STH –SECRETING PITUITARY ADENOMA. ACROMEGALY CASE WITHOUT SUCCESSFUL RESULT

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Head of department: prof. Yabluchansky M.I.
ACROMEGALY is a disorder characterized by growth hormone (somatotropin) hypersecretion, usually caused in more than 98% of cases by a pituitary adenoma.

CLASSIFICATION ACROMEGALY (STAGING):
- Pre-acromegaly
- Hypertrophic (hyperplasia of tissues)
- Tumor (increase of intracranial pressure, blindness)
- Cachectic

There is an approximate 2-fold excess mortality in acromegaly due to the presence of diabetes, hypertension, and cardiovascular, cerebrovascular, respiratory, and some malignancy-related conditions


GH hypersecretion increases insulin resistance, producing impaired glucose tolerance and diabetes mellitus in 15–38% of patients

OUR PATIENT

• Patient P.O.N., woman
• 60 y. old
• unemployed
• city resident
COMPLAINS

- constant headache,
- weakness,
- sweating,
- increased appetites,
- discomfort in the neck area,
- somnolence,
- increasing of BP till 180/110 mm Hg
- partial loss of vision
- squeezing pain in the heart area after some physical load
ANAMNESIS MORBI

- 1999 – first time diagnosed adenoma of pituitary gland, patient refused surgical treatment
- 2006 – polynodular goiter of thyroid, patient refused surgical treatment
- 2008 – diabetes mellitus II type, oral treatment taken constantly (“Oltar” (glimeperid) 3 mg/day in combination with “Diaformin” (metformin) 850mg 2 tabl/2 times a day
- Brother of the patient has thyroid pathology
- 2 delivery, 3 pregnancy
- Menopause from 45 y. old
OBJECTIVE EXAMINATION

- Conciseness - clear, state - severe, height -156 cm, weight - 85 kg, BMI - 34.5 kg/m²
- Patient can orientate himself in place, time, his personality
- Normostenic, with hypertrophied soft tissues of the facial skull with pronounced inion and frontal eminence
- Pale skin and mucosae, clean. Diastema of the teeth.
- Thyroid: diffuse size increasing, small nodulus in 3 cm diameter from the right side
- Musculoskeletal system - no pathological changes
- Chest shape: deformed, cylindrical, thickened ribs. Hypertrophic osteoarthropathy.
- Lung percussion: no clinically significant changes. BR – 16 in min
- Lung auscultation: vesicular breathing
- Borders of the heart: left border – outside of midclavicular left line on 3 cm
- Heart auscultation: rhythmic, heart tones – muffled
- Pulse – rhythmic, 80 bts/min
- BP 150 / 90 mm Hg
- Abdomen: normal size, symmetric, unpainful
- Liver: soft, no pain during palpation in right hypochondrium
- Spleen: normal. Pasternatsky symptom – negative from both sides
- Secondary sex signs – no abnormalities.
- Edemas: pitting of low extremities.
- Pulsation of peripheral vessels is decreased.
# BLOOD COUNT

<table>
<thead>
<tr>
<th></th>
<th>27/08/16</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin, g/l</td>
<td>136</td>
<td>130 – 160</td>
</tr>
<tr>
<td>Red blood cells, 1012</td>
<td>4.4</td>
<td>4.0 – 5.0</td>
</tr>
<tr>
<td>Color index of blood</td>
<td>0.9</td>
<td>0.85 – 1.15</td>
</tr>
<tr>
<td>White blood cells, 109</td>
<td>4.0</td>
<td>4 - 9</td>
</tr>
<tr>
<td>ESR, mm/h</td>
<td><strong>22</strong></td>
<td>1 -10</td>
</tr>
<tr>
<td>Bands</td>
<td>6%</td>
<td>1.06 – 6%</td>
</tr>
<tr>
<td>Segments</td>
<td><strong>40%</strong></td>
<td>47 – 72%</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>2%</td>
<td>0.5 – 5%</td>
</tr>
<tr>
<td>Monocytes</td>
<td>5%</td>
<td>0.1 – 3%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td><strong>47%</strong></td>
<td>19 – 37 %</td>
</tr>
</tbody>
</table>

