Introduction

Obesity represents a significant and growing problem around the globe aside from the impairment of an individual patient, the negative consequences impose a significant economic burden for many health care systems ***(Apovian, 2013)***.

The increase in severe obesity related comorbidities in adolescents, coupled with the present ineffectiveness of non-surgical therapies, have resulted in increased acceptance of weight loss surgery (WLS) as a treatment option during adolescence ***(O'Brien et al., 2014).***

For over a decade, bariatric surgical procedures have established themselves as a way to achieve a permanent weight reduction for a large number of patients ***(Carroll et al., 2013)***.

Bariatric surgery for the treatment of adolescent is becoming more common. Although few prospective or comprehensive studies have been performed to define the comorbitidies, outcomes, and safety of bariatric surgery in adolescents ***(Inge et al., 2014).***

A successful bariatric program for adolescents requires the participation of many pediatric subspecialists including

surgeons familiar with bariatric procedures, anesthesiologists, endocrinologists, gastroenterologists, cardiologists, pulmonologists, and others ***(Samuels, 2005).***

Adolescent patients who wish to explore bariatric surgery must have failed at least six months of an organized attempt at weight management, and must be at, or close to, physical maturity. Patients must also have a BMI of >40 with severe comorbidities, or >50 with less severe comorbidities ***(Inge et al., 2004),*** accordingly, this patient population represents a particular challenge for the anesthiologist ***(Nishiyama et al., 2012)***.

There are various contributions on the anesthetic and perioperative management in this patient group, including the preoperative assessment, especially with regards to the airway management and monitoring strategies, the intraoperative phase, the emergence from anesthesia and the postoperative phase ***(Greenstein et al., 2012).***

Also key factor for the perioperative course of those patients is the duration of anesthesia, which in return consists mostly of the duration of surgery and the expertise of the surgeon ***(Birkmeyer et al., 2013).***

Aim of the Work

The aim of this study is to discuss the effect of obesity in adolescents, the indications and hazards of bariatric surgery for them and to study the hazards and different anesthetic challenges for anesthesia of this category of patients undergoing bariatric surgery.

Chapter (1)
Obesity in Adolescents

Obesity is a chronic, multifactorial condition that results from accumulation of fat, regionally located throughout the body, due to the positive difference between food consumption and energy expenditure. ***(Balistretri et al., 2010)***

Obesity is an independent risk factor for cardio vascular diseases (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 ***(Wang and Dietz, 2002)***.

For adolescents, overweight and obesity are defined using age and sex specific normograms for body mass index (BMI). Adolescents with BMI equal to or exceeding the age, gender specific 95^{th} percentile are defined obese. Those with BMI equal to or exceeding the 85^{th} but are below 95^{th} percentiles are defined overweight and are at risk for obesity related comorbidities ***(Donohoue, 2004)***.

Obesity is commonly associated with a variety of comorbidities, including cardiovascular disease, endocrine-

opathies, and osteoarthritis. The mechanisms underlying these complications are complex and most often are inter-related ***(Klein et al., 2002)***.

One of the most prevalent disorders associated with obesity is type 2 diabetes resulting from insulin resistance and hyperinsulinemia. Hyperinsulinemia frequently results in sodium retention, excessive circulating catecholamines, and increased blood volume. Patients commonly have hypertriglyceridemia and low high density lipoprotein levels, which may contribute to coronary artery diseases (CAD) ***(Cummings and Schwartz, 2003)***.

Epidemiology

Adolescents obesity affects both developed and developing countries of all socio-economic groups, irrespective of age, sex or ethnicity. It has been estimated that worldwide over 22 million children under the age of 5 are obese, and one in 10 children is overweight. A wide range of prevalence levels exist, with the prevalence of overweight in Africa and Asia averaging well below 10 percent and in the Americas and Europe above 20 percent ***(Kosti and Panagiotakos, 2006)***.

Obesity has become a serious public health concern affecting a significant portion of the population in countries like the US. Overall, among adults aged at least 20 years in 1999-2002, 65.1 percent were overweight and 30.4 percent were obese ***(Hedley et al., 2004).***

Obesity prevalence varies across socio-economic strata. In developed countries, children of low socio-economic status are more affected than their affluent counterparts. The opposite is observed in developing countries, adolescents of the upper socio economic strata are more likely than poor children to be obese ***(Wang et al., 2005).***

Measurment of obesity

Several clinical anthropometric measures have been used for time past to assess obesity and overweight but BMI is the most common tool in the assessment of Obesity. BMI has gain international recognition. It has been identified by the World Health Organization as the most useful epidemiological measure of Obesity. Also, it has been considered as a gold standard for defining Overweight and Obesity. BMI is an indicator of overall adiposity**,** it is

calculated as weight/height^2, with weight being in kilograms and height being in meters ***(Odenigbo et al., 2011).***

Waist Hip Ratio (WHR) was suggested and further evaluated in terms of fat distribution via the Waist Hip Ratio WHR, it is an indicator for abdominal adiposity. Studies have indicated that BMI and WHR could be used independently to identify Overweight and Obesity ***(Deurenberg and Yap, 1999)***

Dualenergy X-ray absorptiometry (DXA) scanning is used primarily by researchers to accurately measure body composition, particularly fat mass and fat-free mass. It has the additional advantage of measuring regional fat distribution. However, DXA scans cannot be used to distinguish between subcutaneous and visceral abdominal fat deposits. The current standard techniques for measuring visceral fat volume are abdominal computed tomography (CT) scanning (at L4-L5) and magnetic resonance imaging (MRI) techniques. A simpler technique, using bioelectrical impedance, was recently introduced. ***(Ward, 2012)***

