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Kierownik: dr hab. Teresa Bachanek

IWONA GRAJEWSKA

Ultrastructural study of the dental pulp in patients with adult periodontitis

Badania ultrastrukturalne miazgi zęba u pacjentów z zapaleniem przyzębia dorosłych

Periodontal diseases are long–lasting pathological processes occurring in the gum or other periodontium tissues (9, 11, 22). One of the periodontium diseases is adult periodontitis in whose etiopathogenesis the essential role is played by the inflammation factor related to the bacteria present on the dental plaque (4, 8, 20).

The question which remains still open is the influence of inflammation factors on the dental plaque which is of major importance in impulse receiving and is highly sensitive to numerous factors, both internal and external ones (3, 5, 18, 24). Thus the present work aimed at assessing the structural changes in the tooth pulp taken from the patients suffering from adult periodontitis.

MATERIAL AND METHODS

Patients suffering from adult periodontitis have been examined. Their teeth were extracted because of the advanced inflammation, tooth looseness and for prosthetic reasons. The patients were between 35 and 45 years of age. Two groups were distinguished: group one (I) with the mild course of the disease, and group two (II) revealing the andvanced disease.

Some patients turned up with sound periodontium, and in some cases teeth were extracted for prosthetic reasons or as a result of mechanical injuries of jaw bones – they made a control group.

The teeth were sectioned along their longitudinal axis and the dental pulp, which was to be the subject to ultrastructural studies, was picked out with a preparation needle. The pulp was prepared by fixing it in 4 per cent solution of glutarate alcohol in pH 7,4 phosphatic buffer during the period of 3–5 hrs, and next in 1 per cent solution of osmium tetroxide (OsO_4) for 1 hr. After dehydration the material was embedded in epon resin. Ultrathin specimens were made with the help of Reichert Om–U3 ultramicrotom. Counterstaining with uranyl acetate and lead citrate was applied. The specimens were investigated and pictures were taken in Tesla BS–500 transmission electron microscope.

RESULTS

On ultrathin sections pictures of dental pulp were estimated in the control group and the two groups with adult periodontitis (AP-1 and AP-2): Group I – mild form of the disease – AP-1, Group II – advanced form of the disease – AP-2.

Control group

The dental pulp taken from the people with healthy periodontium did not reveal in their microscope picture any deviation from the normal state (Fig. 1). Attention should be paid to odontoblasts with big oval nuclei, their chromatin accumulated under the nuclear sheath. Their cytoplasm contained mitochondria, endoplasmic reticulum and polyribosomes. It could be observed that in the subodontoblastic area (the zone of Weil) there were numerous nerve fibres with a dark myelin sheath, Schwann cells, and collagen fibres (Fig. 2). In the pulp core an abundant amount of star– or spindle–shaped fibroblasts was present. Their nuclei were oval or elongated and contained agglomerations of chromatin under the nucleus sheath (Fig. 3). In their cytoplasm cell organelles were visible: mitochondria, lysosomes, granular and smooth endoplasmic reticulum. Between the cells there were streaks of collagen fibres and numerous blood vessels.

Group I studied – AP-1

During the initial inflammation period of adult periodontitis, insignificant changes occurred in the dentinogenic cells – odontoblasts. The shape of their nuclei was changed, frequently indentation of the nuclear membrane (Fig. 4) could be observed. The cytoplasm revealed numerous vacuoles, swollen mitochondria and dilated reticulum.

Destructive changes were observed in the nerve fibres. They referred to the myelin sheath in which myelin bodies were formed (Fig. 5). Alterations in the walls of blood vessels applied to endothelium cells whose nucleus was deformed, and their cytoplasm contained numerous vacuoles and single mitochondria. Blood vessels were surrounded by a thick layer of collagen fibres (Fig. 6).

The number of fibroblasts in the pulp core was lower. Their processes were damaged, and the nuclei would change their shape and contain less chromatin (Fig. 7).

Group II studied – AP-2

In the period of advanced adult periodontitis a significant damage to odontoblasts could be observed. In their cytoplasm large vacuoles and swollen mitochon-

dria deprived of their crest occurred. The nuclei of some odontoblasts were deformed (Fig. 8).

