Institute of Experimental Morphology and Anthropology with Museum
Bulgarian Anatomical Society

Acta morphologica et anthropologica, 13
Sofia • 2008

Ultrastructural Peculiarities in the Stroma of the Thyroid Gland with Struma Nodosa

*K. Vidinov, N. Vidinov, M. Kalniev**

Department of Endocrinology and Gerontology, Medical University
**Department of Anatomy, Medical University, Sofia*

The follicules of the thyroid gland are surrounded by connective tissue stroma, which is not well investigated. To fulfill our aim we studied ultrastructurally the operative tissue from Struma nodosa patients. We compared fibroblasts next to active thyrocytes and those near epithelial cells with low activity. Our results point out in stages of thyroid activity the follicular cells show hypertrophy endocytosis complex lysosome activities. The fine basal lamina can be seen. In the fibroblasts from the septum larger and deformed GER, bigger Goldgy apparatus, and number of primary lysosomes can be seen. In the extracellular matrix we found an increased amount of collagen bundles type I as well as proteoglycans. The connective tissue of II group was larger in the septums and in the interlobular space. The number of hypertrophical cells enlargement. The fibroblast from these group were activated with large GER, inormaly large Goldgy complex and lysosomes.

Key words: stroma, thyroid gland, ultrastructure.

The follicules of the thyroid gland are surrounded by connective tissue stroma in which blood and lymphatic vessels are situated. There are many investigations concerned the thyrocytes of the thyroid gland [1, 2, 3, 9, 10], but there are no investigations about connective tissue elements in the gland.

Purpose

Our purpose is to follow the electron microscopical alterations in the connective tissue stroma between the thyroid follicules. To fulfill our aim we studied ultrastructurally the operative tissue from Struma nodosa patients. We compared fibroblasts next to active thyrocytes and those near epithelial cells with low activity.

Material and Methods

In order to do so we used material from 23 patients surgically operated for Struma nodosa (I group) and 4 patients operated for Hashimoto disease [II group]. The

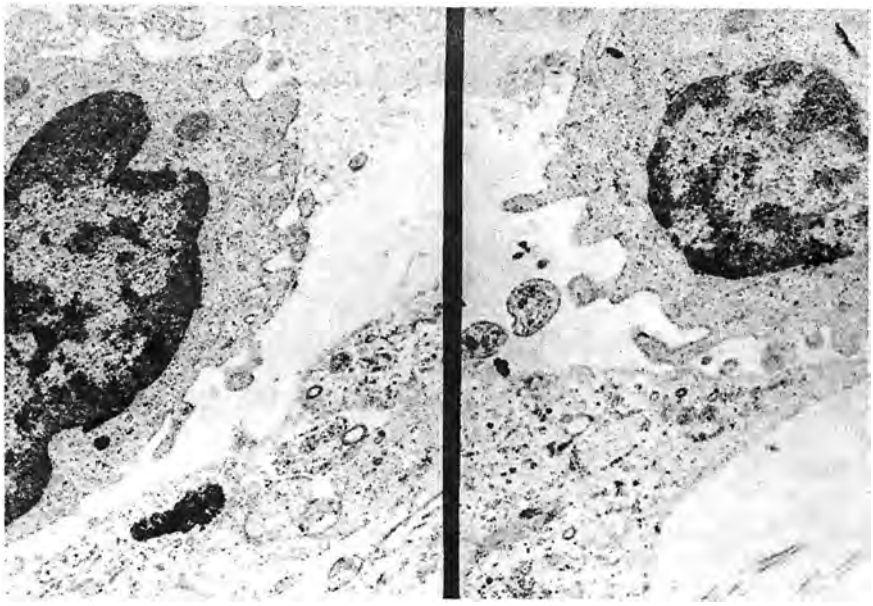

Fig. 1. Cells of I group. A larger and deformed GER and number of primary lysosomes can be seen in them. × 12 000

