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Ultrastructural changes in the syncytiotrophoblast in some types of pathological pregnancy

The syncytiotrophoblast as the external membrane of the placenta barrier plays an essential role in the exchange between the circulating fetal blood in the vessels of villi and the intervillous mother blood.

Only some publications relating to placenta investigations focus on the trophoblastic epithelium (2, 3, 4, 5). Therefore, the estimation of syncytiotrophoblast function disturbance and its reduction with the fetal development is very important for clinical reasons. The observations of changes on the ultrastructural level in some types of pathological pregnancy enable the explanation of the cause of fetal development disturbance at least partially.

MATERIAL AND METHODS

Fragments of 12 mature placentas from pathological pregnancies (gestosis, cholestasis, type A diabetes pregnancies and premature outflow of amniotic fluids) and 3 placentas from normal pregnancies were fixed in glutaraldehyde and OsO_4 and subsequently embedded in Epon 812. Ultrathin sections were contrasted with uranyl acetate and lead citrate according to the Reynold's method. Ultrastructural investigations were made by the electron microscope BS-500 (Tesla).

RESULTS

CONTROL GROUP

In the syncytiotrophoblast of normal placentas the mitochondria and numerous pinocytal vesicles of different sizes were present. In the elongated cell nuclei part of chromatin usually at the nucleus membrane showed great electron density. The syncytiotrophoblast surface was covered with the numerous, well developed microvilli (Fig. 1).

Fig. 1. Normal placenta. The syncytiotrophoblast of the two neighbouring villi. Well developed microvilli on the surface, pinocytic vesicles and cell nucleus are visible. Magn. approx. 6,000x

GESTOSIS

In comparison with the normal placenta the syncytiotrophoblast surrounding numerous villi was very thin and covered with reduced and poorly visible microvilli. Few pinocytic vesicles were visible inside and just under it in the villous stroma – the light spaces, surrounded by membranes.

CHOLESTASIS

The thin syncytiotrophoblast covered with visible microvilli contained very few pinocytic vesicles but a great number of them was observed in the trophoblastic nodules, where the microvilli were well developed. In some places the basal membrane was thicker than in the normal placentas. In the stroma under syncytiotrophoblast irregular spaces of small electron density and a great number of connective tissue fibres were observed.

TYPE A DIABETES PREGNANCIES

In the thick syncytiotrophoblast surrounding the placenta villi from pregnant mothers with the non--insulin dependent diabetes the damage of most mitochondria appearing as the cristae atrophy was found. The electron dense vesicles were present too. In general, the light pinocytic vesicles were not observed and on the syncytiotrophoblast surface the microvilli were absent or they occurred singly. A part of villi was covered with normal syncytiotrophoblast (Fig. 2).

PREMATURE OUTFLOW OF AMNIOTIC FLUIDS

In the placentas from the pregnancy with premature outflow of amniotic fluids the stroma of many villi was electronically very light and showed the increased number of connective tissue fibres. The number of blood vessels and their diameter were considerably smaller than in the normal placentas. These villi were covered with a thin syncytiotrophoblast lying on the considerably thicker basal membrane than in the case of normal placentas. Although the microvilli on the surface of this layer were well developed pinocytic vesicles were not observed inside them (Fig. 3).

DISCUSSION

In all the examined placentas there were found different abnormalities in the syncytiotrophoblast responsible for placenta barrier damage. These were as follows: decrease or disappearance of superficial microvilli, increase in basal membrane thickness, decrease in the number or lack of pinocytic vesicles in general, damage of mitochondria, presence of large lysosomes and diminution or enlargement of syncytiotrophoblast thickness.

These features were present in different combinations and it is unquestionable that they indicated the handicapped exchange between the mother and fetal blood.

In the gestosis, cholestasis and type A diabetes of pregnant women there was observed lack or large decrease in microvilli number on the syncytiotrophoblast surface, and also enlargement of basal membrane thickness. The consequence of these changes is the reduction of resorptive capability from mother's blood and also defect in metabolite transfer to villous capillaries.

