WOUND HEALING AFTER TISSUE INJURY BY CO₂ LASER BEAM

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A study of tissue healing (liver, kidney, and lungs) after injury by a CO₂ laser beam revealed no marked leukocytic infiltration of tissues bordering on the injury. The absence of inflammatory changes during healing is evidence that resorption of injured tissues and proliferation of tissue cells accompanied by inflammation are relatively independent processes and do not constitute a single pathogenetic chain of repair.

KEY WORDS: wounds; healing; CO2 laser.

The use of lasers in modern medicine and biology has opened up wide prospects for the study of some biological effects arising as a result of the action of the laser beam on tissues. One such effect is the absence of any marked leukocytic infiltration in tissues bordering on those which have been injured, and this effect is observed throughout the period of healing. Judging from data in the literature, this effect is not found constantly: some workers [1, 4] observed it in human skin following the use of a neodymium laser with pulsed type of action. Others [4, 7, 8] observed the appearance of leukocytic infiltration in the skin after exposure to lasers of this type. The action of the CO₂ laser beam ("light scalpel") in the experiments of Khromov et al. [6] was accompanied by the development of focal leukocytic infiltration in the tissues of the liver, kidney, and spleen. Zimmermann and Kraushaar [9] resected a pole of the kidney in dogs and rabbits by a CO₂ laser beam and failed to mention the absence of leukocytic infiltration in tissues bordering on the injury, although its absence is clearly visible from the photomicrograph accompanying the paper. Golovnya [3] and Galankin et al. [2] found no leukocytic infiltration after marginal resection of the cat liver by a CO₂ laser beam.

The objects of the present investigation were: 1) to reproduce the phenomenon of noninflammatory healing of tissues in several organs of the rat after injury by a CO_2 laser beam; 2) to assess the degree of organ-specificity of this phenomenon.

EXPERIMENTAL METHOD

Experiments were carried out on 36 male rats weighing 100-120 g. Operations were performed on animals in three series, each containing eight rats, by means of a focused CO₂ laser beam with a power of 200 W; in the animals of series I, the lingual lobe of the liver was resected, in the rats of series II, an area of kidney with a volume of about 4 mm³ was cauterized, and in the rats of series III the upper lobe of the left lung was resected. The animals of three control series, each containing four rats, were subjected to mechanical injury to their organs: in series I the lingual lobe of the liver was resected by scalpel, after which the tissue was sutured to stop bleeding; in series II the same manipulation was done on kidney, with resection of the lower pole; in series III the upper lobe of the lung was resected by means of a scalpel. The rats were killed at different times — up to 25 days later. All operations were performed under ether anesthesia and aseptic precautions were taken, but not strictly.

EXPERIMENTAL RESULTS

Healing of the tissues in the rats injured mechanically (by means of a scalpel) conformed to the classical notions of the basic features of wound healing. For instance, by the end of the first day the injured tissue of each organ (liver, kidney, lung) began to be separated from the deeper, uninjured tissues, by a commencing layer of leukocytic infiltration. The intensity of infiltration increased during the next few days — until the 6th-8th day,

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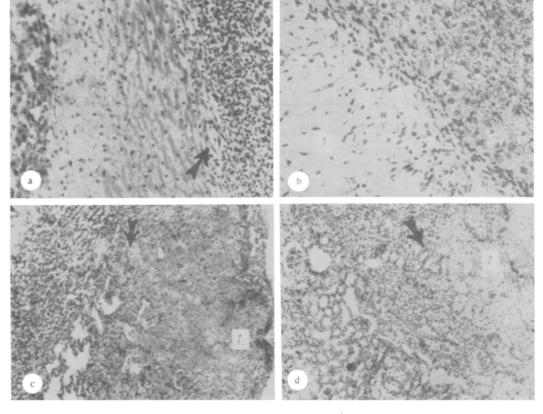


Fig. 1. Healing of tissue after resection by scalpel (a) and laser beam (b, c, d). Hematoxylin-eosin: a) border of the liver on 2nd day after resection; layer of leukocytic infiltration can be seen (arrow), 200×; b) border of liver on 4th day after resection by laser beam; leukocytic infiltration of necrotic tissue absent (1); layer of parallel fibroblasts visible on boundary between injured and uninjured tissue, 250×; c) border of lung resected by laser beam on 3rd day after operation; layer of necrotic tissue (1) on resection surface has no leukocytic infiltration; underlying layer of lung tissue has collapsed; boundary between injured and preserved tissues indicated by arrow. Magnifying glass; d) border of kidney resected by laser beam on 3rd day after operation; layer of necrotic tissue (1) on resection surface has no leukocytic infiltration; boundary between injured and preserved tissues indicated by arrow. Magnifying glass.

and massive destruction of the leukocytes contributed to liquefaction of the necrotic tissues and their subsequent absorption. After about the 6th-10th day, this inflammatory liquefaction reached its peak, and stimulation of connective-tissue cells was observed in the zone boundering on the injured tissues. As a result of completion of absorption and continued proliferation of fibroblasts and collagen formation, healing of the surface of the wound ended with the formation of a layer of fibrous tissue by the 23rd-25th day after the operation. The appearance of leukocytic infiltration toward the end of the 1st or beginning of the 2nd day after the operation signified that healing of the wound would follow its ordinary standard course (Fig. 1a).

In the experiments with resection of the organs by a CO₂ laser beam, the phenomenon of noninflammatory tissue healing was reproduced. The course of healing of the laser wound differed from the ordinary course of healing in many respects. Under the influence of a focused beam, as a result of thermal injury a thin layer of necrotic tissue was formed. This layer, because of the short duration of action of the laser beam, had a distinct boundary with the underlying tissues. Sometimes the boundary consisted of 3-5 layers of cells distended with bubbles, this appearance being due to instantaneous formation of steam within the cells. On the boundary with the uninjured tissues no leukocytic infiltration was present. The process of healing of the laser wound as observed in three organs of the rat (liver, kidney, and lung) showed absence of a leukocytic reaction to tissue necrosis at all stages of repair and in all these organs (Fig. 1b, c, d). It can thus be concluded that the phenomenon of noninflammatory tissue healing is independent of organ-specific features, but is associated with the character of the necrotic tissue formed under the influence of laser radiation. Besides the absence of any

marked leukocytic infiltration on the boundary between the injured and uninjured tissues, activation of connective-tissue cells could be observed in the early stages of healing: by the 3rd or 4th day not only had they appeared in large numbers, but in some places they were arranged in parallel rows, resembling properly formed connective tissue (Fig. 1b), lying on the surface of the preserved tissue. Above this layer there was a structure-less necrotic mass, not undergoing leukocytic liquefaction, but gradually shrinking after the operation. Healing ended with the formation of an even layer of fibrous tissue by the 18th-20th day, sometimes incorporating products of tissue combustion — regions of "soot" surrounded by multinuclear cells, and compact masses of structureless tissue, surrounded by fibroblasts, lay here and there on the surface. The course of healing after exposure to the CO₂ laser beam was the same in all the organs studied: liver, kidney, lung.

Tissue injury by a CO₂ laser beam thus does not lead to the development of marked leukocytic infiltration in tissues bordering on the injured tissues. Healing without inflammatory changes (leukocytic infiltration) after laser irradiation is not a tissue-specific phenomenon but evidently depends on the nature of the tissue rendered necrotic by the laser beam. The phase of inflammatory liquefaction of the necrotic tissues is not invariably found during healing. Inflammatory liquefaction of necrotic tissues and proliferation of cells during regeneration are not pathogenetic links in a single chain but are relatively autonomous in character.

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