ULTRASTRUCTURAL MANIFESTATION OF INTRAVASCULAR COAGULATION DUE TO THERMAL TRAUMA IN THE KIDNEY

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Burn trauma is a symptom-complex accompanied by lowering of the systemic blood pressure and cardiac output and, as a result, by a reduction in the renal plasma flow and the development of acute renal insufficiency [3, 6, 7, 9]. At the same time, disturbances in the clotting system of the blood are an important component in the development of complications arising after burns under clinical and experimental conditions, for shock may itself be both the cause and the consequence of disseminated intravascular coagulation [14]. As regards changes in the reactions of hemostasis in patients, these are observed during the first day, whereas in experimental animals they are found during the first hours after burning [10, 12].

Accordingly, the aim of the investigation described below was to attempt to detect the initial manifestations of intravascular coagulation in the kidney at the earliest possible times by an electron-microscopic method in the course of burn shock.

Fig. 1. Escape of erythrocytes by dispedesia (torpid phase), 3500 X.

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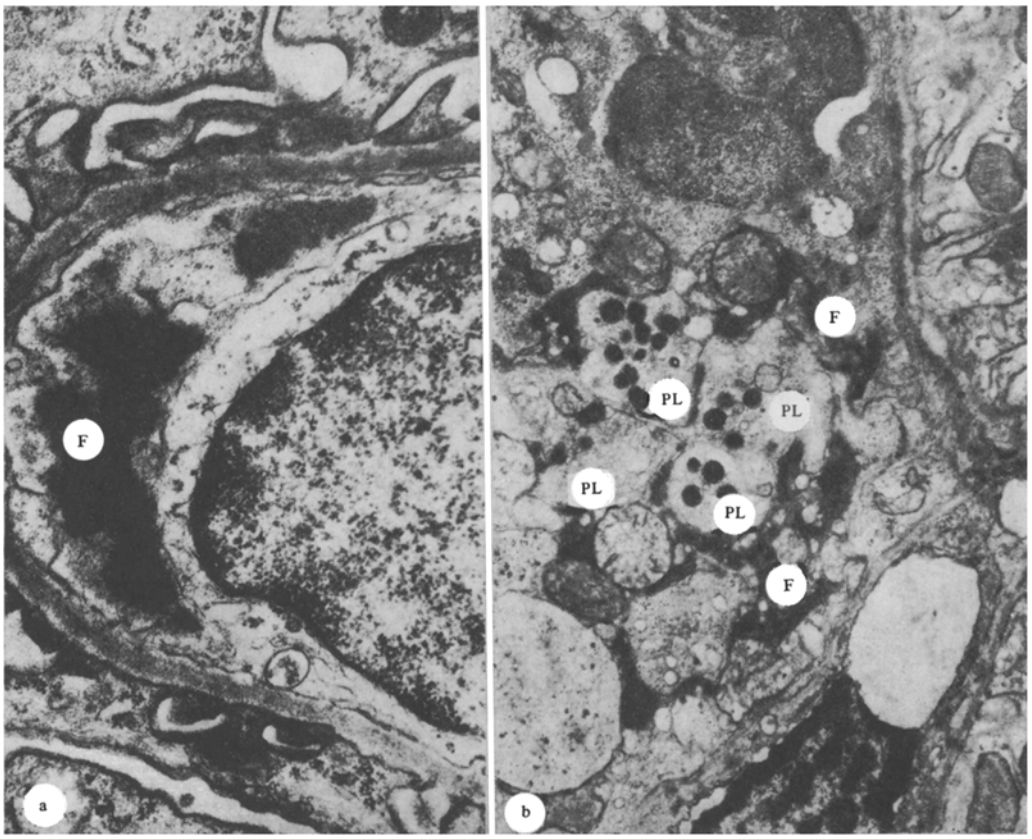

Fig. 2. Intravascular coagulation in renal vessels. a) Precipitation of fibrin (F) in glomerular capillary, 14,000 X; b) aggregation of platelets (PL) and appearance of fibrin (F) in intertubular vein (viscous metamorphosis, torpid phase), 10,000 X.

