PLACENTAL MITOCHONDRIAL RESPIRATION AND MONOAMINE OXIDASE EXPRESSION ARE INCREASED IN ATYPICAL SEVERE PREECLAMPSIA: A CASE PRESENTATION

Anca M. Bînă[1], Adrian Sturza[1], Oana M. Aburel[1], Elena Bernad[2], Claudia Borza[1], Zoran L. Popa[2], Marius L. Craina[2], Danina M. Muntean[1]

[1]Department III Functional Sciences - Pathophysiology, Centre for Translational Research and Systems Medicine, "Victor Babeș" University of Medicine and Pharmacy, Timișoara, Romania
[2]Department XII Obstetrics and Gynecology, "Victor Babeș" University of Medicine and Pharmacy Timișoara, Romania

Preeclampsia (PE) is a major complication of pregnancy defined by a blood pressure increase in previously normotensive women and new-onset organ dysfunctions. One of the presentations of severe PE is the HELLP syndrome (hemolysis, elevated liver enzymes and low platelet count). Mitochondrial dysfunction and increased oxidative stress are central pathomechanisms underlying the abnormal placentation in PE but the source of reactive oxygen species (ROS) is far from being elucidated. We report here a case of a of 28 years old woman, 34 weeks of pregnancy diagnosed with atypical PE/HELLP syndrome due normal blood pressure associated with severe vomiting, hepatocytolysis, hemolysis, low platelet count, proteinuria; the fetus was delivered prematurely at 35 weeks of pregnancy, the fetal weight was normal according to the gestational age. The clinical-biological features improved after the delivery. The placental tissue was assessed after delivery for: i) mitochondrial respiratory function, ii) ROS production and iii) monoamine oxidase (MAO) enzyme expression. Mitochondrial respiration of placental mitochondria was assessed by means of high-resolution respirometry according to a protocol adapted to measure complex I and complex II-dependent respiration. Placental samples were incubated with dihydroethidium for ROS measurement in confocal microscopy. Placental MAO gene (RT-PCR) and protein (immune histochemistry) expressions were assessed. Placental mitochondria showed a significant increase in active and maximal uncoupled respiration for both complexes. Placental ROS and MAO expression were also increased. In conclusion, the atypical PE case was associated with increased placental mitochondrial respiration (compensatory?) and augmented local oxidative stress, at least partially dependent on MAO expression.

Keywords: atypical preeclampsia, placenta, mitochondria, high-resolution respirometry, monoamine oxidase