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# SOME ASPECTS OF MORPHO-FUNCTIONAL CHANGES IN ISCHIADIC NERVE DAMAGED BY E. COLI ENDOTOXIN

**Аннотация**

Бабаева М.Х., Гулиева Н.Т. Некоторые аспекты морфо-функциональных изменений седалищного нерва под действием эндотоксина E. coli

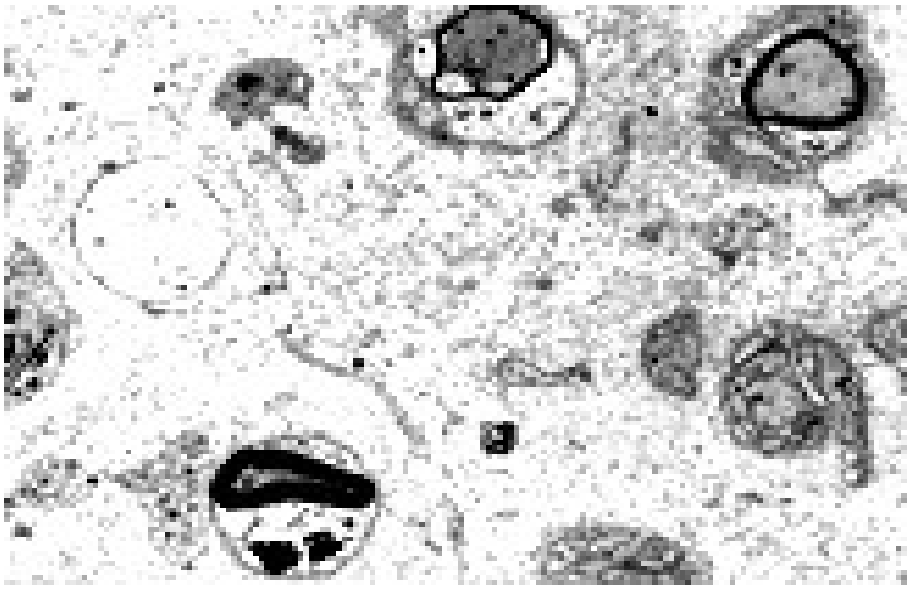
*Цель исследования патологических процессов периферического нерва, является установлением диагноза периферических невропатий, определением, является ли это аксональное повреждение или процесс демиелизации, и нахождением его причину. Изучая 150 волокон седалищного нерва зараженных с E.coli белых мышей, мы обнаружили сегментарный процесс демиелизации в 123 из них и также дегенеративный процесс подобно валерианному дегенерацию и патологические изменения аксонов одновременно с сегментарным процессом демиелизации в остальных 27 из них.*

***Background:*** Spreading of the peripheral nerve diseases shows that, studying of this field is very important. Damages of ischiadic nerve by bacterial endotoxin during the infectious diseases caused by E.coli are widespread too [1]. The goal of the investigation of peripheral nerve changes is to establish the diagnosis of peripheral neuropathy, determine if it is an axonal or demyelinative process, and find its cause.

***Materials and methods:*** For studying the morpho-functional changes in ischiadic nerve we took these nerves via surgical instruments from 150 white mice which were intravenously injected with purified E. coli (serotype 0111:B4; group # 78H4086) lipopolisaccaride (1,0 mg/ kg) and from 20 mice, which consist the control group. Infected mice were decapitated after 2 weeks. We can detect microcirculatory changes by electron microscopy even in Hu nerve fibers of the mice, which are decapitated 2 hours after injection [2]. Then we used hemotoxiline-eosin stain, made ultra thin sections of these nerve fibers and observed these sections microscopically. At the same time we used electron microscopy method to detect ultra structural changes in the nerve sections.

***Discussion:*** The structure of central and peripheral myelin is essentially the same. Myelin is composed of 70% lipids and 30% protein. There are some important differences in myelin proteins between central nervous system and peripheral nerve fibers. These differences explain why an allergic reaction against peripheral nerve myelin does not cause central demyelination and vice versa; and why inherited metabolic disorders of myelin proteins that affect peripheral nerves do not damage central myelin. On the other hand, lipids are similar between peripheral nerves myelin and the myelin of the central nervous system. For this reason, metabolic disorders of myelin lipids, such as metachromatic leucodystrophy, affect both, the central white matter and peripheral nerves.

