Volume 1 | Issue 6 |2022

SMART PEDI UPDATE

Pedismart Newsletter
Vol 1 | Issue 6 | 2022

Pedi smart News Letter
Vol 4| Issue 6 |2022

Dear Doctor,
Greetings from Micro Labs Limited. We are happy to bring you **"PEDI SMART"**which contain latest and interesting news in the field of Paediatrics.

This issue contains:

1. **A Prospective cohort study on Childhood sleep and the risk of obesity**
2. **A study on the relationship between breastfeeding, the parents' BMI, and their child's birth weight and obesity indicators.**
3. **An 11-year experience from a tertiary referral hospital on the long-term effects of lymphatic malformations in children**
4. **A single-center, retrospective study examined the differences between older and younger children's Kawasaki disease's clinical features.**

Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback

Best Regards,

Medical Team, Micro Labs Limited

Volume 1 | Issue 6 |2022

SMART PEDI UPDATE

1.A Prospective cohort study on Childhood sleep and the risk of obesity

Introduction:

Obesity is a major public health concern for both adults and children around the world due to its increasing prevalence. The alarming rise in childhood obesity is seen as a global problem due to its continuous development into adult obesity as well as its relationship with a number of co-morbidities, economic load, and negative effects on quality of life.[1] Obesity in children is becoming more common, which is a serious public health concern. The objective of this study was to determine what are the factors young children's sleep and what links sleep to childhood obesity and overweight.

Short sleep duration may increase the risk of childhood overweight and obesity, as the child's BMI Z-score rises by 0.09 units for every hour less of sleep.

Methods:

In the prospective study, data were gathered using questionnaires that parents completed on behalf of N = 10.840 one-year-old infants who were followed up to the age of eight. The link between factors influencing the children's sleep was evaluated using the chi-squared test and Pearson Correlation. Later, longitudinal mixed model analyses were performed to predict how various aspects of sleep (including bedtime, sleep length, sleep quality, and the frequency of awakenings) will affect BMI Z-scores.

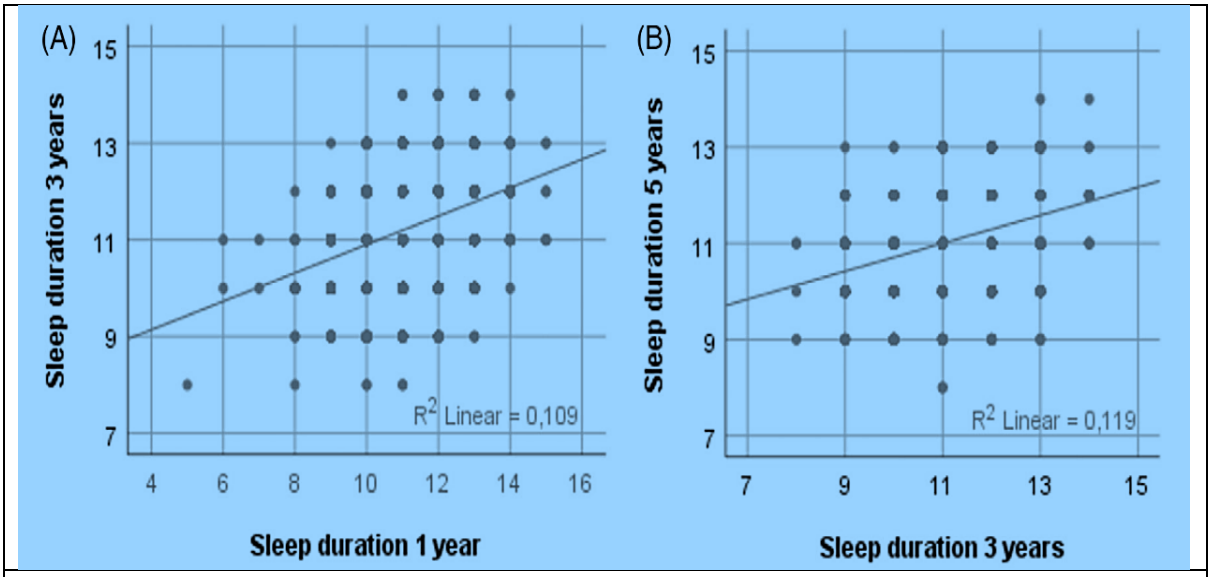
Results:

Children with fewer siblings, non-single parents, non-smoking moms during pregnancy, and children with parents who were born were more likely to have healthy sleep patterns at age one. Shorter sleep duration was associated with more awakenings, more nocturnal feedings, and especially later bedtimes (= 0.544, p <0.0001). Early sleep patterns were linked to lower BMI Z-scores (adjusted effect estimate [95% CI]: = 0.09, [(0.15) - (0.03)], p = 0.005). In addition, children of mothers who smoked while pregnant, smaller size for gestational age, bad eating patterns, and greater birth weight all contributed to higher BMI Z-scores.

