

The Placental Villi During Chronic Hypertension

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Abstract

Two placentas obtained of woman pregnancy at 32 at 39 years old with gestational ages of 37 and 41 weeks of pregnancy were classified as P1 and P2 respectively. Patient P1 with history clinic of three gestations, two caesarian, one abortion and 117 Kg of weight and P2 with seven gestations, 67 Kg of weight, without that history.

Specimens were fixed and processed according H&E stain and the degenerative changes in the placental villi observed with light microscopy. Observations were done in base at necrosis, immaturity, fibrinoid change, edema, hemorrhage, infarcts, chorangiosis, syncytial knots, calcification, fibrosis, polymorphonuclears, changes in the wall of the vessels and intervillous thrombosis. P1 showed degenerative changes in syncytium and stromal region with increased deposition of fibrinoid, edema, tissue death, vessel-contraction, chorangiosis, calcification, infarcts and destruction of the organization of the villi. P2 with dilatation of vessels, infarcts, stem villi with degenerative changes in syncytium and stroma, edema and contracted vessels. Terminal and intermediate placental villi were seen destroyed and erythrocytes are expelled to the intervillous space. Chronic hypertension has provoked severe degenerative changes to the placental villi disorganizing the placental structure with high risk of the good fetal development.

Keywords

Placental villi; High weight; Chronic hypertension

Introduction

Chronic hypertension is defined as blood pressure ≥ 140 mmHg systolic and / or 90 mmHg diastolic before pregnancy or before 20 weeks of gestation and persistence of hypertension for > 12 weeks after delivery [1]. Woman with hypertension are at increased risk for several pregnancy complications, including superimposed preeclampsia, fetal growth restriction, placental abruption, preterm birth, and cesarean section.

Fetal growth abnormalities are often thought of manifestation of placental dysfunction. It is important to note that placental abruption - premature separation of the placenta from the underlying myometrium resulting in pain, bleeding, and potentially clinical significant interruption of fetal gas and nutrient exchange - is more common in women with chronic hypertension [2].

The population of women with chronic hypertension with or without superimposed preeclampsia has a higher incidence of perinatal death and small for gestational age newborns than the general population does [3]. Studies emphasizing hypertensive syndromes in pregnancy as chronic hypertension, gestational hypertension, pre-eclampsia, and preeclampsia superimposed on chronic hypertension have showed positive correlation with respect to the number of syncytial knots, fibrin deposits, and the cases with preeclampsia and chronic hypertension presented a larger number of terminal villi vessels which suggests that in different types of hypertension the final pathway that leads to microscopic lesions in the placenta is the same, with different intensity due to the severity of the disease [4].

Villitis of unknown etiology in placentas of pregnancies with hypertensive disorders has been found in sustained chronic hypertension with a frequency of 32% [5]. This event also is observed when chronic hypertension is superimposed on preeclampsia. Since placental calcifications are associated with maternal history of pregnancy induced hypertension, preeclampsia and placental abruption we want to know if chronic hypertension increases the deposits of calcium in the placental villi [6]. In the hypertensive placenta has been recognized occlusion or narrowing of the utero placental vasculature and placental ischemia.

Besides infarcts, increased syncytial knots, hypovascularity of the villi, cytotrophoblastic proliferation, thickening of the trophoblast basement membrane, obliterated enlarged endothelial cells in the fetal capillaries, atherosclerosis of spiral arteries in the placental bed,

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decreased number of syncytial microvilli and focal syncytial necrosis [7]. This hypertensive disorder is classified in chronic hypertension of any cause and chronic hypertension with superimposed preeclampsia or in mild hypertension and severe hypertension both provoking adverse pregnancy outcomes as preeclampsia, abruption, fetal growth restriction and preterm birth [8,9]. To describe the histopathological changes provoked by the chronic hypertension on the structure of the placental villi is our proposal.

Materials and Methods

Two placentas were obtained by cesarean of two women pregnancy (P1, P2). Patient P1: With 32 years old, 37 weeks of gestation and 3 previous gestations, history of two cesarean, one abortion and 117 Kg of weight. Patient P2: with 39 years old, 41 weeks of gestation, 7 previous gestations, without that history and 67 Kg of weight. Both patients without pregnancy induced hypertension. Samples were obtained after labor in delivery room for their processing in our laboratory. Specimens were fixed and processed according to H&E stain, observing with light microscopy the degenerative changes in the basal plate of human placenta.

Patients have given their informed consent for participation in the research study and there is no conflict of interest in this work.

