**Topic 3. Neuroendocrinological syndroms in gynaecology.**

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| 1. Premenstrual syndrome: etiology, pathogenesis, classification, diagnosis and treatment. |
| There is cyclic appearance of a large number of symptoms during the last 7–10 days of the menstrual cycle. *This means that any cyclic symptoms in the second phase of the menstrual cycle are symptoms of the menstrual cycle.*It should fulfil the following criteria (ACOG) :  Not related to any organic lesion.  Regularly occurs during the luteal phase of each ovulatory menstrual cycle.  Symptoms must be severe enough to disturb the life style of the woman or she requires medical help.  Symptom-free period during rest of the cycle. When these symptoms disrupt daily functioning they are grouped under the name remenstrual dysphoric disorder (PMDD).the clinical picture is very diverse, but there are several forms of PMS that combine certain symptoms: emotional-affective form (neuropsychic), edematous form, cephalgic form, crisis form.**emotional-affective form (neuropsychic) -** irritability, emotionality, aggressiveness, tearfulness, depression, general weakness, drowsiness, forgetfulness, as well as increased sensitivity to sounds and/or smells, numbness of the hands, flatulence and swelling of the mammary glands.**edematous form -** swelling and soreness of the mammary glands, swelling of the face, shins,fingers, weight gain on the eve of menstruation up to 4-8 kg, bloating.**cephalgic form -** headache, dizziness, nausea,vomiting, hypersensitivity to sounds and smells.**crisis form -** panic attacks that may be accompanied by an increase in blood pressure,a violation of the heart rhythm, pain in the heart area, paresthesia, numbness of the extremities, a feeling of lack of air. The attack usually ends with frequent and prolonged urination.*The total number of symptoms* ***to verify the diagnosis*** *should be at least 5. The symptoms should be repeated in at least 2 consecutive menstrual cycles. To clarify the cyclical nature of symptoms, the patient should keep a diary of symptoms.**The scope of additional examinational methods is determined by the form of the disease.*PMS should be differentiated from mental diseases, organic brain lesions, chronic kidney and adrenal diseases, hypothyroidism, a crisis form of arterial hypertension and menstrual migraine.**Treatment.** *Symptomatic therapy*. |
| 2. Climacteric syndrome: etiology, pathogenesis, classification, clinical manifestations, diagnosis, principles of treatment, indications and contraindications to hormone replacement therapy. |
| **Climacteric syndrome** — a complex of symptoms, that complicates a transitional period between premenopause and menopause, caused by hypoestrogenemia.**clinical manifestations.** * ***early symptoms*** vasomotor and psychoemotional disorders
* ***mid-term symptoms*** urogenital symptoms
* ***late-term symptoms*** atrophic processes in the central nervous system, pathology of the musculoskeletal system, pathology of the heart and blood vessels, metabolic menopausal syndrome.

**Treatment.** • hormone replacement therapy* Symptomatic therapy
* treatment of concomitant diseases
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| 3. Post-castration syndrome: clinic, diagnosis, treatment. |
| **Postcastration syndrome** is a complex of pathological symptoms, which appear after surgical ablation of ovaries (ovariectomy). In other words, there is a *climacteric syndrome* associated with a sharp surgical shutdown of estrogen production due to the removal of the ovaries. |
| 4. Polycystic ovarian syndrome: classification, etiology, pathogenesis, clinical forms, diagnosis, therapeutic tactics. |
| **Polycystic ovarian syndrome (Stein-Leventhal Syndrome**) – clinical (functional changes of the ovary) and morphological (structural changes of the ovary) syndrome against the background of insulin resistance (**primary PCOS** ) or other diseases (**secondary PCOS**).*By other diseases, we mean*:* hypothalamic syndrome
* metabolic syndrome
* hyperprolactinemia
* Congenital adrenal hyperplasia

