Hypothalamo-Hypophisis` Diseases

Docent M. P. Kapliyeva, PhD

HYPERSOMATOTROPISM

This syndrom accompanied by hyperproduction of a growth hormone (GH). Growth hormone hyperproduction leads to gigantism in a childhood and to acromegaly in after closed growth zones.

Etiology:

1) Eosinophilic adenoma of hypophisis

2) Hyperplasia of somatotrophs

PATHOGENESIS

Pathophysiological aspect:

- autonomic hyperproduction of growth hormone
- increasing of insulin growth factors (IGF)
- magnification of metabolism in metaphyses and an epiphysis of tubular bones & cartilages
- raised synthesis of a copulative tissue in all organs

PATHOGENESIS

Biochemical aspect:

- growth hormone hyperproduction raised the contrainsular effects by gluconeogenesis and responded hyperinsulinemia
- growth hormone hyperproduction exceed insulinresistancy

PATHOGENESIS

Biochemical aspect:

- increasing of proteoglycans synthesis and exceeding metabolism of proteins in copulative tissue with hyperproduction of growth hormone
- increasing of liposynthesis and lipogenesis, with hypersyntesis of cholesterol and atherogenic lipoproteids

CLINICAL SYMPTOMS OF GIGANTISM

1)exceeded growth in prepubertate age
2)skin is wet & fat. Muscle strength is reduced
3) head is disproportionaly small in comparison with the body
4) splanhomegaly
5) arterial hypertension
6) retardetion of sexual maturity in comparison with the physical development.

CLINICAL SYMPTOMS OF ACROMEGALY

Acromegaly means the growth of distant parts of the extremities (hands & feet) and facial part of the head.

Patient complains to serious headaches, paresthesias, vision disorders, pains in the distant parts of the bones, changing of face: enlarged nose & ears, thickening of eyebrow arches, upper and lower jaws.

CLINICAL SYMPTOMS OF ACROMEGALY

examination data:

1) skin is fat, wet at the expense of a hypertrophy of sebaceous & sweat glands
2) Hypodermic fat – obesity, especially abdominal one
3) changing of facial skull : augmentation of a nose, ears, eyebrows arches, jaws enlargement (comparing with previous photo)
4) Enlargement of distant parts of the extremities (hands & feet widing)

CLINICAL SYMPTOMS OF ACROMEGALY

5) thorax widing
6) arterial hypertention
7) Insufficiency of heart valves
8) Splanhomegaly
9) prediabetes or manifest diabetes mellitus
10) bitemporal hemianopsy, caused by pressure of *hiasma opticum*

ALGORITHM OF DIAGNOSTICS OF HYPERSOMATOTROPISM

1. Visualisation of Hypothalamo-Hypophisis Zone (MRI). Normal dimensions of a Turkish saddle:
   - Sagittal & vertical size – 8-12 mm
   - Horizontal – 10-15 mm.

2. bitemporal hemianopsy researching of an eyeground and fields of vision.

ALGORITHM OF DIAGNOSTICS OF HYPERSOMATOTROPISM

3. growth hormone level investigation in blood serum on a profile within day
4. the most sensitive test is the blood serum level of IGF
5. Glycaemia control
6. Ultrasound diagnostics of internal organs (splanchomegaly)
7. Blood pressure profile

PRINCIPLES OF HYPERSOMATOTROPISM TREATMENT

1. Medicamental treatment

- Somatostatin drugs – octreotide, lanreotide under the IGF & GH control
- Dofaminum agonists

2. Surgical excision of pituitary adenoma (opened trepanation or transsphenoidaly)

3. Radiotherapy (radiation γ-rays exposure)

HYPERPROLACTINEMIA

Hyperprolactinemia is a syndrom with prolactin hormone hyperproduction.

Etiology of hyperprolactinemia:

- pituitary adenoma
- primary hypothyroidism (Van Wik-Ross-Heness syndrome)
- Hypertrophia of prolactotrophes cells
- Medical preparations

PATHOGENESIS OF HYPERPROLACTINEMIA

- the excess of prolactin production leads to galactorrhea, mammaglands enlargement, dismenorrhea & amenorrhea, infertility in women
- symptoms of mammaglands enlargement and sexual disorders could be observed in a male (adolescents & adult men)
- rising of appetite and weight gain
- hirsutism and hypertrichosis

ALGORITHM OF DIAGNOSTICS OF HYPERPROLACTINEMIA

- Investigation of blood serum prolactin hormone level (micromolecular & macromolecular).
- thyroid hormones investigation to find out primary hypothyroidism
- Visualisation of Hypothalamo-Hypophisis Zone (MRI) with contrasting.
- Investigation of blood serum sexual hormones levels (if necessary)

PRINCIPLES OF TREATMENT OF HYPERPROLACTINEMIA

- cabergoline drugs as selective agonists of H_1 dophamine receptors (very effective for treatment of micromolecular hyperprolactinemia)
- Monthy control of prolactin level with titration of dose
- MRI control (one time per six monthes) to control adenoma size
- Van Wik-Ross-Heness syndrome should be treated with levothyroxine

DIABETES INSIPIDES

This disease accompanied by deficiency of antidiuretic hormone (ADH).

- **Etiology:** brain traumas, neuroinfections.
- **Pathogenesis:** ADH deficiency leads to depression of water reabsorption in a distal canaliculus of kidney nephrones that leads to polyuria.

CLINICAL SYMPTOMS OF DIABETES INSIPIDES

- complaints to polyuria and thirst (water consumption from 3-10 l/daily)
- weight loss because of dehydration
- appetite depression
- skin and mucous dry, muscular weakness
- Irritability, tachycardia
- gastroptosis, because of huge volume of water consumption.

DIAGNOSTICS OF DIABETES INSIPIDES

1. counting of balance of water consumption and diuresis
2. daily Zimnitsky test (low specific gravity 1001-1005) in six urine portions
3. Reberg assay
4. test with water reducing (differention diagnostics with a neurogenic polydipsia)
5. Ex juvantibus test with desmopressin (drug of ADH)
6. visualisation (MRI of Hypothalamo-Hypophisis Zone)
7. test blood serum ADH level

PRINCIPLES OF DIABETES INSIPIDES TREATMENT

- replaceable therapy by drugs of ADH (desmopressin & minirin tab. 0,1 mg 2-3 times per day)
- urine specific gravity control