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PATHOLOGICAL STUDIES ON THE PLACENTA FROM PRE-TERM AND TERM MATERNAL TOXICOSIS

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INDEXING WORDS

placenta; pre-term maternal toxicosis; term maternal eclampsia; immunohistoenzymatical study

SYNOPSIS

Fifty one placentae with maternal pre-term and term toxicosis were studied pathologically. In the severe pre-term maternal toxicosis, the placenta reveals hyperplasia of syncytial cytotrophoblasts(S-cells) with knots, fibrin deposit at their surface, fibrinoid degeneration of the stroma, in addition to marked interstitial fibrosis of chorionic villi. The basement membrane is thick and cytotrophoblasts(C-cells) are scattered beneath it. Decidual arteries are hyalinized, but a few decidual arteries are thick and almost obstructed in their lumina. On the other hand, the placenta of maternal term toxicosis show chorionic villi with few C-cells under the thin S-cell lining. Their basement membrane is thin with well developed vasculosyncytial membrane. Decidual arteries are more hyalinized and widened. These pathological changes seen in term maternal toxicosis are the severer than those of placental aging.

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INTRODUCTION

The placenta is an important organ functioning as the site of a metabolic center where material exchanges are made between mother and fetus. Both C-cells and S-cells originates from fetal tissue; these are converted into S-cells in the developmental course of placenta along gestational period. It is accepted that S-cells produce three peptide hormones, hCG, hPL and SP-1, which play an important role in the maintenance of physiological homeostasis during pregnancy.

Toxemia of pregnancy is a severe maternal complication characterized by hypertension and/or proteinuria and/or edema. The sickness which occurs in pre-term period is sometimes accompanied by coma, convulsive seizure (pre-term eclampsia) and other lethal conditions. There have been no etiological clues to explain the pathogenesis in toxemia of pregnancy. However, it its suggested that the placenta may be one of the causative factors for the disease, as the affected pregnant women are usually free from any symptoms after delivery.

Previous reports revealed that placenta of the patient with toxemia in the late period of pregnancy still has a small number of C-cells and thicker basement membrane than that of normal pregnant women. It is also reported that immunostain for beta-hCG are strongly positive in S-cells of the toxemia, while not so in the essential hypertension. On the other hand, these pathological findings seen in the placenta of eclampsia which occurs in term period (term eclampsia) have been suggested to be the mild cases.

The purpose of this study is to clarify the differences in the placental pathology between the placenta of pre-term eclampsia and those of term eclampsia.

MATERIALS AND METHODS

We examined 51 placentae complicated with maternal eclampsia. These cases were divided into two groups: 36 placentae with pre-eclampsia from 28 to 36 gestational weeks, and 15 ones with eclampsia from 37 to 40 gestational weeks. The first first group was, then, subdivided to mild

pre-eclampsia in 16 cases and to severe pre-eclampsia in 20 cases, which showed pregnancy-induced hypertension. This group was compared pathologically to 29 cases of intact placentae from 28 to 36 gestational weeks, without any maternal complications. Another group was composed of eclamptic placentae of the mother with pregnancy induced hypertension from 37 to 40 gestational weeks, and was studied in comparison to histological findings of 52 control placentae between 37 and 42 weeks of gestation. The decidual arteries were obtained from the center of the cotyledon at the decidua. Eight histological blocks were made from respective cases and were treated by routine pathological procedures. Histologic examination was done by HE, EV, PAS, PTAH and Masson trichrome in addition to immunohistoenzymatical stains; hCG, hPL and SP-1.

