Indicaţii pentru operaţii cezariene urgente au fost: hipoxie intrauterină acută a fătului - 68 (48,57%), făt macrosom - 20 (14,28%), insuficienţa forţelor de travaliului - 12 (8,57%), placenta praevia la 3 (2,1%); eclampsia la 12 (8,57%), cordon ombelical scurtat - 5 (3,57%).

Naşterile au fost complicate prin ruperea prematură a pungii amniotice la 57 (40,71%) din gravide, dintre care la 35 (25%) masa probabilă a fătului >3600g + de sex masculin, ceea ce reprezenta o indicaţie strictă pentru operaţia cezariană de urgenţă.

Prezentarea fătului: podalică - 26 (18,57%), fesieră - 106 (75,74%), mixtă 8 (5,6%).
Sexul probabil al fătului: masculin în 69 (49,28%), feminin în 71 (50,71%) cazuri.
Starea nou-născutului la naştere după scorul Apgar: asfixie de gravitate uşoară (5-6 puncte) la 25 (17,85%) din nou-născuţi; medie (5 puncte) la 6 (4,3%); gravă (4 puncte) la 3 (2,1%).
Complicaţiile intraoperatorii al operaţiei cezariene au avut loc la 7 (5%) paciente (hemoragie hipotonă), dintre care la 2 operaţia s-a soldat cu o histerectomie.

Concluzii

1. Operaţia cezariană, realizată pentru păstrarea vieţii a mamei şi a copilului, mai ales la primipare, se va face la timp, respectînd indicaţiile stricte.
2. E important de a determina corect indicaţiile pentru operaţia cezariană la gravidele cu prezentarea pelvină a fătului, mai ales când masa lui probabilă este >3600g şi este de sex masculin.
3. Prezentarea pelvină a fătului se formează mai des la femeile cu o anamneză obstetricală complicată (avorturi, procesele inflamatorii al sistemului reproductiv al femeii, cicatrici uterine ş.a.m.d.).
4. Pentru optimizarea măsurilor chirurgicale, este necesar de a lua în considerare indicaţiile recomandate pentru operaţia cezariană în prezentarea pelvină a fătului.

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POLYCYSTIC OVARIAN SYNDROME

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Summary

This article is dedicated to the particularities of etiology, pathogenesis and treatment of PCOS. The most recent knowledge indicates that abnormal insulin response to glucose stimulus is a key underlying factor in PCOS. Other etiological factors include dearangement of the sympathetic nervous control of the ovaries, estrogen dominance and elevated androgens. Some of the literature suggests a genetic susceptibility to insulin stimulation of androgen secretion, blocking follicular maturation.

Rezumat

Sindromul ovarelor polichistice (revista literaturii)

Articolul prezent este dedicat aspectelor de etiologie, patogeneză şi tratament a sindromului ovarelor polichistice. Datele recente indică faptul că răspunsul insulinic anormal la

glucoză este factorul cheie în dezvoltarea SOP. Alți factori etilogici includ perturbarea controlului nervos simpatic al ovarelor, dominanța estrogenică și nivelul mărit al androgenilor. Unele date din literatura de specialitate sugerează o susceptibilitate genetică a secreției de androgeni la stimularea insulinică, care blochează maturarea foliculară.

Introduction

Proposed criteria (European Society of Human Reproduction and Embryology); 1 May 2003; Rotterdam, the Netherlands include: Polycystic ovary syndrome is diagnosed if there are any two of the following [7]:

- Presence of polycystic ovaries on ultrasound examination
- Clinical or biochemical hyperandrogenism
- Menstrual dysfunction with anovulation

Criteria of the US National Institutes of Health for Polycystic ovary syndrome:

- Presence of menstrual abnormalities and anovulation
- Presence of clinical and/or biochemical hyperandrogenaemia
- Absence of hyperprolactinaemia or thyroid disease
- Absence of late-onset congenital adrenal hyperplasia
- Absence of Cushing's syndrome (www.medlineplus.com)

Other names for this syndrome include: Polycystic Ovary Disease (PCOD); Syndrome O; Functional Ovarian Hyperandrogenism; Hyperandrogenic Chronic Anovulation; Ovarian Dysmetabolic Syndrome; Stein-Leventhal Syndrome.