Discussion:

Poor sleep habits may raise the risk of overweight and obesity in children many years later, according to these linear longitudinal analyses examining the relationship between sleep and BMI Z-scores, which start at age one (8 years of age in this study). With each

hour less of sleep, the child's BMI Z-score rises by 0.09 units on average.[2]The outcomes of other earlier investigations concur with these findings.[3]



The relation of the children's sleep variables at different ages in the study population of 10 840 children. (A) and (B) sleep duration at ages from one to five in hours.

Conclusion:

Every hour less of sleep results in a 0.09-unit increase in the child's BMI Z-score, suggesting that short sleep duration may raise the likelihood of childhood obesity and overweight. When putting measures in place to stop the growth of childhood obesity, parental educational programmes that advise proper sleeping habits should be taken into account.

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Volume 1 | Issue 6 |2022

SMART PEDI UPDATE

2.A study on the relationship between breastfeeding, the parents' BMI, and their child's birth weight and obesity indicators

Introduction:

Children's obesity indicators were correlated with the maternal BMI and birth weight.

Many countries have seen a sharp rise in the prevalence of overweight and obesity over the past few decades, not just in adults but also in children. Public health professionals are concerned about this since juvenile obesity raises the likelihood of adult obesity.[1] Breastfeeding, the BMI of the parents, and birth weight may have an impact on childhood obesity. [2] Therefore, the purpose of this study was to confirm the relationship between breastfeeding, the body mass index of the parents, and the child's birth weight and obesity indicators.

Methods:

In this cross-sectional study, 402 schoolchildren between the ages of 9 and 11 collected data. Parents or guardians provided information on breastfeeding (month), birth weight (kg), parental height and weight (parents' BMI [kg/m2] was computed), and breastfeeding. Indicators of obesity included body mass index (kg/m2), waist circumference (cm), and body fat (%) as evaluated by bio-impedance. The corresponding associations were evaluated using multi-level linear regression models that were adjusted for potential confounders.

Results:

According to children's BMIs, eutrophic, overweight, and obesity were all present in 58.2%, 20.9%, and 17.2% of children, respectively. Breastfeeding, maternal and paternal body mass index, and the children's body mass index, body fat, and waist circumference all showed significant and favourable associations. Waist circumference and birth weight were not related, although body mass index and body fat were slightly and favourably correlated. After adjusting for school, sex, age, race/ethnicity, annual household income, sedentary time, and moderate-to-vigorous physical activity, maternal body mass index and birth weight were positively associated with children's body mass index (β: 0.228; 95%CI: 0.142; 0.314 and β: 0.001; 95%CI: 0.001; 0.002), body fat (β: 0.484; 95%CI: 0.297; 0.671 and β: 0.002; 95%CI: 0.001; 0.003) and waist circumference (β: 0.509; 95%CI: 0.304; 0.715 and β: 0.003; 95%CI: 0.001; 0.005). There was no correlation between breastfeeding and any obesity indices.

Variables	BMI (kg/m^2)	Body fat (%)	WC (cm)	Breastfeeding (month)	Maternal BMI (kg/m^2)	Paternal BMI (kg/m^2)	Birth weight (g)
BMI (kg/m^2)	1.00	0.921*	0.900*	-0.132*	0.320*	0.173*	0.165*
Body fat (%)	-----	1.00	0.834*	-0.112*	0.308*	0.198*	0.161*
WC (cm)	-----	-----	1.00	-0.097*	0.271*	0.153*	0.164
Breastfeeding (month)	-----	-----	-----	1.00	0.054	0.014	0.155*
Maternal BMI (kg/m^2)	-----	-----	-----	-----	1.00	0.189*	0.095
Paternal BMI (kg/m^2)	-----	-----	-----	-----	-----	1.00	0.109
Birth weight (g)	-----	-----	-----	-----	-----	-----	1.00

BMI: body mass index; WC: waist circumference.
*p<0.05 in the Pearson correlation.

