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Use of Dopamine Agonists During Pregnancy and Their Impact on Child Development: A Review of Clinical Data

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Abstract The review is dedicated to the analysis of data on the clinical and hormonal status of children born after pregnancies induced by dopamine agonists. The use of cabergoline, bromocriptine, and quinagolide in women with hyperprolactinemia and prolactinomas during pregnancy planning and management is examined. Information on perinatal outcomes, the frequency of miscarriages, pregnancy complications, and the impact of therapy on the size of pituitary adenomas is summarized. Particular attention is paid to the growth, body weight, nervous system development, and hormonal profile of newborns. Literature data indicate that with proper monitoring, the use of dopamine agonists is safe, does not increase the risk of congenital anomalies, and reduces the frequency of spontaneous miscarriages. The results emphasize the need for systematic monitoring of the mother and child for early detection and correction of possible complications.

Keywords Hyperprolactinemia, Dopamine agonists, Prolactinoma, Pregnancy

1. Introduction

Prolactin plays an important role in the regulation of reproductive function. [6] This hormone is predominantly synthesized by lactotrophs of the pituitary gland. Its secretion is inhibited by dopamine, which is produced in the hypothalamus and reaches the pituitary through the hypothalamic–hypophyseal portal system, where it binds to D2 receptors of lactotroph cells. [3]

Prolactin is synthesized not only by the pituitary gland but also locally in the ovaries, endometrium, and myometrium, where it is regulated by various factors. In addition, prolactin may participate in the carcinogenesis of reproductive organs by increasing the proliferation of endometrial cells and ovarian tumors. [6]

Prolactin is responsible for lactation, corpus luteum function, progesterone synthesis, and reproductive processes. Hyperprolactinemia is caused in about 60% of cases by prolactinomas — lactotroph pituitary adenomas. [3] It affects kisspeptin neurons, regulates ovarian steroidogenesis, and influences the menstrual cycle, which may lead to decreased libido, ovulatory disorders, and infertility in hyperprolactinemia. [6]

Infertility is a widespread problem worldwide. In developed countries, approximately one in seven couples faces infertility, while in developing countries it affects about one in four couples. One of the main risk factors is the woman's age: the probability of infertility increases significantly over time. Among women aged 15–34 years, infertility occurs in 7–9%, in those aged 35–39 years — in about one quarter, and after 40 years — in nearly one third. The highest infertility rates are observed in Eastern Europe, North Africa, and the Middle East. Overall, about 2% of women aged 20–44 years worldwide have never had a live birth, and another 11% were unable to have a second child after a previous birth. [10]

Hyperprolactinemia is detected in approximately 25–30% of couples experiencing infertility, affecting one or both partners. [15]

2. Main Part

Hyperprolactinemia is the most common endocrine manifestation of disorders of the hypothalamic–pituitary system. Hypogonadism that develops on its background is of significant scientific interest because it often occurs during reproductive age and affects metabolic processes. [1]

Prolactin reduces ovarian sensitivity to FSH and LH by blocking their receptors, which disrupts estrogen production. In addition, prolactin suppresses the pulsatile secretion of GnRH, thereby decreasing the release of FSH and LH by the pituitary gland. As a result, ovulation is impaired. [15]

Elevated prolactin levels become a serious factor preventing the normal realization of reproductive function in both men and women. Prolactin affects all components of the reproductive system and simultaneously acts as a metabolically active hormone influencing fat and carbohydrate metabolism. Because of this, the body becomes less prepared

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for pregnancy. [1]

Prolactin may also affect the cardiovascular system: it can cause vasoconstriction and is often elevated in women with arterial hypertension and preeclampsia. Its increased excretion in preeclampsia is considered a possible biomarker of this complication. [15]

Hyperprolactinemia may be physiological or pathological. Under normal conditions, it is caused by pregnancy, lactation, sleep, stress, sexual intercourse, nipple stimulation, and physical activity—in these cases treatment is not required. [8]

Pathological causes include prolactinomas, hypothalamic diseases, infiltrative processes, hypothyroidism, chest wall lesions, renal and hepatic failure, use of antidopaminergic drugs, and rare ectopic prolactin production. Macroprolactinemia is considered separately—an increase in an inactive form of the hormone without clinical symptoms. [8]

