STRUCTURAL FEATURES OF INTRAVITREOUS HEMATOMA

E. G. Rapis, M. D. Shmerling,
E. E. Filyushina, R. A. Gundorova,
and G. P. Yampol'skaya

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Fundamental problems in the pathogenesis of hemophthalmia are those concerned with relations between the blood and the vitreous body, namely: Does blood mix with the vitreous body or does it not mix but form an isolated hematoma; does the blood clot or remain liquid, and how do these relations change with time.

The views of specialists on these matters are inconsistent and even contradictory. For instance, the most widely held view at present is that during hemorrhage, irrespective of the genesis of hemophthalmia, the vitreous body becomes permeated with blood [3, 4, 7, 9, 10]. Meanwhile many clinical and morphological facts point to the presence of a limited single focus of blood isolated from the vitreous. Some workers [1, 10, 11] who have described the structure of the focus of blood, for instance, state that it has a distinct outer border, sometimes resembling a membrane in character. The nature of this membrane and the cause of its appearance has not yet been explained. Some workers consider that the membrane contains fibrin [10], whereas others consider that it is an accumulation of degenerated fibers of the vitreous body [1, 2]. The process of a membrane on the surface of the "thrombus" has also been demonstrated with the electron microscope and it has been suggested that it consists of fibrin and of thin collagen fibers of the vitreous body [8, 9], although no objective data to confirm this view have yet been presented.

A limiting membrane of the hematoma, with the property of birefringence, has also been discovered by one of us [5]. Moreover, the possibility of removal of an isolated hematoma has been demonstrated experimentally [5, 6].

The absence of any general agreement on the structure and nature of the above-mentioned membrane makes it more difficult to understand the essential relations between blood components and the vitreous body in hemophthalmia. The character of formation and the structure of the intravitreous hematoma are of fundamental importance for clinical practice and demand further and more penetrating study. These facts were the motivation for the present investigation.

EXPERIMENTAL METHOD

An experimental model of hemophthalmia was produced in 46 chinchilla and albino rabbits by injection of different quantities of autologous blood (0.2-1.0 ml) into the central part of the vitreous body by puncturing the sclera 4 mm posteriorly to the limbus, followed by enucleation of the eye 1 h to 31 days later. Celloidin and paraffin sections of the enucleated eye were stained for light microscopy with hematoxylin and eosin, for fibrin by the methods of Shueninov, Mallory, and Weigert, and for lipids with Nile blue; the PAS reaction also was carried out for glycoproteins.

Different parts of the hematoma, including its outer parts together with the vitreous body, measuring 1 × 2 mm were removed for electron-microscopic investigation, fixed in a mixture of 2% paraform and 2.5% glutaraldehyde, and then postfixed in 1% OsO_4 solution. Treatment of the material and embedding in Araldite were carried out in the usual manner.

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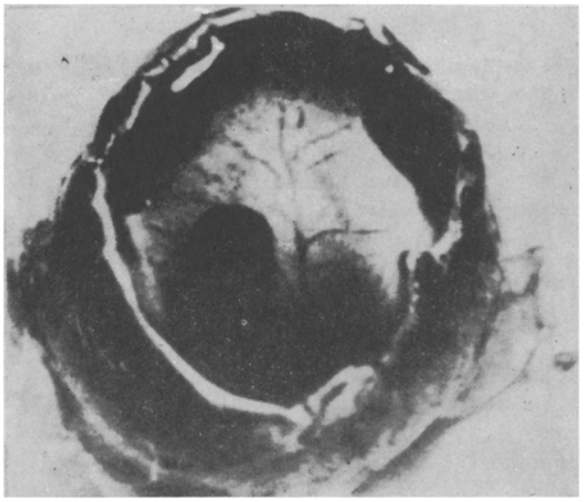

Fig. 1

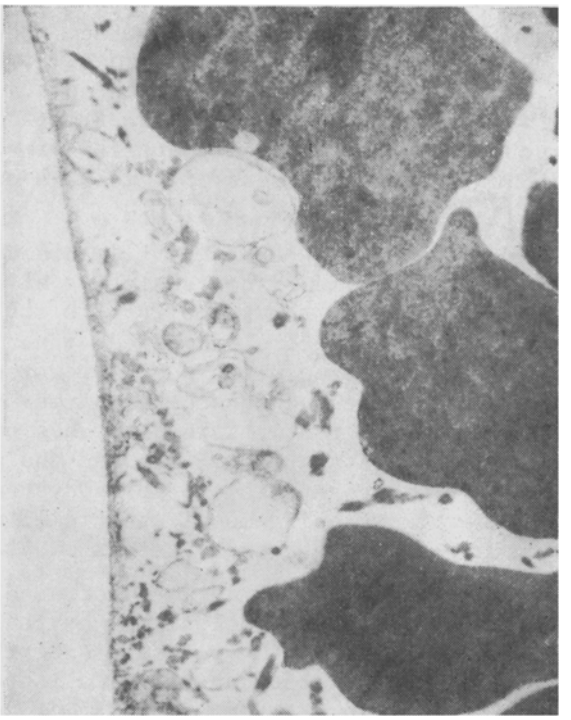

Fig. 2

Fig. 1. Experimental hemophthalmia (2 days). Frontal section through enucleated rabbit's eye. Intravitreous hematoma isolated from vitreous body had distinct outlines. Photograph from life-size specimen.

