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Pediatric Care 2018: Growth faltering Role of insulin like growth factors - Sangita Yadav - Maulana Azad Medical College

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Growth is a sensitive indicator of a child's health, nutritional state and genetic background. Growth of a child is not only controlled by hormones but by many other factors like nutrition and chronic systemic illnesses like hypothyroidism, chronic liver disease, diabetes mellitus, malabsorption, etc. Principal hormones influencing growth are Growth Hormone (GH), thyroid hormones, adrenal androgens, sex steroids, glucocorticoids, vitamin D, leptin and insulin. Growth hormone promotes longitudinal bone growth. GH mediates its effects on target tissues via stimulation of hepatic Insulin like Growth Factor (IGF-1) production. IGF-1 is a single chain polypeptide hormone with structural homology to proinsulin, produced from liver. IGF-1 is at least in part GH dependent and mediates many of the anabolic and mitogenic actions of GH. Alternative hypothesis is the dual effector theory, which is based on the premise that growth is a result of the differentiation of precursor cells, followed by clonal expansion. GH directly promotes the differentiation of cells and the development of IGF-1 responsiveness.

Clonal expansion of these differentiated cells is mediated by local production of IGF-1 in response to GH. IGF-1 appears to be critical for fetal and postnatal growth. Levels of IGF-1 are inversely related to Body Mass Index (BMI). Inadequate calorie intake and/or protein intake is by far the most common cause of growth failure. Protein energy malnutrition is frequently characterized by elevated basal serum GH concentration. In generalized malnutrition i.e. marasmus, GH levels may be near normal or even lower. Under-nutrition could have an effect on GH signal at multiple points on the pathway and cause a state of GH resistance. In animal and cell-based models, caloric restriction is related to a discount in GH receptor messenger RNA transcription. Hypoglycemic agent will increase internal organ GH receptor availableness, and reduced hypoglycaemic agent concentration throughout abstinence could play a job within the reduction of GH receptor transcription. Calorie and macromolecule deficiency disease also can cause GH resistance through effects on post-receptor signal. Embryonic cell protein twenty one (FGF21) is created by adipocytes and hepatocytes, and concentrations are hyperbolic in abstinence. FGF21 reduces STAT5b phosphorylation and will increase Suppressor of protein signal two (SOCS2) expression, each of that decrease IGF-I production. FGF21 additionally will increase IGFBP-1 expression, that additional reduces IGF-I bioavailability for signal. Another potential mechanism involves Sirtuin-1, a deacetylase that mediates the metabolic response to abstinence through its effects on aldohexose and macromolecule metabolism. Sirtuin-1 additionally inhibits the amino acid phosphorylation of STAT5, and represents a further cellular mechanism of GH resistance in deficiency disease. Zn metal and vitamin B complex deficiencies may additionally be related to GH resistance and reduced IGF-I, though the mechanisms of every of those are unknown. In both the conditions, serum IGF-1 concentrations are typically low. Malnutrition is a form of GH Insufficiency (GHI) in which serum IGF-1 concentrations are reduced in presence of normal or elevated GH levels. Elevated GH levels represent an adaptive response whereby protein is spared by the lipolytic and anti-insulin actions of GH.

Reduced serum IGF-1 concentration is a mechanism by which precious calories are shifted from use in growth to survival requirements. Rare causes of IGF-1 deficiency leading to severe growth failure are hypothalamic dysfunction, pituitary GH deficiency and primary or secondary GHI. Low IGF-I concentrations is also an early sign of malabsorptive disorders, even within the absence of epithelial duct symptoms. Disorder could gift with a broad spectrum of symptoms and signs starting from symptomless delicate assimilation (called monosymptomatic celiac disease), wherever the sole symptom is growth failure to severe deficiency disease and secondary failure to thrive. Even in minimally symptomatic kids, IGF-I is under controls at identification and normalizes with a gluten-free diet in parallel to increasing BMI. In kids with established disorder, protein exposure results in a discount in current IGF-I concentration proportional to the degree of little internal organ membrane inflammation. The correlation of IGF-I with disorder activity has been replicated in several pediatric and adult studies, and IGF-I has even been advised as a further marker for watching disorder activity for this reason. Hence these patients with growth failure are evaluated by careful auxologic assessment and appropriate measures of GH-IGF axis. Establishment of deficiency of IGF-1 and IGFBP-1, IGFBP-3 then necessitates a thorough evaluation of hypothalamic-pituitary- IGF function. Hence along with all other factors regulating growth, insulin like growth factors has a crucial role in growth attenuation

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