Prolactin levels can only roughly indicate the cause of hyperprolactinemia: in stress-related forms they are usually below 1000 mIU/L, while in prolactinomas they are often significantly higher. However, hormone levels alone cannot precisely determine the origin of the disease; therefore, all patients with menstrual cycle or fertility disorders require a complete evaluation regardless of prolactin concentration. Medication history must always be clarified, since many drugs, especially antipsychotics, can cause marked hyperprolactinemia (up to 5000 mIU/L or more). TSH and creatinine levels should also be measured in all patients, as hypothyroidism and renal failure may be hidden causes of elevated prolactin, and untreated hypothyroidism can mimic prolactinoma on MRI. [9]

The diagnosis is established when prolactin exceeds the normal range (20–25 ng/mL) in two tests; if the level is >100 ng/mL, one test is sufficient. Before testing, physical exertion and nipple stimulation should be avoided for at least 30 minutes. [8]

The size of the pituitary gland changes throughout life. In childhood, it grows actively, and by reproductive age it reaches approximately 10 mm in length, 5–10 mm in height, and 10–15 mm in width. In women of reproductive age, the pituitary already tends to enlarge, and during pregnancy its growth always occurs. This is associated with hyperplasia and hypertrophy of lactotrophs under the influence of estrogens and progesterone. Prolactin begins to be synthesized in the fetal pituitary from the 5th week of ontogenesis, and from the 10th week its level in fetal blood gradually increases, reaching a maximum by the end of pregnancy. In the last weeks of gestation, hormone concentration in the fetal umbilical vein is higher than in the mother. After birth, prolactin levels in the newborn rapidly decrease. It is assumed that prolactin is important for osmoregulation and lung development in the fetus. [7]

In healthy women, the pituitary enlarges during pregnancy. Since some pituitary adenomas may show similar growth, there is a risk of visual impairment, especially in women with macroadenomas or tumors located near the optic chiasm. Pituitary apoplexy is a rare acute complication of adenomas that requires monitoring and, in some cases, surgical intervention. [4]

In patients with tumor-related hyperprolactinemia, the preferred treatment is dopamine agonists. Cabergoline is recommended as the first-line drug due to its high effectiveness in normalizing prolactin levels and reducing tumor size. Quinagolide has lower safety and is therefore not recommended during pregnancy. [3]

Cabergoline and bromocriptine effectively treat prolactinomas. Cabergoline achieves prolactin normalization in 81–96% of patients and reduces tumor size in 48–83%. In giant prolactinomas, normoprolactinemia is achieved in 60.4% of cases, vision improves in 90.9% of patients, and tumor size decreases by 76.1%. The drug is well tolerated and preserves reproductive potential in women. [6]

Some pituitary tumors are associated with genetic mutations and hereditary syndromes (for example, with AIP gene mutations, where prolactinoma is detected in 10–16% of patients), as well as tumors arising within hereditary syndromes such as MEN-1. Therefore, in hyperprolactinemia it is important to consider family history, age of onset, and response to treatment. Genetic forms usually present early, occur in several generations, grow more aggressively, and respond less well to therapy. [1]

Only isolated cases of prolactinomas are associated with hereditary genetic changes, and when present the disease usually manifests at a younger age than with somatic mutations. In patients with multiple endocrine neoplasia type 1 (carriers of MEN1 gene mutations), macroprolactinomas are more aggressive and often more difficult to treat. At the same time, recent data suggest that microprolactinomas in MEN1 may be less aggressive. Since pathogenic variants of the AIP gene are extremely rare, routine genetic testing for these mutations is not recommended to avoid unnecessary examinations and costs. Children with macroprolactinoma are recommended to undergo genetic testing for germline MEN1 and AIP mutations (high-level recommendation). Prolactinoma in a child should raise suspicion for MEN1 and AIP mutations. [2]

