EXTRAEMBRYONIC STRUCTURES AND PERINATAL COMPLICATIONS

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The pathology of extraembryonic structures (ES) (placental bed (PB), placenta, amniotic fluid (AF), umbilical cord (UC)) and their influence on perinatal outcomes remains poorly understood, despite of the priority role they play in maintenance of fetoplacental system (FPS) homeostasis from nidation until delivery. The intensive study of placenta and its insufficiency (PI) in 70-80th was basically dedicated to investigation of its structure and function in II-III trimesters and has not revealed structural and metabolic features of interrelations of physiological and pathological changes of ES and its influence on maternal and fetal status. In this message the results of long-term investigations of adaptational and homeostatic characteristics of ES are submitted. They had included ultrasound screening of 21840 pregnancies, based on mathematical and sonographical modelling; ascertainment of biochemical hormonal and immunological values of AF (242 samples); histomorphological analysis of PB, placenta and amniotic membranes (AM) with use of electronic microscope (118 cases), investigation of their relationship with FPS homeostasis, perspectives of pregnancy outcome prognosis and assessment of therapy effectiveness. Gross deviations in structure and function of the system PB-placenta-AF-fetus were revealed in pregnancies complicated with olygohidramnion. On the basis of the morphological analysis of PB and AM the common structural mechanisms leading to PI and reduction of AF volume were determined. Ultrasonic criteria are postulated for dynamic quantitative analysis of AF volume in normal pregnancies and those complicated with ES pathology. Signs of different forms of structural and functional insufficiency of PB, FM and UC are classified. Diagnostic and prognostic criteria are determined for assessment of ES status. The role of paraplacental exchange route in normalization of AF volume and possibility (through it) of influence on FPS homeostasis, perspective methods of PI treatment (due to ES pathology) are discussed.

NEOADJUVANT CHEMOTHERAPY WITH EPIRUBICIN / CYCLOPHOSPHAMID HIGH DOSE IN LOCALLY ADVANCED BREAST CANCER (T2-T4/NO-N2/MO)

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Objective: *The timing of chemotherapy (CHT) in relation to surgery has recently been the subject of intensive investigations The response of the primary tumor is a reliable prognostic factor and can in addition be regarded as an "in vivo" chemosensitivity test Is it possible to achieve a higher rate of breast conserving therapy (UCT) on locally advanced breast cancer by neoadjuvant treatment? The efficiency on the primary tumor and on the lymphatic nodes will be presented.*
Method: *61 patients (pts) with breast cancer primarily not suitable for BCT were treated with 3 cycles of Epirubicin (120mg/m) and Cyclophosphamid (600mg/m). To prevent serious neutropenic side effects G-CTS was applied prophylactic. 2-3 weeks after the 3rd cycle surgery was performed. Pathologic evaluation of the tissue followed.*
Results: *After neoadjuvant chemotherapy BCT could be performed in 47 pts. (76%). In 15 pts. a modified radical mastectomy could not be avoided. 458 % of the axillary lymphatic nodes that were clinically positive before treatment converted to pathological negative nodes after CHT. Tumor stage was: 2 times ypTO (3.2%), 3 times ypTis (4.9%), 2] times ypTI (34.6%), 22 times ypT2 (36%), 6 times ypT3 (9.8%), 5 times ypT4 (4.9%) and 2 times ypTx (3.2%). According to the clinical and histological results, the overall response rate (CR+PR) was calculated: RR=68,9%. Clinical complete remissions were found in 14.8% of all tumors, but complete pathological remission were observed in only 34%. 19 breast cancers (31,1%) showed only less than 20 percent decrease after CHT (Stable Disease, SD), but there were still some patients in (his group who could be treated with BCT.*
Conclusion: *Downstaging of locally advanced breast cancer is possible by neoadjuvant chemotherapy, BCT was enabled in 76%. 68.9% of the tumors responded to this therapy with a reduction of the tumor size of more than 20% l.*