Analysis of correlation of independent and dependent variables of children and parents

Discussion:

The complex aetiology of obesity, which has links to social, environmental, behavioural, biochemical, and genetic factors, makes it a chronic inflammatory illness.[3] This study confirmed the favourable correlation between parental BMI and birth weight with several obesity indicators during late childhood based on this and the knowledge of the role of parents in this process.[2] Additionally, breastfeeding has a good correlation with %BF**.** Breastfeeding during the first year of life, as well as supporting and promoting healthy habits during childhood and adolescence, are all ways that parents can help prevent obesity in their children.[4] The data from the current investigation, which showed substantial relationships between maternal BMI and all obesity indices regardless of sedentary time and moderate-to-vigorous physical activity in children, supports this idea.

Conclusion:

This study concludes that children's obesity indicators were correlated with the maternal BMI and birth weight. Therefore, it suggests that the perinatal environment has a significant role in the development of childhood obesity, and public policies must target parental obesity to combat the issue.

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1. Huang H, Gao Y, Zhu N, Yuan G, Li X, Feng Y, Gao L, Yu J. The Effects of Breastfeeding for Four Months on Thinness, Overweight, and Obesity in Children Aged 3 to 6 Years: A Retrospective Cohort Study from National Physical Fitness Surveillance of Jiangsu Province, China. Nutrients. 2022 Oct 6;14(19):4154.
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Volume 1 | Issue 6 |2022

SMART PEDI UPDATE

3.A single-center, retrospective study examined the differences between older and younger children's Kawasaki disease's clinical features.

Introduction:

Acute, self-limiting medium vessel vasculitis with a preference for the coronary arteries is known as Kawasaki disease (KD), also known by the name mucocutaneous lymph node syndrome. It is the main contributor to acquired heart disease in developed countries and is gradually displacing rheumatic heart disease in underdeveloped nations.[1] Compared to younger children under the age of three, children aged >6 years are more likely to develop coronary artery abnormalities in Kawasaki illness.[2] In order to enable early identification of Kawasaki illness and decrease coronary artery anomalies in older children, this study looked into the differences in the clinical characteristics of the disease between older and younger children.

Kawasaki disease should be suspected in children older than 3.0 years with cervical lymphadenopathy and fever

Methods:

This was a retrospective analysis of 405 Kawasaki disease children .A fever that had passed was indicated by a persistent temperature of less than 37.5°C. The day the fever broke was determined to be the day the temperature first dropped below 37.5°C. Both during the diagnosis and two days later, echocardiography was done. The z-score for the coronary artery dimension, as previously established, was used to evaluate coronary artery anomalies. A z-score of more than 2.5 was considered to be a coronary artery lesion. The cutoff age of 3.0 years, as previously reported (5), was used to divide children into two groups: older group (3.0 years) and younger group (3.0 years). None of the treated children received corticosteroid medication during the research period; instead, all received an initial IVIG dose of 2 g/kg.

Results:

The groups of older patients (n = 169) and younger patients (n = 236) were selected from the eligible patients. (112 [66.3%] vs. 229 [97.0%], P < 0.001 in the younger group) Skin rash was discovered in considerably fewer occurrences. Cervical lymphadenopathy was more prevalent in older children (153 [90.5%] vs. 165 [69.9%], P < 0.001) and in patients with incomplete Kawasaki illness (3 or 4 findings) (34 [20.1%] vs. 25 [10.6%], P = 0.0078). (Median: 5.0 days vs. 4.0 days, P = 0.003) Older children had a longer diagnosis delay than younger children. Additionally, the older group had a higher prevalence of fever that was nonresponsive to a single intravenous immunoglobulin (IVIG), and the

duration of the fever was also considerably longer (48 [28.4%] vs. 47 [19.9%], P = 0.0479).

Discussion:

In this retrospective analysis, cervical lymphadenopathy was present in almost 90% of older children (3.0 years) with Kawasaki disease, but only in one-third of those patients. Furthermore, older children than younger children (3.0 years of age) had a higher prevalence of incomplete Kawasaki illness, fever unresolved by a single IVIG, and the tendency to delay treatment initiation during the course of Kawasaki disease.[2] Comparable to this study, the most recent Japanese diagnostic guideline stated that cervical lymphadenopathy affects almost 90% of older children.[3]

