The Fine Structure and Function of the Mechanically Injured Renal Lymphatic Capillary

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Summary

After saline solution was injected into the renal cortex of the rat, the whole kidney was fixed by the intraaortal perfusion method to minimize the artifacts occurring before fixation. The lymphatic capillary in the renal cortex shows wall-defects as well as widely separated endothelial junctions, which induce its increased permeability. These changes are considered to have been caused by a pure mechanical injury due to injection itself, and not by any effect of the injected saline solution nor of blood produced by hemorrhage in the tissue. The injured lymphatics transport the wound products such as fluid, fine fuzzy material, cells and cell-organelles from the lesion; and thus play an important role under the pathological conditions like trauma or wound healing. The micro-injection method should not be used for the investigation of endothelial permeability of the lymphatic, which could be easily changed by the procedure.

It is easily suspected that the micro-injection of any solution into the tissue injures the lymphatic endothelium and changes its permeability. This investigation was aimed to evaluate its damage by the micro-injection and also to provide data about the functions of the lymphatics in case of trauma.

Materials and Methods

Under the general anesthesia by ether, 0.1 ml of warmed physiological saline solution was slowly (0.1 ml in a minute) and manually injected into the renal cortex of the 4 adult rats (Sprague-Dawley), followed by the intraaortal perfusion fixation after 20 minutes. This perfusion fixation as well as the further procedures for electron microscopic examination were performed as described by Ohkuma (7). For the control the same tissues of 2 rats were injured by the same procedures using the injection-needle alone without injecting any solution. For the light microscopic observation, the tissue was fixed by 10% formalin and stained by HE as usual.

Results

The macroscopic observations reveal that the injected area is expanding in size and its margin is not sharply bordered, which is recognized only by means of hemorrhage. The control experiments show only pin-point sized hemorrhagic lesions.

The light microscopic examinations show that some dilated lymphatics with dark or vacant lumina are observed in and around the edematous, damaged and hemorrhagic area. The control specimens reveal the smaller, slightly edematous and hemorrhagic areas with few dilated lymphatics.

Electron microscopic findings are as follows:

Lymphatic lumen contains blood components such as RBC’s (Fig. 1), lymphocytes (Fig. 2), leukocytes, fibrin (Fig. 2 & 3) and platelets (Fig. 1). The fine fuzzy materials are seen now and then in the lumen, specially in the vessels apparently free from damage (Fig. 4).
Fig. 1. A red blood cell, a platelet, fibrin and fine fuzzy material are seen in the lymphatic lumen. 5,400 x.

Fig. 2. Three lymphocytes are observed in the lymphatic capillary with a wide wall defect (>). The blood capillary shows the fenestrated endothelium and the continuous basal lamina. 7,200 x.
Fig. 3. Mitochondria (m) are passing through an endothelial defect of the lymphatic capillary. 9,600 x.

Fig. 4. A lymphatic capillary apparently free from mechanical damage. The lumen is filled with fine electron dense particles. A plasma cell is seen in the connective tissue area. 3,300 x.
The destroyed cells and cell organelles like mitochondria (Fig. 3) are also observed. Some lymphatic endothelia show homogenized chromatin of the nucleus with a slight marginal condensation, fragmented or dilated nuclear envelope, increased or decreased electron density of the cytoplasm and a complete disappearance of the cytoplasm with adhesion of the two plasma membranes (Fig. 5 & 6). There are sometimes widely separated endothelial junctions as well as wall-defects (Fig. 1, 2, 3, 5 & 6). The wall defect of an injured blood capillary is mostly covered and compensated by the platelets (Fig. 7). All those particles and cells observed in the lymphatic lumina are also found in the connective tissue area. Control shows almost the same morphological changes as the injured lymphatic capillary by the injection of saline. Few lymphatics can be found in the less edematous region. They are difficult to find, because the area is small and edema is less in extent.

Discussion

The injury of the lymphatic capillary in this investigation is considered to have been caused by the injection itself and not by an increased pressure due to injected solution or hemorrhage in the tissue, because there sometimes exist apparently intact lymphatics in the edematous and hemorrhagic area and also because the control specimens reveal injured lymphatics. The renal lymphatics dilated by the minimal damage have shown neither separated endothelial junction nor wall defect (3, 7, 9). Such morphological changes of the injured lymphatic endothelium as shown in Fig. 5 and 6 are similar to those of a dying cell or dead cell (1, 2, 6) and also those of a thermally injured lymphatic endothelium (4). All the cells and particulates found in the lymphatic lumina are considered to have entered through the separated endothelial junction or the wide wall-defect. A large quantity of fibrin (Fig. 2 & 3) in the lumen of the lymphatic may get into the blood circulation, inducing or promoting intravascular coagulation (5), if the phagocytic cells of the lymph nodes become overloaded in case of severe trauma. The wall defects of an injured blood capillary are mostly covered and compensated by the platelets (Fig. 7). Thus it is not easy for the large particulates or cells to pass through the injured wall of the blood capillary. On the contrary the endothelial defect of an injured lymphatic capillary remains uncovered with an increased permeability for the large particulates. This is one of the functional differences between the two vessels. If it bleeds to some extent in the control experiments, its condition may resemble that of the tissue after injection of saline solution, which is not looked upon as control in the strict sense of the meaning.
Fig. 6. A higher magnification of the Fig. 5. The endothelium shows a widely separated junction, homogenized chromatin of the nucleus with a slight marginal condensation, fragmented (\(\sim\)) or dilated (\(\sim\)) nuclear envelope, and the cytoplasm with a high or low electron density as well as a complete disappearance of the cytoplasm with two adhered plasma membranes (a). The lumen is not filled with fine electron dense particles. 22,000 x.

Fig. 7. The wall of an injured blood capillary is covered by a number of platelets. A piece of endothelium with a pore (f) as well as a defect of the basal lamina (\(\sim\)) are observed along the capillary wall. 26,000 x.
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General Abbreviations Used in Figures


References

5. McKay, D.G.: Disseminated Intravascular Coagulation, Hoeber Medical Division, N.Y. 1965

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