Observations were done in the different types of placental villi and in the intervillous space. Characteristics were noted in base to necrosis, immaturity, fibrinoid change, edema, hemorrhage, infarcts, chorangiosis, syncytial knots, calcification, stromal fibrosis, presence of polymorphonuclears, changes in the wall of the vessels and intervillous thrombosis. Five fragments of placenta were selected by each placenta and four slides prepared by each one. 20 slides by placenta were taken and compared with the normal placentas. In this work normal blood pressure is 120 over 80 mmHg and hypertension is considered higher than 130 over 80 mmHg. Medication to treat this disorder was used during control. Placentas with hypertensive disorders were studied and compared these with normal placentas which were used without hypertension as controls. Besides we have studied normal placentas. In base to these studies we have the controls very well studied.

Our specimens are not complete organs or complete placenta, our unity of research is one placental villi. This contains trophoblast and stromal region. With the objective of 40x in each field we can see 20 or more placental villi, with the objective 10x we see 80 or 100 placental villi. With these 10 fields we are seeing 1000 villi. But we can't be seen until 100 or more fields in cuts of 4 or 5 μ m. Our tissue - slides have 1 or 3 cuts of tissue which are seen. Each cut is of 10mm x 8mm approximately. we can't be seen with 40 x objectives in 10 camps until 240 villi or more and in 100 camps, 2400 villi. We used 27 tissue slides by each placenta (P1, P2) and we were analyzing millions of placental villi.

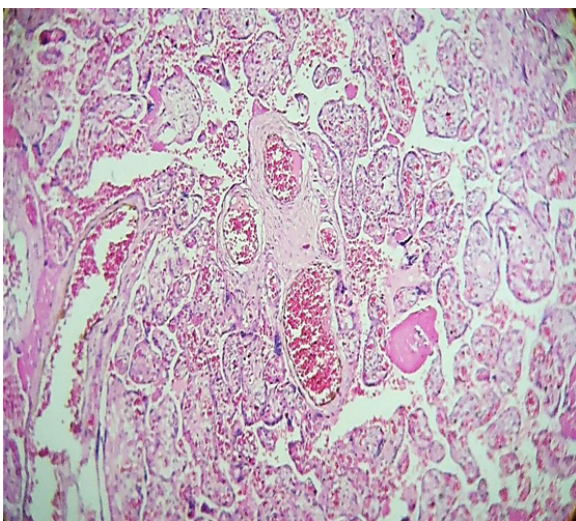


Figure 1: Stem villi is seen in the center with a disorganized structure.400x.H&E.

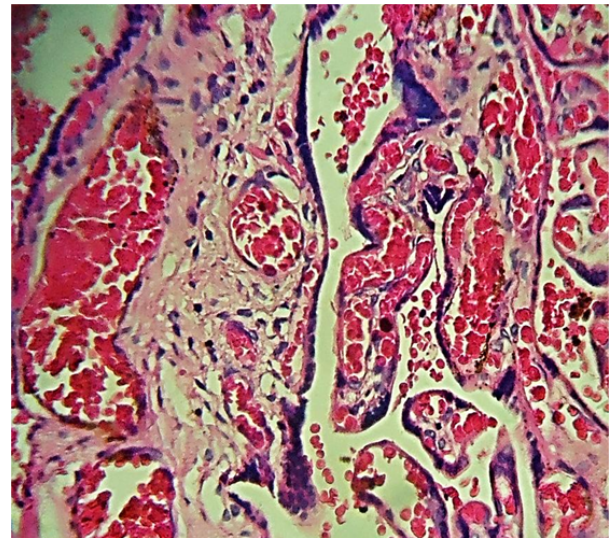


Figure 2: Dilated vessels are noted in two small stem villi.400x.H&E.

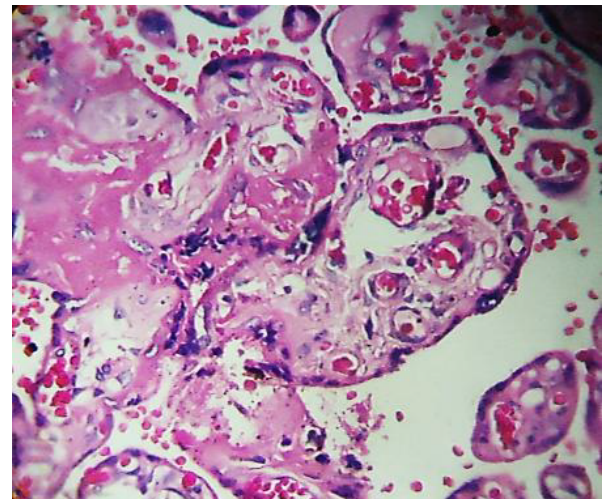


Figure 3: Stem villi shows clear region in stromal region.400x.H&E.

