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Pediatric metabolic syndrome

Abstract: Although the prevalence of obesity and its related complications are increasing among pediatric patients, appropriate management can prevent chronic disease. This article will present an overview of metabolic syndrome, pediatric metabolic syndrome guidelines, pathophysiology, associated risk factors, and clinical practice implications.

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Metabolic syndrome is defined as an adult cluster complex. The definition in children is not clear, although metabolic health concerns have become increasingly prevalent in children and adolescents. It is essential that NPs understand the associated risk factors and pathophysiology in the pediatric population to prevent the development of metabolic syndrome in adulthood.[1]

Obesity can potentially lead to the development of cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) in adulthood.[1] Despite the increasing rates of childhood and adolescent obesity, pediatric metabolic syndrome (PMetS) remains undefined in the US. Rates of pediatric hypertension have increased since 1988 and are now approaching 15% to 19% in boys and 7% to 12% in girls.[2] Nguyen and colleagues reported high total cholesterol, elevated non-high density lipoprotein cholesterol (non-HDL-C), or a low HDL-C in 21% of the children in their study.[3] The prevalence of T2DM has also been rising significantly faster among minority populations than the White population.[4]

Keywords: cardiovascular disease (CVD), counseling, insulin, lifestyle changes, metabolic syndrome, obesity, pediatric metabolic syndrome, type 2 diabetes mellitus (T2DM)

This article will present an overview of PMetS, the existing adult metabolic guidelines, pathophysiology, associated risk factors, and implications for NP practice.

Epidemiology

In the 1980s, researchers found correlations between obesity, hypertension, and hypertriglyceridemia.[1] Insulin resistance and central adiposity are prime elements contributing to PMetS.[1] Obesity, a significant risk factor for CVD, often presents comorbid metabolic abnormalities, including hypertension, dyslipidemia, and insulin resistance.[1] These CVD risk factors are common in the adult population; however, with rising rates of pediatric obesity they are now common in children as well.

The American Academy of Pediatrics' (AAP's) recent review centered on cardiometabolic risk factor reduction as data showed a long-term effect on cardiovascular health.[5] An analysis of the National Health and Nutrition Examination Survey (NHANES) from 1999 to 2002 demonstrated that metabolic syndrome prevalence in obese adolescents increased from 28.7% to 44%.[1] In 2016, the CDC National Center for Health Statistics (NCHS) data brief report identified 18.5% of youth in the US as obese.[6] CDC statistics showcased a steady increase in rates of overweight children in the US. Between 2011 and 2012, rates of overweight children were 14.9% and between 2013 and 2014, rates increased to 16.2%.[7] A 2018 review demonstrated a rise in pediatric obesity from 16.2% in 2007 to 2008 to 18.5% in 2015 to 2016.[6] These authors also found that the prevalence of obesity was higher among males (18.6%) than females (15%).[6]

The NCHS reviewed the incidence of childhood obesity in the US during 2015 to 2016. Data demonstrated that the obesity prevalence was 13.9% among 2- to 5-year-olds, 18.4% among 6- to 11-year-olds, and 20.6% among 12- to 19-year-olds.[6] The 2015 NCHS data brief found an increase in elevated total cholesterol among US children and adolescents from 7.4% in 2011 to 13.4% in 2014 if presenting with a low HDL

Adult metabolic syndrome defining criteria from the NHLBI/NCEP, WHO, and IDF[11-13]

