411-9.
19. Bassel JL PH, Leu R, Kronk R, Visootsak J. Sleep profiles in children with Down syndrome. Am J Med Genet A. 2015;167(8):1830 - 5.
20. Skotko BG, Macklin EA, Muselli M, Voelz L, McDonough ME, Davidson E, et al. A predictive model for obstructive sleep apnea and Down syndrome. American journal of medical genetics Part A. 2017;173(4):889-96.
21. Allareddy V, Ching N, Macklin EA, Voelz L, Weintraub G, Davidson E, et al. Craniofacial features as assessed by lateral cephalometric measurements in children with Down syndrome. Progress in orthodontics. 2016;17(1):1-12.
22. Pinto JA, Godoy LBdM, Marquis VWPB, Sonego TB, Leal CdFA, Ártico MS. Anthropometric data as predictors of obstructive sleep apnea severity. Brazilian journal of otorhinolaryngology. 2011;77(4):516-21.
23. Littleton SW. Impact of obesity on respiratory function. Respirology. 2012 Jan;17(1):43-9.
24. Ryan CM. Bradley TD. Pathogenesis of obstructive sleep apnea. J Appl Physiol. 2005;99(6):2440–50.
25. Punjabi N, O'hearn DJ, Neubauer DN, Javier Neto F, Schwartz AR, Smith PL, Bandeen-Roche K. Modeling hypersomnolence in sleep-disordered breathing. Am J Respir Crit Care Med. 1999;159(6): 1703–9.

26. Chen L, Xiao T, Ng CT. The biomechanical mechanism of upper airway collapse in osahs patients using clinical monitoring data during natural sleep. Sensors (Basel). 2021;21(22):7457.
27. Pahkala R, Seppä J, Ikonen A, Smirnov G, Tuomilehto H. The impact of pharyngeal fat tissue on the pathogenesis of obstructive sleep apnea. Sleep Breath. 2014;18(2):275-82.
28. Schwab R, Gupta KB, Gefter WB, Metzger LJ, Hoffman EA, Pack AI. Upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing. Significance of the lateral pharyngeal walls. Am J Respir Crit Care Med. 1995;152(5 Pt 1):1673–89.
29. Sajkov D, McEvoy RD. Obstructive sleep apnea and pulmonary hypertension. Prog Cardiovasc Dis. 2009;51(5):363-70.
30. Ellulu MS, Patimah I, Khaza'ai H, Rahmat A, Abed Y. Obesity and inflammation: the linking mechanism and the complications. Arch Med Sci. 2017;13(4):851-863.
31. Bonsignore MR, McNicholas WT, Montserrat JM, Eckel J. Adipose tissue in obesity and obstructive sleep apnoea. European Respiratory Journal. 2012;39(3):746-767
32. Obradovic M, Sudar-Milovanovic E, Soskic S, Essack M, Arya S, Stewart AJ, Gojobori T, Isenovic ER. Leptin and obesity: role and clinical implication. Front Endocrinol (Lausanne). 2021;12:585887.
33. Myers MG Jr, Leibel RL, Seeley RJ, Schwartz MW. Obesity and leptin resistance: distinguishing cause from effect. Trends Endocrinol Metab. 2010;21(11):643-651.
34. Romero-Corral A, Caples SM, Lopez-Jimenez F, Somers VK. Interactions between obesity and obstructive sleep apnea: implications for treatment. Chest. 2010;137(3):711-719.
35. Kalra M, Chakraborty R. Genetic susceptibility to obstructive sleep apnea in the obese child. Sleep Med. 2007;8(2):169-75.
36. Hill EA, Williams LJ, Cooper SA, Riha RL. Objective and subjective prevalence of obstructive sleep apnoea/hypopnoea syndrome in uk adults with down syndrome: a strong marker for diurnal behavioural disturbances. Brain Sci. 2021;11(9):1160.
37. Dyken ME, Lin-Dyken DC, Poulton S, Zimmerman MB, Sedars E. Prospective polysomnographic analysis of obstructive sleep apnea in Down syndrome. Archives of pediatrics & adolescent medicine. 2003;157(7):655-60.
38. Chen C-CJ, Spanò G, Edgin JO. The impact of sleep disruption on executive function in Down syndrome. Research in developmental disabilities. 2013;34(6):2033-9.