Impact of Increased Body Mass Index On Critically Ill Patients

An Essay

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CONTENTS

INTRODUCTION

Increased body mass index is a global epidemic increasingly affecting management of intensive care medicine. In America today, more than one third of adults are obese. These individuals have increased risk for a multitude of health disorders including diabetes, coronary artery disease, dyslipidemia, stroke, hypertension, gallbladder disease, certain cancers, osteoarthritis, sleep apnea and other respiratory disorders. As the prevalence of obesity in the population has increased there has also been an increasing trend in hospitalized critically ill patients who are obese and morbidly obese. ***(Goulenok et al.,2010)***

The impact of increased body mass index and obesity on outcome in critically ill patients has not been well studied. There are only a few comprehensive reviews that detail the management of the obese critically ill patient. It is reported that morbidly obese patients have an eightfold higher mortality following blunt trauma than nonobese patients. (***Winkelman and Maloney., 2005)***

It has been shown that hospitalized obese patients are at an increased risk of developing respiratory and other complications. It is likely that obesity increases the incidence of complications in patients admitted to the intensive care unit (ICU) and that these complications

are associated with a longer hospital stay and an overall poorer outcome. ***(Winkelman and Maloney., 2005)***

Critical care staff need to understand what makes these patients different from people of normal size and those who are merely overweight. Obese and morbidly obese patients have increased risks of problems when admitted to the critical care environment. Critical care staff are also at increased risk when caring for these patients. Risks to both patients and physicians has been identified as well as recommendations to address those risks. More research is needed to understand fully changes that should be made in critical care units so that appropriate care is delivered to optimize patient outcomes and promote safety for critically ill obese patients and critical care staff. ***(Grant and Newcombe., 2011)***

The management of the morbidly obese critically ill patient is a challenging and formidable task. A better understanding of the pathophysiologic changes that occur with obesity and the complications unique to this group of patients may improve their outcome. ***(Knaus.,2010)***

AIM OF THE ESSAY

The aim of this essay is to discuss pathophysiology of increased body mass index in critically ill patients, identefication of major risks of increased body mass index and management considerations in obese and morbid obese patients in ICU.

Chapter (1)

Patho physiology of increased body mass index

Items of the chapter

General introduction to BMI
Regulation of energy balance

- Appetite regulation
- Regulation of metabolism
- Role of the central nervous system

Causes of increased BMI

- Inherited causes
- Environmental causes
- Other causes

General introduction to BMI

Body mass index (BMI) is a measure of weight adjusted for height, calculated as weight in kilograms divided by the square of height in meters (kg/m2). Although BMI is often considered an indicator of body fatness, it is a surrogate measure of body fat because it measures excess weight rather than excess fat. Despite this fact, studies have shown that BMI is correlated to more direct measures of body fat, such as underwater weighing and dual energy x-ray absorptiometry (***Whitlock et al .,2013*****)**

In general , two types of obesity are described . Central –android obesity adipose tissue is located predominantly in the upper body and peripheral gynecoid obesity fat is located primarily in the hips , buttocks and thighs (***Whitlock et al .,2013*****)**

A BMI less than 25 is considered normal and is associated with the fewest obesity related illness . A patient with a BMI of between 26 and 29 is considered overweight and a BMI greater than 30 is considered obese ***(Hancox et al ., 2004)***

Morbid obesity is a phrase used to describe extreme obesity that if untreated will significantly shorten that individual's life expectancy ***(Hancox et al ., 2004)***

Definition for morbid obesity vary , some consider any individual who is 50 Kg above his ideal weight to be morbid obese. Other define morbid obesity as doubling of ideal weight . While still others define anyone with a BMI greater than 40 as morbidly obese ***(Hancox et al ., 2004)***

The prevalence of obesity has been increasing for several decades. It is now the most common nutritional disorder worldwide , and its medical, psychological, social and economic effects have major consequences for health ***(Pereira et al ., 2010)***

