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Myelin Fibers of Dental Pulp and their Partial Demyelinization at Acute Pulpitis

Tengis Zhvitiashvili*, Khatuna Davarashvili*

* *Department of Medicine, Zugdidi Teaching University*

(Presented by Academy Member Zurab Kevanishvili)

ABSTRACT. One of the factors of pain is damage of nerve fiber structure. Due to the fiber damage passive penetrability of Na^+ ion is increased, leading to the change of membrane polarization and additional local currents with pain syndrome strengthening. Thus two moments in the occurrence of pain syndrome play an important role: disfibering of myelin and growth of Na^+ -ion penetrability. *© 2011 Bull. Georg. Natl. Acad. Sci.*

Key words*: myelin, pulp.*

Myelin is a compact structure of nerve fiber consisting of membranes, winding around the axon.

Myelin-forming structures are oligodendrocytes and Schwann's cells. The latter take an active part in the creation of myelin peripheral nerve fibers. These cells are today considered to be analogues of oligodendroglyocytes [1]. Myelin fibers have the function of isolator, and have such a structure in which nerve impulses are conducted with larger speed than in nonmyelin fibers [2]. If the impulse distribution on the surface of myelin is uneven, it is connected with myelin fiber structure damage [3], the value of current potential in the nerve fiber itself is not changed. Due to the damage of nerve fiber structure leakage of ions via the damaged parts of the membrane occurs, which leads to the change of diffusion potential [4]. However, it should be noted that impulse transfer is determined not only by the state of membrane itself, but also the whole cell is involved in this process [5].

The aim of the work was to study myelin fibers in the norm and in cases of acute pulpitis. Comparison of the structures of nerve myelin fibers in the norm and clinical material is conducted. It allows to understand better the innervation of tooth and mechanism of pain syndrome.

Material and methods. Clinical material (10 cases), patients with acute pulpitis, was studied. Dental pulp was investigated. Dental pulps in five patients, who needed some orthopedic work to be done, practically healthy persons taken as control. All material was fixed in 1% solution of osmium on buffer. Fiber of the pulp was dehydrated and poured into epon. Blocks poured into epon were cut on ultratom OmU2 (Austria) and looked through under electron microscope BS-500, Tesla, in accelerating voltage 80 kV.

Discussion. Our observations, obtained with the help of electron microscopy, showed that myelin fibers at small increase of control material were represented by straight, parallelly situated lines homogeneous in diameter. Their edges were not coiled, well contoured. With clinical material it is vice versa. The edges of myelin fibers are strongly coiled and heterogeneous in diameter not only in comparison with different fibers but also each fiber separately, the whole length of which was of heterogeneous diameter. Thus strongly swollen places were detected which alternate with narrow constrictions of fiber.

The edges of fibers are strongly coiled. All the fibers are well contoured. Some fibers take larger field of obser-

vation, some have only dotty area. In the control material myelin fibers are uniformly osmiophilled, while in the clinical material nonuniform myelin osmiophility is noted. A great number of white (osmiophobe) places located on the edges of myelin fibers are seen in the clinical material.

Study of the material at big magnification showed that in control cases myelin fibers were represented by a well contoured uniform osmiophil membranes, parallelly directed to each other. The external edge of fibers is hardened. In the clinical material external edge of myelin fibers is contoured nonuniformly, often it is washed out, membranes are of nonuniform osmiophility. Whereas the thickness of myelin layer in the norm for the myelin fiber made up 25-30 A, in the clinical material it made up 18-20 A. It should be underlined that membranes in clinical material were often slightly contoured. Membrane is coiled. In some places myelin fibers form protrusion, and are directed into axon. Whereas in the control the surface of myelin fibers is similar, in the clinical material there are a great number of places with disfibered parts of myelin membrane. Myelin fibers are disfibered nonuniformly. In some places the fibers are agglutinated. In single cases myelin defibration is observed in the inner part of myelin membrane. Between glued fibers there are light osmiophil places. As to the axon, some ellipsoid-form light (osmiophobic), well-contoured formations are detected. In the central part of these formations different nonuniformly contoured multiformed plaques are seen.