Prolactinomas are predominantly benign pituitary adenomas originating from lactotroph cells and secreting prolactin. They account for about 50% of all pituitary adenomas in both men and women. At the age of 25–44 years, these tumors occur significantly more often in women, and the female-to-male ratio ranges from 5:1 to 10:1; however, after menopause this difference nearly disappears and approaches equality. The incidence among women is approximately three times higher than among men. In women, microprolactinomas occur much more frequently than macroforms — about 1:8, whereas in men macroadenomas predominate — about 4:1. [2]

MRI is a mandatory diagnostic method for prolactinomas in all patients with hyperprolactinemia, regardless of prolactin levels and symptoms. Pregnancy should be planned after normalization of prolactin levels and restoration of ovulation; in cases of macroprolactinoma — only after tumor reduction to less than 10 mm. [9]

Microprolactinomas (diameter <10 mm) are the most common tumor variant and only rarely progress into macroprolactinomas (≥10 mm). Giant prolactinomas (>40 mm) are detected rarely. According to studies from the past 20 years, the true prevalence of prolactinomas is higher than previously believed. [2]

Proper management of patients is essential to minimize maternal and fetal complications. Cabergoline (CBG) is considered the most preferred dopamine agonist because it is better tolerated and has higher affinity for D2 receptors compared with bromocriptine (BCR). However, due to its shorter half-life and faster elimination from the body, BCR remains the drug of choice during pregnancy. Even though dopamine agonists are usually discontinued after pregnancy confirmation, the embryo is still exposed to the drug during the critical period of organogenesis, including cases of BCR use. [4]

Pregnancy during treatment with dopamine agonists usually requires discontinuation of therapy, but in women with macroprolactinomas treatment is continued because of the high risk of tumor growth. Microprolactinomas rarely enlarge during pregnancy; therefore, periodic ophthalmologic examinations and monitoring every 3 months are sufficient. In contrast, macroprolactinomas often grow, so such patients require more frequent monitoring; sometimes dopamine agonist therapy is continued, and if symptoms develop, MRI or even surgery in the second trimester may be performed. [15]

In Australia, bromocriptine is considered fully safe during pregnancy (category A), whereas cabergoline belongs to category B, meaning no identified risks in animals but insufficient human data. [4]

Objective of this article: To evaluate the impact of dopamine agonist therapy in women with hyperprolactinemia and prolactinomas on the course of pregnancy, perinatal outcomes, and the clinical and hormonal status of their children.

3. Study

Research shows that children conceived during therapy with Parlodel do not have an increased incidence of congenital anomalies — they occur no more frequently than in the general population. [11]

Most observations indicate that the use of bromocriptine and cabergoline during pregnancy usually does not lead to an increased rate of congenital malformations: among women who continued bromocriptine, only isolated cases of anomalies were reported, and with cabergoline use, most children were also born healthy. Although dopamine agonists could theoretically affect fetal development, serious disorders were extremely rare and their association with therapy has not been proven. [4]

In a small Japanese study (46 women), two groups of patients with idiopathic hyperprolactinemia and recurrent miscarriages were compared. The study included women aged 24–40 years with idiopathic hyperprolactinemia and recurrent pregnancy loss. Half of them received bromocriptine until the 9th week of pregnancy, while the other half received no treatment. The drug showed a possible reduction in miscarriage risk, but the quality of evidence was low. Conception and live birth rates were similar in both groups. The results showed that the dopamine agonist bromocriptine may reduce the risk of miscarriage (low-quality evidence). At the same time, conception and live birth rates did not significantly differ between women who received bromocriptine and those who did not (very low-quality evidence). [5]

Numerous studies demonstrate that dopamine agonists are safe when used during preconception preparation and in early pregnancy. [15] In 2007, Rashidova E.Yu. published results of a study on the health of girls born to mothers with hyperprolactinemic hypogonadism who conceived while receiving bromocriptine. The study included 25 girls aged 14–27 years. It was found that their physical development was normal, and bromocriptine had no negative effect on postnatal development. However, such girls more often exhibited psychological maladaptation and psycho-vegetative disorders, therefore early psychological support is recommended. In addition, they belong to a risk group for developing primary hypothyroidism, which requires long-term monitoring by an endocrinologist from childhood. [10]

Some domestic studies report a higher frequency of hypertensive-hydrocephalic syndrome; however, foreign data do not confirm neuropsychiatric disorders and even note advanced development in such children (parlodel-baby). [11]