RESULTS

A. Placenta of pre-term toxicosis

Histological examination of the chorionic villi of the placentae in pre-term eclampsia revealed intervillous narrowing and poor vasculosyncytial membrane with thick S-cell's lining. There are microfoci of infarctions which consist of collapsed capillaries and perivillous fibrin deposit. Dysmature villi are often encountered with abnormal villous shape(Figure 1). These chorionic villi are swollen and accompanied by an increased number of syncytial sproutus. Some villi exhibit fibrinoid necrosis and syncytial knots without capillaries(Figure 2). The stroma of stem villi are increased in collagenous fibers showing sclerotic changes. Most of chorionic villi are covered by thick S-cells and preserved partly by inner lining of C-cells under the C-cells. A few C-cells exhibit mitotic figures, which is rare in the C-and S-cells of villi in the normal placenta. Both SP 1 and hPL are positive in S-cells, but faintly positive in C-cells(Figure 3). Basement membrane confirmed by PAS stain is thicker in the chorionic villi of this group than characteristic and suggests insufficient formation of vasculosyncytial membrane with thick S-cells' lining. The decidual artery compatible to spiral arteries shows hyalinized media and intimal infiltration by C-cells(Figure 4). However some arteries have markedly thick walls with almost obstructive lumina, which is another characteristic in this group(Figure 5). This thick wall contains few elastic but rich collagenous

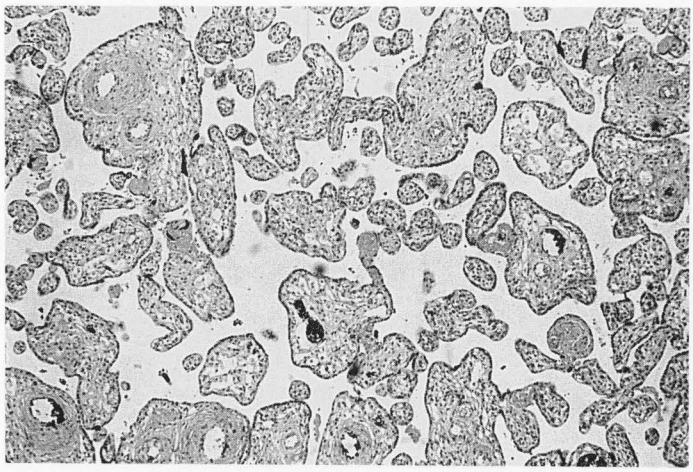

Fig. 1: Dysmature villi of the placenta of 31 gestational weeks seen in pre-term eclampsia. HE, x 40

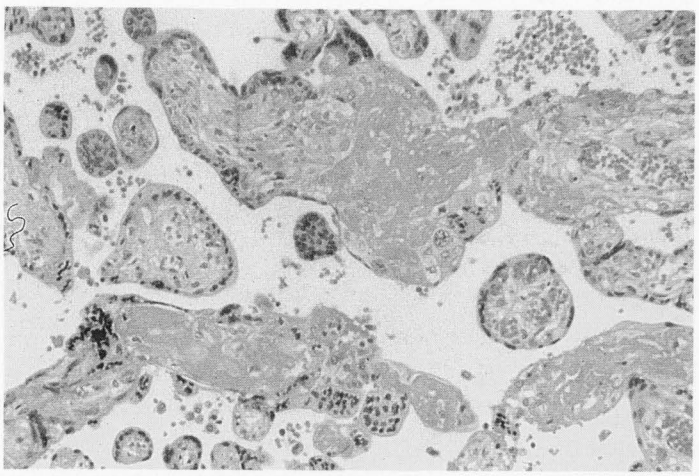

Fig. 2: Fibrinoid necrosis and syncytial knots of the placenta at 31 gestational weeks seen in pre-term eclampsia. HE, x 100

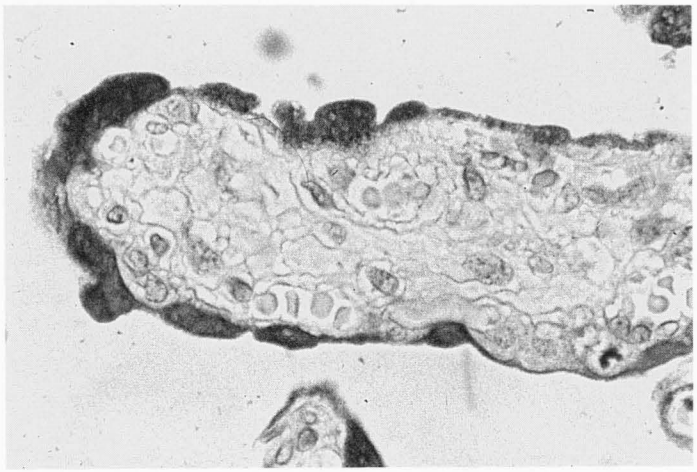

Fig. 3: Syncytial knots and a few C-cells beneath the surface S-cells at 34 gestational weeks seen in pre-eclampsia. SP 1, x 400