Obesity develops when energy intake exceeds energy expenditure over time (usually many years), leading to accumulation of adipose tissue with a corresponding increase in lean body mass (from the necessarily enlarged muscle, bone and connective tissue). It is important to recognize that even a small daily energy imbalance eventually results in significant weight gain; **for example**, a daily excess of 100 kcal (equivalent to a small chocolate bar) leads to an increase of approximately 5 kg of fat over 12 months, or 50 kg over 10 years. Whether an individual develops obesity, when it occurs and its severity depends on a complex interaction of genetic and environmental influences ***(Hancox et al ., 2004)***

In general, severe obesity developing at a young age is considered more likely to be influenced by major genes that influence energy balance. Late-onset obesity, although influenced by minor genes, is more likely to have a strong environmental component ***(Pereira et al ., 2010)***

Obesity is usually defined in terms of body mass index (BMI) but may be considered pathophysiologically to be present when sufficient body fat has accumulated to adversely affect health. This amount may vary between populations and also depends on the distribution within the body of the excess adipose tissue; for example, excess fat in the abdomen is associated with an increased risk of metabolic diseases such as diabetes mellitus. However, fat distribution may be less important for mechanical consequences such as osteoarthritis of the knee ***(Kral , 2006)***

Regulation of energy balance

Energy balance is usually tightly regulated; even in societies where obesity is common, the average weight gain is only about 1 kg per year - reflecting an energy excess of about 20 kcal per day, or less than 1% of daily energy expenditure. This tendency to gain weight throughout adult life probably reflects the fact that the body's regulatory systems have evolved to protect against weight loss rather than prevent weight gain ***(Pereira et al ., 2010)***

Food intake and energy expenditure are both under an important degree of central nervous system control. Afferent neural and hormonal signals arise predominantly from the gastrointestinal tract, liver and adipose tissue, and efferent neural and hormonal signals influence the digestion and metabolism of food ***(Pereira et al ., 2010)***

Appetite regulation

Food intake is under short-term and long-term control. In the short term, hunger develops in response to decreasing circulating concentrations of certain nutrients (e.g. glucose, fatty acids and possibly some amino acids). The hormone ghrelin, which is secreted by the stomach between meals, stimulates food intake and may be an important hunger signal (**Kissebah et al ., 2012)**

Following a meal, nutrient concentrations increase, as do the concentrations of several 'satiety' hormones (e.g. cholecystokinin, glucagon-like peptide 1, oxyntomodulin, pancreatic polypeptide, peptide YY (3-36)); with diminishing hunger signals, these act on the brain to 'switch off' hunger and stimulate a feeling of fullness ***(Mason et al ., 2010)***

Longer-term signals depend on the magnitude of energy stores and include the adipocyte-derived hormone leptin. When adipose tissue mass is low and leptin concentrations decrease below a critical level,

several powerful hunger signals are activated in the hypothalamus ***(Mason et al ., 2010)***

In general, these systems tend to restore energy balance by stimulating food intake and inhibiting thermogenesis. In contrast, the response to energy excess is relatively weak, and though some hunger signals are switched off and dietary thermogenesis increased, food intake is not stopped completely. In general, it has been evolutionarily advantageous to maximize energy stores when food is plentiful ***(Mason et al ., 2010)***

Regulation of metabolism

There are three principal components of energy expenditure : the basal metabolic rate , the thermic effect of food and energy consumed during physical work.