**Conclusion:** lymphocytosis with increased ESR
## BIOTECHNOLOGY TEST DATA

<table>
<thead>
<tr>
<th>Test</th>
<th>Patient’s ranges,</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiolipin antigen plasmatest</td>
<td>negat</td>
<td>negat</td>
</tr>
<tr>
<td>STH, ng/ml</td>
<td>10,4</td>
<td>0 - 4</td>
</tr>
<tr>
<td>Prothrombin index</td>
<td>100%</td>
<td>90 - 105%</td>
</tr>
<tr>
<td>Fibrin</td>
<td>15 mg</td>
<td>9 – 18</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>3,3 g/l</td>
<td>2 - 4</td>
</tr>
<tr>
<td>Fibrinolytic activity</td>
<td>210 min</td>
<td>180 - 300</td>
</tr>
<tr>
<td>Serum Ca, mmol/l</td>
<td>2,3</td>
<td>2,0 – 2,6</td>
</tr>
<tr>
<td>Fasting Glucose, mmol/l</td>
<td>12,5 - 9,6</td>
<td>4,22 – 5,5</td>
</tr>
<tr>
<td>HbA1C, %</td>
<td>9</td>
<td>&lt; 5,6%</td>
</tr>
<tr>
<td>General bilirubin</td>
<td>11 mcmoll/l</td>
<td>8,5 – 20,5</td>
</tr>
<tr>
<td>AST, U/l</td>
<td>44</td>
<td>10 - 45</td>
</tr>
<tr>
<td>ALT, U/l</td>
<td>27</td>
<td>10 - 68</td>
</tr>
<tr>
<td>Creatinine, mcmol/h/ml</td>
<td>0,27</td>
<td>0,1 – 0,68</td>
</tr>
<tr>
<td>Uric acid, mcmol/l</td>
<td>332</td>
<td>150- 350</td>
</tr>
</tbody>
</table>

**Conclusion:** STH - secreting tumor, hyperglycemia, non-adequate DM treatment
# LIPID PROFILE

<table>
<thead>
<tr>
<th></th>
<th>Patient’s ranges,</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol, mmol/l</td>
<td>5,3</td>
<td>≤ 5.2</td>
</tr>
<tr>
<td>LDH - cholesterol, mmol/l</td>
<td>2,82</td>
<td>&lt; 3.5</td>
</tr>
<tr>
<td>HDH - cholesterol, mmol/l</td>
<td>1,2</td>
<td>≥ 0.9</td>
</tr>
<tr>
<td>VLDH - cholesterol, mmol/l</td>
<td>0,71</td>
<td>≤ 1.0</td>
</tr>
<tr>
<td>TAG, mmol/l</td>
<td>5,12</td>
<td>&lt; 2.3</td>
</tr>
<tr>
<td>Atherogenic coefficient</td>
<td>2,66</td>
<td>till 3.00</td>
</tr>
</tbody>
</table>

**Conclusion:** dyslipidemia
Conclusion: Sinus rhythm, left heart axis deviation, hypertrophy of LV
SCULL X-RAY

Structural separation of joints and compaction the sella turcica, the posterior wall is not differentiated, bottom expanded

Conclusion: signs of increased intracranial pressure, pituitary adenoma in sella turcica
Right lobe – V = 35 cm³, left lobe – V= 33,26 cm³. Isthmus – 1,5 cm. Hypoechogenic nodule in the right lobe – 47*35 mm, 3 hypoechogenic nodules in the left lobe – 33*22 mm, 27*32mm, 35*22mm and hyperechogenic nodule – 15 mm in diameter

Conclusion: Polynodular goiter

TSH – 2,0 mME/l (N – 0,3-4,0)
T3 free – 22,0 nmol/l (N - 10-25)
T4 general – 1,3 nmol/l (N - 1,2-2,0)

Conclusion: Euthyroid state
SPECIALISTS CONSULTATIONS:

Cardiologist: Arterial hypertension II grade, acromegalic cardiomyopathy. CHF II A, II func.class by NYHA


Oculist: partial atrophy of ocular nerves of both eyes. Bitemporal hemianopsya.
FINAL DIAGNOSIS

Main:
STH -secreting pituitary adenoma. Acromegaly, tumor stage, chiasmal syndrome, benign clinical course. Polynodular thyroid goiter, euthyroid state. Diabetes mellitus II type, decompensated (HbA1C – 9%).


Concomitant disease: Arterial hypertension II grade. Chronic heart failure IIA stage, II-nd functional class by NYHA
GH < 1.0 μg/L as a therapeutic goal, an age-normalized serum IGF-1 value
TREATMENT

- Somatuline (lantreotid) 0.04g intramuscular 1 time in 14 days
- Diabeton – MR (gliclazid) 500mg 2tabl 2 times a day
- Glucophage (metformin) 1000mg 1 tabl 2 times a day
- Berlipril (enalapril) 5 mg 2 times daily
- Aspecard (acetylsalicylic acid) 75 mg 1 time daily
- Atorvastatin 10mg 1 tabl daily
- Surgical treatment of pituitary adenoma and polynodular goiter
NEW OPPORTUNITIES IN TREATMENT

The objective of treatment in acromegaly is not primarily the normalization of GH, but rather to normalize life expectancy and quality of life.

Notable interdependences between the acromegaly, the glucose metabolism of predisposed patients and their treatment with pegvisomant were observed. Pegvisomant, GHRA has positive influence on the quality of diabetic metabolic status and it is only significant for patients under monotherapy. Support recent findings suggest that intra-portal insulin levels determine the GH receptor expression in the liver underlined by the fact that patients with concomitant diabetes mellitus, in particular those receiving insulin therapy, require higher pegvisomant doses to normalise IGF1.

SUCCESSFUL TREATMENT = REDUCED IGF1 = GOOD PROGNOSIS
THANKS FOR YOUR ATTENTION