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体外受精对多囊卵巢综合征不同表型的有效性

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内分泌不孕症的结构中, 多囊卵巢综合征占主导地位。文章讨论了多囊卵巢综合征表型的内分泌和代谢特征, 关于使用辅助生殖技术的有效性和并发症的现代观念, 具体取决于具体的表型。多囊卵巢综合征对辅助生殖技术方法选择的影响, 以及体外受精过程中可能出现的并发症, 妊娠发病过程的特点等问题仍未解决。考虑到荷尔蒙特征的差异和多囊卵巢综合征每种表型的代谢紊乱特征, 该方法的个体化似乎很有希望, 这可能会进一步提高体外受精的效率并降低并发症的发生率。

关键词: 多囊卵巢综合征; 表型; 胰岛素抵抗; 高雄激素血症; 肥胖; 妊娠糖尿病; 流产; 体外受精; 卵巢过度刺激综合征; 效率。

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IVF efficiency in different phenotypes of polycystic ovary syndrome

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Polycystic ovary syndrome occupies a leading place in the structure of endocrine infertility. This article presents the endocrine and metabolic features of the polycystic ovary syndrome phenotypes, as well as modern concepts of efficiency and complications of the use of assisted reproductive technologies, depending on the specific phenotype. The issues of polycystic ovary syndrome influence on selecting the method of assisted reproductive technologies, as well as possible complications that occur during *in vitro* fertilization and the features of the pregnancy course remain unresolved. The individualization of the approach seems to be promising when taking into account the differences in the hormonal profile and the features of metabolic disorders in each polycystic ovary syndrome phenotype. That may allow us to take one more step towards improving the effectiveness of *in vitro* fertilization and reducing the frequency of complications in patients with polycystic ovary syndrome.

Keywords: polycystic ovary syndrome; phenotype; insulin resistance; hyperandrogenemia; obesity; gestational diabetes mellitus; miscarriage; *in vitro* fertilization; ovarian hyperstimulation syndrome; efficiency.

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Эффективность экстракорпорального оплодотворения при различных фенотипах синдрома поликистозных яичников

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В структуре эндокринного бесплодия синдром поликистозных яичников занимает лидирующее место. В статье рассмотрены эндокринные и метаболические особенности фенотипов синдрома поликистозных яичников, современные представления об эффективности и осложнениях применения вспомогательных репродуктивных технологий в зависимости от конкретного фенотипа. Вопросы влияния синдрома поликистозных яичников на выбор метода вспомогательных репродуктивных технологий, а также возможных осложнений, возникающих в ходе процедуры экстракорпорального оплодотворения, особенностей течения наступившей беременности остаются нерешенными. Перспективным видится индивидуализация подхода с учетом различий в гормональном профиле и особенностях метаболических нарушений при каждом фенотипе синдрома поликистозных яичников, что, возможно, позволит сделать еще один шаг на пути к повышению эффективности экстракорпорального оплодотворения и снижению частоты осложнений.

Ключевые слова: синдром поликистозных яичников; фенотип; инсулинорезистентность; гиперандрогенемия; ожирение; гестационный сахарный диабет; невынашивание беременности; экстракорпоральное оплодотворение; синдром гиперстимуляции яичников; эффективность.

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多囊卵巢综合征(polycystic ovary syndrome, PCOS)是一种广泛存在的多基因多因素疾病,具有多种特征性症状,表现为生殖功能受损和代谢紊乱[1]。

今天PCOS是育龄妇女中最常见的妇科疾病,可导致内分泌不育[2]。PCOS的发病率差异很大—从4%到13%[3,4]。2016年研究的系统评价和元分析结果发表在科学门户Pubmed和OvidSP上。对几千名妇女进行了调查。根据美国国立卫生研究院(NIH)、鹿特丹共识和AE-PCOS协会的诊断标准,总体PCOS患病率(95% CI)为6% (5-8%, $n=18$ 项研究)、10%(8-13%, $n=15$ 研究)和分别为10% (7-13%, $n=15$ 项研究)。根据鹿特丹共识的标准,PCOS的发生率可达21%[5, 6]。

