

Structural Changes in Abdominal Aorta and Vena Cava Inferior after Experimental Microwave Destruction

Yu. I. Denisov-Nikol'skii, V. V. Shafranov*, E. N. Borkhunova,
A. A. Doktorov, L. M. Mikhaleva**, and A. P. Mikhalev

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We studied morphological changes in rat abdominal aorta and vena cava inferior induced by endovascular microwave destruction. This treatment induced thrombosis and necrosis of the vascular walls, but fibrous framework was not completely destroyed. Then we observed irreversible changes in vessels: obliteration, atrophy of the aorta walls, and sclerosis of venous walls. The preserved fibrous framework of the vessel walls probably plays a role of a framework for the formation of fibrous tissue.

Key Words: *endovascular microwave destruction; abdominal aorta; vena cava inferior*

Endovascular occlusion of the artery supplying blood to the tumor with the help of microwave electromagnetic field (EMF) is an effective method of treatment of hemangiomas of complex anatomical localization [2]. This method was effectively used for the treatment of 22 children at the Clinics of Child Surgery (Russian State Medical University). The method is also effective for the treatment of venous malformation (phlebectasia). Endovascular occlusion is based on thermocoagulation of the vascular wall induced by microwave EMF [2], which is known to produce a thermal effect [1,4]. The procedure was carried out using a bipolar probe with fluoroplast coating preventing adhesion of the coagulated walls to the probe. The regimens of microwave destruction of arteries and veins and subsequent venous obliteration were described in a single study and histological changes in veins were examined on hematoxylin/eosin-stained preparations [2]. However, microwave-induced structural changes in arteries and veins were not studied in details.

Here we studied morphological changes in rat abdominal aorta (AA) and vena cava inferior (VCI) after microwave destruction.

MATERIALS AND METHODS

Albino rats were subjected to lapatomy under ketalar narcosis and AA and VCI were exposed. Vascular fragments between the renal arteries and veins and bifurcation of AA and VCI into the common ileac arteries and veins were prepared. AA and VCI were ligated under a microscope proximally and distally from the studied region, a 0.8-mm endovascular probe was inserted in vessels, and endovascular microwave destruction was performed at 10 W for 10 sec using a Yakhta device. Wall coagulation and thrombus formation were evaluated macroscopically, then the ligatures were removed. No bleeding was observed. The operative wound was sutured layer-by-layer using a Vycryl synthetic thread. The animals were killed by ketalar overdose 1 and 24 h, 7, 14, and 30 days after surgery.

For histological examination, the specimens were fixed in 10% neutral paraformaldehyde (pH 7.4) and embedded in paraffin. The sections were stained with hematoxylin-eosin by the method of Van Gieson and with resorcin-fuchsin by the method of Weigart [3] and examined under a Nu microscope. For scanning electron microscopy [3], the specimens were fixed in 2.5% glutaraldehyde on cacodylate buffer (pH 7.2), washed in distilled water, dehydrated in increasing

Research Center for Biomedical Technologies; *Russian State Medical University; **Institute of Human Morphology, Russian Academy of Medical Sciences, Moscow

concentrations of acetone, and by the method of transition over a critical point on a Hitachi device. The preparation were dusted with copper on a JOEL JEE device and examined under a Philips SEM 515 microscope.

RESULTS

The walls of control AA consisted of adventitia, media (6 layers of smooth muscle cells separated with 7 elastic membranes), and intima presented by a thin light layer. The endothelium was clearly outlined. VCI walls consisted of adventitia, two smooth muscle cell layers, inner elastic membrane of the media, and intima. Endothelial cells were clearly seen. Morphometric parameters of AA and VCI walls are presented in Table 1.