Etiology

Despite the extensive investigations, the etiology of PCOS remains poorly understood. The most recent knowledge indicates that abnormal insulin response to glucose stimulus is a key underlying factor in PCOS. Other etiological factors include derangement of the sympathetic nervous control of the ovaries, estrogen dominance and elevated androgens. Some of the literature suggests a genetic susceptibility to insulin stimulation of androgen secretion, blocking follicular maturation [1].

Pathogenesis

In the normal state, the hypothalamus secretes gonadotropin-releasing hormone (GnRH) in a pulsatile manner. The pituitary gland responds to GnRH by releasing luteinizing hormone (LH) and follicle-stimulating hormone (FSH) in a similar cycle. In the follicular phase of the menstrual cycle, LH acts primarily on the theca cells of the ovary to increase the production of androgenic precursors. Concurrently, FSH acts on the granulosa cells to promote conversion of the androgens into estrogens, particularly estradiol, which assists in follicular development. During the follicular phase, increasing levels of estradiol lead to an LH surge. In a complex interaction, the LH surge, the elevated levels of estradiol and an increase in the circulating progesterone level trigger the midcycle surge of FSH [6,10,11,12].

In polycystic ovarian syndrome, the above cycle is disturbed. Any of several possible precipitating factors may contribute to the imbalance. Evaluation of blood serum levels typically reveals elevated LH levels and normal or low FSH levels. Patients also have increased levels of free estrogen, primarily estrone and estradiol. Estrogens exert a complex feedback effect on the pituitary gland that results in the suppression of FSH secretion and the increased release of LH. Thus, the production and release of androgen precursors by ovarian theca cells is increased [3,5,7]. The peripheral conversion of androgens to estrogens, primarily estrone, strengthens the feedback effect on the pituitary gland (fig. 1).

