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Tenny Parker change A reflection of placental pathology

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Abstract:

Pre-eclampsia is a disorder of pregnancy characterized by hypertension and proteinuria. It is now thought that abnormal placentation and placenta function is a strong predisposing factor for pre-eclampsia. Syncytial knots are aggregates of nuclei at the surface of terminal villi and their number increase with increasing gestational age. Increased syncytial knots (Tenny parker change) are associated with conditions of utero-placental malperfusion and are important in placental examination. Vasculo-syncytial membrane (VSM) is the primary site of feto-maternal exchange in placenta. In pre-eclampsia, VSM thickness is increased causing fetal hypoxia.
We here report a case of an autopsy finding in a pre-eclamptic female. On histopathological examination, we found terminal chorionic villi with excessive syncytial knots (Tenny Parker change). Syncytial knots were seen in more than 40% of chorionic villi. Vasculo-syncytial membrane (VSM) thickness was also increased. Thus, histopathological findings of Tenny Parker change and increased vasculo-syncytial membrane (VSM) thickness are the clues for placental pathology in pre-eclampsia.

Key words: pre-eclampsia, placenta, syncytial knots, Tenny Parker change, vasculosyncytial membrane

Introduction:

The fetus, placenta and mother form a composite triad of dynamic equilibrium, and dysfunction of any one of them can affect the others. Preeclampsia is a condition unique to pregnancy and characterized by sudden onset of hypertension and proteinuria with maternal dysfunction after 20 weeks of gestations with no previous history of hypertension.[1]

Syncytiotrophoblast is a multinucleated epithelium of the placenta. Although many nuclei are dispersed within the syncytioplasm, others are aggregated into specializations referred to as true and false syncytial knots, and syncytial sprouts. Nuclei within the true knots display highly condensed chromatin and are thought to be aged and effete. True knots increase in frequency with gestational age. Excessive formation (Tenny Parker change) is associated with placental pathology, and a knotting index is used to assess severity. However this index is potentially

confounded by the creation of artifactual appearances (false knots) through tangential sectioning. In addition, knots must be distinguished from syncytial sprouts, which are markers of trophoblast proliferation.[2] The vasculosyncytial membrane (VSM), primary site of feto-maternal exchange is formed when syncytiotrophoblast surround the terminal villi and make a close contact with capillaries. Undoubtedly there is a clear-cut inverse relation between villous VSM and fetal hypoxia.[3]

Case Report:

An autopsy specimen of uterus with attached placenta of 30 years old female received for histopathological examination. The female was 9 months ANC with pre-eclampsia with gravida 3, para 3, one live baby with history of death of one baby. On post-mortem examination, uterus measured 52×25×21cms with fetus in situ. Fetus was in vertex position and weighed 2500 gms. Placenta was attached postero superiorly to uterus and umbilical cord measured 41 cm in length. On gross examination, part of uterus with attached placenta was received. Placenta was easily detachable from uterus. No gross abnormalities were noted in placenta.

On histopathological examination, we found term chorionic villi with excessive syncytial knots (Tenny Parker change). Syncytial knots were seen in more than 40% of chorionic villi. We studied further serial sections to rule out false syncytial knots because of tangential cuts.

Also, VSM thickness was increased which was confirmed on PAS staining.

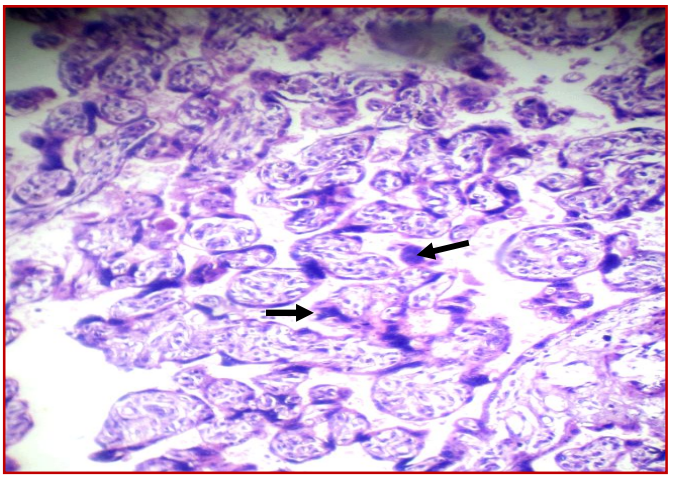

Figure No. 1. Microphotograph showing term chorionic villi with Tenny Parker change.