Some major alterations were observed in the nerve fibres. Numerous, very big myelin bodies appeared in them (Fig. 9).

In the pulp core fibroblasts were destroyed. These cells were frequently decomposed. Their nuclei were homogeneous with a small amount of chromatin, the nuclear sheath was plicated, cytoplasm vacuolised, and the vacuoles themselves revealed various density degrees (Fig. 10).

DISCUSSION OF THE RESULTS AND CONCLUSIONS

The specific position of the dental pulp and its direct contact with the periodontium through the apical foramen and lateral and ventricular–periodontium canals suggest that the pathological processes taking place in periodontium may influence the pulp structure (5, 12, 14, 25).

While analysing the problem of periodontium diseases, and adult periodontitis in particular, attention was paid to ultrastructural alterations in the dental pulp resulting from this disease entity. The studies excluded the cases involving caries alterations. What was taken into consideration was not only the type of disease but also the degree of its progression.

In the case of adult periodontitis morphological alterations in the pulp were related to the degree of disease progression and to its course in individual patients. When the degree of progression was high, alterations of inflammatory character occurred in superficial and deep layers of the pulp as well as inflammatory focuses appeared. In several cases pulp stones and also large denticles were observed, which could be the symptoms of degenerative alterations of the pulp. Similar denticles were mentioned by Rydasz et al. (21) discussing the periodontium diseases, mainly as intraventricular denticles.

Particular consideration was given to the odontoblasts lying on the external surface of the pulp, which take part in the process connected with dentine creation and therefore provide the tooth with protection against inflammatory processes. The investigation of odontoblasts run by Barańska–Gachowska et al. (1), Grigorian et al. (7), Jansen (10), Negm et al. (17), and other authors (2, 6, 15, 16, 23) suggests that the former react to the alterations occurring in periodontium, as well as to other factors affecting the tooth (malocclusion, filling materials).

In our investigations the layer of odontoblasts in the course of adult periodontitis was damaged and limited to a small number of cells. It was observed that in the initial period of adult periodontitis – AP-1 alterations in odontoblasts involved the

shape of the nuclei, their chromatin location and indentation of the nuclear sheath. Within the cytoplasm of these cells vacuoles appeared. The mitochondria were swollen and the endoplasmic reticulum was widened. Slight destruction of the nerve fibres with myelin bodies occurring in them was observed. The fibroblasts of the pulp core revealed the signs of damaged processes and cell organelles. Their nuclei contained a small amount of chromatin nodules. Also the cells of the vascular endothelium were subject to degenerative alterations, which was described by C h e n et al. (3) and other authors (13, 19), with the pulp being ischemic.

Much more serious structural alterations were observed in the pulp cells, both odontoblasts and fibroblasts in the state of intensive, long–lasting inflammation (AP-2). The odontoblasts revealed major structural alterations in the nucleus and cytoplasm, which often led to their complete destruction. The destruction of nerve cells related to the presence of numerous myelin bodies took place. In the pulp core the destruction of fibroblasts occurred, along with vacuolisation of the cytoplasm bringing about the cell's decomposition.

On the basis of the obtained morphological, pictures of the pulp it should be concluded that at the initial stage of adult periodontitis – AP-1 alterations within the pulp are not significant and there is a possibility of their regression after the inflammation has disappeared. Whereas in the case of the disease in progression – AP-2 alterations of the pulp are significant and irreversible.

The present investigations have enabled us to explain to some extent the influence of adult periodontitis on morphological alterations in the pulp, and the range of these alterations may be different and depending on manifold factors.

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STRESZCZENIE

Badano wpływ zapalenia przyzębia dorosłych na zmiany ultrastrukturalne w miazdze zęba. Wyniki badań wskazują, że w początkowym okresie zapalenia przyzębia dorosłych (AP-1) zmiany morfologiczne w miazdze są niewielkie i istnieje możliwość ich cofnięcia się po ustąpieniu procesu zapalnego. Natomiast w przypadku zaawansowanego stanu chorobowego (AP-2) zmiany w miazdze są znaczne i nieodwracalne.