The myelin sheath acts as an electrical insulator, preventing short-circuiting between axons. More important, it facilitates conduction. The nodes of Ranvier are the only points where the axon is uncovered by myelin and ions can be exchanged between it and the extracellular fluid. Depolarization of the axonal membrane at the nodes of Ranvier boosts the action potential that is transmitted along the axon and is the basis of salutatory (jumping) conduction**.**



**Demyelinization process in the nerve fiber.**

During the microscopically investigation in hemotoxiline-eosin stained sections which were taken from the control group mice we didn't detect any destructive change. We detected lympho-leucosyte infiltration and inflammatory changes in microcirculatory system of the nerve fibers from mice which injected with lipopolisaccaride [6]

Studying 150 nerve fibers, we detected segmental demyelination process in 123 (82%) of them and also degenerative process like Wallerian degeneration and pathologic changes of axons at the same time with segmental demyelination process in remaining 27 (18%).

In our investigations we detected the breakdown and loss of myelin over a few segments (as shown above in the picture). In 123 of our nerve fiber sections the axon remains intact and there is no change in the neuronal body as above-mentioned. But although the axonal axes are intact in this case salutatory conduction of nerve impulse is lost because of segmental demyelination. All these processes lead to decrease of conduction velocity and conduction block**.**

Deficits develop rapidly but are reversible because Schwann cells make new myelin. However, in many cases, demyelination leads to loss of axons and permanent deficits. The nerve, in segmental demyelination, shows demyelinated axons, thin - regenerating-myelin, "onion bulbs" and, in severe cases, loss of axons [4].

The status of myelin is evaluated with teased fiber preparations of peripheral nerves and by electron microscopy.

"Onion bulb" formations are concentric layers of Schwann cell processes and collagen around an axon. This proliferation is caused by repetitive segmental demyelination and regeneration of myelin and can cause gross thickening of ischiadic nerve. The central axon is often demyelinated or has a thin layer of myelin.

We also detected degenerative changes at the same time with demyelination in 27 of our nerve fibers. In these sections the neuronal cell body maintains the axon through the axoplasmic flow. When an axon is transected, its distal part, including the partly destroyed myelin sheath, had undergone a series of changes leading to its complete structural disintegration and chemical degradation.

Degeneration process of axon and myelin develops first in the most distal parts of the axon and, if the inflammatory process persists, the axon "dies back". Neurofilaments and organelles accumulate in the degenerating axon (probably due to stagnation of axoplasmic flow). Eventually the axon becomes atrophic and breaks down. Severe distal axonopathy resembles above mentioned degenerative process**.** At an advanced stage, there is loss of myelinated axons. Many clinically important neuropathies caused by drugs and industrial poisons such as pesticides, acrylamide, organic phosphates, and industrial solvents are also characterized by degenerative processes of axons [5].

Degenerative process is thought to be caused also by pathology of the neuronal body resulting in its inability to keep up with the metabolic demands of the axon. This explains why the disease begins in the most distal parts of nerves, and large axons that have the highest metabolic and nutritional demands are more severely affected as we detected in ischiadic nerve in our investigation. However, this question is not settled. It is hard to imagine how the relatively miniscule neuronal body can keep up with the metabolic demands of the enormous mass of the axon. Furthermore, the neuronal body is just as dependent on the distal axon and its synapses for trophic interactions that keep it alive and functioning [3].

We decided that the neuronal cell bodies are also damaged by E. coli endotoxin at the same time with the degradation processes in the nerve fibers. Demyelination process shows that pathologic process goes not only in the peripheral nerve fibers, but also in the neuronal cell bodies.

***Results****:* Studying 150 nerve fibers of ischiadic nerve we detected segmental demyelination process in 123 of them caused by E.coli endotoxin. And also degenerative processes like Wallerian degeneration at the same time pathological changes in axonsin 27 of them. We had come to conclusion that in the ischiadic nerves of infected by E.coli mice was gone both disintegrative, chemical degradative process in axons and demyelinating process at the same time with degeneration. And these kind of processes must be appeared in human organism by neurological symptoms.

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