Results

Placenta P1

Stem villi were seen with degenerated syncytium, thinner, suffering necrosis and interrupted. Prominent vessel - dilatation and disorganization stromal was found [Figure 1]. These stem villi showed increased deposition of peripheral fibrinoid with presence of x-cells and degenerative changes in the wall of stromal vessels. Conspicuous edema was noted [Figure 2] and clear regions in the stroma of stem villi are frequent indicating zones of tissue or cellular death [Figure 3].

In some stem villi vessel - contraction was observed with changes in endothelial cells [Figure 4]. Many placental villi have stayed in a large deposition of fibrinoid [Figure 5] and ghost regions of stromal regions are noted. In numerous mature intermediate and terminal villi the vessel-dilatation has occupied the stromal region. With frequency deteriorated or destroyed villi are observed in the intervillous space.

Some immature intermediate villi also were seen with dilated vessels exploding near the syncytial surface. Mature intermediate villi could be seen with three lineal vessel-contractions in short distance increasing the possibility of thrombosis and aneurysmal

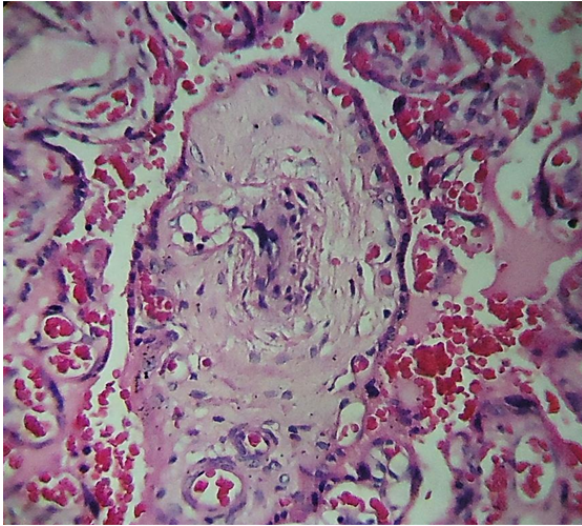


Figure 4: Stem villi with vessel contracted.400x.H&E.

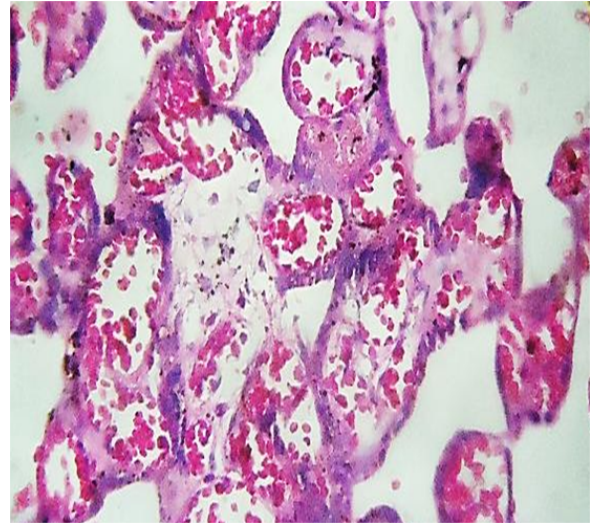


Figure 7: Placental villi are associated by syncytial contact 400x.H&E.

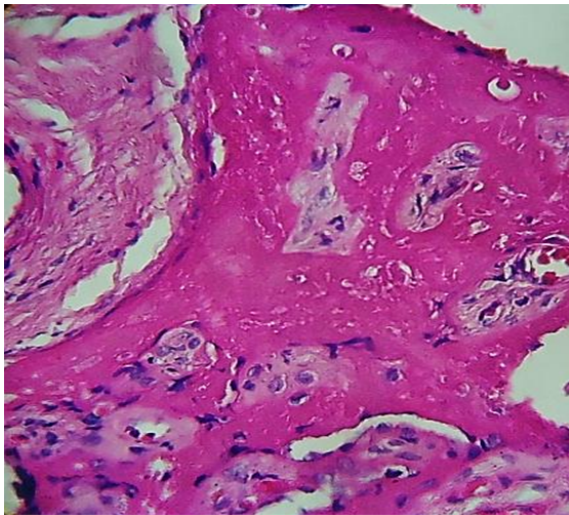


Figure 5: Stromal regions of placental villi are seen surrounding by a deposition of fibrinoid great. At to the left a degenerated vessel is observed.400x.H&E.