EXPLANATIONS TO FIGURES

Fig. 1. Control group. A layer of odontoblasts with oval nuclei (N) and chromatin nodules (Ch) at nuclear membrane. Capillary vessel (CA). Magn. x 4500.

Fig. 2. Control group. Nerve fibres with dark myelin sheath (Nm). Schwann cell (Le) with big oval nucleus (N). Collagen fibres (cF). Magn. x 4500.

Fig. 3. Control group. The pulp core. Fibroblasts (F) with elongated nuclei (N). Bundles of collagen fibres (oF). Magn. x 3600.

Fig. 4. Group I (AP-1). The odontoblast layer, nuclei with their shape alterned (N). Partial vacuolisation of the cytoplasm (V). Magn. x 3600.

Fig. 5. Group I (AP-1). Nerve fibre with a myelin body (CM). Cell vacuolisation (V). Magn. x 4500.

Fig. 6. Group II (AP-2). Change in the shape of the odontoblast nuclei (N), chromatin condensation (Ch), cytoplasm vacuolisation (V). Magn. x 3600.

Fig. 7. Group II (AP-2). Huge myelin bodies (CM) occur in the myelin sheath. Degenerated fibroblasts (F), abundance of collagen fibres (cF). Magn. x 5400.

Fig. 8. Group II (AP-2). Destruction of fibroblasts (F), nucleus (N), vacuolisation of cytoplasm (V). Magn. x 3600.

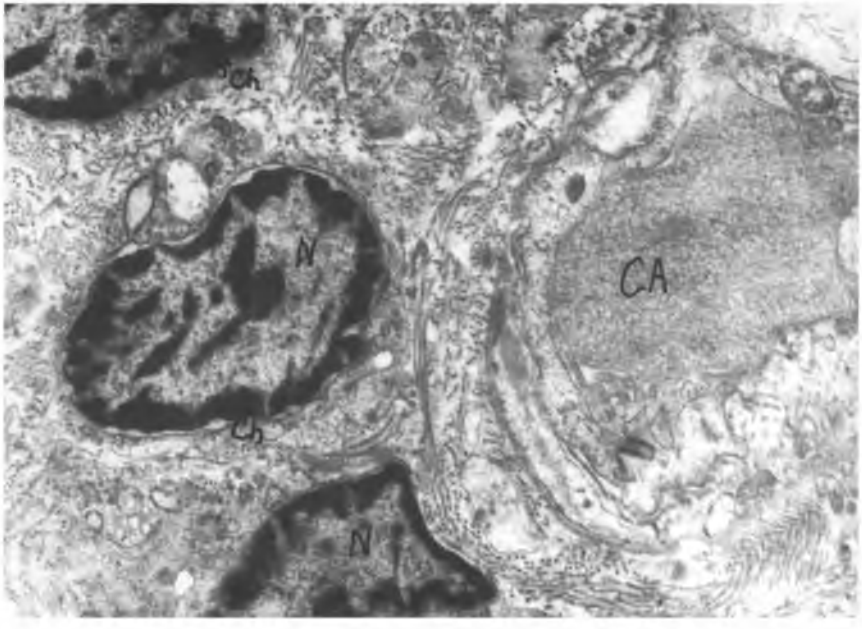
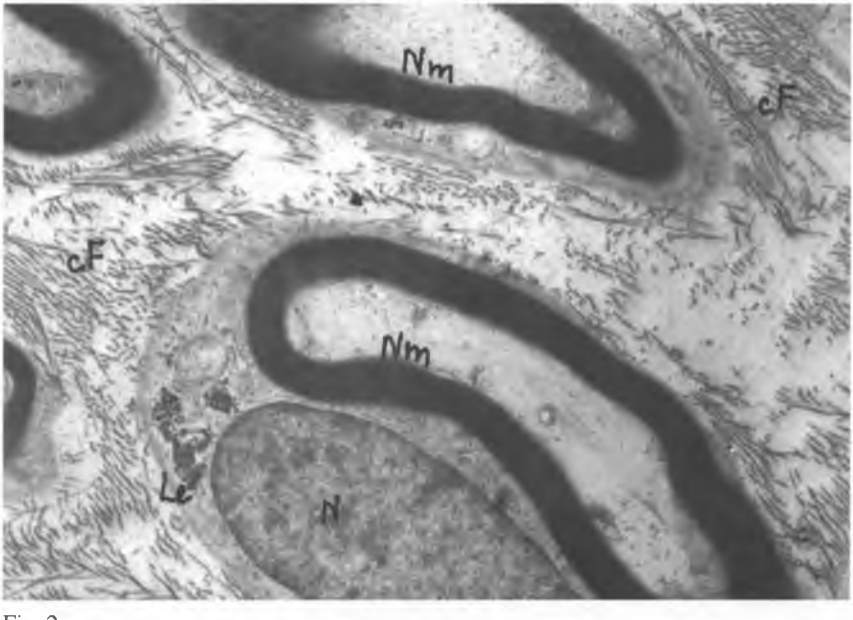


