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FUNDUS CHANGES IN HYPERTENSION

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Abstract:

The frequency of eye fundus lesions in patients with essential hypertension (AH), according to various authors, varies from 50 to 95% [1]. This difference is partly due to age and clinical differences in the studied group of patients, but mainly due to the difficulty of interpreting the initial changes in the retinal vessels in hypertension. Internists attach great importance to such changes in the early diagnosis of GB, determining its stage and phase, as well as the effectiveness of the therapy. The most interesting in this regard are the studies of R. Salus. Under the conditions of a well-organized experiment, he showed that the diagnosis of HD, made by him according to the results of ophthalmoscopy, turned out to be correct only in 70% of cases. Errors in diagnosis are associated with significant individual variations in retinal vessels in healthy individuals, and some of the variants (relatively narrow arteries, increased tortuosity of vessels, the "crossover" symptom) can be misinterpreted as hypertensive changes.

Keywords: According to the observations of O.I. Shershevskaya [7], with a single check of an unselected group of patients with GB, specific changes in retinal vessels are not detected in 25–30% of them in the f functional period of the disease and in 5–10% in the late phase of the disease.

The vessels of the retina and optic nerve and the Central retinal artery (CAS) in its orbital section has a structure typical of medium-sized arteries. After passing the cribriform plate of the sclera, the thickness of the vascular wall is halved due to the thinning (from 20 to 10 microns) of all its layers. Within the eye, the CAC divides repeatedly dichotomously. Starting from the second bifurcation, the branches of the CAS lose their characteristic features of the arteries and turn into arterioles.

The supply of the intraocular part of the optic nerve is carried out mainly (with the exception of the neuroretinal layer of the optic disc) from the posterior ciliary arteries. Posterior to the cribriform plate of the sclera, the optic nerve is supplied with centrifugal arterial branches coming from the CAC and centropetal vessels coming from the ophthalmic artery.

The capillaries of the retina and optic disc have a lumen with a diameter of about 5 μm. They start from precapillary arterioles and join into venules. The endothelium of the capillaries of the retina and the optic nerve forms a continuous layer with tight junctions between cells. Retinal capillaries also have intramural pericytes, which are involved in the regulation of blood flow. The only blood collector for both the retina and the ONH is the central retinal vein (CRV). The adverse effect of various factors on the retinal circulation is smoothed out due to vascular autoregulation, which ensures optimal blood flow using local vascular mechanisms. Such blood flow ensures the normal course of metabolic processes in the retina and optic nerve.

Pathomorphology of retinal vessels in GB

Pathological changes in the initial transient stage of the disease are hypertrophy of the muscle layer and elastic structures in small arteries and arterioles. Stable arterial hypertension leads to hypoxia, endothelial dysfunction, plasma impregnation of the vascular wall, followed by hyalinosis and arteriolosclerosis [3]. In severe cases, fibrinoid necrosis of arterioles is accompanied by thrombosis, hemorrhages and microinfarctions of the retinal tissue.

Retinal vessels in GB.

Two vascular trees are clearly visible in the fundus: arterial and venous. It is necessary to distinguish: (1) the severity of each of them, (2) branching features, (3) the ratio of the caliber of arteries and veins, (4) the degree of tortuosity of individual branches, (5) the nature of the light reflex on the arteries.

The severity and richness of the arterial tree depend on the intensity of blood flow in the CAS, refraction, and the state of the vascular wall. The more intense

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the blood flow, the better the small arterial branches are visible and the more branched the vascular tree. In hypermetropia, retinal vessels appear wider and brighter on ophthalmoscopy than in emmetropia, while in myopia they become paler. Age-related thickening of the vascular wall makes small branches less noticeable, and the arterial tree of the fundus in the elderly looks depleted.

With GB, the arterial tree often looks poor due to tonic contraction of the arteries and sclerotic changes in their walls. Venous vessels, on the contrary, often become more pronounced and acquire a darker, more saturated color (Fig. 4, 1, 5). It should be noted that in some cases, provided that the elasticity of the vessels is preserved, in patients with HD, not only venous, but also arterial plethora is observed. Changes in the arterial and venous vascular bed are also manifested in a change in the arteriovenous ratio of retinal vessels. Normally, this ratio is approximately 2:3; in patients with GB, it often decreases due to narrowing of the arteries and dilatation of the veins (Fig. 1, 2, 5).

Narrowing of the retinal arterioles in HD is not an obligatory symptom. According to our observations [3], pronounced narrowing, which can be determined clinically, occurs only in half of the cases. Quite often only separate arterioles are narrowed (fig. 2, 5). Characterized by the unevenness of this symptom. It is manifested by the asymmetry of the state of the arteries in paired eyes, the narrowing of only individual vascular branches, and the uneven caliber of the same vessel. In the functional phase of the disease, these symptoms are caused by unequal tonic contraction of blood vessels, in the sclerotic phase - by uneven thickening of their walls.

Much less often than the narrowing of the arteries, with GB their expansion is observed. Sometimes both narrowing and dilation of arteries and veins can be seen in the same eye and even on the same vessel. In the latter case, the artery takes the form of an uneven chain with swellings and interceptions (Fig. 5, 7, 9).