PCOS的主要临床表现是雄激素过多症(HA)、少排卵和无排卵、以及超声检查中的多囊卵巢形态(美国)(ESHRE, 2018年)。PCOS中,常见的病理状况包括胰岛素抵抗(IR)、2型糖尿病、缺血性心脏病、动脉粥样硬化性高脂血症、脑血管功能不全、焦虑和抑郁症、子宫内膜增生过程、卵巢癌和子宫内膜癌[1, 2, 5, 7-12]。

PCOS患者中,72.1-82.0%的病例发生GA。其在此类患者中的主要临床表现是雄激素依赖性皮肤病,通常表现为多毛症、痤疮、皮脂溢,很少表现为棘皮症和斑秃。去女性化(乳腺减少、骨骼结构改变、外生殖器官发育不全)和男性化(男性型脂肪沉积、重音、肌肉质量增加)不是PCOS的典型症状,但可能会出现在不及时纠正GA[3, 13]。Gurkan Bozdogan等人的荟萃分析中(2016),表明在PCOS中,GA的频率平均为13% (8-20%, $n=14$) ,

高胰岛素血症(GI) - 11% (8-15%, $n=9$) ,多囊卵巢形态学 - 28%(22-35%, $n=12$),寡或无排卵 - 15% (12-18%, $n=9$) [6]。

1990年,美国国立卫生研究院(NIH)召开了一次会议,会议通过了PCOS诊断的标准化标准,包括由于无排卵导致的月经不规律和HA的临床或生化体征,而没有考虑到PCOS的形态学变化。卵巢。后来在鹿特丹,来自美国生殖医学学会和欧洲人类生殖与胚胎学会(ESHRE/ASRM, 2003)的一组专家用多囊卵巢形态的超声征象补充了PCOS的诊断标准[14, 15]。

作为替代方案,雄激素过多症和PCOS协会(AE-PCOS协会, 2006年)建议考虑诊断该综合症的两个方面: GA临床或生化和卵巢功能障碍(根据超声波的寡或无排卵和/或多囊形态)。图1显示了用于诊断PCOS的标准的比较描述。

多中心随机试验证明了PCOS临床表现的多态性。事实证明,这种病理学涵盖了广泛的症状,以及它们的组合,显著超过了建立诊断的初始标准。修订了当时存在的PCOS诊断方法(NIH, 1990年)。2012年以来,ESHRE/ASRM标准(2003年)与综合征表型的强制性指示被认为是正确的。由于采用了这些补充,有可能在多囊卵巢形态和HA以及月经不规律与多囊卵巢形态相结合的女性中诊断PCOS[1, 5, 16]。在对患有PCOS的女性进行多方面评估时,鉴别诊断和排除月经不调和GA的其他病因很重要,上述所有共识也指出了这一点[16]。

PCOS的结构中,习惯上区分四种表型。表2显示了它们的比较特征,具体取决于当前的特征。

表1 多囊卵巢综合征的诊断标准

| 美国国立卫生研究院, 1990 | 鹿特丹共识, 2003 | 雄激素过多症和PCOS协会, 2006 |
|---|--|--|
| <ul style="list-style-type: none"> 慢性无排卵。 临床和/或生化高雄激素血症 | <ul style="list-style-type: none"> 寡核苷酸和/或无排卵。 雄激素过多症的临床和/或生化体征。 多囊卵巢形态 | <ul style="list-style-type: none"> 雄激素过多症的临床和/或生化体征。 卵巢功能障碍(寡核苷酸、无排卵和/或多囊卵巢形态) |
| 所有标准都是必需的 | 必须满足三个条件中的两个 | 所有标准都是必需的 |