Fig. 1



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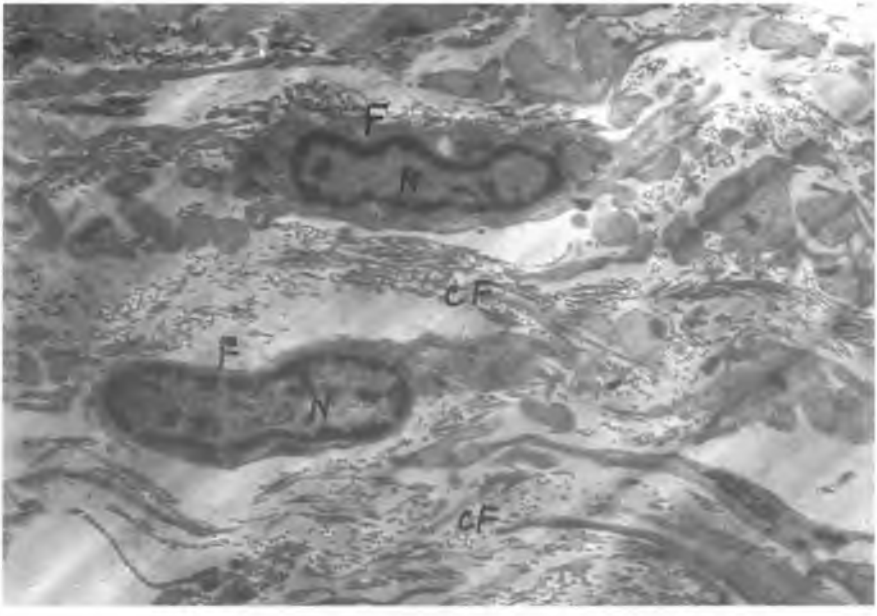