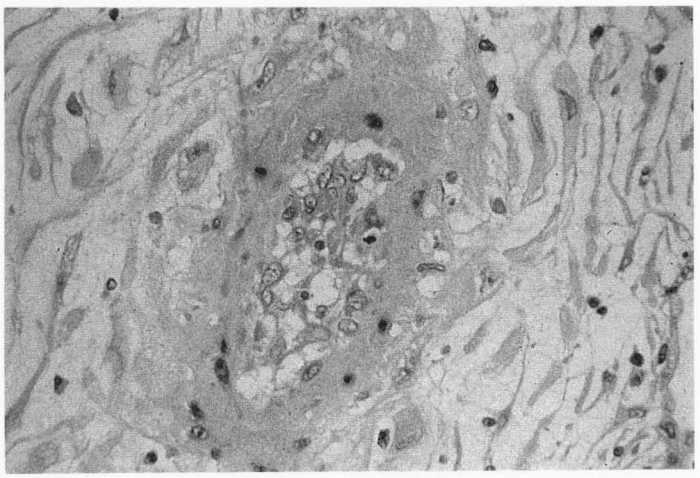

Fig. 4: Sprial artery showing hyalinization and C-cell infiltration in the placenta of 34 gestational weeks seen in pre-term eclampsia. EV, x 200

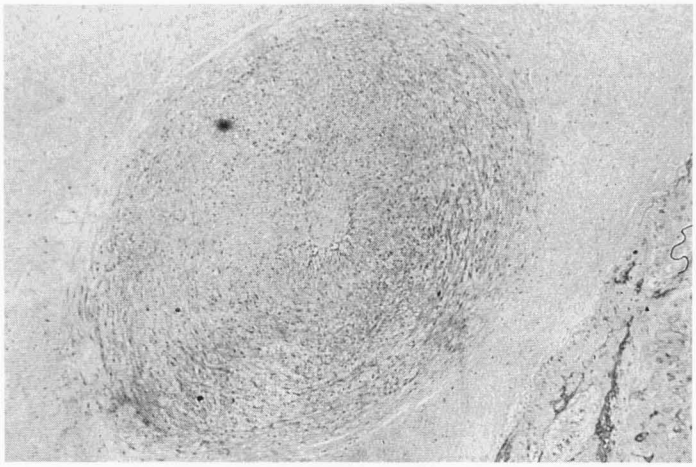

Fig. 5: Thick spiral artery of the placenta at 31 gestational weeks seen in pre-term eclampsia. PTAH, x 40

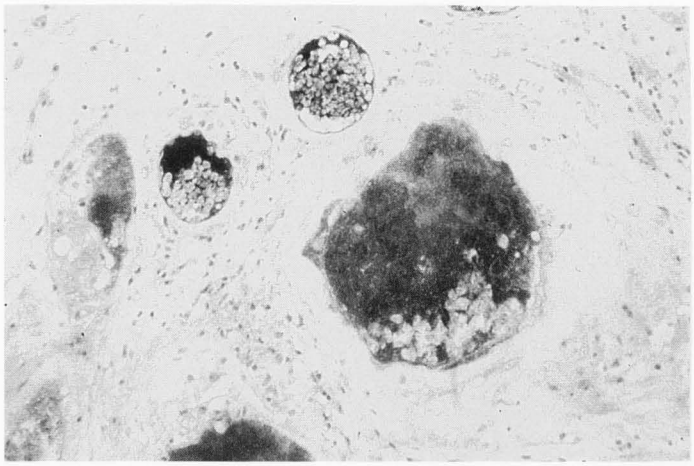

Fig. 6: Spiral arteries of the placenta at 34 gestational weeks seen in pre-term eclampsia . SP 1, x 100

tissues as shown by Azan stain. Their walls are negative by hPL and SP-1 and hCG stains, but their lumina positive for SP 1 and hPL(Figure 6).