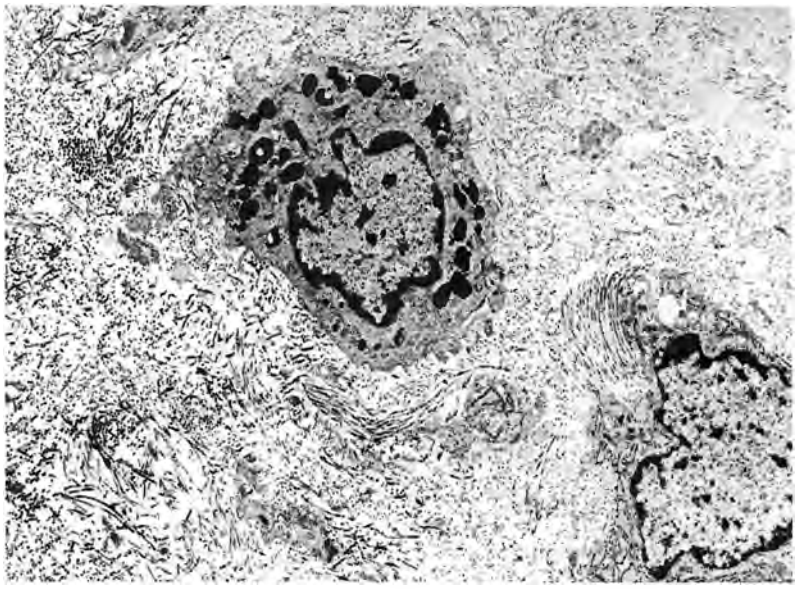

Fig. 2. Cells from II group. The fibroblast from these group were activated with large GER, large Goldgy complex and many complex lysosome. × 8400

materials were used for light microscopical investigations staining with hematoxilin-eosin and by Masson. Electron microscopical investigations was made by standard electron microscopy as well as Safranin O staining [8].

Results

The follicules of the thyroid gland from the I group were with very different size. The connective tissue stroma was a thin layer in the septa of the follicules and as a group of cells in the interlobular space. Our results point out in stages of thyroid activity the follicular cells show hypertrophy endocytosis complex lysosome activities. The fine basal lamina can be seen. In the fibroblasts from the septum larger and deformed GER, bigger Goldgy apparatus, and number of primary lysosomes can be seen (Fig.1). In the extracellular matrix we found an increased amount of collagen bundles type I as well as proteoglycans. The connective tissue of II group was larger in the septums and in the interlobular space. The number of hypertrophical cells enlargement. The fibroblast from these group were activated with large GER, abnormally large Goldgy complex and lysosomes (Fig. 2).

Conclusion

The results from the ultrastructural findings have to be considered in regard to the participation of the connective tissue in the gland metabolic changes (5, 6, 7) and in the alterations of the epithelial cells [4].

References

1. Beamont, A., P. Fragu.Ultrastructural changes in rat thyroid gland during iodine deficiency. — Bioll. Cell, **54**, 1985, 177-180.
2. Chen, H., D. Hayakawa, S. Emura. Effects of ethanolol on the ultrastructure of the hamster thyroid cells. — Histol. Histopathol., **15**, 2000, 469-474.
3. Davidson, N. Ultrastructure of thyroid C cells in sheep treated with vit D3. — J.Vet. Med Sci., **53**, 1991, No5, 921-927.
4. Hoang-Vu, C., G. Barabant, A. Leitolf, H. Von Muhlen. Functional and morphological changes of the thyroid gland following 5 days of pulsative TRH stimulation in male rats. — Journal of Endocrinology, **146**, 1995, 339-348.
5. Kniec, B. Histoenzimatic investigations of the thyroid gland of guinea pig. — In: Proc. 33 Conference of the Polish Histochemical Soc.
6. Lawson, V., F. Carrick. Morphology of thyroid in coastal and noncoastal population of the koala in Queensland. — General and Comparative Endocrinology, **110**, 1998, 295-306.
7. Lee, T., S. Kuo, T. Yah. Reversible systolic heart failure in hyperthyroidism. — Am. J. Med. Sci., **312**, 1996, 246-248.
8. Shepard, N., N. Mitchel. The location of proteoglycans by light and electron microscopy using Safranin O. — J. Ultruct. Res., **54**, 1976, 451-460.
9. Shin, W., B. Aftalion, E. Hotchkiss, E. Shenkman. Ultrastructure of a primary fibrosarcoma of the human thyroid gland. — Cancer, **44**, 1979, 584-591.
10. Werthington, B., C. Enwonwu. Functional variations in the ultrastructure of the thyroid gland in malnutrished infant monkeys. — Am. J. Clin. Nutrition, **28**, 1975, 66-75.

