

• 科研论著 •

产前抑郁对子代早期脑发育及 1 岁时神经行为发育的影响

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摘要: **目的** 探究孕母产前抑郁 (AD) 对子代早期脑发育及 1 岁时神经行为发育的影响, 为促进母婴健康提供科学依据。 **方法** 前瞻性选取 2022 年 1—12 月在广州中医药大学第一附属医院产科门诊进行早孕建册 (孕周 < 13 周) 的 821 例孕母, 于孕晚期 (≥ 28 周) 诊断为 AD 的孕母子代 124 例 (15.1%) 进行出院后追踪随访, 排除 23 例 (18.5%) 失访或未定期完成相关评估者, 最终纳入 101 例 AD 孕母子代作为 AD 组, 并随机选取同期健康产检孕母子代 101 例作为对照组, 观察并比较两组新生儿生后 24~48h 脑结构超声测量值及随访 1 岁时神经行为发育状况。 **结果** AD 组胼胝体长度、小脑蚓部高度和小脑蚓部前后径测量值低于对照组, 差异有统计学意义 ($t=6.901, 7.548, 2.320, P<0.05$)。AD 组 1 岁时适应性、语言和个人社交发育落后于对照组, 差异有统计学意义 ($t=2.147, 1.016, 4.246, P<0.05$)。在校正混杂因素后发现, 孕母存在 AD 的子代语言 ($OR=2.124, 95\%CI: 1.624 \sim 7.264$) 和个人社交 ($OR=2.016, 95\%CI: 2.014 \sim 6.998$) 发育异常的风险增高 ($P<0.05$)。 **结论** 孕母患 AD 与子代大脑早期微观结构改变有关, 并可导致子代 1 岁时神经行为发育异常风险增高, 临床需加强孕母心理健康评估, 并重视其子代出院后随访管理, 促进母婴健康。

关键词: 产前抑郁; 脑结构; 神经行为发育; 子代

中图分类号: R179 文献标识码: A 文章编号: 1008-6579(2025)06-0603-05 doi: 10.11852/zgetbjzz2024-1340

Effects of antenatal depression on early brain structure and neurobehavioral development of infants at 1 year of age

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Abstract: **Objective** To investigate the impact of antenatal depression (AD) in pregnant women on early brain development in offspring and neurobehavioral development at 1 year of age, in order to provide scientific basis for promoting maternal and child health. **Methods** A prospective study was conducted on 821 pregnant women who registered for early pregnancy care (gestational age < 13 weeks) at the Obstetrics Clinic of the First Affiliated Hospital of Guangzhou University of Chinese Medicine between January and December 2022. Among them, 124 (15.1%) were diagnosed with AD in late pregnancy (≥ 28 weeks). After excluding 23 cases (18.5%) due to loss to follow-up or incomplete assessments, 101 offspring of AD mothers were included in the AD group. Additionally, 101 offspring of healthy pregnant women from the same period were randomly selected as the control group. Brain structure ultrasound measurements were taken within 24 - 48 hours after birth, and neurobehavioral development was assessed at 1 year of age. **Results** The AD group showed significantly lower measurements in corpus callosum length, cerebellar vermis height, and anteroposterior diameter of the cerebellar vermis compared to the control group ($t=6.901, 7.548, 2.320, P<0.05$). At 1 year of age, the AD group exhibited delays in adaptive behavior, language, and personal-social development compared to the control group ($t=2.147, 1.016, 4.246, P<0.05$). After adjusting for confounding factors, offspring of mothers with AD had an increased risk of language ($OR=2.124, 95\%CI: 1.624 - 7.264$) and personal-social ($OR=2.016, 95\%CI: 2.014 - 6.998$) developmental abnormalities ($P<0.05$). **Conclusions** Maternal AD is associated with early microstructural changes in offspring brain development and increases the risk of neurobehavioral abnormalities at 1 year of age. Clinically, it is essential to strengthen mental health assessments in pregnant women and enhance postnatal follow-up management to promote maternal and child health.

Keywords: prenatal depression; brain structure; neurobehavioral development; offspring

基金项目: 广东省医学科研基金 (A2020616)

