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Hemorrhagic Endovasculitis is a Proliferative and Time-Dependent Lesion Occurring Secondary to Stoppage of Fetal Placental Vessel Flow.

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Background: Hemorrhagic endovasculitis (HEV) is a lesion of the placenta most often found in association with intrauterine fetal death, where it's reported distribution is multifocal (10% – 25%) or extensive (> 25%). This is in contrast to live-born infants where the lesions are mostly (98%) focal and associated with fetal placental thrombosis. We hypothesize that HEV is a progressive, proliferative lesion occurring in response to low/no shear conditions in the fetal vasculature such as in fetal death and stem vessel thrombosis. Multiple observations have shown that endothelial cells, which are kept flat when subjected to flow, become nearly cuboidal and proliferate when shear stress is removed. A similar process is seen in the organization of a thrombus and in states of chronic boundary layer separation in vivo.

Methods: We reviewed the autopsy and slides of 60 cases of intrauterine fetal death. Death-to-delivery interval was determined by histologic examination of fetal tissues as described by Genest DR, et al. in 1992. The placental slides of these fetuses were then reviewed to assess the degree of development of HEV. Fetal placental vessels were classified as having contraction, proliferation, septation, or obliteration of the vascular lumen.

Results: HEV correlates strongly with the death-to-delivery interval, moving from contraction to proliferation (>50% at 24 to 48 hrs) to septation (>50% at 4 days to 1 week) and finally obliteration of the vascular lumen, in a progressive manner. This process results from endothelial and vascular smooth muscle cell proliferation achieving luminal obliteration between 1 and 4 weeks of death-to-delivery time in 58% of the cases.

Conclusions: These observations support the concept that HEV is a proliferative process secondary to low/no shear stress on the endothelium resulting from cessation of fetal blood flow as seen in intrauterine fetal demise and stem vessel thrombosis. This is permitted because the ongoing maternal placental circulation maintains viability of fetal placental tissues. As vascular septation progresses erythrocytes are trapped into progressively smaller lumina, giving the appearance of escape into the surrounding tissue. No hemorrhagic, endovasculitic, or thrombotic components of HEV were evident in the present study.