Classifiation of obesity

According to body mass index, obesity classified as the following:

- Grade 1 overweight (commonly and simply called overweight) - BMI of 25-29.9 kg/m ^{2}.
- Grade 2 overweight (commonly called obesity) - BMI of 30-39.9 kg/m ^{2}.
- Grade 3 overweight (commonly called severe or morbid obesity) - BMI greater than or equal to 40 kg/m $^{2.}$

The cut-off for each grade varies according to an individual's ethnic background. For example, a BMI of 23 kg/m^2 or higher may define grade 1 overweight and 27.5 kg/m^2 or higher may define grade 2 overweight (obesity) in many Asian populations, in which the risk was shown to be high and extremely high for grade 1 and 2 overweight at these levels, respectively. Other BMI cutoffs identified as potential public health action points in these populations are 32.5 and 37.5 kg/m^2. ***(Shiwaku et al., 2011)***

Etiopathogenesis of Adolescents obesity

Etiopathogenesis of adolescents obesity is multi-factorial. Interactions between genetic, neuroendocrine, metabolic, psychological, environmental and sociocultural factors are clearly evident in childhood obesity ***(Raj andKumar, 2010)***.

Factors of obesity

Energy balance is determined by the interplay between food intake, energy expenditure and energy storage. Obesity is a multifactorial disorder resulting from combination of several environmental and genetic factors. Reduction in physical activity, metabolic rate and thermogenesis eventually decrease energy expenditure leading to increased energy storage and obesity. Availability of palatable food as well as hypothalamic injury and different drugs stimulate food intake. A growing list of genetic factors including dysmorphic syndromes, leptin/receptor mutation ***(Berthoud, 2002).***

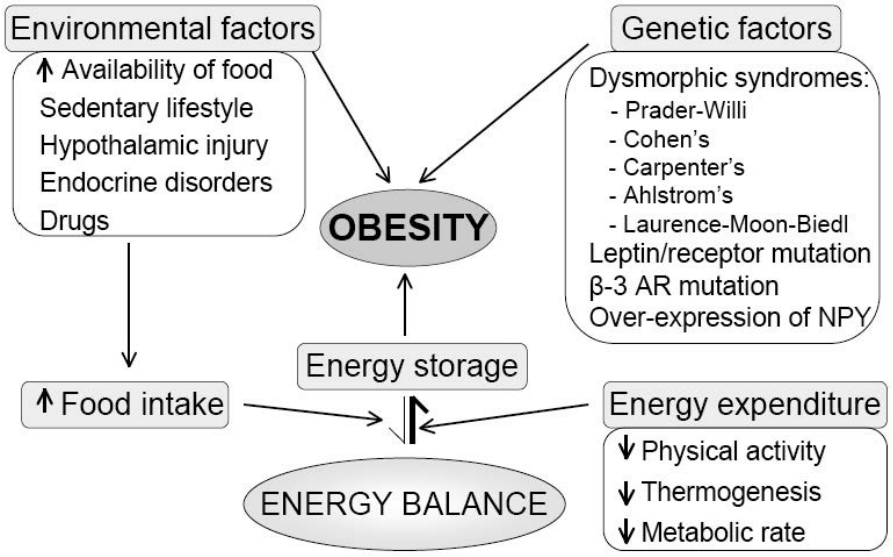

Figure (1): Relation between obesity and energy storage ***(Berthoud, 2002)***

i) 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, Lep^{ob}) and diabetes (db/db, $Lepr^{db}$) 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 Lep^{ob}mice encodes a protein termed leptin, which is deficient in these animals. Leptin deficiency has been documented in subsets of human obesity ***(Raj and Kumar, 2010)***.

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 ***(Kosti and Panagiotakos, 2006)***.

Genetic conditions known to be associated with predilection for obesity include Prader Willi syndrome, Bardet Biedl syndrome, and Cohen syndrome. Obesity clearly demonstrates a familial tendency ***(Reilly et al., 2005)***.

ii) 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 pathways ***(Donohoue, 2004)***.

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 ***(Raj and Kumar, 2010)***.

iii) 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. There is 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 ***(Reilly et al., 2005)***.

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 ***(Ibáñez et al., 2006)***.

Catch up growth and early adiposity rebound increase the odds of children as well as adults becoming obese significantly. The combination of lower birth weight and higher attained BMI is most dangerous as it is associated with extreme CVD risk in later life ***(Dietz, 2004).***

The nature and duration of breastfeeding have been found to be negatively associated with risk of obesity in later childhood ***(Raj and Kumar, 2010)***.

Breastfeeding seems to have a small but consistent protective effect against obesity in children ***(Arenz et al., 2004).***

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 85^{th} percentile ***(Adair and Larsen G, 2001).***

The risk of obesity persisting into adulthood is higher among obese adolescents than among younger children. Observations suggest that up to 80 percent of overweight adolescents will become obese adults ***(Daniels et al., 2005)***.

iv) Environmental risk factors for obesity

Environmental risk factors for overweight and obesity are very strong and inter-related. Suboptimal cognitive stimulation at home and poor socio-economic status predict development of obesity ***(Strauss and Knight, 1999).***

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 ***(Kennedy and Powell, 1997)***.

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 ***(Nixon et al., 2008).***

v) 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