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Obesity Disease and its Prophylactic among Children and Adolescents

Makhamatov U. Sh, To'lqinov I. T
Ferghana Medical Institute of Public Health, Fergana, Uzbekistan

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ABSTRACT

Childhood obesity is a serious medical condition that affects children and adolescents. It's particularly troubling because the extra pounds often start children on the path to health problems that were once considered adult problems — diabetes, high blood pressure and high cholesterol. Childhood obesity can also lead to poor self-esteem and depression.

One of the best strategies to reduce childhood obesity is to improve the eating and exercise habits of your entire family. Treating and preventing childhood obesity helps protect your child's health now and in the future.

When to see a doctor

If you're worried that your child is putting on too much weight, talk to his or her doctor. The doctor will consider your child's history of growth and development, your family's weight-for-height history, and where your child lands on the growth charts. This can help determine if your child's weight is in an unhealthy range.

Worldwide, disease profiles are transforming at a rapid pace catching the attention of medical professionals and policy makers alike. This is particularly true in low and middle-income countries that form the major chunk of global population. The emerging epidemics of obesity, cardiovascular disease (CVD) and diabetes form the crux of this phenomenal change. Among these entities, obesity has become a colossal epidemic causing serious public health concern and contributes to 2.6 million deaths worldwide every year. Obesity is an independent risk factor for CVD. Obesity is associated with an increased risk of morbidity and mortality as well as reduced life expectancy. The last two decades of the previous century have witnessed dramatic increase in health care costs due to obesity and related issues among children and adolescents.

Childhood obesity often causes complications in a child's physical, social and emotional well-being.

Societal changes and obesity

Dramatic and rapid societal changes during the last decades have contributed significantly to childhood obesity. There is evidence stating that individual's eating and physical activity behaviours are heavily influenced by surrounding social and physical environmental contexts both for adults and children. Urbanization related intake behaviours that have been shown to

promote obesity include frequent consumption of meals at fast-food outletsconsumption of oversized portions at home and at restaurants, consumption of high calorie foods, such as high-fat, low-fiber foodsand intake of sweetened beverages These behaviours are cultivated in an environment in which high calorie food is abundant, affordable, available, and easy to consume with minimal preparation as is the case of urban cities throughout the country. Television viewing and other sedentary activities have also been related to childhood obesity 3Unfortunately this habit is growing exponentially in developing countries as well. Low levels of physical activity is definitely promoted by an automated and automobile-oriented environment that is conducive to a sedentary lifestyle Community design and infrastructure characteristics are also becoming increasingly important in determining levels of obesity in populations42. Such factors include availability of safe walkways, bicycle paths, playgrounds and other avenues for physical activity related recreation.

Gene mutations and obesity

Single and polygenic gene mutations that occur naturally can produce obesity in rodents like mice and rats. The prototypic obese mice with single gene defects are the obese (ob/ob, Lepob) and diabetes (db/db, Leprdb) autosomal recessive mutations. These mutations produce phenotypes of severe hyperphagia, obesity, type 2 diabetes, defective thermogenesis, and infertility. The mutant gene responsible for the phenotype in Lepobmice encodes a protein termed leptin, which is deficient in these animals. Leptin deficiency has been documented in subsets of human obesity. Severe early-onset human obesity caused by a mutant leptin receptor has also been identified. In the fatty (fat/fat) mouse, the recessively inherited mutation causes hyperinsulinaemia without hyperglycaemia and post-pubertal obesity that is less severe than that seen in ob/ob or db/db mice. The yellow mutation of agouti mice is a dominant trait that causes yellow coat colour, obesity, and diabetes. The polygenic mouse models of obesity closely resemble the human obesity phenotypes than single gene models and have mutations that influence obesity, plasma cholesterol levels, body fat distribution, and propensity toward development of obesity on a high-fat diet.