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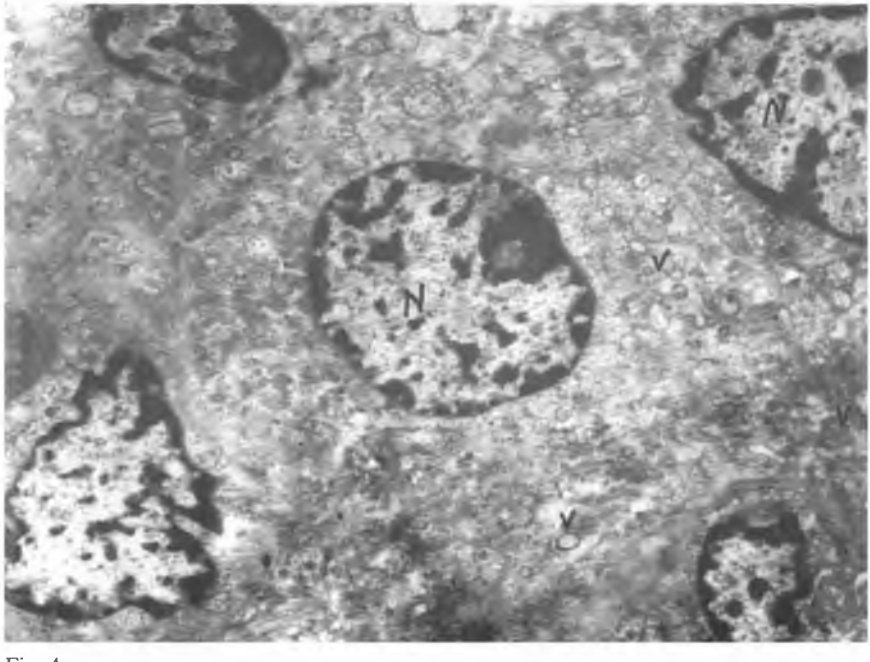
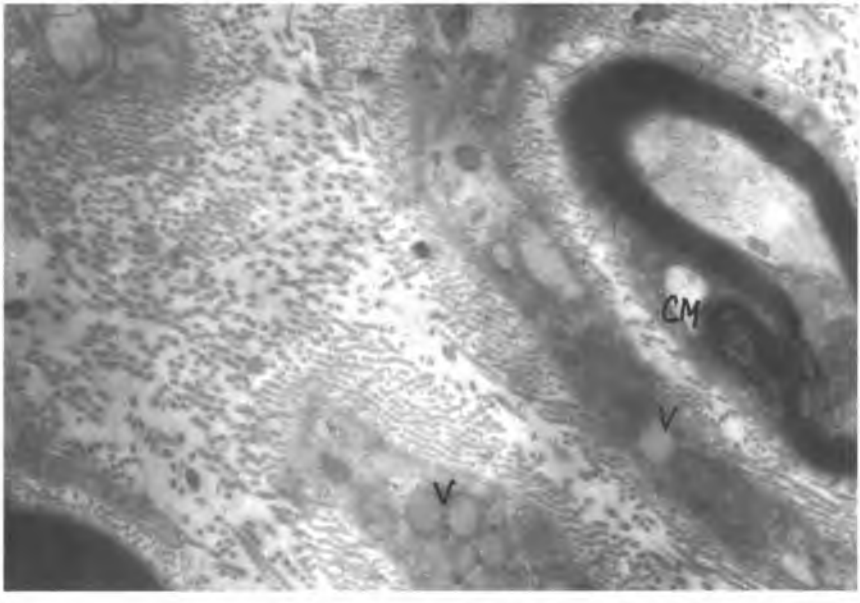


Fig. 4



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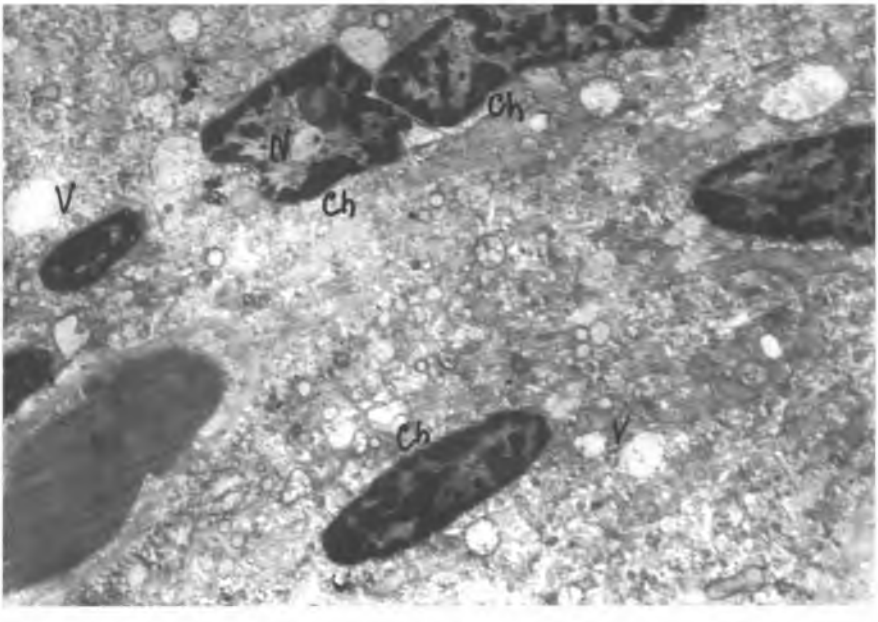