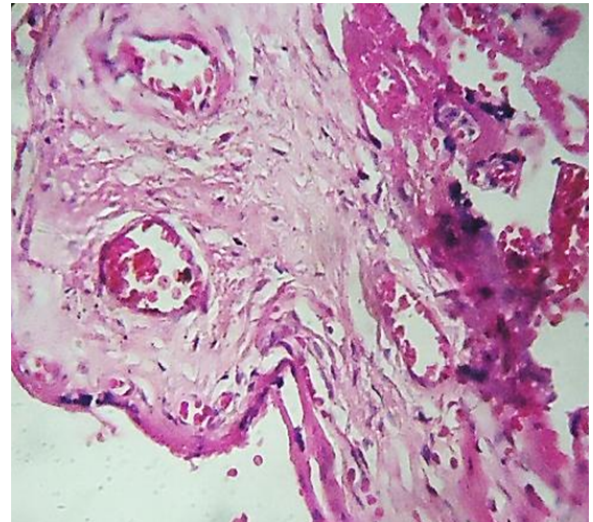


Figure 8: A stem villi is seen with great destructive effect.400x.H&E.

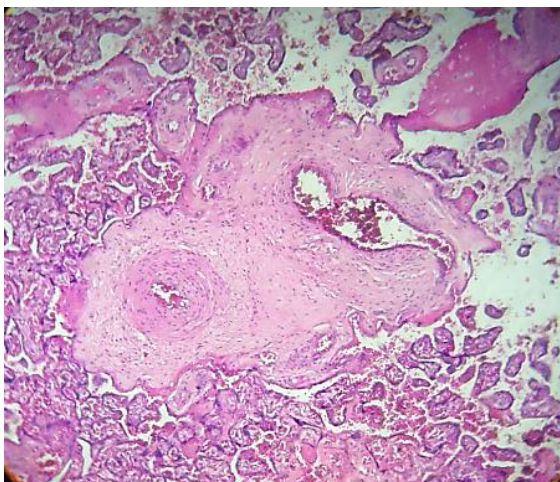


Figure 6: Aneurysmal dilatations in stem villi are noted.100x.H&E.

prolongations. Chorangiosis, calcification, infarcted extensive regions and aneurysmal prolongations of vessels were present. The explosion of vessels, occasionally, destroys all the villous hystoarchitecture.

Clinical data: Blurred vision, nose bleeds, trouble breathing and pain in back.

Laboratory data: 140 mmHg to 159 mmHg systolic pressure and 90 mmHg to 99 mmHg diastolic pressure.

Placenta P2

The vessel-dilatation in stromal region has been maximum and deforms the general morphology of the placental villi [Figure 6], these have the tendency of form groups and they take contact, syncytium with syncytium, giving origin to infarcts [Figure 7].The stem villi suffers severe degenerative changes that affect the syncytium and the stromal region [Figure 8]. These villi are seen with vessels in vessel-contraction and numerous small placental villi are associated to these villi which were seen in extensive infarcted regions [Figure 9].

The vessels of small stem villi, mature intermediate villi or of terminal villi, all dilated, are moved toward the surface of the placental

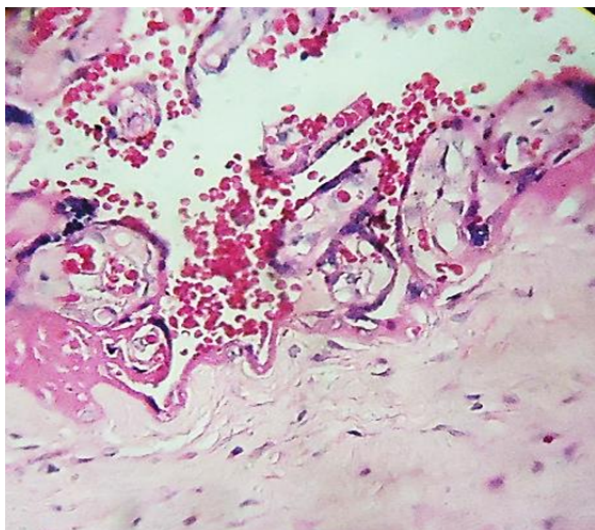


Figure 9: A region of stromal zone of stem villi is associated to small placental villi.400x.H&E.

