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Krukenberg tumor which cause virilization female fetus – case report

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Summary:

We present a case of a patient diagnosed in the 3rd trimester of pregnancy with a Krukenberg tumor being a metastasis of stomach cancer. Due to severe pre-eclampsia, the pregnancy was solved by Caesarean section, a female child was born with severe virilization.

Key words: Krukenberg tumor, stomach cancer, virilization of the fetus

Case report

A 35-year-old woman was hospitalized in the 29th week of pregnancy with symptoms of pre-eclampsia. In addition, the right ovary tumor of 15x21 cm was diagnosed in the ultrasound examination. Due to hypertension and increasing proteinuria, the patient was qualified for the Caesarean section. A female child with a body weight of 1160 g was born with increased virilization features of the external genital organs. Due to prematurity and the need for mechanical ventilation, the newborn was transferred to the Intensive Care Unit. Initial diagnosis of congenital pneumonia, respiratory failure, anemia, and later in the hospitalization of bronchopulmonary dysplasia and first order retinopathy. Due to the traits of virilization of the child - hypertrophic clitoris, hypertrophy of the labia outer lips and their dark color, a cytogenetic examination was carried out that confirmed the correct female karyotype (46, XX).

During the Caesarean section, a large tumor emerging from the appendages of the right one, located partially retroperitoneally, with a smooth surface and limited mobility. Due to the location of the tumor and difficulties with its mobilization, it was decided to remove changes after childbirth and ending with involution of the uterus. Due to the child's condition, the levels of androgens in the mother were measured (DHEA-S - 125 µg / dl, progesterone - 13.52 ng / ml, androstendione > 10,000 ng / ml, 17-OH progesterone -> 19.00 ng / ml, DHEA - 8.16 ng / ml, CA-125 - 66 U / ml, CA 15-3 - 24.7 U / ml, CA 19-9 - 59.0 U / ml, CEA antigen - 1.6 ng / ml, estradiol (E2) - 790 pg / ml, follitropin (FH) - <0.30 mIU / ml, luteotropin (LH) - <0.10 mIU / ml, prolactin (PRL) - 124.80 ng / ml, testosterone (TTE) - 8,598.9 ng / ml, alpha-fetoprotein (AFP) - 54.3 ng / ml).

The patient was re-hospitalized in the planned mode, right-sided adnexectomy was performed, the major network was removed and the left ovary dissected. The patient was discharged home in good general condition. The next hospitalization became necessary after obtaining the result of histopathological examination of the left ovary tumor specimen. A Krukenberg tumor was identified, which is probably a metastasis of gastric adenocarcinoma. Laparotomy was performed to remove the stomach and the inferior adnexa. In the postoperative period, symptoms of gastrointestinal obstruction appeared and the need to carry out relaparotomy, during which the hematoma from the pelvis was removed and an artificial double-barrel anus was established on the transverse. The patient in good general condition was discharged home.

Discussion

Tumor metastases to the ovary constitute from 6 to about 28% of all tumors of this organ. The starting point of the primary tumor is usually the stomach and large intestine, appendix and breast gland [1,2]. The Krukenberg tumor, which is the most frequently diagnosed metastatic tumor of the ovary, is diagnosed in a microscopic examination based on characteristic morphological features, within the stroma there is the presence of multiple signaling cells and spilled infiltration of the stroma, giving a sarcoma-like picture [1,2].

Metastatic tumors of the ovary usually occur on both sides. Same as in the patient described, the primary neoplastic disease may not show specific symptoms, and the diagnosis is made only after the surgery. Prognosis in these patients is disadvantageous due to the

dissemination of cancer and the aggressive nature of primary cancer [1,2]. Interesting in the described patient was the secretion of a significant amount of androgens through the tumor-stained ovary, which led to virilization in the fetus. Hyperandrogenism during pregnancy is extremely rare and is most often caused by the occurrence of primary gonadal tumor, metastatic tumor (Krukenberg tumor), thecalutein cysts or luteoma gravidarum as well as casuistic deficiency of placental aromas and adrenal pathology [4].

In normal pregnancy there is a significant increase in maternal plasma testosterone levels. At the same time, however, protective mechanisms are activated that reduce the risk of excessive exposure to androgens in the mother and the fetus. The activity of the placental aromatase converting the excess of androgens to estradiol increases, which is ultimately transformed in the liver of the mother into estriol and excreted in the urine. Unfortunately, in severe maternal hyperandrogenism, the above protective mechanism turns out to be insufficient and virilization of the fetus [3,4]. The hyperandrogenism states in pregnancy can also be associated with the presence of non-tumoral ovarian tumors such as thecalutein cysts or luteoma gravidarum. Their etiology is associated with stimulation by HCG, however, the literature does not provide any examples of virilization of fetuses born by mothers with the above changes in the ovaries [3,4].

In conclusion, the significantly elevated level of androgens in pregnant women leading to virilization of female fetuses is a very rare phenomenon associated with the presence of severe ovarian pathology. Precise clinical diagnosis requires in these cases a series of imaging, laboratory tests, however, only the histopathological examination of the removed tumor allows to determine exactly whether the source of excess androgens is primary or secondary ovarian cancer.

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