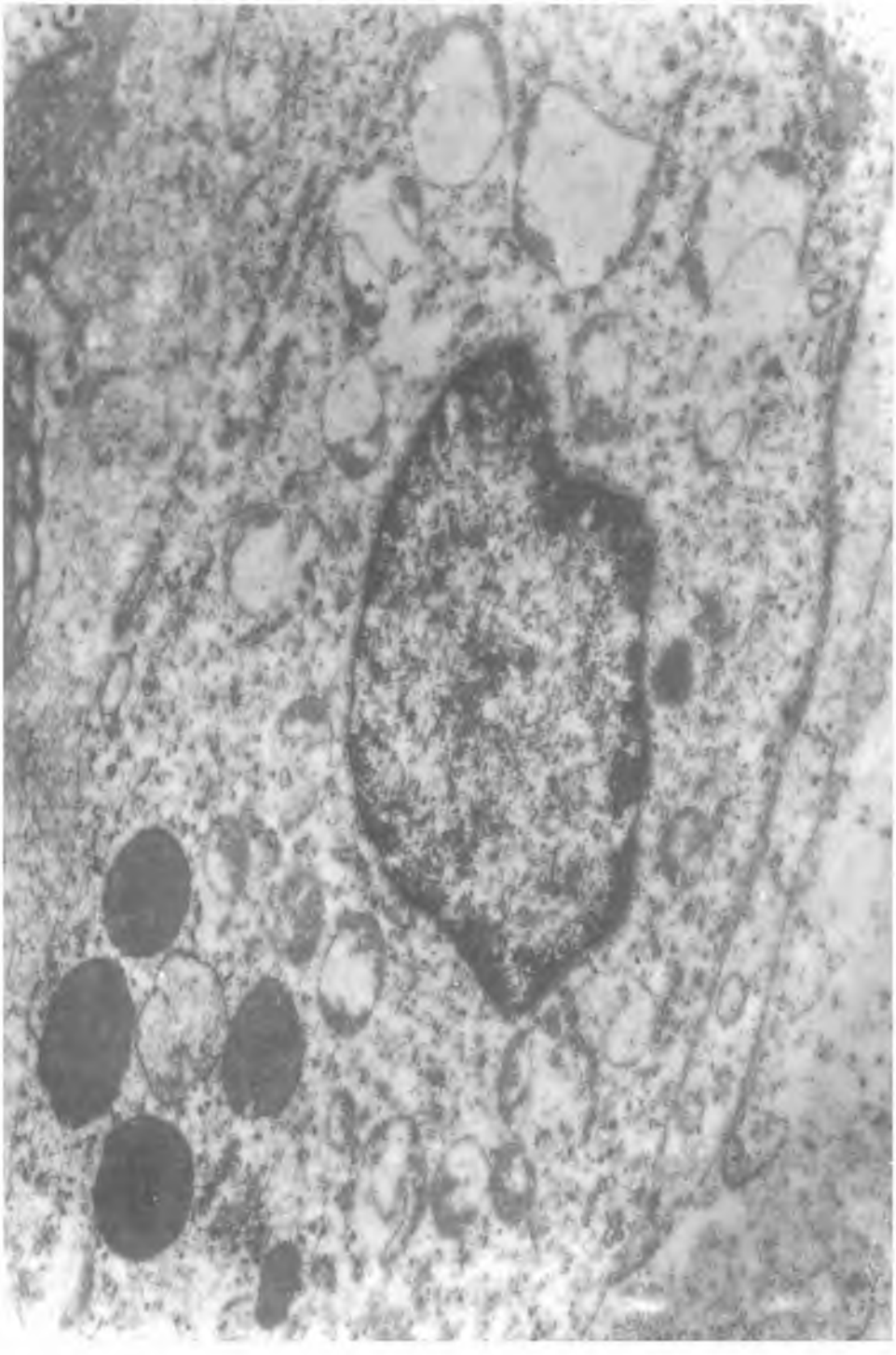

Fig. 2. Type A diabetes pregnancies. In the thick syncytiotrophoblast numerous damaged mitochondria and electron dense lysosomes are visible. Magn. approx. 8,000x

