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Pregnancy related diabesity and poor fetal outcome: the key role of inflammation

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Context. Over the recent 10 years the physiopathology and etiology similarities between obesity and diabetes, have led Dr. Francine Kaufman to define the notion of diabesity, as a heterogeneous metabolic dysfunction ranging from mild blood sugar intolerance to overt type 2 diabetes mellitus. The disease involves the association of abdominal obesity, dyslipidemia, high blood pressure, high serum glucose, a systemic inflammatory syndrome and hypercoagulability.

Objective. We searched the link between pregnancy diabesity phenotype and fetal outcome, in order to show that high levels of inflammation cause lipid peroxidation, endothelial dysfunction and promote insulin resistivity.

Methods and Patients. We designed an animal interventional experiment, using diet induced obese female Wistar rats, whom were submitted to dietary changes during pregnancy. We analyzed markers of inflammation from maternal serum, placental homogenates and fetal tissue homogenates (liver, pancreas, brain), and compared them between groups.

Interventions. The dietary interventions consisted in administering normal diet in one group of rats, Omega 3 fatty acids in the second, Omega 6 fatty acids in the third, Sea buckthorn berries in the fourth and continuing the high fat high sugar diet in the last one.

Main outcome measures. We analyzed lipid peroxidation markers as proinflammatory molecules, antioxidation levels and histologic aspects of the placenta and fetal organs. Fetal birth weight, adaptation, monthly weight gain, adult life weight and lifespan were assessed. A link between fetal metabolic environment alterations and adverse fetal outcome was searched.

Results. We proved that gestational diabesity causes fetal dysfunctions mediated through a chronic proinflammatory status, generating an altered fetal outcome, which is maintained during adult life.

Conclusions. The link between maternal diabesity and poor fetal outcome seems to be intermediated by inflammation, which generates lipid peroxidation, alteration of the membrane cell and endothelium, glucose intolerance and last but not least, epigenetic changes with expression in adulthood.

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