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Oculomotor Disorders in Patients with Diabetes

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Introduction

The article focuses on oculomotor disorders in diabetic patients. Quite often diabetic patients have oculomotor neuropathies, both as mononeuropathy and multiple cranial neuropathies. 25% of all cases of paralysis of the eye muscles are due to diabetic neuropathy. The frequency of lesions of a group of oculomotor nerves in diabetes exceeds similar violations in patients without diabetes. Recently, due to several reasons the interest of neurologists to diabetes mellitus (DM) has greatly increased. The number of patients with damage to both central and peripheral nervous system caused by DM has also increased, which is associated with the rapid increase in the incidence of diabetes in the world and its transformation into a global epidemic. Diabetic patients have oculomotor neuropathies quite frequently, both as mononeuropathy and multiple cranial neuropathies. 25% of all cases of paralysis of the eye muscles are due to diabetic neuropathy [1].

Oculomotor muscles are innervated by three pairs of cranial nerves, the loss of any of them can cause diplopia in one or several gaze directions. An oculomotor nerve plays the most important role in the eye movement because it innervates the upper, lower and medial rectus muscles, the lower oblique muscle and the muscle that lifts the upper eyelid, and it also innervates the sphincter of the pupil and the ciliary muscle thus contracting the pupil and providing accommodation. So, with the damage of all fibers of oculomotor nerve most eye motor functions cease, with incomplete lesions some of the functions remain.

Complaints of double vision in a horizontal or oblique plane (no diplopia in ptosis) are very characteristic. Isolated lesion of abducens can be most easily detected. It is manifested through paresis of the lateral rectus muscle and restriction in eye abduction. The patient has horizontal diplopia that is worse while looking in the affected side.

Violation in eye abduction becomes visible when the patient looks in the affected direction. Under block nerve paralysis the patients complain of double vision in a vertical or oblique plane, which intensifies when looking down. Forced position of the head is also characteristic (at turn and lean in non-affected side), when diplopia weakens. The pathogenesis of diabetic neuropathy remains unclear. PNS defeat in diabetes is associated with microangiopathy which causes ischemic damage to nerves and is the main reason for acutely developing asymmetric neuropathies, and/or with metabolic disorders in neurons and lemmocytes which are characterized with accumulation of sorbitol or myoinositol deficient as a result of activation of polyol path in glucose utilization. This is particularly important in the development of symmetric slowly increasing polyneuropathies [2]. As a rule, the severity of diabetic neuropathy increases with the degree and duration of hyperglycemia.

Perhaps, decrease in the synthesis and violation of axonal transport of proteins to peripheral neurons play a certain role, as well as reduction in the activity of ion channels, violation in protein synthesis of myelin, non-enzymatic glycosylation of proteins in peripheral nerves. It is possible that genetic factors also play a certain role in the genesis of neuropathy, the importance of which in the pathogenesis of diabetes itself seems certain [3].

The first description of paresis of group of oculomotor nerves in diabetes was made in 1887 by J. Hirschberg, when the author mentioned a more frequently defeated abducens, and by 1905 Dieulafoy was able to generalize about 55 cases published in the literature of the defeat of this group of nerves and described 3 of his own observations. Dieulafoy and many other authors proved that most often abducens suffers, and less often the block one [4,5]. Under diabetes the frequency of lesion of a group of oculomotor nerves is according to various authors from 0.5 to 5% [6]. Among the examined 6520 patients the mentioned defeat was in 29 (0.44%): abducens in 18, oculomotor in 10 and the block one in 1 patient. Such a low percentage of defeat of this group of nerves (as well as the facial one) in the study is apparently because 3710 (57.2%) out of 6520 diabetic patients were under the age of 40.

The pathology of the mentioned nerves mainly occurs in patients over 50 (24 out of 29 patients mentioned above were over 50), which dominated in the majority of publications of most authors. The frequency of lesions in group of oculomotor nerves in diabetes exceeds similar violations in patients without diabetes where it is 0.2% [6].

This is also confirmed by high incidence of diabetes, which is, according to some authors, detected in 15.4% of patients with abducens paresis, in 20% of patients with lesions of the oculomotor nerve, and in 4.5% of patients with paresis of the eye muscles [7]. Paresis of the eye muscles in diabetes usually occur quickly, they are usually unilateral and are often accompanied by pain in the temporal region [8]. The authors have conducted a retrospective study of diabetic patients with ophthalmoplegia for 10 years, in total 6765 patients were hospitalized, ophthalmoplegia was found in 27 patients (0.40%) [9].

Isolated lesion of III nerve was observed in the majority of patients (59.3%), paresis of the VI cranial nerve occurred in 29.6% of patients and was observed more frequently than the lesion of all oculomotor nerves (11.1%) [9]. Typically, the lesion occurs in patients with non-severe diabetes, and often in latent diabetes. The forecast, according to most authors, is favorable enough, usually there is remission within a few weeks to 4 months, but cases of recurrent flow can also be observed [9,10]. As for the disorders of pupillary reactions, they were described in patients with diabetes by many authors [11-13].

These disorders are manifested through the symptom of Argyll Robertson (including one-way), an isolated violation of reaction to the accommodation, uneven pupils and a weak response to drugs that dilate the pupil.

The frequency of these violations, according to different authors, varies in patients with diabetic polyneuropathy from 9 to 24% [12].

The mechanism of onset of Argyll Robertson symptom in diabetes may be associated with retinal lesions. Thus, this symptom is observed in a few patients with proliferative retinopathy in which it could be "caused both by retinal disorders and lesions of the optic nerve fibers and the tract, as it is known that loss of light reflex may occur even at mild changes in the optic nerve [13,14].

Histopathological studies conducted in 1957 by P. Dreyfus et al. at the patient autopsy with a lesion of the oculomotor nerve found fusiform thickening of this retro-orbital part of the nerve. In the nerve center the necrosis was identified with the destruction of myelin and axial cylinders, as well as an increased amount of connective tissue. In the nucleus of the oculomotor nerve only changes of the retrograde nature were noted.

The authors found thickening of intraneural vessels and called for ischemic genesis of nerve lesion [14], a similar conclusion was reached by other authors [15].

Conclusion

It should be noted that the current understanding of the various forms of diabetes, detection and leveling of risk factors for diabetic complications, early detection of diabetic polyneuropathy, clinical observation and timely treatment of complications make it possible to maintain those functions for a long time.

Acknowledgement

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Conflict of Interest

No conflict of interest.

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