
ICU Volume 9 - Issue 4 - Winter 2009/2010 - Cover Story

Obese Paediatric Patients Requiring Intensive Care

Childhood obesity is increasing in prevalence worldwide. In the United States, studies estimate that among children aged six to nineteen, 16.5% are overweight (Body Mass Index [BMI] 85th to 94th percentile for age and gender) and 17.1% are obese (BMI \geq 95th percentile for age and gender) (Ogden et al. 2006). This trend will likely lead to an increase in obese paediatric patients requiring intensive care. Studies have shown that obese adults in intensive care units (ICUs) have increased length of stay, poorer outcomes, and increased mortality (Pieracci et al. 2006); however, similar studies in children are lacking. Obesity is associated with physiologic derangements similar to critical illness including chronic inflammation, impaired immunity, insulin resistance, and hypercoagulability. Excess body mass requires increased work from all major organ systems, causing a decrease in the physiologic reserve (Pieracci et al. 2006). Practitioners in the paediatric intensive care unit (PICU) should be familiar with some of the basic physiologic derangements that may be seen in these patients.

The metabolic syndrome describes a cluster of cardiovascular risk factors, including hypertension, dyslipidemia, insulin resistance, and central adiposity, caused by the release of inflammatory and vasoactive mediators from adipose tissue (Jolliffe et al. 2006). Approximately one-third of obese adolescents have the metabolic syndrome, compared to 7% of overweight adolescents and 0.6% of adolescents with normal BMI (Cook et al. 2003). Obesity is an independent risk factor for hypertension in children, defined as systolic and/or diastolic blood pressure \geq 95th percentile for gender, age, and height. Obese children have reduced peripheral artery dilatation due to impaired endothelial and smooth muscle function (Aggoun et al. 2008) and decreased parasympathetic nervous system activity (Carchman et al. 2005). Increased left ventricular mass occurs in obese children regardless of blood pressure classification (Maggio et al. 2008). Uncontrolled hypertension may cause seizures, encephalopathy, stroke, and congestive heart failure (National High Blood Pressure Education Program 2004). Critically ill obese children should have close blood pressure monitoring using appropriate-sized cuffs that cover two-thirds the length of the upper arm with the bladder length encompassing the entire arm circumference (Aggoun et al. 2008).

Childhood obesity is associated with the release of catecholamines, cortisol, glucagon, and proinflammatory mediators, leading to impaired glucose tolerance and insulin resistance. Stress-induced hyperglycaemia is often seen in critically ill patients, and may be more pronounced in children with impaired glucose tolerance at baseline. While initially advantageous, providing energy to organs and tissues with increased demand, prolonged hyperglycaemia leads to free radical formation, cellular damage, and impaired immunity, ultimately causing poorer outcomes and increased mortality (Clark et al. 2008). Although there is a paucity of data on the relationship between glucose control and outcomes in critically ill children, studies in adults suggest that strict glucose control in ICUs improves morbidity and mortality (Wintergerst et al. 2006). A subset of children with impaired glucose tolerance will develop type 2 diabetes (Velasquez-Mieyer et al. 2007). The symptoms are often subtle, which may lead to a delay in diagnosis. Children may present with Hyperglycaemic Hyperosmolar Syndrome (HHS), characterised by hyperglycaemia, elevated serum osmolality, and mild metabolic acidosis without ketosis. They can lose 15-20% of total body water, leading to severe hypovolemia and requiring aggressive fluid resuscitation. The adult mortality rate for HHS is 10-15%; the mortality rate in children is unknown, although case reports have shown poor outcomes, including rhabdomyolysis, multiorgan failure, and death (Carchman et al. 2005).

Asthma has increased in prevalence parallel to the rise in childhood obesity. Elevated BMI is associated with higher rates of asthma and wheezing (Shaheen et al. 1999). Leptin, a hormone produced by adipose cells, has a positive correlation with BMI and asthma; this may be due to its proinflammatory effects (Guler et al. 2004) or its association with hypoventilation in obese individuals (Phipps et al. 2002). In addition, obesity causes a restrictive lung pattern due to increased pulmonary blood volume and increased chest wall mass from adipose tissue. Obese children admitted to the PICU for status asthmaticus have increased lengths of stay and require longer courses of intensive therapy, including the need for supplemental oxygen and administration of continuous beta-agonists and intravenous steroids (Carroll et al. 2006).

Obesity is a risk factor for obstructive sleep apnoea (OSA) and correlates with more severe symptoms. Approximately 37% to 46% of obese children have OSA (Shine et al. 2006). Obese children with OSA who undergo tonsillectomies and adenoidectomies have more intra-operative respiratory complications, including multiple intubation attempts and post-induction oxygen desaturations. They are more likely to be hospitalised postoperatively and have increased lengths of stay (Nafiu et al. 2009). Postoperative upper airway obstruction may be caused by airway compression from excess neck tissue, hypoventilation due to decreased chest wall compliance and upward displacement of the diaphragm, and decreased airway tone after anaesthesia. Preoperative polysomnography is recommended for all obese patients to determine OSA severity, which can guide the need for intensive care monitoring postoperatively (Shine et al. 2006). If left untreated, OSA can cause pulmonary hypertension and cor pulmonale due to chronic nocturnal hypoxemia (Bower et al. 2000).

Obese children undergoing surgery have an increased risk of complications. They have significantly higher American Society of Anaesthesiology (ASA) scores, given their medical comorbidities (Nafiu et al. 2007). Vascular access can be difficult to obtain and control of the airway is more challenging. Medications, especially sedatives and narcotics, should be dosed based on ideal body weight. Postoperatively, there is increased risk of upper airway obstruction and desaturations (Veyckemans et al. 2008). Obesity is an independent risk factor for deep venous thrombosis; therefore, obese children in particular should receive DVT prophylaxis postoperatively (Wurtz et al. 1997).

As the prevalence of childhood obesity increases, there will continue to be what were formerly considered "adult" diseases manifesting in children and adolescents. It is important to be aware of these comorbidities in order to anticipate potential complications in critically ill obese

children. More research is needed to better understand the effect of obesity on outcomes in the PICU and to determine strategies to improve morbidity and mortality for these patients.

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