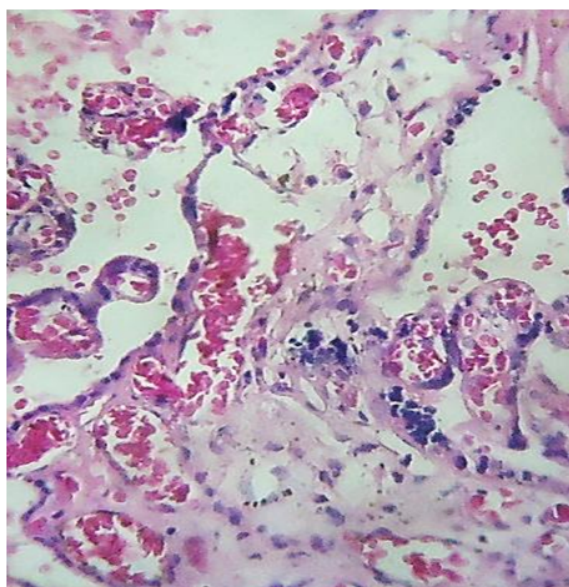


Figure 10: Stromal vessel of stem villi has exploited at the surface of stromal zone.400x.H&E.

villi, indent it, and explode. So, the erythrocytes are expelled to the intervillous space [Figure 10].

With frequency debris of placental villi are found in the intervillous space of P1 and P2.

Clinical data: Headache, chest pain, dizziness or weakness, pain in neck.

Laboratory data: 140 mmHg to 149 mmHg systolic pressure and 99 mmHg to 110 mmHg diastolic pressure.

Discussion

In our cases the chronic hypertension has not been superimposed to pregnancy induced hypertension. Nevertheless in our study of placenta has been showed a significant number of syncytial knots, fibrinoid necrosis, calcification and hyalinization which correspond with results of pregnancy induced hypertension [10]. Villous hypermaturity has been observed significantly more often in the hypertensive placenta which is in agreement with this study by the presence of numerous mature intermediate villi [11].

Chronic high blood pressure has been cause of placental insufficiency and this condition maintains in high risk the good fetal development. Woman pregnancy of high weight has reduced blood flow since her obesity and related atherosclerosis of utero-placental arteries in decidual region leads to low blood flow. This reduced flow under high blood pressure in the placenta causes the disaster in the cytoarchitecture of the placental villi here observed [12]. There is tendency of fetal death when mother is older (defined as ≥ 40 years), has high weight, chronic hypertension and multiparity as in patient of placenta P2 [13].

Most studies about chronic hypertension in pregnancy not found adverse pregnancy outcomes nonetheless caution should be used in cases of impaired uteroplacental perfusion as here presented and patients should undergo serial measurements of high blood pressure [14].

Chronic hypertension is caused by essential hypertension and vascular, renal and endocrine diseases. Mild and severe chronic hypertension produces preeclampsia, abruptio, fetal growth restriction and preterm birth. It could be possible that when the blood pressure decreases placental vessels normally dilate whereas when blood pressure increases, they constrict to maintain constant placental blood flow in the structure of the placental stroma. So, would be explained the presence of vessels constricted or dilated here seen [15].

This reduction of blood supply can age the placenta prematurely and the maternal hypertension blocks the normal nutritional exchange of the fetus endangering their organs. The physiological mechanism contributing with reduced vascular diameter is due to increased vascular contraction of smooth muscle in the placental villi [16]. In obese mothers it has been observed an increased muscularity in the vessel wall altering the placental vascular function [17]. The aging of the placenta prematurely can provoke a reduction of their weight as has been observed in placentas affected by chronic hypertension [18].

Chronic villitis elevated nucleated red blood cells and chronic inflammation was not seen by us being reported in placentas associated at early IUGR with concomitant hypertensive disease [19]. A before work reported by us also describes not inflammation in placental villi affected by obesity and hypertension [20]. A progressive decreasing in total lymphocyte and monocyte before term of pregnancy could to explain the absence of inflammatory cells in that paper, or an exhaustive control urinary infection [21].

Others authors have written that decreased placental weight was inconsistently related to blood pressure elevation and that this relation only is possible when severe proteinuria is diagnosed [22].

Chronic hypertension six fold increases the risk of placental lesions by vascular bad perfusion when compared with others hypertensive disorders [23]. Massive perivillous fibrin or fibrinoid deposition is associated to maternal hypertension; if this event is observed occupying >30 -50% the intervillous space would be potentially lethal to the fetus [24, 25].

The placenta plays important roles in control of blood pressure in response to this ischemia or hypoxia which provokes fetal suffering or developmental pathologies [26].

In conclusion, a general histologic disorganization of the placental villi has been observed in placentas of patients with high weight at term and prolonged pregnancy whose chronic hypertension has provoked severe placental lesions that maintains in high risk the good fetal development.

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