One of the frequent symptoms of hypertensive angiopathy is a violation of the normal branching of the retinal arteries. Arteries usually branch dichotomously at an acute angle. Under the influence of increased pulse beats in hypertensive patients, this angle tends to increase, and it is often possible to see branching of the arteries at a right and even obtuse angle ("bull horns symptom", Fig. 3). The greater the branching angle, the greater the resistance to blood flow in this zone, the stronger the tendency to sclerotic changes, thrombosis and disruption of the integrity of the vascular wall. High arterial pressure and large pulse amplitude are accompanied by an increase not only in lateral, but also in longitudinal stretching of the vascular wall, which leads to elongation and tortuosity of the vessel (Fig. 5, 7, 9). In 10–20% of patients with GB, tortuosity of the perimacular venules is also observed (Guist symptom).

Significant significance for the diagnosis of hypertonic fundus is the symptom of the Gunn-Salus chiasm. The essence of the symptom lies in the fact that at the site of intersection with the compacted artery of the venous vessel, a partial squeezing of the latter occurs. There are three clinical degrees of this symptom (Fig. 4). The first degree is characterized by a narrowing of the lumen of the vein under the artery and near the junction of the vessels. A feature of the second degree is not only partial squeezing of the vein, but also its displacement to the side and into the thickness of the retina ("arc symptom"). The third degree of decussation of the vessels is also characterized by a symptom of the arch, but the vein under the artery is not visible and seems to be completely compressed. The symptom of decussation and venous compression is one of the most frequent in HD. However, this symptom can also be found in retinal arteriosclerosis without vascular hypertension.

Symptoms pathognomonic for retinal arteriosclerosis in HD include the appearance of side bands ("cases") along the vessel, symptoms of "copper" and "silver" wire (Fig. 5). The appearance of white side stripes is explained by the thickening and decrease in the transparency of the vascular wall. The streaks are visible along the edge of the vessel, as there is a thicker wall layer and a thinner layer of blood compared to the central part of the vessel. At the same time, the light reflection from the anterior surface of the vessel becomes wider and less bright.

Symptoms of copper and silver wire (terms proposed by M. Gunn in 1898) are interpreted ambiguously by various authors. We adhere to the following description of these symptoms. The symptom of copper wire is found mainly on large branches and is distinguished by an expanded light reflex with a yellowish tint. The symptom indicates sclerotic changes in the vessel with a predominance of elastic hypertrophy or plasma impregnation of the vascular wall with lipoid deposits. The silver wire symptom appears on arterioles of the second or third order: the vessel is narrow, pale, with a bright white axial reflex, often it seems to be completely empty.

Retinal hemorrhages

Hemorrhages in the retina in GB occur by diapedesis of erythrocytes through the altered wall of microvessels, rupture of microaneurysms and small vessels under the influence of high pressure or as a consequence of microthrombosis. Especially often hemorrhages occur in the layer of nerve fibers near the optic disc. In such cases, they look like radial

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strokes, stripes or flames (Fig. 9). In the macular zone, hemorrhages are located in the layer of Henley and have a radial arrangement. Much less often, hemorrhages are found in the outer and inner plexiform layers in the form of irregularly shaped spots.
Retinal "exudates"
For GB, the appearance of soft exudates resembling cotton wool is especially characteristic. These greyish-white, loose-looking anteriorly protruding lesions appear predominantly in the parapapillary and paramacular zones (Fig. 8, 9). They arise quickly, reach their maximum development within a few days, but never merge with each other. During resorption, the focus gradually decreases in size, flattens and fragments.
A cotton-wool focus is an infarction of a small area of nerve fibers caused by occlusion of microvessels [8, 9]. As a result of blockade, axoplasmic transport is disturbed, nerve fibers swell, and then fragment and disintegrate [10]. It should be noted that such foci are not pathognomonic for hypertensive retinopathy and can be observed with congestive discs, diabetic retinopathy, CVD occlusion, and some other retinal lesions in which necrotic processes develop in arterioles.
Unlike cotton-wool lesions, hard exudates in GB have no prognostic value. They can be punctate and larger, rounded or irregular in shape (Fig. 7, 8), located in the outer plexiform layer and consist of lipids, fibrin, cellular debris and macrophages. It is believed that these deposits result from the release of plasma from small vessels and the subsequent degeneration of tissue elements. In the macular region, solid lesions are banded and radially arranged, forming a complete or incomplete star figure (Fig. 8, 9). They have the same structure as other solid foci. With the improvement of the patient's condition, the figure of the star may resolve, but this process takes a long time - for several months or even several years.
Edema of the retina and optic disc
Edema of the retina and optic disc, combined with the appearance of soft foci, indicates a severe course of GB (Fig. 7, 9). Edema is localized mainly in the peripapillary zone and along the large vessels. With a high content of proteins in the transudate, the retina loses its transparency, becomes grayish-white, and the vessels are sometimes covered with edematous tissue. Edema of the optic disc can be expressed in varying degrees - from a slight blurring of its contour to a picture of a developed congestive disc.

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