FOUR COMPLICATIONS OF PREGNANCY AFFECTING THE PLACENTAL VILLI

Olivar C Castejón S¹, Ángela López G²


Correspondence should be addressed to Olivar C Castejón S

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ABSTRACT

Preeclampsia severe, megaloblastic macrocitic anemia, infection by zika virus and marginal insertion of umbilical cord affected to patient of 26 years old who showed fetal death to 22 weeks of pregnancy. Light microscopy was applied to the study of her placenta. Stem villi were observed with syncytial hyperplasia, congested vessels, dilated regions in their trajectory, changes in the thickness of the syncytium or interruptions, scanty development of muscular layer in stromal vessels, immature intermediate villi with aneurysms, mature intermediate villi with scarcity of terminal villi, terminal villi with interrupted syncytium and bad development of ramifications of the placental villi which impede the normal interchange of gases or nutrients. These complications of the pregnancy have provoked severe degenerative changes in the placental villous tree that affected the immature and mature intermediate villi with scarcity of terminal villi which have contributed with the death early of the fetus

KEY WORDS: Preeclampsia. Macrocitic anemia. Zika virus. Marginal insertion.

INTRODUCTION

The placenta can be affected by changes that occurs in the mother or in the formation of their structure. Maternal diseases and eccentric or marginal cord insertion are factors that can to change the histo-architecture of the placental villi (1). Preeclampsia severe, megaloblastic macrocitic anemia, infection by zika virus (Zikv) at 14 weeks of pregnancy and marginal insertion of the umbilical cord affected to patient of 26 years old who showed fetal death to 22 weeks of pregnancy. It has been described that during preeclampsia the following placental lesions have been mentioned in the literature: infarcts, intervillous thrombi, abruptio, decidual necrosis, abnormal cord insertion, Tenney-Parker changes and retarded growth (2).

With hematological disorders the placenta can to present Tenney-Parker changes, infarcts, increased fibrin, abruptio, and villous edema (3). Increased fibrinoid deposition intravillous or perivillous, trophoblastic necrosis, stroma with dilated and replete vessels, endothelial necrosis, sclerosis, stromal disorder by hidropic edema, calcification and terminal villi deficiency were observed when there are placental perfusion bad (4). The effect of anemia on the placenta results in compensatory placental hypertrophy or highly significant placental enlargement in some cases (5). Chronic hypoxia can to provoke capillaries more numerous, thinner and villous length reduced (6).
Zika virus produces in stem villi peripheric degenerative changes with collapsed vessels, Tenney-Parker changes or conglomerate of syncytial knots, immature intermediate villi and fibrinoid deposition with macrophages together in Hoffbauer channels, necrosis of the syncytiotrophoblast and stromal fibrosis. Koyloctic cell, destroyed villi, damage in the wall of the vessels and mature intermediate villi with scarcity of terminal villi were also found (7).

Marginal insertions may be more susceptible to vessel rupture or compression (8) and have been associated with fetal growth retardation and stillbirth when placental maturation is unevenly accelerated because of low uteroplacental blood flow (9). Peripheral insertion is related with thrombosis, and these can lead to mural necrosis of vessels with calcification (10). This type of chorionic insertion is associated with fewer penetrating arteries, fewer cotyledons and abnormal umbilical artery blood flow (11). To describe the changes provoked by these four factors on the structure of the placental tissue with light microscope is our proposal.

MATERIAL AND METHODS

Two groups of population of placental villi were taken of placenta study and placenta normal. The group study proceed from hospitable institution whose placenta was obtained to the 22 weeks of pregnancy. Woman pregnancy of 26 years old in come to the hospital with renal insufficiency, preclampsia severe, megaloblastic macrocitic anemia, infection by Zikv at 14 weeks of pregnancy and with eccentric insertion of the umbilical cord observed after delivery. Zikv was confirmed by nucleic acids detection by RT-PCR. The serology of patient with placenta study was negative for hepatitis B, C, cytomegalovirus, Epstein Barr virus, rubella and toxoplasmosis.

The newborn was born alive at 22 weeks and died 4 hours after. The infected woman pregnancy persisted with Zikv infection during five days and had knowledge of informed consent and approval by the ethical committee of the hospitable institution for the realization of this investigation according to the Helsinki declaration. The placenta normal was obtained at 38 weeks of patient with an increase of weight of 10 kg without antecedent of disease.