Institute of Experimental Morphology and Anthropology with Museum
Bulgarian Anatomical Society

Acta morphologica et anthropologica, 13
Sofia • 2008

Ultrastructural Characteristics of the Connective Tissue Elements in PVR and PDR

C. Vidinova, N. Vidinov, K. Michailova*

Clinic of Ophthalmology, Military Medical Academy, Sofia
Department of Anatomy, Medical University, Sofia

Diabetic retinopathy (PDR) and Proliferative vitreoretinopathy (PVR) are characterized by the formation of fibrous epiretinal membranes (ERMs) at the vitreoretinal interface that are due to the excessive proliferation, migration and differentiation of several cell types and they are causes of blindness. Much investigated PVR is still poorly understood. Its pathological mechanisms are not yet very clear. Similar pathological changes occur at the end stage of Proliferative diabetic retinopathy.
The purpose of our study was to compare the ultrastructure of the epiretinal membranes in PVR with those of the proliferations in PDR patients. The results from our investigations showed that in the membranes of the patients with PVR mainly two types of cells prevailed: the elongated fusiform-shaped, fibroblasts and the retinal pigment epithelial cells. Occasionally transitional forms with mesenchymal origin, glial cells and macrophages were seen in the membranes. In the proliferations of PDR patients we found significant differences. The main cell types comprising these membranes unlike PVR were: macrophages, fibroblasts, glial cells and other elements of the blood. In contrast to the previous group pigment epithelial cells were very rare to find. In these fibrovascular proliferations different types of capillaries were seen embedded in the fibrous tissue.

Key words: ultrastructure, PVR, PDR.

Introduction

One of the most common causes for ablations retinae are Diabetic retinopathy (PDR) and Proliferative vitreoretinopathy (PVR). PVR is characterized by the formation of fibrous epiretinal membranes (ERMs) at the vitreoretinal interface that are due to the excessive proliferation, migration and differentiation of several cell types [1, 5, 6, 8]. They usually tend to contract and provoke tractional retinal detachments, leading to impairment of the visual acuity and often blindness. Much investigated PVR is still poorly understood. Its pathological mechanisms and risk factors are not yet very clear.

Similar pathological changes occur at the end stage of Proliferative diabetic retinopathy, which is one of the major causes of blindness all over the world [10, 11]. It usually affects young people with type I diabetes and its hallmarks are the neovascular proliferations in the posterior pole, often related with bleeding, retinal tractions and visual loss.

Purpose

The purpose of our study was to compare the ultrastructure of the epiretinal membranes in PVR with that of the proliferations in PDR patients.

Material and Methods

In our prospective study 32 patients, 21 with PVR CP 1-4 and 11 with PDR were included. All of them after undertaking a precise ophthalmologic examination were operated with pars plana vitrectomy. During the operation proliferative tissue was collected and used for transmission and scanning electron microscopy according to the routine techniques.