Neuroendocrinology of energy metabolism

Energy metabolism is controlled by complex neuroendocrine interactions, which influence food intake and energy expenditure. Leptin, almost exclusively produced by the adipose tissue is the major hormone in this mechanism that acts centrally in the hypothalamus. Low plasma concentrations of leptin and insulin as found during fasting and weight loss increase food intake and decrease energy expenditure by stimulating neuropeptide Y synthesis, and perhaps by inhibiting sympathetic activity and other catabolic pathways3. High leptin and insulin concentrations found during feeding and weight gain decrease food intake and increase energy expenditure through release of melanocortin and corticotropin-releasing hormone, among others. The major peptides that stimulate feeding are orexins A and B, which are secreted by the hypothalamus, and ghrelin, which is secreted by the stomach.

Fundamental phases in evolution of obesity

There are critical phases in the evolution of obesity. Intrauterine growth patterns play a significant role in the evolution of obesity by modifying fat and lean body mass, neuroendocrine appetite control mechanisms, and pancreatic functional capacities. Longitudinal studies have identified a strong relationship between birth weight and BMI attained in later life. Increasing birth weight was independently and linearly associated with increasing prevalence of childhood obesity in the

Avon Study

In addition, low birth weight babies show a dramatic transition to central adiposity and insulin

resistance very early in life. These two factors are known to increase cardiovascular risk manifold. Catch up growth and early adiposity rebound increase the odds of children as well as adults becoming obese significantly. [1] The combination of lower birth weight and higher attained BMI is most dangerous as it is associated with extreme CVD risk in later life.

The nature and duration of breastfeeding have been found to be negatively associated with risk of obesity in later childhood. [2] A systematic review of nine studies has concluded that breastfeeding seems to have a small but consistent protective effect against obesity in children. The normal pattern of insulin resistance during early puberty may be a natural cofactor for unnecessary weight gain as well as various co-morbidities of obesity.[3] Early menarche is clearly associated with extent of obesity, with a two-fold increase in rate of early menarche associated with BMI greater than the 85th percentile. The risk of obesity persisting into adulthood is higher among obese adolescents than among younger children. Observations suggest that up to 80 per cent of overweight adolescents will become obese adults.

Environmental risk factors for obesity

Environmental risk factors for overweight and obesity are very strong and inter-related. Sub-optimal cognitive stimulation at home and poor socio-economic status predict development of obesity. Parental food choices significantly modify child food preferences, and degree of parental adiposity is a surrogate for children's fat preferences. Children and adolescents of poor socio-economic status tend to consume less quantities of fruits and vegetables and to have a higher intake of total and saturated fat. Early rebound of BMI is linked to glucose intolerance and diabetes in adults. Short sleep duration in children is also associated with an increase in the odds of becoming obese as well as an increase in body fat per cent.[4]

The trigger factor for initiation of events leading to metabolic syndrome in obesity is not clearly identified. Two schools of thought predominate, one focusing on intra-abdominal fat depots and the other on insulin resistance as starting points. Accumulation of visceral fat is characterized by high lipid turnover resulting in higher levels of free fatty acids (FFA) in the portal circulation46. This could lead to enhanced lipid synthesis, gluconeogenesis, insulin resistance and activation of sympathetic nervous system. [5] Activation of sympathetic nervous system can contribute to elevation of blood pressure through its effects on vascular tissue as well as renal handling of sodium and water. Insulin resistance can independently lead to increased hepatic synthesis of very low-density lipoprotein (VLDL), resistance of the action of insulin on lipoprotein lipase in peripheral tissues, enhanced cholesterol synthesis, increased high-density lipoprotein (HDL) degradation, increased sympathetic activity, proliferation of vascular smooth muscle cells, and increased formation and decreased reduction of plaque. The prevalence of metabolic syndrome in obese children and adolescents vary with the type of diagnostic definition used as well as the population studied. Evidence from large international studies suggests that it could range from 10 to 40 per cent depending on the levels of obesity. Similar trends were reported from adolescent Indian population as well.