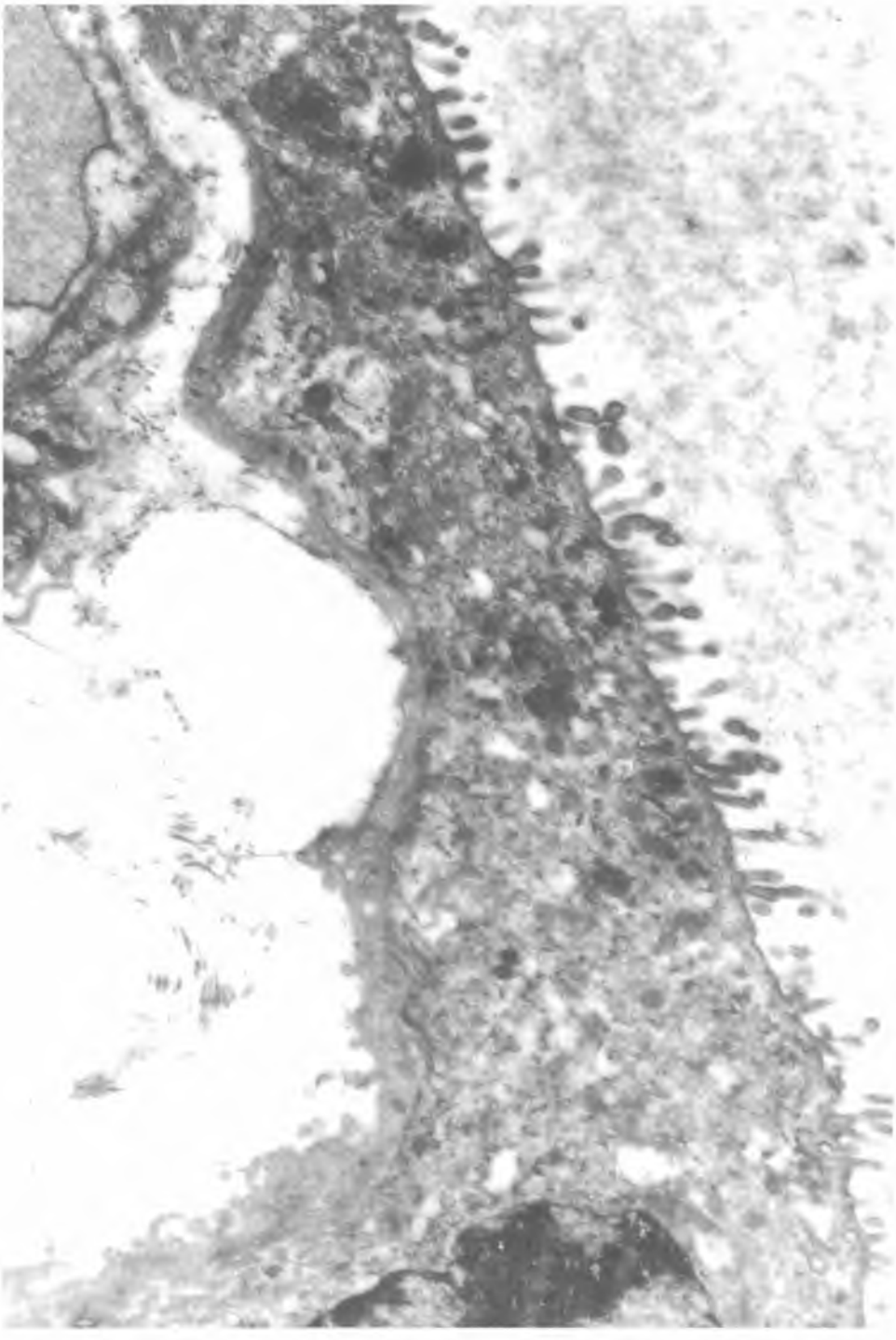

Fig. 3. Premature outflow of amniotic fluids. In the thin syncytiotrophoblast well developed microvilli and lack of pinocytic vesicles are observed. The thick basal membrane separates the syncytiotrophoblast from the electron loose stroma containing a lot of connective tissue fibres. Magn. approx. 8,000x

Basal membrane thickening and reduction of syncytiotrophoblast thickness is often observed in pathological placentas mainly in pre-eclampsia (5). These changes and also microvillous abnormality were observed in the placenta of smoking mothers (2).

One may think that basal membrane thickness growth is caused by the presence of toxic compounds in mother blood, howerer thickness growth of syncytiotrophoblast in mothers with non-insulin dependent diabetes may be associated with the disturbance of processes leading to its maturation (3, 4, 5), or cytotrophoblast reparative hyperplasia (1).

The presence of pathological mitochondria and of large lysosomes indicates damage of this part of the placenta barrier (2).

CONCLUSIONS

1. In different types of pathological pregnancy the following changes in the syncytiotrophoblast layer are observed: a) microvillous atrophy, b) enlargement or diminution of syncytiotrophoblast thickness, c) enlargement of basal membrane thickness, d) decrease in the number or lack of pinocytic vesicles, e) damage of mitochondria and increase in lysosome activity.

2. These changes appear in different combinations depending on the pregnancy pathology.

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SUMMARY

The studies were performed on 12 placentas from pathological pregnancies (gestosis, cholestasis, type A diabetes pregnancies, premature outflow of amniotic fluids) and 3 placentas from normal pregnancies. Ultrathin sections were stained by the Reynold's method and evaluated under the electron microscope BS-500 (Tesla). The changes found in syncytiotrophoblast were the following: the increase

or the decrease in its thickness, increase in basal membrane thickness, decrease in the number or lack of pinocytic vesicles and superficial microvilli, damage of mitochondria.

Zmiany ultrastrukturalne syncytiotrofoblastu w niektórych typach patologicznej ciąży

Badania wykonano na 12 łożyskach pochodzących z patologicznych ciąż (gestoza, cholestaza, cukrzyca ciężarnych typu A, przedwczesne odpływanie wód płodowych) i 3 łożyskach prawidłowych. Ultracienkie skrawki barwiono według metody Reynoldsa i oceniano w mikroskopie elektronowym BS--500 firmy Tesla. W syncytiotrofoblaście stwierdzono następujące zmiany: zwiększenie lub zmniejszenie jego grubości, zwiększenie grubości błony podstawnej, zmniejszenie liczby lub brak pęcherzyków pinocytarnych i mikrokosmków na powierzchni, uszkodzenie mitochondriów.