The basal metabolic rate is the energy required to maintain normal metabolism. Under some circumstances, it is possible to 'uncouple' oxidative phosphorylation within mitochondria, dissipating excess energy as heat. In rodents, BAT (brown adipose tissue) is fat rich in mitochondria that are specialized for this purpose; uncoupling occurs following activation of BAT via the sympathetic nervous system (the b3 adrenoceptor is specific to BAT). Activation of BAT is an important part of the response to cold, but it may also help determine ability to resist weight gain in response to overfeeding (**Kissebah et al ., 2012)**

There is evidence that this may also occur in humans, but there is controversy about the magnitude of the effect and whether it contributes to the tendency of some individuals to gain weight (**Kissebah et al ., 2012)**

The thermic effect of food (dietary thermogenesis) is the energy used in digesting and storing a meal. This is greatest for protein-rich meals, intermediate for carbohydrates and very low for fats, which may partly

explain why a high fat intake is likely to lead to weight gain (**Kissebah et al ., 2012)**

Energy consumed during physical work accounts for a variable amount of total energy expenditure. However, obese individuals use a greater amount of energy than lean individuals in walking the same distance, contributing to the greater energy expenditure seen in the obese (**Kissebah et al ., 2012)**

Role of the central nervous system (CNS)

Within the CNS, at least 50 different neurotransmitters respond to the circulating nutritional, neural and hormonal signals described above, determining feelings of hunger or satiety and thereby food intake, and influencing metabolic rate (via hormones and the sympathetic nervous system) ***(Sarlio-Lahteenkorva et al ., 2005)***

Signals that increase food intake tend to decrease metabolic rate (favouring conservation of energy), and vice versa. Multiple circuits exist that may be influenced at different levels, though the complete system is not fully understood. For example, leptin concentrations in blood decrease when body fat mass decreases, activating hunger signals in the hypothalamus (e.g. neuropeptide Y) and inhibiting other neurons, including those producing pro-opiomelanocortin (POMC) ***(Sarlio-Lahteenkorva et al ., 2005)***

Once fat mass increases again, this process is reversed, thereby maintaining body weight homeostasis. Other systems, including endogenous opiates and cannabinoids, may influence energy intake and the hedonic responses to food ***(Sarlio-Lahteenkorva et al ., 2005)***

Causes of obesity

(1) Inherited causes

(table 1)

Single-gene defects causing obesity are rare in humans. Examples include rare mutations in the gene encoding leptin or its receptor. Affected individuals suffer uncontrollable hunger and develop severe obesity at a young age; unlike similar syndromes in rodents, there is no evidence that these children have a defect in thermogenesis. Other syndromes have been described involving defects in signals downstream of leptin, notably in the POMC gene and the melanocortin-4 receptor ***(Fawcett and Barroso ,2015)***

Severe obesity is a feature of several inherited syndromes. The most common is PradereWilli syndrome. These children typically fail to thrive in the first 2 years of life but then develop a voracious appetite, leading to severe obesity and often causing complications such as type 2 diabetes and obstructive sleep apnoea ***(Fawcett and Barroso ,2015)***

Other features of the syndrome include learning difficulties, short stature, small hands and feet, and almond-shaped eyes. The syndrome is usually associated with a mutation in a paternally imprinted gene on chromosome 15 but the biological explanation for the increased appetite and obesity is not known ***(Fawcett and Barroso ,2015)***

Such inherited syndromes account for only a very small proportion of the 20% of the population who are obese (though single-gene defects, notably melanocortin-4 receptor mutations, may account for up to 1 in 20 cases of severe early-onset childhood obesity). There is evidence from family and twin studies that body weight and the tendency to develop obesity are partly inherited, with an overall heritable contribution of 20 - 70% to the variance between individuals. The highest heritability figures arise in twin studies, and are likely to be influenced by non-genetic factors e.g. differential up-

bringing of identical vs non-identical twins ***(Fawcett and Barroso ,2015)***

Bouchard et al. have estimated the genetic contribution of variability in BMI to be about 30%. Genome-wide association studies have identified several hundred genes that may influence body weight, the most significant of which is the FTO gene. This is
relatively common (about 16% of the population have one variant allele); its presence is associated with an average 1.5 kg increase in
weight. Thus, of the common gene variants identified so far, it appears that even the most powerful effects may contribute very little to the overall heritability of body weight ***(Bouchard et al ., 2010)***