Type 2 diabetes mellitus

The association of obesity with type 2 diabetes in adolescents and children is very strong and confirmed by various studies. Evidence entail that obesity driven type 2 diabetes might become the most common form of newly diagnosed diabetes in adolescent youth within 10 years.Evidence is accumulating which suggests a global spread of type 2 diabetes in childhood. [6]Traditionally type 2 diabetes mellitus had been a disease of adults; however, the same now occurs in increased numbers among obese adolescents.Studies demonstrate an increased risk of nephropathy and retinopathy compared to young people with type 1 diabetes, while recent data indicate early signs of cardiovascular disease in youth with type 2 diabetes. Evidence is emerging of a growing prevalence of type 2 diabetes among urban Indian children as well.

Pharmacological treatment

Data supporting the use of pharmacological therapy for paediatric obesity are limited. The drugs sibutramine, orlistat and metformin are currently in use among obese children and adolescents with varying results.[7] Sibutramine, a serotonin non adrenaline reuptake inhibitor enhances satiety and has been shown to be the most effective drug in treating adolescent obesity. This drug may be associated with side effects including increases in heart rate and blood pressure limiting its use in obese adolescents with higher blood pressure.[8] Orlistat, which is a pancreatic lipase inhibitor, acts by increasing faecal fat loss. It is associated with flatulence, diarrhoea, gallbladder diseases, malabsorptive stools and requires fat-soluble vitamin supplementation and monitoring. [12] Orlistat appears to be less effective in those who follow diets which are low in fats as is the case of many Indian diets. Metformin is a valuable adjuvant to the treatment of obese adolescents with severe insulin resistance, impaired glucose tolerance or polycystic ovarian syndrome. Pharmacotherapy should be reserved as a second line of management and should be considered only when insulin resistance, impaired glucose tolerance, hepatic steatosis, dyslipidaemia or severe menstrual dysfunction persist inspite of lifestyle interventions.

Surgical treatment

Many cases of severe adolescent obesity warrant aggressive approaches including surgical treatment. Adolescent candidates for bariatric surgery should be very severely obese (defined by body mass index of > 40), have attained a majority of skeletal maturity (generally > 13 yr of age for girls and > 15 yr of age for boys), and have co-morbidities related to obesity that might be remedied with durable weight loss. More severe elevation of BMI (>50 kg/m2) may be an indication for surgical treatment in the presence of less severe co-morbidities. The bariatric procedures preferred in adolescents are Roux-en-Y gastric bypass and adjustable gastric banding. Late complications include small-bowel obstruction, incisional hernias, weight regain, as well as vitamin and micronutrient deficiencies. [9] These patients warrant meticulous, lifelong medical supervision. Current evidence suggests that after bariatric surgery, adolescents lose significant weight and co-morbidities are appreciably reduced. [13] Bariatric surgery performed in the adolescent period may be more effective treatment for childhood-onset extreme obesity than delaying surgery till adulthood.

Prevention of obesity

The ideal preventive strategy for obesity is to prevent children with a normal, desirable BMI from becoming overweight or obese. Preventive strategies should start as early as newborn period. The strategies may be attempted at the individual, community or physician's level. [10] Those at the individual level backed by consistent evidence include limiting sugar sweetened beverages, reducing daily screen time to less than two hours, removing television and computers from primary sleeping areas, eating breakfast regularly, limiting eating out especially at fast food outlets, encouraging family meals and limiting portion sizes.[11] Encouraging diets with recommended quantities of fruits and vegetables have been supported by mixed evidence. Healthy behaviours derived from this evidence include consuming a balanced diet rich in calcium and fiber, initiating and maintaining breastfeeding, accumulating 60 min or more of moderate to vigorous physical activity per day and limiting consumption of energy dense foods

What can I do if my child is very overweight?

If your child is very overweight, there's lots you can do to help them become a healthy weight as they grow.

Very overweight children tend to grow up to be very overweight adults, which can lead to health problems such as type 2 diabetes, heart disease and certain cancers.

Research shows children who achieve a healthy weight tend to be fitter, healthier, better able to learn, and are more self-confident. They're also less likely to have low self-esteem and be bullied.

As a parent, there's lots you can do to help your child become a healthier weight. Getting them to be more active and eat well is important.

Listen to your child's concern about their weight. Overweight children often know they have a weight problem, and they need to feel supported and in control of their weight.

Let them know that you love them, whatever their weight, and all you want is for them to be healthy and happy.

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