Going beyond growth with the growth hormone deficiency (GHD) patient

**Growth hormone (GH) signaling**

GH secretion from the anterior pituitary into the blood activates GH receptors on the cell membrane of several cell types and induces insulin-like growth factor I (IGF-I) production.

**GHD**

A rare cause of growth failure that can be congenital or acquired

- Affects 1:3500 children

**Impacts on multiple systems and organs**

Is a lifelong condition

**Transition for childhood-onset GHD patients**

Transition period is defined as the time from late puberty to establishment of adult muscle and bone composition

Transition typically begins in the mid-late teens and continues for 6–7 years past adult height

**Pediatric phase (also called ‘adapting to early adolescence’)**

- Reassessment of Endocrinology and disease management
- Assessment of patient and caregiver readiness
- Establish MDT
- Discuss transfer plan with patient and family
- Increase patient self-management
- rhGH treatment
- Periodic assessment of IGF-I, clinical symptoms, BMI
- Plan first adult care appointment

**Adult phase**

- Full care by adult endocrinologist
- Plan lifelong care
- MDT involvement

**Steps in transition for childhood-onset GHD**

BMI, body mass index; MDT, multidisciplinary team; rhGH, recombinant human growth hormone

**GHD and carbohydrate metabolism**

- Reduced glucose tolerance
- Impaired insulin sensitivity
- Increased insulin resistance

**Impact of GHD on carbohydrate metabolism**

- Episodic fasting hypoglycemia
- Increased insulin sensitivity
- Increased insulin sensitivity, diminishes with age as a result of: Hyperglycemia (ex-steroid effects)
- Body composition changes disease

**GHD and the cardiovascular system**

- Increased vescular adiposity
- Decreased lean body mass
- Hypertension
- Atherosclerosis
- Premature cardiovascular morbidity and mortality

**GH and bone**

- Normal bone mineral content and density, when corrected for stature
- GH replacement has expected effect for change in size
- Increased markers of bone turnover
- Short term: BMD reduced
- Long term: Bone mass and cortical thickness increased

**Monitoring of glycemic status is not indicated for multiple systems and organs a affects production cell types and induces insulin-like growth factor I (IGF-I)**

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**Characteristics of GHD by age**

**Infancy**

- Increased appetite
- Unfavorable CVD risk
- Decreased BMI

**Childhood**

- Hypoglycemia
- Poor feeding
- Delayed milestones
- Signs of pituitary disease, eg nystagmus, mid-line defects, microopen

**Adulthood**

- Increased adiposity
- Delayed/disturbed puberty
- Failure to reach peak bone mass

**LATE ADOLESCENCE TO TRANSITION**

**Effect of GHD on bone**

- Increased BMD
- Decreased BMD
- Post-receptor GH signaling

**Skeletal growth via growth plate**

- Bone mineral and strength via osteoblast
- Short stature, growth failure
- Decreased BMD

**Oral function for both males and females**

- Reduced fertility in females

**Kidney growth and function**

- Adrenal cortical function
- Low GFR

** CNS development**

- Impaired memory, cognition, quality of life

**Body composition: Direct and indirect effects on fat and skeletal muscle**

- Decreased lean body mass
- Increased fat mass

**Cardiovascular and lipid metabolism**

- Dysglycemia
- Dyslipidemia

**Hypoglycemia
**

- Fever

**Skeletally mature**

- Height

**Peak bone mass**

- Height

**Decrease in fracture risk**

- Bone mass

**BMI, body mass index; CVD, cardiovascular disease; MDT, multidisciplinary team; rhGH, recombinant human growth hormone**

**References**

1. Lindsay R et al. Pediatr 1994;103:29-32