It has also been shown that maternal hormones, including prolactin, influence the formation of hormonal regulation in the child, especially pituitary function. Such children more often demonstrate enhanced compensatory responses of the body, manifested by changes in pituitary hormones and imbalance in the pituitary–thyroid and pituitary–adrenal systems, but this is adaptive rather than pathological. [11]

Bromocriptine, the most extensively studied drug, is not associated with an increased risk of congenital malformations or pregnancy termination even when more than 6000 cases were analyzed. Cabergoline has also demonstrated a frequency of spontaneous abortions and congenital anomalies comparable to the general population; long-term follow-up of children (up to 16 years) revealed no developmental disorders. Pregnancy occurring during cabergoline therapy does not require termination, since the drug does not increase risks for the mother or child. [15]

According to a study by Argentine authors, a retrospective analysis of cabergoline's effect on pregnancy course and embryo-fetal development showed that when cabergoline was used, mainly in the first trimester, no significant pregnancy complications were identified. Spontaneous abortions, preterm births, low birth weight, and neonatal anomalies occurred within expected rates for the general population. Long-term development of children, evaluated in 61 cases, also did not show a significantly increased risk: isolated cases of epilepsy and pervasive developmental disorders corresponded to population levels. [10]

It is assumed that Parlodel, as a drug acting on the central nervous system, may influence fetal nervous system and behavioral development. Some domestic studies reported more frequent neurological disorders and adaptation difficulties in children conceived during Parlodel therapy. However, foreign studies, on the contrary, demonstrated earlier neuropsychological development in such children compared with controls. Since the dopaminergic system is associated with the development of serious neurological and psychiatric disorders, its influence is considered clinically significant and requires further investigation. [11]

The use of bromocriptine during pregnancy in women with prolactinomas significantly reduces the risk of miscarriage, missed pregnancy, and threatened abortion compared with women who did not receive the drug. Particularly low rates of pregnancy loss were observed in women with macroadenomas whose treatment was continued due to high prolactin levels.

Labor in women with prolactinomas generally proceeds similarly to that in healthy women; however, premature rupture of membranes occurs more often and fetal hypoxia is slightly more frequent. The rate of emergency obstetric complications does not differ significantly, but cesarean section is performed more often, mainly electively, due to severe gestosis and the presence of adenoma.

Children are born with normal length, but birth weight in newborns of mothers with prolactinomas is lower, which is associated with gestosis and earlier delivery. In children born to mothers with prolactinomas, the average Apgar score does not differ from the control group. Birth asphyxia was noted in only three newborns and was associated with prematurity or placental insufficiency rather than directly with prolactinoma.

In women with prolactinomas, pregnancy often proceeds with complications, especially due to threatened miscarriage and nephropathy. At the same time, the severity of pregnancy complications does not depend on tumor size or prolactin level.

Taking bromocriptine in the first trimester reduces the risk of miscarriage and missed pregnancy but more often causes toxicosis.

Pregnancy and breastfeeding do not worsen the course of the disease; on the contrary, they usually lead to tumor shrinkage and decreased prolactin levels after delivery. [12]

Early studies of quinagolide did not reveal negative effects on pregnancy course or fetal development. However, later analysis of 176 pregnancies in which quinagolide therapy was continued for an average of the first 37 days recorded 24 spontaneous miscarriages, 1 ectopic pregnancy, and 1 case of intrauterine fetal death at 31 weeks. Additionally, 9 cases of congenital malformations were reported in the same study, including spina bifida, trisomy 13, Down syndrome, and foot formation defects. [14]

Hyperprolactinemia during pregnancy increases the risk of obstetric and perinatal complications, primarily chronic placental insufficiency; therefore, such women require modern prenatal diagnostic methods and timely treatment. Dopamine agonist therapy before pregnancy is effective and safe for restoring fertility. During pregnancy, patients should remain under continuous supervision by both an obstetrician and an endocrinologist. [13]

4. Conclusions

Available data allow dopamine agonists to be considered an effective means for restoring fertility and managing pregnancy in women with hyperprolactinemia. However, the long-term consequences of intrauterine exposure to these drugs on child development remain insufficiently studied. Although most published studies indicate the relative safety of dopamine agonist use during pregnancy, the question of their impact on the long-term stages of child development remains open. Small sample sizes, the retrospective nature of studies, and heterogeneity of assessment methods limit the possibility of definitive conclusions. Therefore, further accumulation of clinical data and large-scale studies are required to evaluate the physical, hormonal, and neuropsychological development of children at different age periods.