	All (N = 405)	Older group (≥3.0 years old) (N = 169)	Younger group (<3.0 years old) (N = 236)	*P*-value
Male (%)	249 (61.5%)	109 (64.5%)	140 (59.3%)	0.29
Family history (%)	15 (3.7%)	7 (4.1%)	8 (3.4%)	0.69
<Clinical findings>				
Fever (%)	405 (100.0%)	169 (100.0%)	236 (100.0%)	1.00
Conjunctival injection (%)	360 (88.9%)	149 (88.2%)	211 (89.4%)	0.70
Oral cavity changes (%)	356 (87.9%)	147 (87.0%)	209 (88.6%)	0.63
Extremity changes (%)	351 (86.7%)	141 (83.4%)	210 (89.0%)	0.14
Rash (%)	341 (84.2%)	112 (66.3%)	229 (97.0%)	<0.001
BCG inoculation (%)	155 (38.3%)	15 (8.9%)	140 (59.3%)	<0.001
Lymphadenopathy (%)	318 (78.5%)	153 (90.5%)	165 (69.9%)	<0.001
Incomplete Kawasaki disease (3 or 4 findings) (%)	59 (14.6%)	34 (20.1%)	25 (10.6%)	0.0078

Abbreviations: *BCG*, Bacille Calmette–Guérin

Comparison of patient characteristics and clinical features at the diagnosis of Kawasaki disease between the older and younger groups

Conclusion:

In spite of the lack of a skin rash, Kawasaki disease should be suspected in children older than 3.0 years with cervical lymphadenopathy and fever. Children older than three years are also more likely to have incomplete Kawasaki disease, fever that is unresponsive to a single IVIG infusion, and a propensity to delay treatment initiation.

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4.A case of paediatric atypical progeroid syndrome with focal segmental glomerulosclerosis

Introduction:

Numerous conditions, including muscular dystrophies, cardiomyopathies, and progeroid syndromes, are now referred to as laminopathies and are caused by mutations in the LMNA gene. Progeroid syndromes often fall under the category of atypical progeroid syndrome (APS), which is primarily linked to LMNA mutations.[1] The progeroid syndrome known as atypical progeroid syndrome (APS) is a rare condition mostly brought on by heterozygous missense mutations in the LMNA (MIM 150330) gene. Although APS includes a variety of clinical symptoms, renal symptoms, particularly in children, are rarely reported. The first paediatric instance of APS with focal segmental glomerulosclerosis (FSGS) is reported here.

Screening for proteinuric nephropathy is essential for managing atypical progeroid syndrome patients

Case Presentation:

A 10-year-old boy had further evaluation of accidentally discovered hyperuricemia at the nephrology clinic. With a full-term vaginal delivery and a birth weight of 3.64 kg in the past,he experienced four episodes of urinary tract infection while he was an infant. He received nitrofurantoin prophylaxis till 22 months after having bilateral vesicoureteral reflux (grade 4 on the left and grade 1 on the right) was discovered at three months. After a year, there were no further urinary tract infections, thus it was stopped. At the age of 28 months, his vesicourethrogram revealed no reflux following the discontinuance. When he was 2 years old, he complained of cold-induced discolouration of his extremities, but tests for autoimmune disease and an arteriography of his upper and lower extremities turned up nothing unusual. At the age of 7, his knee and hip flexion were limited, and he had a waddling stride. On orthopaedic evaluation two years later, prominent coxa valga with relative coxa magna and a tiny pelvis were discovered. At the age of nine, he saw a genetics expert. He had normal development, decreased subcutaneous fat tissue in his trunk, and both extremities with pale and dark skin, despite growing to a height and