Defining criteria	Central adiposity	Hypertension	Dyslipidemia: hypertriglyceridemia	Dyslipidemia: low HDL	Hyperglycemia	Urinary albumin
NHLBI/NCEP[12]						
T2DM, insulin resistance, or impaired fasting glucose (110–125 mg/dL) plus any three of the following	Men: Waist circumference >40 in (>102 cm) Women: >35 in (>88 cm)	BP ≥130/ ≥85 mm Hg	Triglycerides >150 mg/dL	Men: <40 mg/dL Women: <50 mg/dL	Fasting glucose: ≥110 mg/dL	Not applicable
WHO[11]						
>3 criteria must be present	Men: waist to hip ratio of >0.90 Women: waist to hip ratio >0.85 and/or a BMI >30 kg/m^2	SBP ≥140 mm Hg or DBP ≥90 mm Hg or treatment with antihypertensive medication	Triglycerides >150 mg/dL	Men: HDL <35 mg/dL Women: HDL <39 mg/dL	Impaired fasting glucose of 110-125 mg/dL	Urinary albumin excretion ≥20 mcg/min or albumin/creatinine ratio ≥30 mg/g
IDF[13]						
Central obesity plus at least two out of four criteria	In US, reference includes 102 cm for male and 88 cm for female but measurements for ethnic differences should be considered BMI >30	SBP ≥130 mm Hg or DBP ≥85 mm Hg or treatment with antihypertensive medication	Triglycerides >150 mg/dL	Men: HDL <40 mg/dL Women: HDL <50 mg/dL or on treatment for low HDL	Fasting blood glucose ≥100 mg/dL or previously diagnosed T2DM	Not applicable

cholesterol.[3] In 2017, Flynn and colleagues found an increased prevalence of hypertension in children, with higher rates in boys (15% to 19%) than in girls (7% to 12%).[2] The NHANES from 2001 to 2016 researched the prevalence of hypertension in youth ages 12 to 19 and found a decrease in the rates of hypertension. However, 795,000 US youths were reclassified as having hypertension using the new guidelines.[8] Children who were male, older, and obese accounted for a disproportionate amount of those reclassified with hypertension.[8]

The lack of an established definition of PMetS is problematic, and means that there are no clear guidelines for management.[5] Research indicates that an excessive amount of weight gain in the first years of life can alter developing neural and metabolic systems and increase the risk of obesity and chronic conditions including T2DM, CVD, hypertension, stroke, osteoarthritis, asthma, and certain cancers later in life.[9] Further, early infant weight gain may be a predisposition for the development of obesity and metabolic syndrome later in life.[10] Given the increasing incidence of obesity, diabetes mellitus, hyperlipidemia, and hypertension in the pediatric population, a clear definition of PMetS could improve identification and management to prevent long-term sequelae.

Adult and PMetS guidelines

Several organizations, including the World Health Organization (WHO), the National Heart, Lung, and Blood Institute/National Cholesterol Education Program (NHLBI/NCEP), and the International Diabetes Foundation (IDF), have established various definitions for adult metabolic syndrome.[11-14] The suggested criteria for metabolic syndrome in adults vary by organization but generally include the following: hypertriglyceridemia, hyperglycemia, central adiposity, elevated BP, and low HDL-C.[5] (See *Adult metabolic syndrome defining criteria from the NHLBI/NCEP, WHO, and IDF.*) Given the fluctuating changes in fat, BP, and body composition that occur with age and development, adult definitions cannot be applied in the pediatric population.[1] However, understanding the criteria for adult metabolic syndrome can help the NP identify and support high-risk children. The IDF has created standards for PMetS for children ages 6 to 16 years and older. The definitions are divided according to age groups: age 6 years to younger than 10 years (the IDF suggest that metabolic syndrome should not be diagnosed in this age group); age 10 years to younger than 16 years; and age 16 years and older (the IDF recommends using metabolic syndrome criteria for adults in those age 16 years and older).[14] (See *IDF PMetS definition in children ages 10 to younger than 16.*) Further research may enable refinement of this criteria.[14]

IDF PMetS definition in children ages 10 to younger than 16[14]

Obesity
- ≥90th percentile or adult cutoff if lower

Triglycerides
- ≥150 mg/dL

HDL-C
- <40 mg/dL

BP
- Systolic ≥130; diastolic ≥85 mm Hg

Glucose
- A fasting glucose of 100 mg/dL or greater
- If known T2DM, recommend an oral glucose tolerance test