These histological changes were found in 12 placentae with severe signs of pre-term maternal toxicosis, but the other 4 were almost the same as those of the control cases. On the other hand, 8 placentae showed findings mentioned above in mild signs of maternal toxicosis, but the other 12 showed almost same histological changes as those of control placentae from 28 to 36 weeks of gestation.

B. Placenta of term toxicosis

The placentae of term eclampsia had often multiple infarctions or hematomas, but were not so large in general. Infarcted lesions are occupied by collapsed villi with fibrin deposit. It is suggested that no circulation existed in the infarcted areas. Chorionic villi are swollen by capillary congestion. The formation of vasculosyncytial membrane is well developed in this group. Intervillous narrowing is found the same as in the placenta of pre-term group. There are chorangiosis containing many capillary vessels per unit area of a villous(Figure 7). Furthermore, there are many syncytial knots, calcification and fibrin deposit around or in the villi(Figure 8). But collagenous fibers are fewer than those of pre-term eclampsia in villous stroma. Only a few basement membrane of villous capillaries show even thick structure by PAS stain. S-cells are positively stained by SP 1 and and hPL, but C-cells are seldom seen in this group. The decidual arteries compatible to spiral arteries have totally hyalinized media infiltrated by foamy cells, and its lumen is almost obstructed by fibrin thrombi(Figure 9). The infiltrated cells are degenerative. The morphological changes of decidual arteries are marked hyalinization of the wall with many foamy cells. There are no positive stains in the wall of decidual arteries and foamy cells.

These histological characteristics were found in 8 placenta of term maternal toxicosis, but 2 were similar to those seen in the placenta seen in mothers with severe pre-term toxicosis, the other 5 were almost the same as those seen in intact placenta of control from 37 to 40 gestational weeks.

The morphological results of the two groups were summarized in Table1.

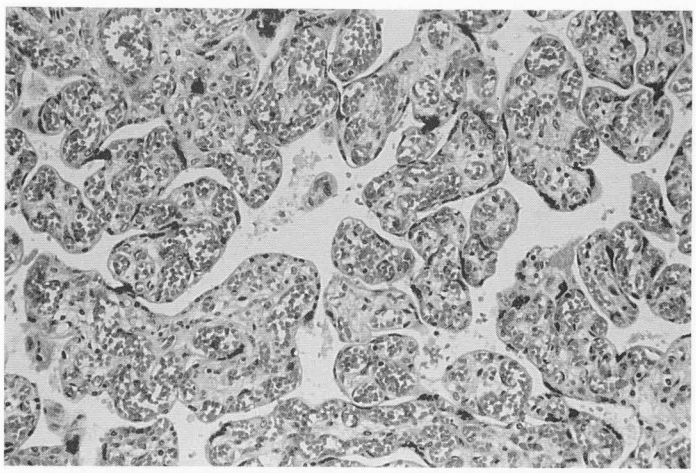

Fig. 7: Chorangiosis in the placenta of 40 gestational weeks seen in term eclampsia. HE, x 100

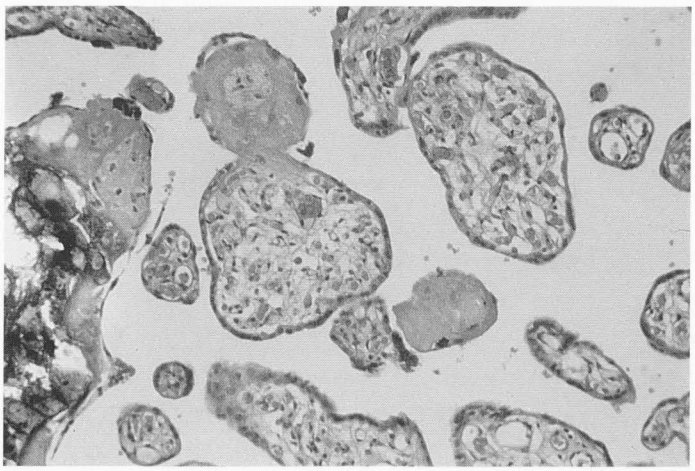

Fig. 8: Chorionic villi of the placenta at 37 gestational weeks, showing fibrinoid degeneration and calcification seen in term eclampsia. HE, x 100