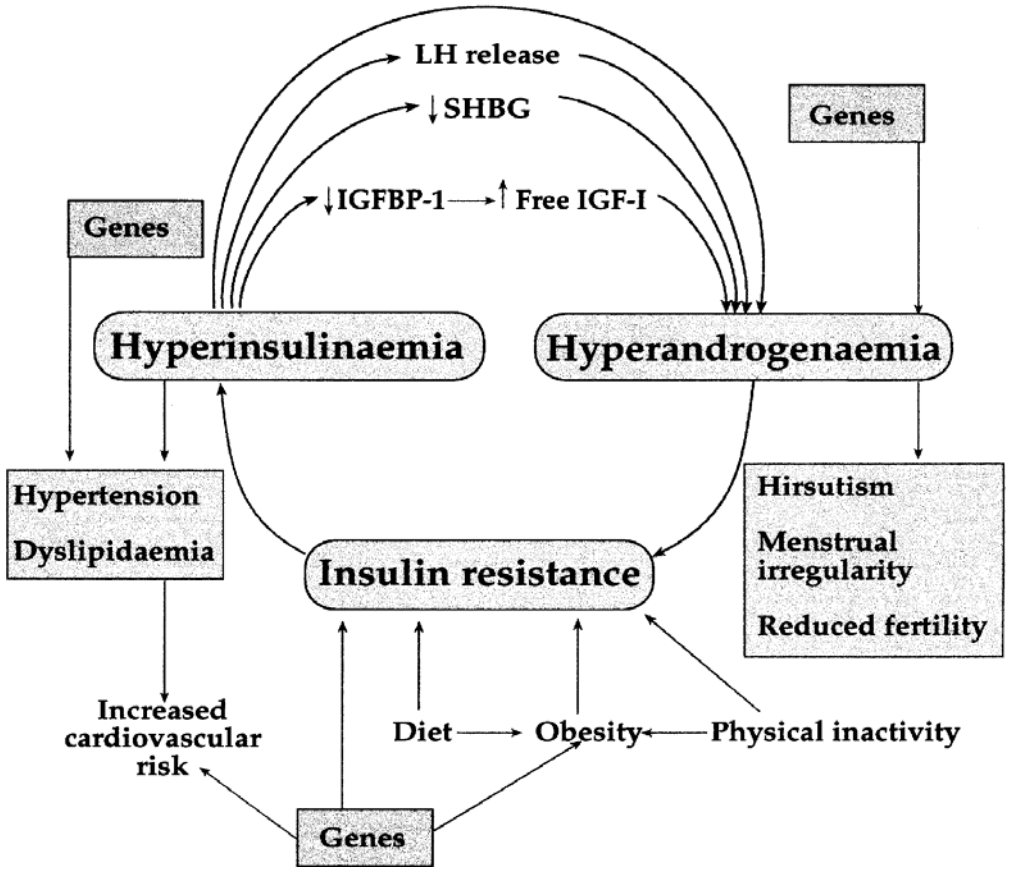

Figure 1. Relationship between androgen levels and insulin resistance in PCOS

Manifestation of PCOS. PCOS is a lifelong condition which may have effects at all ages, not just in the reproductive years. The condition may have its origins in fetal life, with either intrauterine growth retardation or post-term birth. Researchers have claimed that these children are more prone to hyperinsulinism, premature pubarche and signs of PCOS early in reproductive life.

Teenagers will often have oligo- or amenorrhoea, hirsutism, acne and weight disorders. It is controversial whether patients with PCOS suffer from a raised prevalence of bulimia.

Women seeking to become pregnant will have difficulties because of anovulation and later may be concerned about overweight and hirsutism. It is controversial whether miscarriage is increased in PCOS, or whether pregnancy loss is a result of excess body weight [14,15].

Common symptoms of PCOS include:

- Oligomenorrhea, amenorrhea: irregular, few, or absent menstrual periods.
- Infertility, generally resulting from chronic anovulation (lack of ovulation).
- Hirsutism: excessive and increased body hair, typically in a male pattern affecting face, chest and legs.
- Androgenic alopecia: male-pattern baldness.
- Acne, oily skin, seborrhoea.
- Acanthosis nigricans: dark patches of skin, tan to dark brown or black, a sign of insulin resistance, which is associated with PCOS.
- Acrochordons (skin tags): tiny flaps of skin.
- Prolonged periods of PMS-like symptoms (bloating, mood swings, pelvic pain, backaches).
- Depression

Mild symptoms of hyperandrogenism, such as acne or hyperseborrhea, are frequent in adolescent girls and are often associated with irregular menstrual cycles. In most instances, these

symptoms are transient and only reflect the immaturity of the hypothalamic-pituitary-ovary axis during the first years following menarche [3].

It is important to know that PCOS can present in any age. Many can be diagnosed as young children, some might not present until after menopause [6,10].

Risks. Women with PCOS are at risk for the following:

- Endometrial hyperplasia and endometrial cancer are possible, due to over accumulation of uterine lining, and also lack of progesterone resulting in prolonged stimulation of uterine cells by estrogen. It is however unclear if this risk is directly due to the syndrome or from the associated obesity, hyperinsulinemia, and hyperandrogenism.
- Insulin resistance/Type II diabetes
- High blood pressure
- Dyslipidemia (disorders of lipid metabolism — cholesterol and triglycerides)
- Cardiovascular disease
- Strokes
- Weight gain
- Miscarriage [6,10,11].

Diagnosis. Not all women with PCOS have polycystic ovaries (PCO), nor do all women with ovarian cysts have PCOS; although a pelvic ultrasound is a major diagnostic tool, it is not the only one. Diagnosis can be difficult, particularly because of the wide range of symptoms and the variability in presentation (which is why this disorder is characterized as a syndrome rather than a disease).

Standard diagnostic assessments:

- History-taking, specifically for menstrual pattern, obesity, hirsutism, and the absence of breast discharge. A clinical prediction rule found that these four questions) can diagnose PCOS with a sensitivity of 77.1% (95% CI 62.7%–88.0%) and a specificity of 93.8% (95% CI 82.8%–98.7%) [4].
- Gynaecologic ultrasonography, specifically looking for ovarian cysts. These are believed to be the result of failed ovulation, reflecting the infrequent or absent menstruation that is typical of the condition. In normal menstruation, eggs are released from follicles - essentially cysts that burst to release the egg. One dominant follicle emerges with each menstrual cycle, and after ovulation the follicle remnant shrinks and disappears. In PCOS, failure of ovulation means that the follicles remain in the ovaries for many months. There may be 10 or more in each ovary, and on ultrasound examination they may give the appearance of a 'string of pearls'. The numerous follicles mean that the ovaries are generally 1.5 to 3 times larger than normal [2, 9,13].
- Laparoscopic examination may reveal a thickened, smooth, pearl-white outer surface of the ovary. This would usually be an incidental finding if laparoscopy were performed for some other reason, as it would not be routine to examine the ovaries in this way to confirm a diagnosis of PCOS [12,14].
- Elevated serum levels of androgens, including dehydroepiandrosterone sulfate (DHEAS) and testosterone: free testosterone is more sensitive than total; free androgen index is often used as a substitute.
- Some other blood tests are suggestive but not diagnostic. The ratio of LH (Luteinizing hormone) to FSH (Follicle stimulating hormone) is greater than 1:1, as tested on Day 3 of the menstrual cycle. The pattern is not very specific and was present in less than 50% in one study. There are often low levels of sex hormone binding globulin.

Common assessments for associated conditions or risks:

- Fasting biochemical screen and lipid profile
- 2-hour oral glucose tolerance test (GTT) in patients with risk factors (obesity, family history, history of gestational diabetes) and may indicate impaired glucose tolerance (insulin resistance) in 15-30% of women with PCOS. Frank diabetes can be seen in 65–68% of women with this condition. Insulin resistance can be observed in both normal weight and overweight patients [8].

Differential diagnosis. Other causes of irregular or absent menstruation and hirsutism, such as congenital adrenal hyperplasia, Cushing's syndrome, hyperprolactinemia, androgen secreting neoplasms, and other pituitary or adrenal disorders, should be investigated. PCOS has been reported in other insulin resistant situations such as acromegaly [8].

Treatment. Medical treatment of PCOS is tailored to the patient's goals. Broadly, these may be considered under three categories:

- Restoration of fertility
- Treatment of hirsutism or acne
- Restoration of regular menstruation, and prevention of endometrial hyperplasia and endometrial cancer

Treatment of infertility. Not all women with PCOS have difficulty becoming pregnant. For those who do, clomiphene citrate and metformin are the principal treatments used to help infertility. The major complication of clomiphene was multiple pregnancies. The overall success rates for live birth remained disappointing, even in women receiving combined therapy. Though surgery is usually the treatment option of last resort, the polycystic ovaries can be treated with surgical procedures such as laparoscopy electrocauterization or laser cauterization; ovarian wedge resection (rarely done now because it is more invasive and has a 30% risk of adhesions, sometimes very severe, which can impair fertility) and ovarian drilling

Treatment of hirsutism and acne. When appropriate (e.g. in women of child-bearing age who require contraception), a standard contraceptive pill may be effective in reducing hirsutism. The most common choice of contraceptive pill is one that contains cyproterone acetate. Cyproterone acetate is a progestogen with anti-androgen effects that blocks the action of male hormones that are believed to contribute to acne and the growth of unwanted facial and body hair.

Treatment of menstrual irregularity, prevention of endometrial hyperplasia. If fertility is not the primary aim, then menstruation can usually be regularised with a contraceptive pill. The purpose of regularising menstruation is essentially for the woman's convenience, and perhaps her sense of wellbeing; there is no medical requirement for regular periods, so long as they occur sufficiently often. Most brands of contraceptive pill result in a withdrawal bleed every 28 days. Dianette® (a contraceptive pill containing cyproterone acetate) is also beneficial for hirsutism, and is therefore often prescribed in PCOS.

If a regular menstrual cycle is not desired, then therapy for an irregular cycle is not necessarily required - most experts consider that if a menstrual bleed occurs at least every three months, then the endometrium is being shed sufficiently often to prevent an increased risk of endometrial abnormalities or cancer. If menstruation occurs less often or not at all, some form of progestogen replacement is recommended. Some women prefer a uterine progestogen implant such as the Mirena® coil, which provides simultaneous contraception and endometrial protection for years, though often with unpredictable minor bleeding. An alternative is oral progestogen taken at intervals (e.g. every three months) to induce a predictable menstrual bleed [14].

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