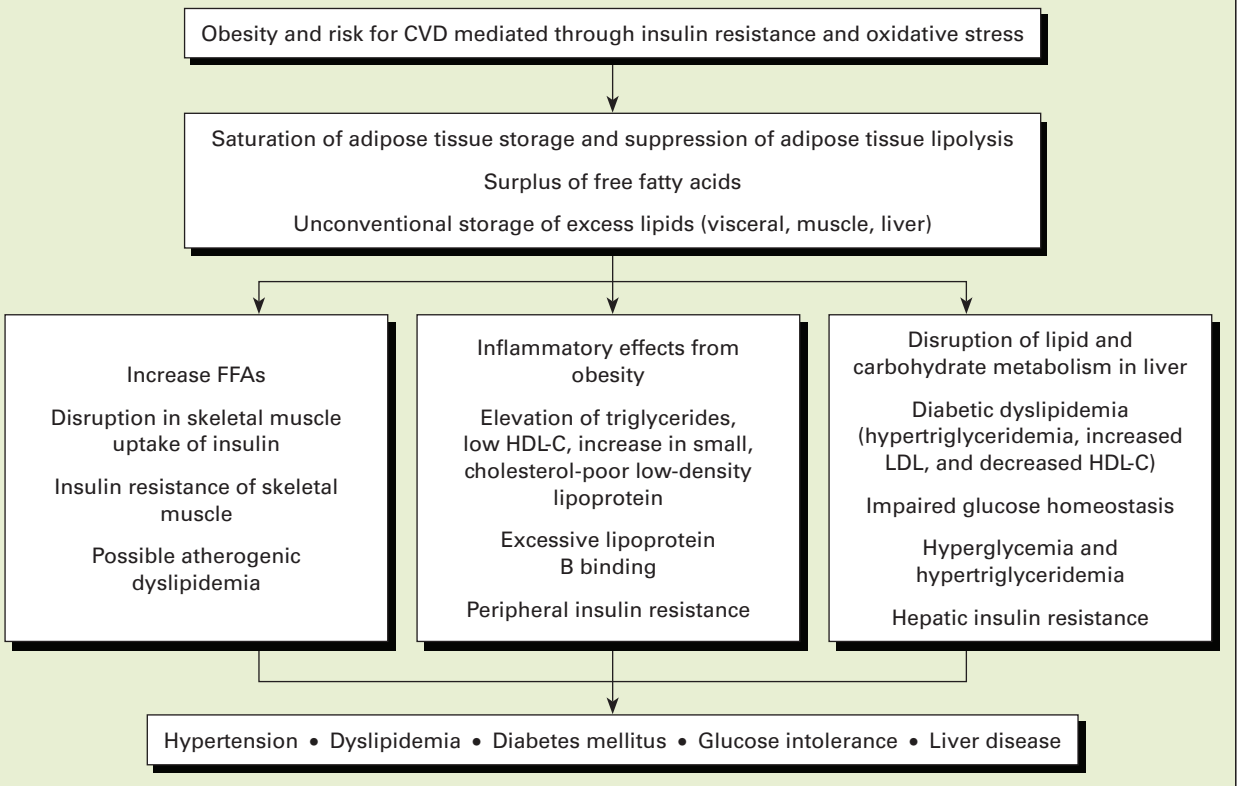
New clinical guidelines for cardiovascular risk reduction in high-risk patients

The American Heart Association published new guidelines to reduce the risk of CVD in pediatric patients with high-risk conditions, which include obesity. Severe obesity is considered a moderate-risk condition, whereas obesity is regarded as an at-risk condition.[15] A body mass index (BMI) of at least 120% of the 95th percentile is the definition of severe obesity. Current estimates are that 6% of children ages 2 years to 19 years fall into this classification.[15] The guidelines focus on the gradual change of lifestyle with improving the quality of foods, reducing calorie intake, and increasing physical activity; adding pharmacotherapy and bariatric surgery to the management should be considered when lifestyle changes fail.[15]

Pathophysiology

The pathophysiology in PMetS needs further clarification to develop practical pediatric recommendations for identification and management of PMetS. There are several studied mechanisms proposed to influence the pathophysiology of metabolic syndrome.[16] Obesity and the subsequent inflammation is a significant contributor to insulin resistance. Inflammation induced by obesity increases the CVD risk by increased triglycerides, lowering HDL-C, and increasing small

