
INTRAUTERINE GROWTH RETARDATION: CTG, ULTRASOUND BIOMETRICS AND DOPPLER BLOOD FLOW

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Intrauterine fetal growth retardation (IUGR) is still a problem in obstetrics. The (IUGR plays an important role in the perinatal morbidity and mortality. IUGR is a multifactorial process having heterogeneous causes. A causal treatment is not yet available. At the Hamburg University Hospital the incidence of IUGR in premature babies and newborns was 19,1% over five years (1993-97). The percentage of premature babies with IUGR is 18,6%. 220 Patients were examined between January 1996 and July 1997. The overall mortality was 4,1%, the perinatal mortality was 3,6% and the neonatal mortality was 0,5%. In 1,4% of the 220 patients IUGR occurred. All cases with IUGR presented with severest IUGR associated with a weight below the 3rd percentile. $21 \pm 1,6$ cardiotocograms (CTG) per patient were registered on average. In 59,5% of the patients we detected noticeable CTG-findings (FSC7), in 40,5% CTGs were unremarkable till the delivery (FSC 8-10). Regression analysis showed that the pathological CTGs of 7 fetus with (IUGR were more often accompanied by a pH7,20 and Apgar7 in the intrapartal phase (odds-ratio 3,1, $p < 0,01$ and odds-ratio 2,4, $p < 0,05$). The time difference between the first pathological Doppler flow findings (gestation age - 33w6days) and the first borderline CTG recording (FSC 5-7 at 35w5days) was 13 days on average. In cases with pathological Doppler flow the time till a pathological CTG (FSC4) was detected 18 days on average. The time between the first detection of pathological Doppler flow findings and delivery was 21 days on average compared to 16 days on average after initial diagnosis of borderline CTGs (FSC 5-7) and 7 days on average (median 1,0 day) after initial diagnosis of pathological CTGs (FSC4). In 46 (21%) of the examined patients pathological Doppler flow findings occurred before the first noticeable CTG. In 22 (10%) of all cases with pathological Doppler flow findings no pathological CTG was recorded during the further course of pregnancy. In 21 cases (9,5%) a pathological CTG was present before the first noticeable Doppler flow findings. In 28 fetus (12,7%) GR was symmetrical, in 192 fetus (87,3%) the IUGR was detected by ultrasound biometry. In 124 pregnant women with pathological Doppler flow findings 15 fetus (12,1%) had an "enddiastolic zero flow" in the fetal aorta and 16 fetus (12,9%) in the umbilical artery. "Reverse flow" in the fetal aorta was detected in 5 (4%) fetus and in the umbilical artery was found in 4 (3,2%) fetus.

HORMONAL BACKGROUND IN WOMEN WITH SECONDARY OVARIAN SCLEROCYSTOIS IN HYPERPLASTIC PROCESSES OF ENDOMETRIA

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We have observed the hormonal background in 160 patients with endometrial hyperplasia on the background of the secondary ovarian sclerocystosis. The investigation has been carried out in the proliferative stage of the menstrual cycle. It should be mentioned, that if the disease didn't exceed 3 years, the data showed the affection of hypothalamic-hypophyseal system, which was accompanied with the increase of gonadotropic secretion, strengthening of gonadotropic secretion was due to the increase of consideration FOG (27,12 mE/ml) control (16,1) and relative insufficiency LG (11,2 mm/E/ml) control (7,9) the decrease of LG/FOG state also showed the same. Increased stimulation of ovarian function by mean of FOG was accompanied with a certain increase of estradiol concentration (91,9 ne/ml) control (59,5) in the blood plasma. Clinically these phenomena were estimated by incomplete ovulation with the deficiency of yellow bodies in the form of incomplete secretion. Long-term (more than 3 years) stimulation of ovaries by mean of FOG resulted in progressive pathological process in endometria as a result of development of anatomical changes in ovaries with the formation of expressed secondary sclerocystosis. In this case hormonal background of the patient FOG (10,1 mm/Eml), LG (11,2), estradiol 68,5 was also changed. Clinically hormonal dislocations were characterized by the development of anovulation of DOB, with recurrent hyperplastic processes in endometria.