Anatomy of the normal and polycystic ovary 1990 NIH diagnostic criteria include both 1 and 2 – chronic anovulation, clinical and/or biochemical hyperandrogenism. 2003 Rotterdam diagnostic criteria (two out of three): oligo- or anovulation, clinical and/or biochemical hyperandrogenism, polycystic ovaries.**Сlinic.** 1. **functional changes of the ovary** (Disorder of the menstrual cycle and infertility)
2. **structural changes of the ovary (**bilaterally enlarged ovaries with a compacted capsule (sclerosed protein shell) and a cystically altered stroma (multiple follicular cysts). Changes in the follicles include cystic atresia, hypoplasia of follicular cells and hyperplasia of tec cells)
3. **clinical (**symptoms of masculinization**) and/or biochemical hyperandrogenism**
4. **Metabolic syndrome:**This appears as a tendency towards central obesity and other symptoms associated with **insulin resistance**

**Diagnosis** • General examination • gynecological examination • ultrasound of the pelvic organs • ultrasound of the adrenal glands • hormonal examination**The treatment** includes lowering of insulin resistance levels (in case of excessive body weight – weight loss), restoration of fertility, treatment of hirsutism or acne , restoration of regular menstruation, and prevention of endometrial hyperplasia and endometrial cancer. |
| 5. Adrenogenital syndrome: classification, etiology, pathogenesis, clinic, diagnosis, treatment. To study Survey methods of gynecologic patients |
| Congenital adrenal hyperplasia - a group of autosomal recessive genetic diseases caused by defects in steroidogenesis enzymes.**Etiology and pathogenesis**. Congenital, inherited by autosomal recessive type, a defect of the CYP21B gene, which encodes the formation of the enzyme 21-hydroxylase. This enzyme controls the formation of cortisol from 17-OH-progesterone. Due to the absence of this enzyme, cortisol is not formed - and ACTH is produced in the pituitary gland by the feedback mechanism.ACTH again stimulates the production of cortisol from 17-OH-progesterone, however, due to the lack of the necessary enzyme,androgens are formed instead of cortisol,mainly dihydroepiandrosterone (DHEA), which is a precursor of androstenedione and testosterone. Thus, the absence of the enzyme 21-hydroxylase leads to an increased formation of androgens. *ACTH hypersecretion----adrenal hyperstimulation----adrenal hyperplasia----hyperandrogenism----violation of foliculogenesis----secondary polycystic ovary syndrome*.**Classification**:* ***The classic form*** (simple virile form) - a form of adrenogenital syndrome caused by a defecate of the enzyme 21hydroxylase and a violation of cortisol production.-🡪 **symptoms of virilization**
* ***The salt-losing form*** - a form of adrenogenital syndrome caused by a violation of aldosterone production🡪 **symptoms of virilization+ hypovolemia, hyponatremia, hyperkalemia and acidosis. Children die during the first year of life**
* ***The hypertonic form*** - a form of adrenogenital syndrome caused by a violation of corticosterone deficiency with compensatory increase in deoxycorticosterone🡪 **symptoms of virilization+arterial hypertension with the development of cardiac and renal decompensation, sometimes stroke. Children die within the first decade.**

The more the enzyme's deficit, the earlier the disease manifests itself and the more the severity of the patient's condition.Thus. there are the following forms of adrenogenital syndrome, depending on the age of the patient:* ***Antenatal🡪*** symptoms of virilization at birth
* ***Pubertal🡪*** violation of puberty +symptoms of masculinization
* ***post-pubertal🡪*** minor deviations from puberty , the main symptoms appear in the reproductive age: miscarriage, menstrual disorders, infertility.

**Diagnosis** * General examination
* gynecological examination
* ultrasound of the pelvic organs
* ultrasound of the adrenal glands
* hormonal examination

**Treatment.** * hormone replacement therapy (glucocorticoids)
* Combined oral contraceptives with an antiandrogenic effect.
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| 6. Dispensary observation of patients with neuroendocrine syndromes in the women's clinic. |
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