Pathophysiology of obesity[1,13-15]



low-density lipoprotein (LDL) particles.[15] The inflammation triggers insulin resistance to keep glucose high to meet activated immune system requirements, but this leads to impaired lipoprotein lipase activity, increased triglyceride production, and the overproduction of hepatic lipoproteins including smaller LDL and HDL particles.[15] The decreased HDL-C promotes the accumulation of cholesterol, which in turn promotes the synthesis of steroid hormones, especially cortisol. Higgins and colleagues suggest that the following are also contributors to the pathophysiology of metabolic syndrome: lipid partitioning and inflammation, adipose tissue insulin resistance and free fatty acid (FFA) flux, muscle insulin resistance and glucose intolerance, hepatic insulin resistance and fasting dyslipidemia, and intestinal insulin resistance and postprandial dyslipidemia.[1] The mechanism of insulin resistance is the best pathologic factor understood.[15]

Insulin resistance. Insulin is a polypeptide hormone secreted from the pancreatic beta cells. Insulin acts through glycoprotein receptors located in the liver, skeletal muscle, intestines, and adipocytes.[16] Insulin plays a variety of roles in the human body but, most important, it regulates glucose and fat. It promotes gluconeogenesis, glycogenolysis, promotes glucose storage, stimulates enzymes involvement in glycolytic and fatty acid synthesis pathways, and directly inhibits hepatic gluconeogenic enzymes.[16] Insulin resistance is considered a decreased tissue response to cellular actions that are mediated by insulin.[1] Because of insulin's effects on various organ tissues, its resistance can cause significant metabolic dysfunction throughout the body, contributing to the metabolic abnormalities associated with metabolic syndrome.[17] (See *Pathophysiology of obesity*.)

It is critical to understand the factors influencing insulin function in the pediatric population. Puberty and obesity play a role in insulin sensitivity and action. Pubertal maturation was found to be associated with a decrease in insulin sensitivity.[18] Puberty impacts the distribution of fat and insulin secretion, including a 25% to 50% reduction in insulin sensitivity, which recuperates upon pubertal completion.[1] The physiologic changes that occur during puberty cause shifts

in insulin sensitivity, growth hormone, sex steroid hormones, and body composition.[18] Faster weight gain in BMI during childhood is associated with greater abdominal adiposity, insulin resistance, and elevated systolic BP (SBP).[19] Further, rapid growth or weight in infants and children results in the development of early puberty.[19] Obesity and early pubertal development are interlinked. Early onset of adrenarche in girls is associated with insulin resistance, dyslipidemia, and obesity later in life.[19] Moreover, early-onset menarche is associated with obesity, insulin resistance, hypertension, and CVD in adulthood.[19]

Koyama and colleagues determined that early adiposity rebound and a high BMI during childhood is associated with an increased risk of T2DM and coronary heart disease in adulthood.[10] Children who exhibit early adiposity rebound are predisposed to the development of insulin resistance because of various physiologic factors, including higher BMI, triglycerides, atherogenic index, apolipoprotein, and BP; lower HDL in boys; and higher apolipoprotein in girls.[10] It is critical to note that insulin resistance can occur in the absence of other signs of metabolic syndrome.[20]

Patient identification and management

Identification of risk factors. Initially, screening for risk factors to identify at-risk children is vital. Risk factors for PMetS include a family history of MetS, Hispanic or Black ethnicity, tobacco smoke exposure, physical inactivity, and dyslipidemia.[20]

History and physical. The NP should take a complete history to look for potential risk factors, signs of obesity, and associated diseases including sleep apnea, intermittent limp, hip pain (evaluating slipped capital femoral epiphysis), and right upper quadrant pain (fatty liver disease). Other common comorbidities associated with obesity further include liver disease and mental health disorders.[5] The AAP guidelines include depression screening starting at age 12.[21] The NP must be sensitive to the child's mental health and use appropriate screening tests along with the history gathered to evaluate for mental health conditions.

Obesity screening using BMI in children ages 2 to 18 years and height for length measurement in children under age 2 is essential. The growth curve is the best way to note changes in a child's growth parameters. Growth rate and pubertal timing are critical to the evaluation of obese children. If there is a fall in the growth rate, the NP should consider other differentials such as thyroid disease, cortisol excess, growth hormone deficiencies, or genetic syndromes. A careful physical exam should include evaluation for any genetic syndromes such as Prader Willi or Bardet-Biedl syndrome. The Endocrine Society recommends that genetic testing should be considered in children with extreme early-onset obesity.[22]

It is also essential to evaluate for an enlarged liver, which may or may not be present in patients with obesity and fatty liver disease. Darkened, thickened, velvety skin or acanthosis nigricans frequently accompany obesity and are correlating factors involved in insulin resistance.[23] Annual BP screening with auscultatory methods should start at age 3 years for healthy children.[2] Carefully evaluate the child for violaceous striae with central obesity and a buffalo hump that can indicate cortisol excess. However, the majority of children with overweight or obesity have a routine physical exam.