注: PCOS是多囊卵巢综合征。

表2 多囊卵巢综合征表型的比较特征

| 表型变异 | A表型(经典) | B表型(无排卵) | C表型(排卵期) | D表型(非雄激素) |
|-------------|---------|----------|----------|-----------|
| 发生的频率 | 44-65% | 8-33% | 3-29% | 18.5-23% |
| 临床/生化高雄激素血症 | 存在 | 存在 | 存在 | 缺乏 |
| 寡核苷酸和/或无排卵 | 存在 | 存在 | 缺乏 | 存在 |
| 多囊卵巢形态 | 存在 | 缺乏 | 存在 | 存在 |

可以假设PCOS中的GA、超重和严重月经不调是代谢紊乱的独立预测因素[17, 18]。因此, 在R. Azziz (2018) 的研究指出, 月经不调的严重程度与IR的水平直接相关[1]。

研究结果表明, A和B表型的PCOS患者表现出更明显的月经不规律, 他们以GI和IR为特征, 他们患代谢综合征的频率是D表型患者的2倍[19-26]。PCOS雄激素表型女性的体重过重更为典型: 经典表型占53.1%, 无排卵表型占33.3%, 非雄激素表型占30.0%[19, 22]。A表型患者的肥胖率达到86.0%, B表型 - 27.9%, C表型 - 46.6%, D表型 - 38.8%。A表型的患者中观察到最明显的致动脉粥样硬化性高脂血症(超过65.9%); 这种表型的特征还在于高胆固醇血症和低 α 蛋白血症[19]。一组意大利研究人员表明, 排卵表型更具有社会经济地位的PCOS患者的特征[27]。或许, 这种依赖性可以用胰岛素水平和体重指数值的差异来解释[27]。然而, 其他研究人员没有观察到不同PCOS表型的高体重指数、IR和血脂异常的发生率有任何显著差异。C.N. Wijeyaratne和合著者(2011)和A.S. Melo和合著者发现具有不同PCOS表型的女性代谢综合征的患病率没有差异[28, 29]。

具有该综合征雄激素表型的患者糖耐量减低的患病率在A表型中为62.5%, 在B表型中为55.6%, 在C表型中为20.0%。相反, 非雄激素D表型在碳水化合物代谢受损的预后方面是有利的: 在此类患者中几乎从未发现糖耐量受损, 分别观察到8%和12%的GI和IR。这些具有经典(分别为14.1、27.1、30.6%)、无排卵(分别为9.1、9.1、9.1%)和排卵(分别为14.3、28.6、35.7%)表型的指标的百分比证实了这一判断。当比较所有PCOS表型的碳水化合物代谢指标时, 没有发现显著差异, 但在雄激素表型中发现空腹时免疫反应性胰岛素水平增加28.5-34.3%, 特别是在经典表型中。D表型组中, 免疫反应性胰岛素的血液水平增加了14.3%[24, 26]。

数据显示, 与非雄激素性PCOS表型患者和健康女性相比, A表型和B表型PCOS患者发生脂肪肝变性的风险增加[30]。

评估激素谱时, 注意到A和B表型中的GA主要由于硫酸脱氢表雄酮和游离睾酮含量的增加而产生。每三分之一患者的睾酮-雌激素结合球蛋白(TEBG)水平降低, 因此所有表型的游离雄激素指数均增加。排卵C表型中与其他雄激素表型相比, 发现游离睾酮的血液水平较低。据信, 卵巢

GA会对卵子质量、胚胎质量及其发育成功产生不利影响[31]。

A. Jami1和合著者(2016)表明黄体生成素(LH)/促卵泡激素(FSH)的比率在A表型中显著高于D表型和没有PCOS的女性。与其他PCOS表型患者和健康女性相比, A表型的血清总睾酮水平较高[32]。PCOS患者血清中抗苗勒管激素(AMH)水平超过8.7纳克/毫升[33]。血液中AMG含量最高的是A表型。同时进行了研究, 对FSH、AMG、催乳素、雌二醇、双氢睾酮的血液含量无可靠差异, 17 α -羟基孕酮和孕酮在睡眠中具有厌食性表型[3, 33]。