Of each placenta were taken five small specimens of the maternal surface selected at random from the region central parabasal in the vertical plane. Three slides by specimen were prepared for light microscopy and 30 histological slides in total were stained with H&E for their observation.

RESULTS

Stem villi near of decidual region are observed with notable syncytial hyperplasia. Stromal vessels are replete of erythrocytes and others are emptied (Fig. 1). Any stem villi showed congested vessel with dilated region in their trajectory (Fig. 2). Small stem villi exhibit changes in the syncytiotrophoblast and their stromal region is noted in degeneration (Fig. 3). Immature intermediate villi were observed with aneurysms in their vessels which are observed dilated (Fig. 4). Interruptions of the syncytiotrophoblast in small stem villi can be seen with frequency and scanty development of muscular layer in stromal vessels (Fig. 5). Bad development of small stem villi with dilated vessels occupying almost all the stromal region are noted (Fig. 6). Small stem villi was seen with degenerated syncytiotrophoblast and dilated vessels in stromal region emptied (Fig. 7). Terminal villi also were seen with interruption of the syncytiotrophoblast and stromal erythrocytes being extruded to the intervillous space (Fig. 8). A general observation of the placental villi permit to us to see bad development of the stem villi (Fig. 9).

Figure 1: Stem villi is seen with increased thickness of the syncytiotrophoblast associated a decidual region H&E: 400X
**Figure 2:** Stem villi showing a vessel which shows dilated region to the right. H&E: 400X

**Figure 3:** Placental villi that shows changes in the thickness of the syncytium and severe changes in stromal region. H&E: 400X
Figure 4: Immature intermediate villi with aneurysm in one of their vessels. Degenerative villi are observed in their periphery. H&E: 400X

Figure 5: Small stem villi are observed with vessels interrupted and notable changes to level of the syncytium. Muscular layer is reduced in some vessels. H&E: 400X
Figure 6: Stem villi with stromal region emptied and dilated vessels. The syncytium shows degenerative changes. H&E: 400X

Figure 7: Bad development of small stem villi is seen with reduction of the stromal zone by dilated vessels. The syncytium is degenerated. H&E: 400X
Figure 8: Terminal villi in the center of the microphotography is noted with syncytial interruption and expelling erythrocytes. H&E: 100X

Figure 9: Stem villi exhibiting bad development of ramifications of placental villi. H&E: 100X

DISCUSSION

During preeclampsia the typical feature of the placental villi is of preterm maturation with long, twisted, partly branched terminal villi that are aggregated around the hidden mature intermediate villi. Severe alteration of terminal villous capillarization occurs showing local variations of non-branching to prevalent branching
Zika virus contains molecules that produces a destructive effect on the placental structure (7) and leads to apoptosis induction (14). Zika viral proteins NS2A, NS2B and NS3 induce apoptosis and protein NS1 possibly damages the endothelial cells through anti-NS1 antibodies (15). Stromal region which was observed almost emptied in some placental villi showed this viral activity. Some stromal vessels have exploded by high blood pressure provoking severe degenerative changes. The combined effect of Zikv and high blood pressure have produced a general disorganization of the structure of the stem villi.

Severe damage in the vessels of stem villi also has been observed in placenta infected by viruses of the togaviridae family which belong the Chikungunya virus (16). In general the viruses appears affect the structure of vessels as has been showed in others works where the placental villi was infected by HIV-HPV (17) or by Cytomegalovirus infection (1).

Lysis of the syncyntial plasma membrane by the Zikv could produce holes on it and the entrance of fluids to the stromal region since the intervillous space disorganize it. Zika viral proteins functioning as proteases can to lysis the stromal region. Zika viral proteins NS2A, NS2B and NS3 induce apoptosis and protein NS1 possibly damages the endothelial cells through anti-NS1 antibodies (15). Stromal region which was observed almost emptied in some placental villi showed this viral activity. Some stromal vessels have exploded by high blood pressure provoking severe degenerative changes. The combined effect of Zikv and high blood pressure have produced a general disorganization of the structure of the stem villi.

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