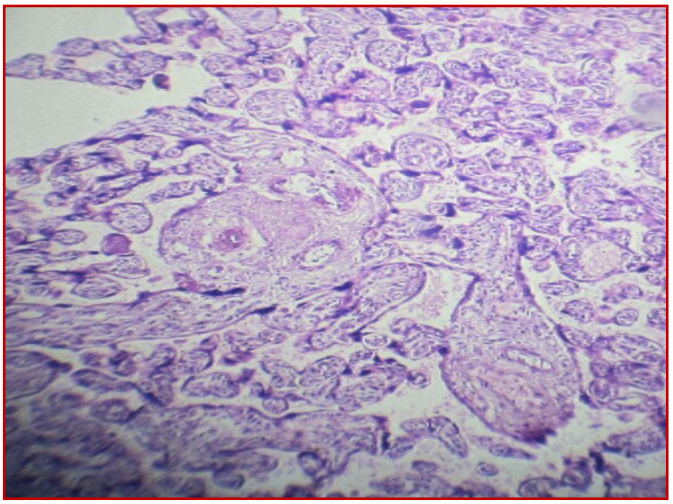

Figure No. 2. Microphotograph showing increased syncytial knots and VSM thickness.

Discussion:

Worldwide preeclampsia is estimated to occur in about 5-8% of all pregnancies and is a major contributor of maternal morbidity and mortality. The precise etiopathogenesis of preeclampsia still remains under extensive research; while some believe it to be a multifactorial. However the presence of placenta is mainly responsible for the onset and severity of preeclampsia. Preeclampsia is associated with shallow placentation caused by inadequate trophoblast invasion into the maternal spiral artery leads into hypoxic state.[4]

In preeclampsia the hypoxia injury disrupts the syncytial architecture which in turn initiates other complications of preeclampsia.[1]

In normal gestation as age advances, the terminal villi are reduced in diameter. The cytotrophoblast in the terminal villi gradually subtle and they contribute their cell mass to growing syncytium. The syncytiotrophoblast is the outer layer of placenta which is in direct contact with maternal blood and is uniquely positioned to alter maternal hemostasis and endothelial function.[5]

An uneven distribution within the syncytiotrophoblast results in clusters of nuclei, the syncytial knots (SKs) which is a focal aggregation of syncytial nuclei on the outer surface of a tertiary placental villous. Syncytial knots at term are present between 10-30% of the terminal villi.[1]

Although 30% of terminal villi with syncytial knots at term are often reported, no reference values have been developed for the percentage of villi with syncytial knots at different gestational ages. According to one study of syncytial knots at different gestational ages ranging from 20-40 weeks, there was a significant positive correlation of gestational age with percentage of villi with syncytial knots. Term placenta (37-40 weeks) showed an average of 28% syncytial knots.[6]

The mode of formation and the function of syncytial knots are still far from clear, for they have been variously considered as a degenerative phenomenon, an aging change, a syncytial hyperplasia and as a response to trophoblastic ischemia or hypoxia.[7]

The placental villous membrane (syncytioplasm) and the fetal capillaries remain separate but functionally act as a single unit, the so called vasculosyncytial membrane (VSM) which is the only physical barrier between fetal and maternal blood. The most significant properties of the VSM are maintenance of exchange surface area and effective diffusion distance of feto-maternal surfaces. Increase thickness of VSM leads to fetal hypoxia and appears to subject the fetus to considerable risk. Deficient of VSM in hypertensive placental villi can be considered as a failure of trophoblastic differentiation.[3]

In the present case, syncytial knots density and the diameter were found to be increased in preeclampsia. VSM thickness was also found to be increased in preeclampsia.

Eternally the oxygen in the intervillous space reaches the terminal villus diffused in the fetal capillaries through VSM and finally reaches the fetus via the fetal vascular network. In the present case, the increase in thickness of the VSM in case of preeclampsia, reduces the feto-placental circulation and the release and accumulation of syncytial knots, even makes the condition worst. Both of these changes are associated with maternal uteroplacental vascular pathology resulting in intrauterine distress of fetus.[8,9]

Conclusion:

Structural placental changes cause functional placental changes. In preeclampsia, hypoxia injury disrupts the syncytial architecture resulting in the increased density of syncytial knots and VSM thickness. The increased thickness of VSM causes impaired maintenance of feto-maternal exchange initiating the aponecrosis of syncytiotrophoblast as syncytial knots, subsequently culminating the systemic inflammatory response of the mother. These factors are suggested to pathologically activate the maternal endothelium, leading to preeclampsia.

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