大多数研究发现, 睡眠时非雄激素表型D的患者内分泌和代谢紊乱程度较低, 分别为: 代谢综合征发病率低[26, 34, 35]。与典型的睡眠表型相比, 这些女性的LH/FSH比率几乎没有变化, 一般和自由睾酮的血液含量较低, 此外, 自由雄激素指数值较低, 睾酮雌激素结合球蛋白水平较高[32]。表型D中与其他PCOS表型相比, 倒阴漏更频繁地与规律的月经周期交替出现[36]。

总体而言, 已发表的数据表明, 超过一半的PCOS患者具有A表型, 而其他三种表型(B、C、D)几乎同样常见。表型A和B的患者约占PCOS患者总数的三分之二[37]。

了解PCOS表型的分布对于确定人群中该综合征的流行病学以及在体外受精(IVF)方案中应用个性化方法治疗和管理该综合征患者具有重要意义。其目的是降低并发症的风险并提高手术的有效性。

与PCOS相关的主要问题之一是这些患者的生育能力丧失。作为内分泌不孕的原因, PCOS占55-91%[38]。同时高达70%的PCOS病例仍未得到证实[39]。由于辅助生殖技术的快速进步, IVF是实现生殖功能最有效的方法[38]。尽管在选择方案时有多种替代方法, 但在临床实践中, PCOS患者存在许多问题, 例如未成熟的卵母细胞、卵巢过度刺激综合征(OHSS)以及产科和新生儿并发症的风险增加[40]。PCOS对IVF后妊娠并发症的频率和严重程度增加的影响仍然存在争议和争议。

试管婴儿的有效性可以通过考虑许多指标来评估: 抽吸卵母细胞的数量和质量(成熟度)、获得的胚胎数量、怀孕和早孕的频率、自然流产的频率。

刺激患有PCOS的女性的卵巢具有挑战性, 因为卵巢往往反应过度。许多系统评价和荟萃分析表

明PCOS患者发生OHSS的风险增加。已经确定了轻度与重度形式的OHSS[41, 42]。假设这种并发症与选择的IVF协议和PCOS表型有关[43]。已经根据PCOS表型描述了不同的卵巢反应:表A型与发生OHSS的较高风险相关, B表型与发生这种并发症的风险较低[42]。在T. Sha和合著者(2019)已经表明, 当新鲜胚胎被移植到PCOS患者的子宫腔中时, OHSS会更频繁地发生[38]。

L. S. Tselkovich和合著者(2017)揭示代谢综合征对PCOS中IVF结果的负面影响。因此, 从没有代谢紊乱的PCOS女性身上获得的优质胚胎总数为72.5%, 在存在代谢紊乱的情况下, 其分别降至61.4%、50.7和30.0%适合冷冻保存[43]。V. Cela和合著者研究(2017)转移胚胎和冷冻胚胎的数量和质量没有发现统计学上的显著差异。然而, 在A表型的PCOS患者中获得并冷冻的胚胎数量略多[42]。以多囊卵和合著者巢形态为特征的表型组中与表B型相比, 着床、生化和临床妊娠的发生率最高[44]。使用促性腺激素释放激素激动剂作为排卵触发剂的刺激周期中成熟MII卵母细胞的百分比为65.0%, 而当使用人绒毛膜促性腺激素时, 该指标不超过49.9%[42]。PCOS中存在代谢紊乱的情况下, IVF后的住院频率增加了三倍, 达到70.6%的病例[43]。

PCOS患者和非PCOS患者在辅助生殖技术计划中的妊娠发生率没有差异[38]。与此同时无代谢紊乱的PCOS女性的妊娠率(50.7%)明显高于患有此类疾病的女性(33.3%) [43]。发现表B型的存在与胚胎移植后未怀孕之间存在相关性[42]。与其他原因的不孕症相比, 患有这种综合征的患者的妊娠发生率更高[38]。然而, M. De Vos和合著者(2018)观察到与表D型(33.7%)相比, 高雄激素A表型和C分别为16.7%和18.5%)的妊娠率较低[45]。PCOS合并输卵管不孕因素的自然流产发生率达到64.7%, 而没有这一因素的健康女性与对照组无显著差异[38]。PCOS包括代谢紊乱是34.6%的流产原因, 在没有代谢紊乱的情况下不超过5.9%[43]。健康女性和患有上述综合征的女性的异位妊娠频率没有差异[38]。