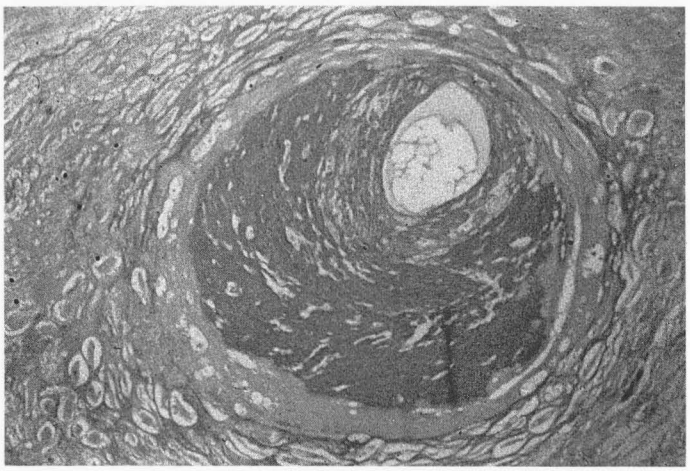

Fig. 9: Hyalinization and fibrin thrombi in spiral artery at 40 gestational weeks seen in term eclampsia. PAS, x 100

Table 1 : Characteristic tendencies seen in severe pre-eclamptic and eclamptic placentae

		Severe pre-eclamptic placentae	Eclamptic placentae
Gestation		28 to 36 weeks	37 to 40 weeks
CV	S-cells	thick, SK(++)	thin, SK(+)
	C-cells	persistent, but a few	few
	Interstitium	fibrosis(++)	fibrosis(+)
	Capillary	congestion(+),VSM(+)	congestion(++),VSM(++)
SV	Artery	sclerotic wall(+)	sclerotic wall(-)
	Vein	congestive	more congestive
IVS		narrower	narrow
Decidual Artery		hyalinization(+) foamy cells(+)	hyalinization(++) foamy cells(++)

S-cells: Syncytiotrophoblasts
C-cells: Cytotrophoblasts
IVS: Intervillous space
CV: Chorionic villi
SV: Stem villi
SK: Syncytial knot

DISCUSSION

Maternal toxemia and severe eclampsia such as convulsive attack have been discussed concerning the placental capacity or hypofunction from old times, and it is not clearly understood even now. With development of chorionic villi as placental ageing, C-cells are precursors of S-cells, which disappear apparently after 16 gestational weeks, whereas about 20% of C-cells per total villous trophoblasts exist at full term[13]. In the placenta of early gestation, intervillous space is the highest in PO2 concentration, and the peripheral stroma of villi is in contact with high PO2 blood, but the central stroma is low in PO2. C-cells are most likely distributed under S-cell's lining and at the central areas. It is speculated that the mitotic activity of C-cells is seen under low oxygen tension, and S-cells undergo respective fusion under high oxygen concentration[5]. As the persistence of C-cells and retarded capillary exposure at the terminal villi can be highly seen in the placenta, the pre-term eclamptic placentae hypofunctional circulation usually observed in the placenta of early gestation may especially continue in these conditions.

Recent pathological studies on the eclamptic placentae are few[8,11,12]. Spiral arteries named as ureteroplacental vessels are related to the perfusion of maternal blood into the placenta[9]. Some investigators describe that the endothelial lining is originated from C-cells[5,14], others from S-cells[4,6]. They show a reduction of elastic fibers and become fibrinoid rich, then increase in their lumina as development. At the pre-term eclampsia, their physiological changes are inadequate and may cause maternal hypertension during pregnancy and subsequently are followed by foetal growth retardation[1,2,7]. From our examination, foamy cells and invaded cells to the wall were both negative for hCG, hPL and SP-1, suggestting that it is not to be S- but probably to be C-cells' origin. The decidual arteries, which are mostly composed of spiral(coiled) arteries, branch by a sharp angle from radial and basal arteries of the myometrium, and play important roles such as control of blood supply to the placenta by the vasoconstriction and dilatation as resistant arteries. Another importance point is the sensitivity to hormones and peptides such as prostaglandin series. As those arteries can be rather easily obtained from the decidua of the central cotyledon at earlier gestation but difficult at full term, it may be possible that we observed superficial arteries from the placenta at full term maternal

toxicosis.

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