Fig. 2. Experimental hemophthalmia (1 h): superficial part of intravitreous hematoma. Thin membrane-like layer of fibrin threads and finely granular electron-dense material can be seen on surface. Inner zone of hematoma contains erythrocytes and platelets. Electron micrograph, 16,000 ×.

Ultrathin sections were cut on the LKB-8800 ultramicrotome, stained with uranyl acetate and lead citrate, and examined in the electron microscope.

EXPERIMENTAL RESULTS

The experiments showed that at the first contact with the vitreous body, blood forms an isolated round focus, which can be detected macroscopically and microscopically at all periods of observation (Fig. 1).

Light microscopy revealed an amorphous surface layer with the appearance of a membrane on the boundary separating the vitreous body and the focus of hemorrhage, which contained a compact mass of erythrocytes. On histochemical examination this layer showed the staining properties of fibrin and also reacted positively for lipids, lipoproteins, and glycoproteins. As regards the hematoma itself, starting with the 3rd-4th day the first signs of hemolysis appeared; erythrocytes were destroyed first in the central zone, later at the periphery. The fibrin network was preserved throughout the period of observation.

Electron-microscopic examination revealed a very thin layer (50-100 nm) of chaotically arranged fibrin threads and finely granular material of average electron density, resembling a membrane and separating the focus of hemorrhage from the vitreous body, on the surface of the hematoma (Fig. 2). Unlike other investigators, we did not find collagen fibrils of the vitreous body either in the limiting membrane or in the region of the hematoma itself, whereas outside the hematoma a delicate network of thin collagen fibrils could be clearly distinguished in the vitreous body.

One hour after creation of the model of hemophthalmia and at all subsequent times of observation the inner zone of the hematoma contained all the components of a thrombus: erythrocytes, platelets, fibrin, and solitary white blood cells. Besides normal erythrocytes, in the earliest stages destroyed erythrocytes containing a loose, crumbly debris of low

electron density could be observed. Often the cell membrane was ruptured and debris "scattered." With time the number of destroyed erythrocytes increased, but until the late stages of hemophthalmia (31 days) erythrocytes with the normal structure still remained. Platelets were constantly found at all times of observation, and starting from the 3rd day of the experiment some of these platelets were in various stages of injury. These observations indicate that the process of hemolysis can be discovered rather earlier by electron than by light microscopy.

On entering the vitreous body, whole blood thus forms a hematoma which is separated from the vitreous body by a membrane consisting of blood proteins similar in composition to fibrin. These experiments show, contrary to the general opinion of ophthalmologists, that the vitreous body does not become permeated with blood, for the two interact as two immiscible fluids. These facts agree with modern views on adsorption of proteins on the partition boundaries between heterogeneous high-polymer solutions or, in other words, "on the boundary between phases of different nature." It has been shown that during interaction of this kind adsorption layers are formed between the phases, with transition from the liquid state, with Newtonian flow properties, to solid systems. These findings, obtained for the first time by Soviet workers using model artificial membranes [2, 7], explain the physicochemical nature of relations between the vitreous body and blood in hemophthalmia.

The presence of a hematoma, i.e., of a collection of blood isolated from the vitreous body, is also demonstrated conclusively by the fact that no collagen fibrils of the vitreous body were ever found among the components of the hematoma. The results of this investigation showed that as a rule a clot containing all the components of a thrombus is formed in the hematoma, but unlike in a thrombus, the clot is formed immediately at the moment of contact between the vitreous body and blood. It can be tentatively suggested that this is due to the action of the collagen of the vitreous body, which is one of the factors initiating clotting and giving rise to biochemical and structural changes in platelets.

The rapid development of massive hemolysis of erythrocytes on the 2nd-3rd day in the central zone of the hematoma (data of light microscopy) may evidently be connected with structural features of the hematoma resulting in dense packing of the erythrocytes preventing access of oxygen. This is combined with the long duration of hemolysis and its frequent failure to reach completion at the periphery of the hematoma, in the zone of contact between erythrocytes in the vitreous body.

Finally, the membrane discovered on the surface of the hematoma, formed from the moment of contact between vitreous body and blood, prevents the two from mixing and is responsible for the relative stabilization of the structure of the hematoma.

These observations thus provide pathogenetic justification for the need to carry out isolated removal of a hematoma as early as possible (in the period of its stabilization) from the vitreous body without vitrectomy.

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