Plaques on myelin fibers are represented in two type structures: dense (dark) and light (honeycomb). Dense (dark) plaques were mostly noticed on the surface of myelin fibers. Light (honeycomb) plaques were observed only along the inner edges of myelin fibers, deeply entering the axon. They consisted of partitions of different diameters and looked like bee honeycombs. On the surface of honeycombs there were tiny dark spots at equal distance from each other. Honeycombs were united into bunches, which were surrounded with close band. Bunches were also joined with each other into one general cell.

The presence of plaques changes the diameter of axon, which leads to a change of speed and rate of impulses running along the myelin fiber.

Plaques are represented by elements of cytoskeleton: micropipes, microfilaments and neurofilaments. Neurofilaments consist of proteins NF-L (light), NF-M (middle) and NF-H (high). The tinctorial properties of plaques are determined by the number of neurofilaments. Neurifilaments, micropipes and microfilaments form three-dimensional network of cytoskeleton. Cytoskeleton creates firmness and ensures intracellular transport, etc. Micropipes are of dynamic structure, they consist of α and β tubulins and kinesins. Micropipes are characterized by polarity, i.e. in each micropipe there is an end to which new subunits of tubulin profilaments and n(-) end where tubulin subunits are divided, creating new structures, can join.

Analysing the data obtained from the position of functional abilities of myelin fibers it follows that impulse conduction along them in norm and clinical material must be different, which, in its turn, is connected with changes of the fiber itself. Disfibered places of myelin fiber, observed with clinical material, most probably, must perform the same function as Ranvier's constrictions. It should be assumed that in the area of Ranvier's constrictions strengthening of nerve impulse occurs. According to H.Jost [5] constrictions play the role of "relay" of local current. In clinical material due to damage of myelin structure, the number of relay points increases (Ranvier's constrictions+places with distfibered myelin playing the role of constrictions), which leads to the growth of relay places and then to the value of saltatory, i.e. jumping relay current, perceived by us as a pain syndrome.

Due to damage of structure of nerve fiber passive penetrability for Na^+ ions is increased, leading to the change at membrane polarity. Additional local currents appear and pain the syndrome increases. Thus, two points are very important: damage (disfibering of myelin) and growth of penetrability of ions of Na.

Some authors [6] connect pulpitis with acute enteric infections, caused by microbes and virus antigens. Penetrating into the organism they usually cause sickness, temperature increase, etc.

Todays enteric infections take second place in the world as to frequency after respiratory diseases. Parents are considered to be their infection sources. Antigen is propagated in epithelial fiber, the infection often assures latent character.

Patients often do not pay attention to caries and then pulpitis, odontitis, diseases which they can have for years. They should know that those diseases can be precursors of more complicated cases such as demyelinization of neuroglial elements in CNS, leading to grave pathologies.

სამედიცინო მეცნიერებანი

კბილის პულპის მიელინური ბოჭკოები და მათი ნაწილობრივი დემიელინიზაცია

თ. ჟვიტიაშვილი*, ხ. დავარაშვილი*

** ზუგდიდის პედაგოგიური უნივერსიტეტი, მედიცინის განყოფილება*

(*წარმოდგენილია აკადემიის წევრის ზ. ქევანიშვილის მიერ*)

ნაშრომში შესწავლილია კბილის პულპა მწვავე პულპიტის დროს. ნაჩვენებია, რომ მწვავე პულპიტის დროს აღინიშნება მიელინის ბოჭკოების ნაწილობრივი დემიელინიზაცია. ტკივილი დაკავშირებულია მიელინის ბოჭკოების დანგრევასთან და ნატრიუმისა და კალიუმის იონების პასიური განვლადობის მომატებასთან. ამ შემთხვევაში მატულობს სალტატორული მუხტის რაოდენობა. მიელინის განბოჭკოება მიუთითებს ციტოპათოგენურ ეფექტზე, რომელიც მკაფიოდ წარმოდგენილია შვანის უჯრედებში.

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