Results

The results from our investigations showed that in the membranes of the patients with PVR (CP 1-4) mainly two types of cells prevailed: the elongated fusiform-shaped, fibroblasts and the retinal pigment epithelial cells. Occasionally transitional forms with mesenchymal origin, glial cells and macrophages were seen in the membranes. The cells were usually situated close to one another and the connections between them were strong, the so called "tight junction" type. The retinal pigment epithelial cells (RPE) were either among the fibroblast-like or in clusters. They were more or less cubuoid or oval in shape. Various amounts of electron microscopically dense granules, with average size — melanin granules were seen in the cell cytoplasm. In the extracellular matrix we observed collagen bundles with different length —

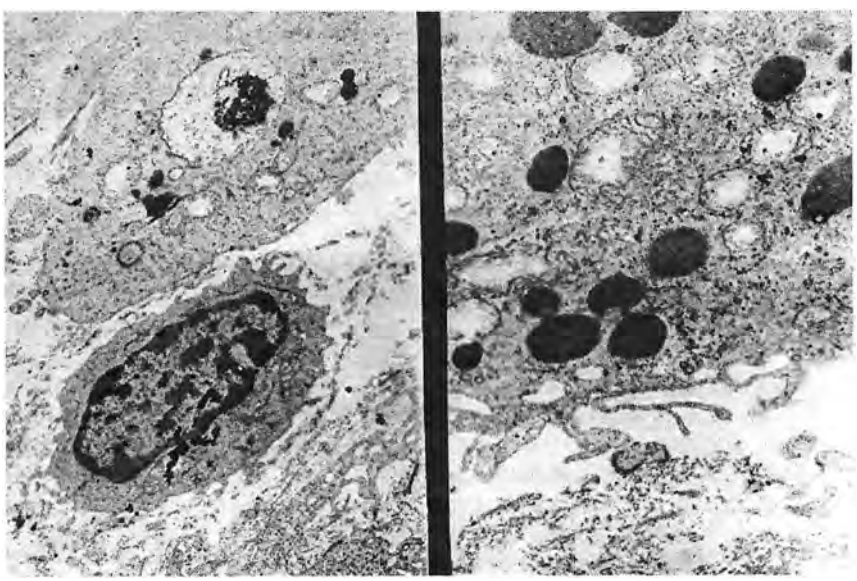

Fig. 1. Macrophages from PVR-membranes. There are many complex lysosomes and phagosomes. × 10 000

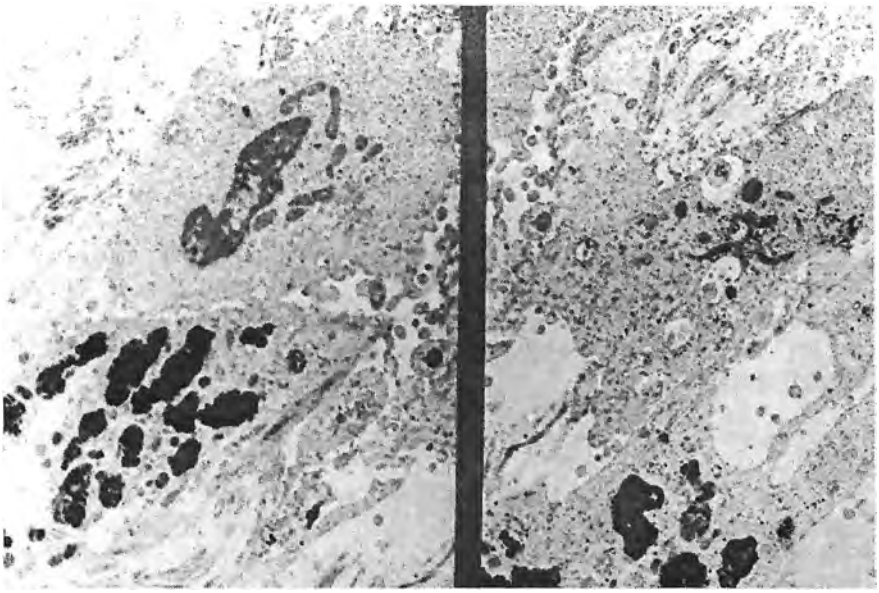

Fig. 2. Cells from PDR membranes. Degenerated fibroblasts and pigment cells can be seen. × 10 000

