

Adrenal Diseases

Glucocorticoid`s disfunctions. Primary and secondary hypercorticism. Aetiology, pathogenesis. Clinical symptoms, diagnostics, differential diagnostics. Modern principles of treatment.

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HYPOTHALAMIC-PITUITARY-ADRENAL AXIS

Hypothalamus
(Corticoliberin)



Adenohypophysis (ACTH)



Adrenal Cortex
(Cortizole, sexual steroids,
Particularly mineralocorticoids)

HYPERCORTICISM

- This state accompanied by excess of adrenal cortex hormones (first of all glucocorticoids & mineralocorticoids)
- Primary hypercorticism is a lesion of adrenals (Cushing-syndrom)
- Secondary (central or hypothalamic-pituitary) hypercorticism (Cushing disease)
- Ectopic ACTH-syndrome

ETIOLOGY OF HYPERCORTICISM

- **PRIMARY:**

1. Adrenal tumor (glucocorticosteroma)
2. A hyperplasia of adrenal cortex

- **SECONDARY:**

1. Pituitary tumour (a basophilous adenoma)
2. A hyperplasia of corticotrophs

- **ECTOPIC ACTH-SYNDROME:**

1. Lung cancer
2. Tumours of testicles
3. Carcinoid

PATHOGENESIS OF HYPERCORTICOIDISM

PATHOPHYSIOLOGICAL ASPECT:

- Primary hypercorticism: cortisol level is increased and ACTH level is decreased.
- At the secondary hypercorticism: ACTH level is increased and cortisol level is increased too

PATHOGENESIS OF HYPERCORTICISM

BIOCHEMICAL ASPECT:

- In an exchange of carbohydrates it is raised gluconeogenesis and a glycogenolysis (a hyperglycaemia, "steroid,, diabetes mellitus).
- Exchange of lipids metabolism leads to central obesity and exceeded synthesis of cholesterol and dislipidemia.
- Exchange of protein metabolism leads to catabolism of muscles and increasing of creatinine and urea levels in blood serum.
- Mineral metabolism – a hypernatremia and a hypopotassemia, a hypocalcemia.

CLINIC OF HYPERCORTICISM

- Complaints to fatigability, headaches, the emotional lability, the raised appetite, weightgain.
- Skin: dystrophics, with acnae vulgaris and folliculitis and plethora of the face (red cheeks). There are purple stria on the breast, abdomine, hips, in axillary recesses. Hypertrichosis.
- Central obesity with the thin extremities, "moon" face, "buffalo hump" over 7th cervical vertebra.

CLINICAL SYMPTOMS OF HYPERCORTICISM



CLINIC OF HYPERCORTICISM

- Osteal system: a system osteoporosis, compression fractures of vertebra, "the fish" vertebra, reduction of the height
- Cardiovascular system: hypervolemia, arterial hypertension, tachycardia and myocardium dystrophia.

CLINICAL SYMPTOMS OF HYPERCORTICISM

Cushing's Syndrome



red cheeks

moon face

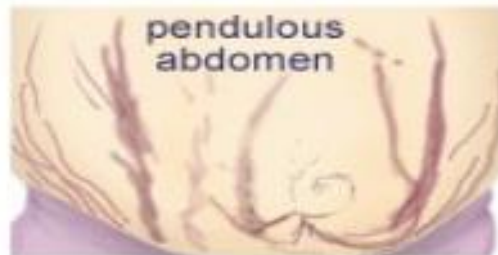
Osteoporosis;
compressed
(codfish)
vertebrae

Excessive Cortisol

fat pads
(buffalo
hump)



pendulous
abdomen



high
blood
pressure

thin
skin

thin
arms
and
legs

bruisability
ecchymoses

pendulous
abdomen

red
striae

poor
wound
healing



CLINIC OF HYPERCORTICISM

- GASTROINTESTINAL TRACT: the raised appetite, a hyperacid gastritis, "steroid" ulcers and hepatosteatoses.
- Manifest diabetes mellitus or pre-diabetes of specific type.
- Nerve system: irritability, aggressiveness, emotional lability.
- Sexual system: a dysmenorrhea, an amenorrhea at female; depression of a libido at male.

DIAGNOSTICS OF HYPERCORTICISM

1. Anamnesis & clinical examination
2. Plasma cortisol test in 8.00 and 21.00 (cortisol increasing in the morning and daily allowance violation rhythm's)
3. The daily urine excretion of free cortisol is increased
4. Low dexamethasone suppressive test (negative test - no suppression cortisol secretion; normal - decreasing cortisol by 50% or more of the basic level)

DIAGNOSTICS OF HYPERCORTICISM

- The stages of diagnostics:
 1. The identification of hypercortisolism
 2. Differential diagnosis forms of hypercorticism
 3. Establishment of localization the main pathological process

DIAGNOSTICS OF HYPERCORTICISM

1. Low dexamethasone test (daily Liddle test):

8:00 - Initially free urine cortisol and dexamethasone tab. - 0.5 mg x 4 times a day.

Next morning 8:00 - cortisol urine

2. Low dexamethasone test (night test)

8:00 – cortisol in plasma, then at 24.00 - dexamethasone tab. 1.0 mg

Next day at 8:00 – cortisol in plasma

DIAGNOSTICS OF HYPERCORTICISM

Investigation of ACTH in plasma 8:00 & 21:00 reduced
– with primary, increased - with a secondary
hypercorticism.

ACTH-ectopic syndrome: ACTH level is elevated, no
daily rhythm

DIAGNOSTICS OF HYPERCORTICISM

Big dexamethasone test:

- positive (decreasing of cortisol level more than 50% from basic level) allow to diagnose secondary hypercorticism (Cushing disease)
- negative – no significant changing of high cortisol level (Cushing syndrome).

DIAGNOSTICS OF HYPERCORTICISM

- Big dexamethasone test

8:00 – plasma cortisol investigation

24.00 - dexamethasone tab. 8.0 mg

8:00 next day - plasma cortisol

DIAGNOSTICS OF HYPERCORTICISM

Visualisation:

- Adrenal glands (computer tomography) with contrasting.
- Hypophysis (magnetic resonance imaging) with contrasting.

TREATMENT OF HYPERCORTICISM

Primary hypercorticism:

1. Surgical treatment – tumor resection or adrenalectomy (less invasive surgery using laparoscope)
2. Medicamentous treatment – steroidogenesis blockers (ketoconazole)

Doses of drugs are selected individually.

TREATMENT OF HYPERCORTICISM

The secondary hypercorticism:

1. Radiotherapy (protonotherapy, telegammatherapy).
2. Operative treatment (under indications).
3. Medicamentous treatment – Dopamine agonists.
Doses of drugs are selected individually.

TREATMENT OF HYPERCORTICISM

Symptomatic treatment:

1. Osteoporosis should be treated with calcium, VitD₃ and other.
2. Diabetes mellitus treatment.
3. Arterial hypertension treatment.
4. Dyslipidemia treatment.