REFERENCES

[1] Dzeranova L.K., Vorotnikova S.Yu., Pigarova E.A. Evolution of pregnancy planning in hyperprolactinemia. https://doi.org/10.14341/omet13144 Vol. 21, No. 2 (2024).

[2] Petersenn S., Fleseriu M., Casanueva F.F., et al. Diagnosis and treatment of prolactin-secreting pituitary adenomas: an international consensus statement of the Pituitary Society. Nat Rev Endocrinol 19, 722–740 (2023). https://doi.org/10.1038/s41574-023-00886-5.

[3] Dedov I.I., Melnichenko G.A., Dzeranova L.K., Andreeva E.N. Clinical Guidelines "Hyperprolactinemia" (Draft). https://doi.org/10.14341/omet13002 Vol. 20, No. 2 (2023).

[4] Luger A., Broersen L.H.A. ESE Clinical Practice Guideline on functioning and nonfunctioning pituitary adenomas in pregnancy. European Journal of Endocrinology, Volume 185, Issue 3, Sep 2021, Pages G1–G33. https://doi.org/10.1530/EJE-21-0462.

[5] Chen H., Fu J., Huang W. Dopamine agonists for preventing future miscarriage in women with idiopathic hyperprolactinemia and recurrent miscarriage history. Cochrane Database of Systematic Reviews 2016, Issue 7. Art. No.: CD008883. DOI: 10.1002/14651858.CD008883.pub2.

[6] Dzeranova L.K., Vorotnikova S.Yu., Pigarova E.A. Evolution of pregnancy planning in hyperprolactinemia. https://doi.org/10.14341/omet13144 (2024).

[7] Larina A.A., Grigoryan O.R. Hyperprolactinemia and pregnancy (literature review). https://www.mediasphera.ru/issues/problemy-reproduktsii/2013/3/031025-7217201333.

[8] Coniaris C., Benadiva C. Macroprolactinemia: mini-review and updated clinical practice information. DOI: 10.1016/j.xfre.2023.05.005.

[9] Ilovaiskaya I.A. Hyperprolactinemia in obstetric and

gynecological practice. https://dx.doi.org/10.18565/aig.2017.4.149-54.

[10] Nasirova H.K., Muzaffarova D.M. Current state of the issue of hyperprolactinemic infertility and pregnancy outcomes in mothers treated with dopamine receptor agonists. Journal (2023), Vol. 2, No. 2.

[11] Sudakova O.D., Kurysheva E.V., Kurlyandskaya R.M. Health status of children born to mothers with persistent galactorrhea-amenorrhea syndrome treated with Parlodel. https://doi.org/10.14341/probl199844239-42 Vol. 44, No. 2 (1998).

[12] Kasumova A.R., Bondarenko M.V., Potin V.V. Prolactinoma and pregnancy. 2006. Research Institute of Obstetrics and Gynecology named after D.O. Ott, St. Petersburg. https://cyberleninka.ru/article/n/prolaktinoma-i-beremennost/viewer.

[13] Zhukova E.V., Melnichenko G.A. Pregnancy and childbirth in patients with hyperprolactinemic hypogonadism. 2009. https://cyberleninka.ru/article/n/beremennost-i-rody-u-bolnyh-s-giperprolaktinemicheskim-gipogonadizmom/viewer.

[14] Dzeranova L.K., Bykanova N.S., Pigarova E.A. Hyperprolactinemia and pregnancy: main achievements and unresolved issues. 2011. https://doi.org/10.14341/brh2011116-21.

[15] Adamyan L.V., Yarmolinskaya M.I., Suslova E.V. Hyperprolactinemia syndrome: from theory to practice. Journal: Problems of Reproduction. 2020; 26(2): 27–33. DOI: 10.17116/repro20202602127.