Identification of lab markers

The NP must consider which diagnostic labs to order after taking the child's history and performing a physical. Current recommended practices can be obtained using resources such as the Choosing Wisely website (www.choosingwisely.org) as well as the regularly updated AAP periodicity table.[19,24]

Recommended screenings. The AAP has recommended that all children be screened for hyperlipidemia during well-child checks at ages 9 to 11 years, and again at ages 17 and 21 years.[19] However, in the child with obesity, yearly screening for lipid disorders with a nonfasting non-HDL-C, followed by a fasting lipid profile for initial cholesterol greater than 200 mg/dL, an HDL less than 45 mg/dL, or a non-HDL-C >145, is recommended.[13] The American Diabetes Association (ADA) 2019 criteria for pediatric T2DM screening include overweight (BMI greater than the 85th percentile) and at least two of the following risk factors:[25]

- family history of T2DM in either a first- or second-degree relative
- Native American, Hispanic, Asian American, Pacific Islander, or Black ethnicity/race
- maternal history of gestational diabetes or maternal history of T2DM
- signs or conditions associated with insulin resistance such as acanthosis nigricans, dyslipidemia, polycystic ovary disease, or small for gestational age at birth.

A fasting plasma blood glucose, hemoglobin A1C (A1C), or 2-hour plasma glucose during a 75-g oral glucose tolerance test are first-line tests for identification of the patient at risk for or with diabetes mellitus.[25] The 2019 guidelines recommend yearly fasting plasma glucose or an A1C.[25] The NP should keep in mind that the use of A1C may be falsely low in ethnic minority or patients with a hemoglobinopathy (such as sickle cell disease). The hemoglobin A1C is a more expensive test.[25]

Liver involvement secondary to obesity can cause a range of hepatic abnormalities including nonalcoholic fatty liver, nonalcoholic fatty liver disease (NAFLD), nonalcoholic steatohepatitis, or NAFLD with cirrhosis. The AAP recently endorsed the 2017 recommendations from the Expert Committee on NAFLD and the North American Society of Pediatric Gastroenterology, Hepatology and Nutrition to screen obese children with an alanine aminotransferase (ALT) starting at ages 9 to 10 years and repeat screening every 2 to 3 years.[26] Early screening is done in the face of severe obesity, excessive weight gain, or other obesity-related complications. An ALT greater than 80 U/L is considered clinically significant and requires further testing and referral.[26]

The AAP's Section on Endocrinology does not recommend routine screening for insulin levels or routine thyroid screening.

Screenings not recommended. The AAP's Section on Endocrinology does not recommend routine screening for insulin levels or routine thyroid screening. Insulin levels are considered a research tool.[22] Unless there is another reason, routine testing for thyroid disease in obese children is not recommended. Thyroid-stimulating hormone levels are slightly elevated because of the increase in leptin levels, and free T4 or free thyroxine will be within normal range.[25,27]

Screening for vitamin D deficiency is not needed unless there is a reason to suspect vitamin D deficiency. Evidence is not sufficient to support routine testing of obese or overweight children for vitamin D deficiency.[24] If there is a reason to suspect vitamin D deficiency, 25-hydroxyvitamin D would be the correct test to further assess.[22,24]

The NP's role

The treatment and prevention of obesity should be the first-line approach to reduce cardiometabolic risk.[5] However, lifestyle changes are the first-line approach for obesity. Emphasis should be placed on tailoring the care to the patient's and family's wishes and focusing on the risk reduction of developing metabolic syndrome.[5] The use of lifestyle changes in the child with severe obesity is limited, but parental coaching and motivational interviewing shows promise; however, more studies are needed.[13]