mainly type II collagen. In the proliferations of PDR patients we found significant differences. The main cell types comprising these membranes unlike PVR were: macrophages, fibroblasts, glial cells and other elements of the blood. In contrast to the previous group pigment epithelial cells were very rare to find. In these fibrovascular proliferations different types of capillaries were seen embedded in the fibrous tissue. They were generally two types: young — with a small lumen, no basal lamina, thin layer of fenestrated endothelial cells, and the mature capillaries with a medium size lumen — comprised of thin layer endothelial cells, developed basal membrane, containing formal elements of the blood.

Discussion

Our results point out that the proliferative tissues in PVR and PDR differ considerably in their cellular and extracellular content. This to a certain extent is expected as the pathogenesis of the two illnesses differs considerably. While in PVR mainly processes of cell migration, cell dedifferentiation and proliferation are having a key role in the pathogenesis, in PDR the triggering mechanism is the formation of the new vessels (2, 3, 4). That is why PVR membranes in their cytoarchitecture are richer of fibrous elements, migrated RPE cells and cells undergoing transdifferentiation (5, 7, 9). On the other hand, in PDR membranes fibrous tissue follows the developing new vessels and is richer of elements typical for the blood: macrophages, segment nuclear leucocytes etc.

References

1. Ayo, S. H., R. Radnik. W. F. Glass II, J. A. Garoni, E. R. Rampt, D. R. Appling, J. R. Kreisberg. Incresed extracellular matrix synthesis and mRNA in mesangial cells grown in high-glucose medium. — Am. J. Physiol., **260**, 1991, F185-F191.
2. Balian, G., E. M. Click, P. Bornstein. Location of a collagen binding domain in fibronectin. — J. Biol. Chem., **255**, 1980, 3234-3236.
3. Cagliero, E., T. Roth, S. Roy. Characteristics and mechanisms of high glucose-induced overexpression of basement membrane components in cultured endothelial cells. — Diabetes, **40**, 1991, 102-110.
4. Casaroli Marano, R. P., S. Vilaro. The role of fibronectin, laminin, vitronectin and their receptors on cellular adhesion in PVR. — Invest. Ophthalmology Vis. Sci., **35**, 1994, 2791-2801.
5. Charteris, D. Proliferative vitreorethinopaty: pathology, surgical managment, and adjunctive treatment. — British J.Ophtalm., **79**, 1995, 953-960.
6. Hiscott, P. S., I. Grierson, D. McLeon. Natural history of fibrocellular epiretinal membranes: a quantitative, autoradiographic, and immunohistichemical study. — British J. Ophtalmol., **69**, 1985, 810-823.
7. Hiscott, P. S., I. Grierson, C. J. Trombetta, A. H. S. Rahi, J. Marshall, D. McLeon. Retinal and epiretinal glia: An immunohistochemical study. — British J. Ophthalmol., **68**, 1984, 698-707.
8. Kampik, A., K. Kenion, R. Michels, W. Green, Z. Criz. Epiretinal and vitreous membranes: Comparative study of 56 cases. — Arch. Ophthalmol., **99**, 1981, 1445-1454.
9. Koteliansky, V. E., M. A. Glukhova, M. V. Bejanian. A study of the structure of fibronectin. — Eur. J .Biochem., **119**, 1981, 619-624.
10. Pillat, M., A. Kapetanios, G. Donati, M. Redard. TGF-b1, TGF-b receptorII and ED-A fibronectin expression in myofibroblast of vitreoretinopathy. — IOVS, **41**, 2000, 2336-2342.
11. Sayon, R., E. Cagliero, M. Lorenzi. Fibronectin overexpression in retinal microvessels of patients with Diabetes. — Invest. Ophth. Vis. Science, **37**, 1996, p. 2.