EXPERIMENTAL METHOD

Experiments were carried out on 40 mongrel dogs weighing 9-13 kg. Burn shock was produced in animals anesthetized with pentobarbital (40 mg/kg) by the method described previously [1]. Material for electron-microscopic study was taken in the erectile (5 min) and torpid (60 min) phases after decapitation. Pieces of kidney were fixed by the usual method with glutaraldehyde and osmium and embedded in a mixture of Epon with Araldite. Ultrathin sections cut on the LKB-8800 ultramicrotome were stained with uranyl acetate and lead citrate and examined in the JEM-100S electron microscope.

EXPERIMENTAL RESULTS

The writers showed previously that ultrastructural disturbances characteristic of developing intracapillary glomerulonephritis are observed in the erectile phase of burn shock. Degenerative changes were found at the same time in the proximal and distal portions of the nephrons, together with an increase in obligatory and facultative reabsorption, but no signs of intravascular coagulation were present [4].

Electron-microscopic investigations of the kidneys in the torpid phase of burn shock showed that the general direction of the structural changes remained basically the same as that characteristic of the erectile phase, but the changes were more severe [2].

The present investigation showed considerable injury to the endothelial cells in the glomerular and intertubular capillaries as early as within a few minutes after burn trauma. In particular, the outlines of their luminal surface were found to be uneven and their cytoplasm contained well developed organelles. Chromatin distributed irregularly in the nuclei and the perinuclear space was widened. The lamellar complex most frequently appeared intact. The greatest changes were found in the rough endoplasmic reticulum, the cisterns of which were dilated. Mitochondria were few in number, and most were round in shape with poorly developed cristae.

Dense bodies, differing in diameter, appeared in the lumen of the capillaries; the larger ones morphologically resembled lysosomes of endothelial cells, whereas the smaller resembled lysosomes of polymorphonuclear neutrophils. Considering that most have been shown electron-histochemically to have acid phosphatase activity [15] it is logical to assume that lysosomal

enzymes participate in injury to membranous structures. It must also be emphasized that the transient enzymopathy is reflected in the state not only of the intracellular organelles, but also of the cells as a whole, and it plays a definite role in the change in permeability of the tissue-blood barriers in kidney [5].

In the torpid phase of burn shock a progressive increase in severity of the vascular disturbances was observed, as revealed by destruction of the basement membranes of the glomerular and intertubular capillaries. Injury to these structures can be explained by the combined action of toxic substances formed in burns and activation of lysosomal enzymes, as a result of which foci of extravasation were often observed. Mitochondria, fragments of cytoplasmic reticulum, and various inclusions could be seen in the lumen of the capillaries, and some were also perivascular in arrangement. The defect in the basement membrane could be absent, very small, or very wide. Electron-microscopic analysis revealed that blood cells escape chiefly by diapedesis (Fig. 1). In that case, the endothelium is damaged initially, then the basement membrane; the severity of the hemorrhages correlates with structural disturbances of the capillaries [8, 11].

The signs of intravascular coagulation, first established 1 h after burning, deserve particular attention. The presence of fibrin was observed in the glomerular and intertubular capillaries (Fig. 2a, b). Ultrastructural changes also were recorded in the platelets (vacuolation of the cytoplasm, swelling of mitochondria, destruction of the plasma membrane), accompanied by their standard reaction, namely liberation of a special platelet component, the so-called thromboplastic factor. The process of adhesion and aggregation of platelets, combined with their motphological and functional changes, together constitute the phenomenon of viscous metamorphosis (Fig. 2b).

The investigations revealed signs of increased coagulability of the blood, in agreement with experimental and clinical data obtained previously [10, 12]. In recent years the development of intravascular coagulation has come to be regarded as one of the chief factors responsible for irreversibility in various shock states, including burn shock [13]. The inclusion of a coagulating component supplements and aggravates the disturbance of the intrarenal hemodynamics and contributes to a disturbance of glomerular filtration and tubular reabsorption, one result of which is acute renal failure arising after burns.

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