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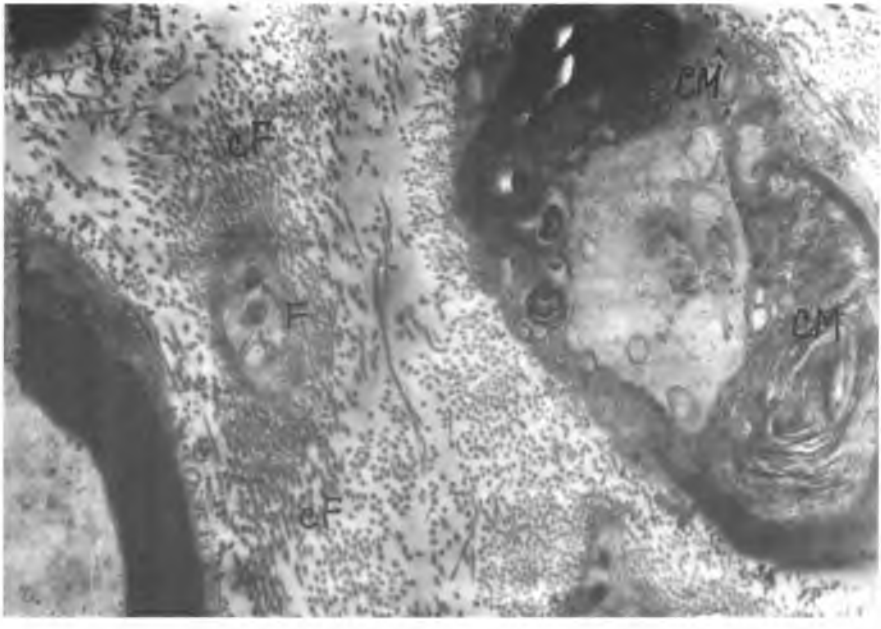

Fig. 7

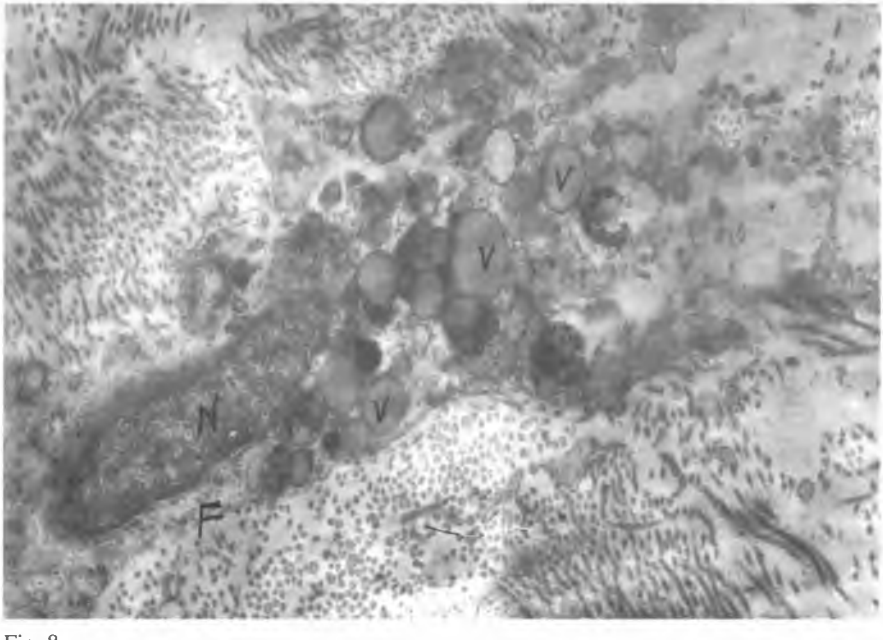

Fig. 8