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围生期抑郁 (perinatal depression, PND) 是指孕母从妊娠开始到产后 4 周内发生的心理疾病, 包括产前抑郁和产后抑郁^[1]。其中, 产前抑郁 (antenatal depression, AD) 是指女性在孕期因对胎儿健康和分娩过程的过度担忧或恐惧, 无法适应孕期变化而产生的良性精神疾病综合征, 以沮丧、悲观和焦虑为主要表现, 伴有社会功能丧失^[1-2], 通常在妊娠早期和妊娠晚期较为明显^[3]。近年来, AD 发生形势严峻, 全球总检出率高达 20.7%^[4], 我国 AD 患病率为 19.7%^[5]。随着健康与疾病发育起源理论的提出, 越来越多的研究显示, AD 不仅会影响胎儿宫内发育和妊娠结局, 还会引发子代的内化和外化障碍等一系列健康问题^[6]。目前, AD 对子代早期脑结构及神经系统发育影响的研究不多, 其相关性尚不明确。因此, 本研究对诊断 AD 的母亲子代进行出院后随访观察, 探讨孕母 AD 对子代早期脑结构及神经行为发育的影响, 旨在为改善其神经发育预后提供理论依据。

1 对象和方法

1.1 研究对象 前瞻性选取 2022 年 1—12 月期间在广州中医药大学第一附属医院产科门诊进行早孕建册 (孕周 < 13 周) 的 821 例孕母开展孕晚期 (≥ 28 周) 抑郁情绪问卷调查, 对诊断为 AD 的孕母子代 124 例 (15.1%) 进行追踪随访, 排除 23 例 (18.5%) 失访或未定期完成相关评估者, 最终纳入 101 例患 AD 的孕母子代作为研究对象, 并随机选取同期健康孕母子代 101 例为对照组。入选标准: 1) 孕母产前诊断 AD; 2) 子代生后无严重合并症及并发症; 3) 出院后定期在本院儿童保健门诊随访满 1 岁。排除标准: 1) 严重先天畸形或脑发育不良; 2) 有家族性遗传代谢性疾病或精神病家族史; 3) 中途失访或随访资料不全。研究已经过监护人知情同意及本院伦理委员会审核批准 (TCMF1-2021010)。

1.2 方法

1.2.1 AD 诊断标准 在产科门诊采用抑郁自评量表 (Self-Rating Depression Scale, SDS) 于孕晚期对孕母进行抑郁症状评估, 内含 20 个反映抑郁主观感受的项目, 每个项目按症状出现的频率分为 4 级评分, 其中 10 个为正向评分, 10 个为反向评分。综合评分 ≥ 53 分考虑存在 AD 状态, 分值越高说明抑郁症状越严重^[7]。若得到阳性结果, 由心理专科医生对个体进行全面的临床晤谈, 以最终确诊 AD。

1.2.2 脑结构发育检测 新生儿生后 24~48h 行头颅彩色多普勒超声检查, 于正中矢状切面观察胼胝体形态正常、无缺失后, 于正中矢状切面测量胼胝体长度、胼胝体-顶点长度、小脑蚓部高度、小脑蚓部前后径, 均测量 3 次, 取其平均值。

1.2.3 神经行为发育评估 AD 孕母子代在 1 岁时使用 Gesell 发育量表进行神经行为发育评估, 包括适应性、大运动、精细运动、语言和个人社交 5 个维度, 每个维度最终评分以发育商 (development quotient, DQ) 表示。DQ ≥ 85 分为神经发育正常, DQ < 85 分为神经发育异常。

1.3 统计学方法 应用 SPSS 22.0 统计软件对数据进行录入与分析。以 Shapiro-Wilk 检验计量资料的正态性, 符合正态分布的计量资料以 $\bar{x} \pm s$ 表示, 组间比较采用 t 检验; 计数资料以例数 (%) 表示, 组间比较采用 χ^2 检验。采用多因素 Logistic 回归分析孕母 AD 对子代 1 岁时神经行为发育的影响, 校正相关混杂因素的影响。P < 0.05 为差异有统计学意义。

2 结果

2.1 一般情况 两组间孕母年龄、剖宫产、胎龄、出生体重、出生头围、1min Apgar 评分、小于胎龄儿的比例、孕母学历大学及以上的比例以及家庭平均月收入 ≥ 7 000 元的比例比较, 差异均无统计学意义 (P > 0.05), 两组儿童性别/女和纯母乳喂养 > 6 月的比例比较, 差异有统计学意义 (P < 0.05)。见表 1。

