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PLACENTAL PATHOLOGY IN WOMEN WITH PRE-ECLAMPSIA & ECLAMPSIA

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Pre-eclampsia (PE), a multi-systemic disorder of pregnancy with a global prevalence of approximately 4.6%, and eclampsia (with a prevalence around 0.3%), both present as serious conditions in the modern practice of obstetrics. It is possible to identify significant macroscopic and microscopic differences, as well as biochemical and histological factors that differ, between placentae of women with pre-eclampsia and eclampsia in comparison to those of healthy women.

Macroscopic differences are mainly a reflection of malperfusion, presenting in a multifarious manner – as infarcts, fibrin deposition, villous free placental lakes, and retroplacental hemorrhages. Minimal infarcts, in general, were more common in PE/eclampsia placentae compared to healthy controls. The mean placental weight was significantly lower in PE/Eclampsia patients and numerous studies have shown that low placental weight was related to PE/eclampsia severity. Orabona R. et al. (2016) noted differences present between early-onset (EO-PE) and late-onset pre-eclampsia (LO-PE) placentae as well; in LO-PE, the placentae were smaller, thinner and had higher opacity while macroscopic infarcts were rarer compared to those in EO-PE.

Microscopically, decidual arteriopathy was the most consistent histological finding across all placentae in women with PE/Eclampsia. Accelerated villous maturation with an absence of intermediate villi, cytotrophoblastic proliferation, avascular terminal villi similarly, had statistically significant differences from the control groups. As with macroscopic findings, differences can be noted between placentae of EO-PE and LO-PE. Those with greatest statistical significance included distal villous hypoplasia, syncytiotrophoblast ‘knots’ and villous infarcts – all of which were more prevalent in EO-PE.

Guo F. et al. (2020) observed that the transcriptomes of placentae of EO-PE showed significant difference from controls, whereas it was the transcriptomes of peripheral blood of LO-PE that showed differences from healthy controls. New biomarkers for EO-PE (EBI3, IGF2, ORMDL3, GATA2 and KIR2DL4) were identified paving new pathways for early diagnosis, while traditional biomarkers such as FLT1, EG, LEP and PAPPA2 were concluded to only be upregulated in EO-PE placentae.

From the biochemical point of view, an increased lipid content of the placentas in PE, both in total and in individual phospholipid classes were seen, compared to controls. Linked with the pathogenesis of pre-eclampsia, placental growth factor (PLGF) was expressed lower in severe PE as opposed to mild PE and normotensive patients; VEGF and hypoxia-inducible factor-1α (HIF-1α) were expressed significantly higher in PE placentae.

Understanding the prevalent placental pathologies seen in pre-eclampsia and eclampsia will allow for better diagnosis and management of the condition to reduce overall morbidity and mortality.