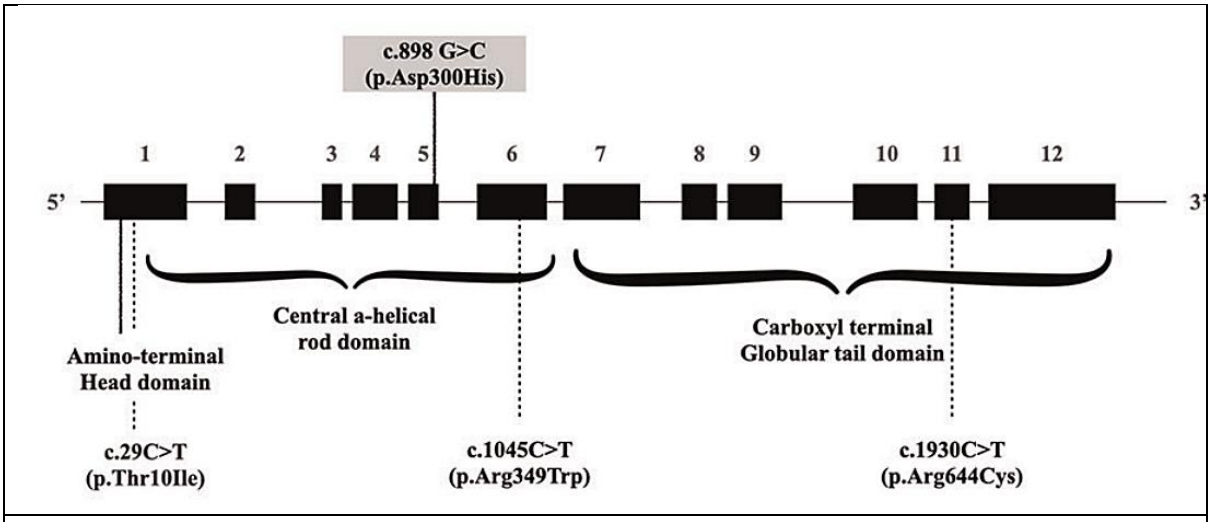
weight of 143 cm (88.6 percentile) and 33 kg (47.5%), respectively. At the age of nine, he saw a genetics specialist. While his weight and height were typical for his age (143 cm and 33 kg, respectively), there was decreased subcutaneous fat tissue in his trunk and pale and dark skin on both of his limbs. He also had a beaked nose, a short neck, prominent eyes, and both axillary patches of acanthosis nigricans. The effects of Raynaud's were still noticeable. Liver transaminases and cholesterol levels were elevated, indicating impaired liver function. His fasting blood glucose level was 83 mg/dl, his HbA1c was 6.5%, and his insulin levels were higher. He was referred to an endocrinologist for treatment of dyslipidemia and elevated HbA1c, but insulin and HbA1c levels remained high (6.1%, 55.7 IU/ml), indicating insulin resistance. Hepatocyte fatty changes and portal and periportal fibrosis were seen in a liver biopsy performed at age 10. Mild mitral and tricuspid valve regurgitation was observed; however, it grew worse throughout follow-up and was accompanied by the emergence of hypertrophic cardiomyopathy (HCMP), necessitating mechanical mitral valve replacement at the age of 13. Despite multisystem symptoms, his diagnosis was elusive. There have been numerous studies aimed towards progressive storage illnesses, such as glycogen storage disease, but none have produced any significant findings. He eventually had a de novo heterozygous mutation of LMNA with the amino acid sequence change c.898G > C (p.Asp300His), which allowed for a genetic diagnosis of APS. His urine protein to creatinine ratio was just 0.23 mg/mg during his initial visit to the nephrology clinic, but it grew worse throughout the course of the subsequent appointments. His urine protein to creatinine ratio and N-acetyl-b-D-glucosaminidase to creatinine ratio both rose to 1.52 and 18.7 in three years, respectively. The results of the kidney biopsy were similar with those of FSGS, peri-hilar type, demonstrating segmental sclerosis in 1 (5%) of the 21 glomeruli. To control his proteinuria (0.7 mg/kg), an Angiotensin receptor blocker (Losartan) was administered, but the dizziness made it difficult to continue. Losartan was reintroduced because his proteinuria got worse during the follow-up visit. His proteinuria fluctuated with his heart status, and the dosage (up to 1.2 mg/kg) was changed in response to the symptoms. His renal function remained stable while on losartan, and his proteinuria did not worsen. When he was 15 years old and had his last follow-up, he did great and worked out every day. At the nephrology clinic, benzbromarone, losartan, and sodium bicarbonate have all been recommended for him. He was given a pacemaker for postoperative sinus node dysfunction and warfarin for the repair of his mitral valve. A beta-blocker, a diuretic, amlodipine for HCMP, and omega-3 for hyperlipidemia were additional medications he was taking.

Discussion:

This is the first instance of FSGS in a child with proven APS, lipodystrophy, HCMP, steatohepatitis, and progeroid characteristics. The patient also had heart valve failure. Kidney involvement in APS is a very uncommon finding because it is such a rare disease. In earlier kidney manifestation reports, proteinuria, including the nephrotic range, was

SMART PEDI UPDATE

present.[2] The renal involvement was not mentioned in the two cases where the mutation of our case, c.898G > C (p.Asp300His) in LMNA (Figure 3), was previously reported. Given that the mutation has already been published, it is not a novel mutation. However, it is a very uncommon mutation.[3,4]



Schematic structure of the LMNA gene. The mutation of the patient is indicated with a solid line, and the locations of mutations associated with FSGS are shown in dotted lines. FSGS: focal segmental glomerular sclerosis.

Conclusion:

This is the first Pediatric case with APS who had FSGS. Although the disease's kidney presentation has not previously been highlighted, associated proteinuria and FSGS may worsen the prognosis, especially if discovered later on after the progression of sclerosis. As a result, testing for proteinuria and kidney function should be taken into account while treating APS patients.

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