表 1 AD 组和对照组一般情况比较

Tab. 1 Comparison of general characteristics between AD group and control group

项目	AD 组 (n=101)	对照组 (n=101)	t/χ^2 值	P 值
孕母年龄 (岁, $\bar{x} \pm s$)	27.4 ± 3.6	26.4 ± 3.8	0.009	0.993
剖宫产 (n, %)	38 (37.6)	40 (39.6)	0.294	0.588
性别/女 (n, %)	55 (54.5)	48 (47.5)	10.124	0.035
胎龄 (分, $\bar{x} \pm s$)	40.0 ± 0.6	39.1 ± 0.5	0.607	0.356
出生体重 (kg, $\bar{x} \pm s$)	3.34 ± 0.35	3.28 ± 0.17	0.748	0.579
出生头围 (分, $\bar{x} \pm s$)	33.0 ± 1.0	33.0 ± 0.9	0.029	0.895
小于胎龄儿 (n, %)	8 (7.9)	6 (5.9)	0.198	0.360
1min Apgar (分, $\bar{x} \pm s$)	9.2 ± 0.8	9.3 ± 0.7	0.764	0.547
纯母乳喂养 > 6 月 (n, %)	47 (46.5)	65 (64.4)	13.659	< 0.001
孕母学历大学及以上 (n, %)	49 (48.5)	51 (50.5)	0.116	0.733
家庭平均月收入 ≥ 7 000 元 (n, %)	30 (29.7)	32 (31.7)	0.607	0.436

2.2 AD 组和对照组婴儿生后 24~48h 脑结构发育情况 AD 组和对照组婴儿生后 24~48h 胼胝体-顶点长度超声测量值差异无统计学意义($P>0.05$),AD 组胼胝体长度、小脑蚓部高度和小脑蚓部前后径超声测量值低于对照组,差异有统计学意义($P<0.05$)。见表 2。

表 2 AD 组和对照组生后 24~48h 脑结构超声测量值比较(mm, $\bar{x}\pm s$)

Tab. 2 Comparison of brain structure ultrasound measurements within 24 - 48h after birth between AD group and control group(mm, $\bar{x}\pm s$)

项目	AD 组	对照组	<i>t</i> 值	<i>P</i> 值
胼胝体长度	35.96±2.84	37.82±2.37	6.901	0.011
胼胝体-顶点长度	41.47±2.34	42.05±2.18	0.768	0.549
小脑蚓部高度	19.04±1.85	20.17±2.40	7.548	0.009
小脑蚓部前后径	11.78±2.14	12.51±2.06	2.320	0.048

2.3 AD 组和对照组 1 岁时神经行为发育状况 AD 组和对照组在 1 岁时大运动、精细动作 DQ 比较,差异无统计学意义($P>0.05$),AD 组适应性、语言和个人社交 DQ 落后于对照组,差异有统计学意义($P<0.05$)。见表 3。

表 3 AD 组和对照组 1 岁时神经行为发育商比较(分, $\bar{x}\pm s$)

Tab. 3 Comparison of neurobehavioral DQ between AD group and control group at 1 year of age(score, $\bar{x}\pm s$)

项目	AD 组	对照组	<i>t</i> 值	<i>P</i> 值
适应性	86±15	93±16	2.147	0.010
大运动	94±14	95±13	0.085	0.783
精细动作	90±17	91±15	0.351	0.545
语言	86±16	90±15	1.016	0.049
个人社交	85±20	93±14	4.246	<0.001

2.4 孕母 AD 对子代 1 岁时神经行为发育的影响 采用多因素 Logistic 回归分析孕母患 AD 对子代 1 岁时神经发育的影响,以神经发育 5 个能区是否正常为因变量(是=0,否=1),以孕母是否发生 AD 为自变量,在校正性别/女、纯母乳喂养>6 月等混杂因素影响后发现,孕母 AD 的子代语言和个人社交发育落后的风险增高,OR 值分别为 2.124 和 2.016 (95%CI:1.624~7.264、2.014~6.998, $P<0.05$),而适应性、大运动、精细动作和语言发育均不存在落后风险($P>0.05$)。见表 4。

3 讨 论

3.1 孕母 AD 对妊娠结局及子代发育具有重要影响 随着现代社会对孕母精神心理健康的不断重视,PND 已成为全球关注的公共卫生问题之一。PND

表 4 孕母 AD 对子代 1 岁时神经发育的多因素 Logistic 回归分析

Tab. 4 Logistic regression analysis of the effect of maternal AD on the neurobehavioral development of offspring at 1 year of age

发育能区	未校正		校正后	
	OR 值(95%CI)	<i>P</i> 值	OR 值(95%CI)	<i>P</i> 值
适应性	2.164(1.042~4.868)	0.046	1.956(0.184~4.692)	0.223
大运动	0.997(0.334~2.173)	0.896	1.165(0.315~2.253)	0.531
精细运动	3.157(0.107~5.398)	0.253	2.795(0.424~6.283)	0.674
语言	2.327(1.554~8.657)	0.011	2.124(1.624~7.264)	0.039
个人社交	2.822(1.985~5.875)	0.007	2.016(2.014~6.998)	0.025

不仅损害母婴的身心健康,也会对其家庭及社会等多方面产生危害。尽管国内外对 PND 已有大量研究,但主要集中在产后抑郁上,对于 AD 的研究相对较少,其早期识别及对母婴的危害性也未受到足够重视。研究表明,AD 与多种不良妊娠结局的发生有关,包括早产、低出生体重、新生儿出生窒息等^[8-9],并且可能对子代的脑结构及神经系统发育造成不良影响^[10-11],需加强孕母妊娠期心理健康评估,并重视患 AD 孕母子代出院后随访管理及早期干预,以促进母婴健康。

