**EP02.11 Thrombophilia and pregnancy: a risk factor for placenta mediated complications?**

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**Objective**

Through this study, we aim to monitor the associations between the different types of thrombophilia and the complications that may occur during their development, and we are also attempting to review the literature on aspirin and heparin use to prevent these complications. We aim to study the influence of pregnancy – related thrombophilia in the IUGR determinism and the risk of thrombosis in these pregnant women, and the effectiveness of treatment methods in the prevention of complications.

**Patients & methods**

• A retrospective study involving 161 pregnant women with a history of complicated pregnancy: maternal thrombosis, placental vascular pathology (intrauterine growth restriction, preeclampsia, recurrent miscarriages), who were admitted to the “Cuza Voda” Maternity, between 2015 and 2017. Genetic tests were performed to identify the factor V Leiden mutations, the prothrombin gene mutation, the MTHFR C677T and A1298C genes mutation.

**Results**

• Of the 161 patients included in the study, 27 of them were diagnosed with IUGR. This group was compared to a control group – 27 women, with a normal pregnancy, without IUGR. The prevalence of maternal thrombophilia was monitored in the 2 groups. The prevalence of hereditary thrombophilia was 62% in the IUGR group and 29% in the control group. The frequency of the MTHFR gene mutation and of protein S was significantly higher in the IUGR group compared to the control group.

• The risk of thrombosis in patients with mutation in the factor V gene was 2.52 times higher than in patients who didn’t have this mutation. No statistical association was found for the patients with MTHFR gene mutation.

• Pregnant women with a family medical history of thrombosis showed a 2.17 times higher risk of thrombosis than female patients with no medical history of this type.

• Of the 161 patients included in the study, 21 of them showed thrombotic events: deep venous thrombosis, pulmonary embolism, cerebral thrombosis or myocardial infarction.

• No statistically significant difference was found between the groups with prothrombin or protein C genes mutations. There was no significant connection between factor V Leiden mutation and preeclampsia or between factor V Leiden and IUGR.

**Conclusion**

• Pregnant women with factor V Leiden thrombophilia and those with a family medical history of thrombosis have a higher risk of developing thrombosis than pregnant women who don’t have these issues. In their case, thrombosis prophylaxis is justified.

• No statistical association was found between thrombosis and the MTHFR gene mutation in the case of pregnant women with thrombophilia. This result could help reduce the excess of anticoagulant treatment in the groups of pregnant women with MTHFR gene mutation.

• The prevalence of hereditary thrombophilia was 62% in the IUGR group and 29% in the control group.

• The frequency of the MTHFR gene mutation and of protein S was significantly higher in the IUGR group compared to the control group. This result may help a more thorough investigation of the non-followed-up cases of IUGR pregnancies, with subsequent investigation of the thrombophilia profile and detection of new cases of hereditary thrombophilia, with appropriate management for a future pregnancy.

• Accurate antepartum diagnosis of IUGR cases in pregnant women with MTHFR and protein S genes mutations could improve fetal prognosis.