

Obstetrics and gynecology: Is Lipoprotein(a) involved in pre-eclampsia?

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Pre-eclampsia could affect 5-15% of pathologic pregnancies and has an obvious influence on maternal and fetal mortality. One of the known causes of this disorder is an endothelial dysfunction with a wide range of mechanisms like platelet deposition, thrombosis, infarction, atherosclerosis. The main reason could be located in lipid metabolism leading to endothelial dysfunction.

One of the interesting points in this chain is Lipoprotein(a) – Lp(a), one of acute-phase proteins present in the inflammatory process. Its genetic expression is independent from LDL and diet. Lp(a) is 90% similar to plasminogen, which could explain its competitive role in reducing clot lysis. Elevated levels of this protein were reported in patients with an atherosclerotic process as well as in obesity and/or with other lipid disturbances.

Basak et al [1] performed an interesting study among 48 mild and 43 severe pre-eclampsia patients and compared them to healthy pregnant controls. All patients were between 32 and 40 gestational weeks and were qualified to subgroups according to the American College of Obstetricians and Gynecologists guidelines. The main interest was focused on peripheral blood Lp(a) and lipids with all other clinical measurements. The subgroups were not statistically different regarding age, weight, parity, birth weight. Obviously, they differed by blood pressure (as the criteria of ACOG were responsible for subdivision) and also by platelet count, total protein, albumin and fibrinogen.

The total cholesterol and LDL-fraction were not different in healthy women and those with mild to severe pre-eclampsia. Pre-eclamptic patients had higher levels of triglycerides and VLDL and lower levels of HDL fraction. The intensity of changes was dependent on the severity of pre-eclampsia. The peripheral blood concentrations of Lp(a) were not different in all 3 subgroups. The endothelial damage is crucial in the pathogenesis of pre-eclampsia, but it could be caused by many factors in lipid metabolism. Pregnancy changes hemostasis toward fibrinolysis, but pre-eclampsia activates coagulation. In not many in-vitro studies Lp(a) was responsible for coagulation and it was hypothesized that it could also play an important role in pregnancy induced hypertension. Moreover, the typical atherosclerosis and pregnancy endothelial lesions look similar and an identical pathway was suggested. In the previously performed studies Lp(a) was elevated in pre-eclamptic patients, and a Lp(a) induced plaque on placental blood vessels was found. The question is also, whether the elevated Lp(a) formation could be accompanied by urine protein loss that occurs in pre-eclampsia. In other study no correlation of Lp(a) levels with severity of the disorder was found, in spite of the correlation with the ability of molecules to accumulate in the arterial wall.

The results from the few studies with Lp(a) in pre-eclampsia are controversial and sometimes different. They still need to be confirmed and new designed complex studies are desired, especially that the promising in vitro and less optimistic in vivo studies are inconsistent.

References

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