Clinical practice

The first step in the evaluation of a child with obesity is monitoring the BMI and BP. Calculating, plotting, and reviewing the BMI annually during well-child visits are suggested for monitoring weight and signs of severe obesity.[15,21] Patients with severe obesity may be more challenging to manage, so referral to a multidisciplinary team can be beneficial for treatment. In 2018, Burton and colleagues reviewed efforts of prevention and reduction of obesity through an established interdisciplinary approach, which includes a medical team, behavioral health, nutritionist or dietitian, exercise physiologist, occupational therapist, and administrative staff.[28] This approach can be considered when working with severely obese children or children who are at higher risk, such as minority youth.[28] The measurement of waist circumference, which is part of the IDF criteria is not recommended in the 2019 guidelines. Research suggests that waist circumference over the 70th percentile in obese children may be an indication of an increased risk of metabolic syndrome.[29]

Monitoring the patient's BP during a yearly checkup and plotting the results on the AAP 2017 charts to evaluate for hypertension are reccomended. If the diastolic or systolic BP is greater than or equal to the 90th percentile, then two additional readings need to be completed. After lifestyle counseling, the child is followed up within 3 months and every 3 to 6 months after the initial follow-up visit.[15]

Counseling regarding diet modification includes the recommendations from the DASH diet. The DASH diet emphasizes a target daily sodium consumption of less than 2,300 mg, encouraging a diet high in fruits

and vegetables, and an increase in foods containing dietary fiber.[15] NPs should encourage patients to avoid foods with added sugar and sugar-sweetened beverages and consume more lean sources of protein.[5] Encouraging consumption of whole fruits, rather than fruit juices, and avoidance of high-sodium processed foods and high-fructose corn syrup can prevent development of or reduce obesity.[22] To reduce the risk of hyperlipidemia, foods that contain polyunsaturated and monounsaturated fats instead of trans fats and saturated fats are reccomended.[15] A low glycemic diet with an intake of added sugar of 5% or less is recommended and can serve as protective factors in maintaining a healthy weight and reduce metabolic and cardiovascular risk.[15]

Exercise recommendations should be written in a prescription and include 5 or more hours of moderate or vigorous exercise a week.[15] Assessing screen time and evaluation of psychosocial comorbidities should be part of patient management.[22] Screening for comorbidities in obese children and adolescents should be kept in mind when gathering a thorough history. Taking a careful sleep and menstrual history will help identify patients with obstructive sleep apnea and polycystic ovary syndrome. All management should be individualized and tailored in an age-appropriate, culturally sensitive, and family-centered manner.[22]

For children whose lifestyle modifications have not been efficacious in weight reduction and management, orlistat is FDA-approved for weight loss in children ages 12 years and older.[5] The patient and family must understand the benefits and risks of the drug including the adverse reactions associated with orlistat. Other drugs, such as metformin, have been studied in pediatric clinical trials for weight reduction; however, it is not FDA-approved for use in managing obesity in pediatric patients.[28] If pharmacologic measures and lifestyle changes fail, or if the adolescent is severely obese, referral to a pediatric surgical multidisciplinary team specializing in obesity is the next step.

Education and research

NPs can attend seminars to improve motivational interviewing and counseling skills. These skills should be practiced and enhanced in NP programs using simulation. Staying current with clinical practice guidelines is vital for practicing NPs. Research is needed to determine the most effective methods to teach these skills to NPs. Further research is also necessary to define metabolic syndrome risk in pediatrics to tailor treatment for this patient population. NPs can partake in clinical research to identify effective methods for reduction of metabolic syndrome development in children and adolescents.

Conclusion

The NP should be aware of health disparities including low socioeconomic status, race, and ethnicity as well as children at higher risk for cardiometabolic health conditions. Understanding the criteria for adult metabolic syndrome can help the NP evaluate children for risk factors. PMetS and its fundamental understanding is a work in progress in the field of pediatrics. Current literature is focused on the reduction of CV risk. Knowledge of PMetS must be elucidated to provide adequate treatment and management in children. Further, there are guidelines for obesity and cardiometabolic risk reduction in the pediatric population. As rates of obesity steadily increase in the pediatric population, rates of chronic conditions will increase as well. Lifestyle changes, counseling, and close follow-up are the best tools available at present. NP

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