PCOS患者的峡部宫颈功能不全和早产的发生率高达28%。这种结果的可能致病原因之一可能是支持慢性炎症的促炎细胞因子(肿瘤坏死因子- α 、白细胞介素-1和-6)含量增加。分之一的流产病例发生在妊娠中期[40]。

早期研究表明, 多囊卵巢转化、IR、GA可能是妊娠期糖尿病、先兆子痫和妊娠期动脉高血压的潜在原因[40]。根据2019年元分析结果T. Sha和合著

者在患有PCOS的女性中, TEGS浓度低与妊娠糖尿病风险增加有关[38]。根据L. E. Kjerulff和合著者结合球蛋白-1的低浓度胰岛素样生长因子可导致动脉高血压的发生[46]。

2015年队列回顾性研究的作者比较了产科并发症的患病率, 包括妊娠糖尿病、妊娠动脉高血压、蛋白尿、诊断为PCOS的女性胎儿生长迟缓综合征、孤立的多囊形态, 以及接受IVF的其他不孕症患者协议。各组间无统计学差异[47]。

颗粒细胞产生的AMH是卵巢功能储备的标志物, 也称为小生长卵泡池, 最终决定IVF获得的卵母细胞/胚胎的数量和质量。AMH是卵巢对受控卵巢刺激反应的可靠标志物。血清中AMH的基础水平与窦卵泡计数的超声数据密切相关[48]。A经典表型的特点不仅是雌激素水平较高, 卵泡数量8-12毫米, 而且AMH水平较高—根据一些资料, A型患者血液中AMH水平与B表型的患者相比, 增加了三倍。与患者的年龄和体重指数相比, 血清AMH浓度是更可靠的预测卵巢反应和发生OHSS可能性的标志物[49]。因此, 高AMH水平的先验知识允许制定降低OHSS风险的策略。与此同时, G. Bozdogan合著者(2019)证明AMH测定在诊断三种PCOS表型(B、C、D)中的重要性较低, 但经典A表型除外[53]。

结果表明甲状腺素通过多种机制促进排卵恢复[50]。这些包括增加胰岛素敏感性、直接抑制产生雄激素的卵巢酶以及血管内皮生长因子的分泌减少, 后者在OHSS的病理生理学中起关键作用[51]。接受IVF和二甲双胍治疗不孕症的PCOS女性组中, 妊娠频率增加, OHSS发生率降低, 同时卵子数量无显著差异获得的刺激天数或取消周期的频率, 以及怀孕、流产的频率[51]。

2016年, 一组美国研究人员充分详细地描述了以前未选择的PCOS样表型, 其特征是AMH水平高, 但睾酮水平异常低。这种病理影响没有肥胖的年轻女性与低水平的硫酸脱氢表雄酮有关。这种情况下, 低睾酮很可能是肾上腺来源的, 并且是由于自身免疫性肾上腺功能不全, 因为它伴随着低皮质醇水平。作者指出, 在这些患者中添加脱氢表雄酮可使雄激素水平正常化并改善IVF周期的结果[33, 52]。

结论

迄今为止PCOS的问题在文献中被广泛覆盖: 提出了许多工作, 其中考虑了病理学的发病机制、临床表现、诊断和治疗等问题。迄今为止正在进行的研究可能会扩大PCOS的表型范围。

然而,对我们综述中提供的文献数据的分析使我们得出结论,迄今为止发表的大多数关于PCOS患者生育障碍治疗特征的著作,特别是辅助生殖技术的使用,显示出相互矛盾的结果,这表明对问题的了解程度不足,研究数量较少。

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