

Pathophysiology Effects of Acute Malnutrition in Children

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DESCRIPTION

Acute malnutrition is an absence of nutrients brought on by insufficient protein or energy intake. An imbalance between nutrient requirement and intake, resulting in cumulative deficits of energy, protein, or micro-nutrients that may adversely affect growth, development, and other relevant outcomes are known to be paediatric malnutrition. Repeated exposure to environmental pathogens leads to the colonisation of small intestinal bacteria, which causes accumulation of inflammatory cells in the small intestinal mucosa that damages the intestinal villi which results malabsorption of nutrients causes the malnutrition. Secondary acute malnutrition is typically caused by increased energy expenditure, or decreased food intake leads to abnormal nutrient loss. This condition is frequently present in conjunction with underlying, mostly chronic diseases like cystic fibrosis, chronic renal failure, chronic liver diseases, childhood cancers, congenital heart disease, and neuromuscular diseases.

Pathophysiology

Physiological adaptations lead to insufficient energy intake and causes several symptoms like growth restriction, loss of fat, muscle, and visceral mass, a decrease in basal metabolic rate, which ultimately leads to decrease in overall energy expenditure. Acute malnutrition causes biochemical alterations in the metabolic, hormonal, and glucoregulatory systems. Thyroid hormones, insulin, and Growth Hormone (GH) are the main hormones that are impacted. Tri-iodothyronine (T3), insulin, and Insulin-like Growth Factor-1 (IGF-1) levels are decreased, whereas GH and cortisol levels are increased.

Initially, glucose levels are frequently decreased due to the depletion of glycogen reserve and cause rapid gluconeogenesis and the subsequent loss of skeletal muscle due to the use of amino acids, pyruvate, and lactate characterizing the early phase of malnutrition. Following the phase of protein conservation, fat

is mobilized, resulting in lipolysis and ketogenesis, and also causes major electrolyte changes, such as sodium retention and intracellular potassium depletion, which will attribute to increased cell membrane permeability in kwashiorkor and decreases the activity of the glycoside-sensitive energy-dependent sodium pump. Acute malnutrition impairs various organ systems like Thymus, lymph node, and tonsil atrophy which will all show an impact on cellular immunity. Decrease in secretory immunoglobulin A, loss of delayed hypersensitivity which impaired phagocytosis, and a reduced Cluster of Differentiation (CD) 4 are present with normal CD8-T lymphocytes. Villous atrophy with ensuing loss of disaccharidases, crypt hypoplasia, and altered intestinal permeability results in malabsorption, which in turn increases susceptibility to invasive infections (urinary, gastrointestinal, septicemia, etc). Other frequent features include bacterial overgrowth, pancreatic atrophy leading to fat malabsorption, and fatty liver infiltration. Drug metabolism may be affected by low plasma albumin levels and low fractions of the glycoproteins that bind drugs. Cardiac myofibrils become thin and show a decrease in contractility, due to weight loss the cardiac output also decreases proportionally. In severe cases, bradycardia and hypotension are also frequent. Arrhythmias are more likely to occur when bradycardia, decreased cardiac contractility, and electrolyte imbalances are present. Decreased minute ventilation and a compromised ventilatory response to hypoxia may be caused by reduced thoracic muscle mass, a slowed metabolic rate, and electrolyte imbalances (hypokalemia and hypophosphatemia). Acute malnutrition reduces the number of neurons, synapses, dendritic arborizations, and myelinations, all of which lead to a reduction in brain size, and therefore brain growth starts becoming slow, and the cerebral cortex is thin. Malnutrition also has been linked to delay in cognitive, motor, and overall function. After 3-4 years of age, the effects on the developing brain may become permanent.

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