3.2 孕母 AD 与子代生后早期脑结构发育有一定关系 本研究结果显示,AD 组婴儿在生后 24~48h 超声测量胼胝体长度、小脑蚓部高度和小脑蚓部前后径低于对照组,差异有统计学意义($P<0.05$),提示 AD 与子代大脑早期微观结构改变有关。妊娠期是胎儿生命周期中对外界环境最为敏感的阶段,孕期母亲心理应激水平升高后会直接影响子宫内环境,从而干扰胎儿的正常发育编程,并可引发产后级联效应^[12]。Rifkin-Graboi 等^[13]研究使用磁共振弥散张量成像分析了 157 例孕母患 AD 与新生儿 6~14 日龄时大脑杏仁核体积及微观结构之间的关联,在校正了可能的混杂因素后发现,与正常孕母的新生儿相比,孕母存在 AD 症状的新生儿右侧杏仁核轴向扩散率(轴突完整性的指标)和各向异性分数(脑纤维结构的连贯性)均显著减少。另外一项前瞻性研究评估了 52 例孕产妇在妊娠期的抑郁症状,其子代在 2.6~5.1(3.6±0.5)岁时接受头颅核磁共振检查,在校正了潜在混杂因素后发现孕母发生 AD 与其子代右侧额叶下区和颞中皮质厚度减少有关。皮质变薄和扩散率下降是大脑成熟的过程,过早出现神经元突触修剪及髓鞘形成限制了大脑结构及功能的可塑性^[14]。此外,有研究显示,孕母 AD 与子代脑结构改变之间的关联可能存在性别差异,女童大脑早期微观结构的形成对 AD 似乎更为

敏感^[15]。

3.3 孕母 AD 对子代 1 岁时神经行为发育有一定影响 本研究结果还显示,AD 组 1 岁时语言、适应性和个人社交 DQ 落后于对照组,差异有统计学意义($P < 0.05$)。校正性别、母乳喂养等混杂影响因素后发现,与对照组比较,AD 组 1 岁时语言和个人社交发育异常的风险分别增加了 1.124 和 1.016 倍。相关研究表明,AD 影响胎儿神经连接的充分利用,与子代各年龄段的神经系统发育均具有相关性^[16]。Borchers 等^[17]研究显示,孕母 AD 可能会影响子代胼胝体的发育,进而影响其 18 月龄时社交能力和行为问题。国内一项前瞻性队列研究结果显示,孕母 AD 症状会显著增加子代 2 岁时情感反应、退缩行为、攻击行为、内化及外化行为问题的发生风险^[18]。在语言发育预后方面,O'Leary 等^[19]研究采用贝利婴幼儿发育量表第三版(BSID-III)评估孕母 AD 对子代 12 月龄时精神运动发育状况的影响,结果显示 AD 组语言发育得分显著低于对照组(87.33 ± 10.54 vs. 95.06 ± 11.78 , $P = 0.037$),与本研究结果基本一致。Deave 等^[20]研究也发现,AD 孕母子代在 18 月龄时出现语言技能落后等发育迟缓风险增高($OR = 1.5$, 95% $CI: 1.2 \sim 2.0$)。AD 对子代神经系统发育的不良影响机制涉及多个方面。其中,孕母下丘脑-垂体-肾上腺皮质轴激素(如促肾上腺皮质激素释放激素)调节异常导致子代皮质醇暴露增加在此过程中发挥关键作用^[21]。此外,代际遗传、胎盘和脐带血中基因的表现遗传学改变(如 DNA 甲基化)、神经信号通路改变、免疫炎症因子改变、肠道菌群紊乱以及抗抑郁药物的使用导致母亲和胎儿中枢神经系统的 5-羟色胺功能改变等均可能介导了 AD 子代神经发育预后不良的病理过程^[6]。

综上,孕母 AD 与子代大脑早期微观结构改变有关,并可导致其 1 岁时神经行为发育异常风险增加,临床需加强对孕母妊娠期心理健康评估,并重视 AD 子代出院后随访管理,及时有效干预,改善其神经发育预后。但本研究为单中心小样本量研究,今后仍需更大样本量的多中心研究来进一步评估 AD 对子代早期脑结构及神经行为发育的影响。

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- 收稿日期: 2024-11-05 修回日期: 2025-03-25 本文编辑: 李少闻

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- 收稿日期: 2025-05-03 修回日期: 2025-05-19 本文编辑: 李少闻