Immediately after endovascular microwave destruction AA walls at the site of its contact with the probe collapsed, thrombosis was seen at the site of contact and up- and downstream. Wall collapse probably results from spasm of the aorta occurring at 43°C [2] and subsequent coagulation at higher tem-

peratures. Thrombosis was also observed in VCI (Table 1). These changes were seen 24 h after the procedure (Fig. 1). At this term the adventitia was infiltrated with segmented neutrophils, smooth muscle cells in the media underwent necrosis, but the fibrous framework was preserved and tinctorial properties of collagen and elastic fibers remained unchanged. The endothelium was desquamated into the lumen. On day 7 after treatment (Fig. 2), AA at the site of treatment was narrowed, AA and VCI were occluded with mixed thrombi with signs of organization. The adventitia was infiltrated with leukocytes and macrophages. In AA and VCI media, only fibrous framework was preserved. Collagen and elastic fibers retained their tinctorial properties. All elastic membranes of AA except the inner one were fragmented. The media was infiltrated with macrophages, fibroblasts, and some segmented neutrophils. The cell infiltrate was organized and located interfibrillary. The endothelium was absent. On day 14 after the procedure (Fig. 3, *a, b*), a fine connective tissue bundle enriched with fibroblasts was seen in AA at the site of destruction. The vascular

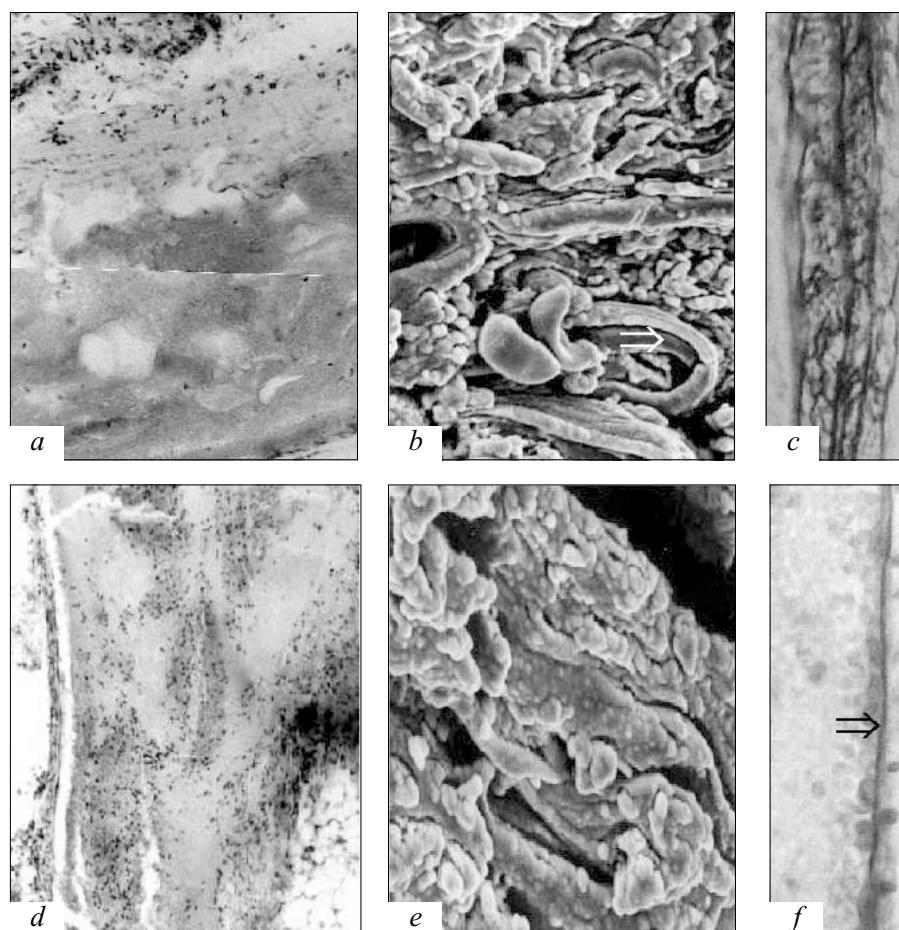


Fig. 1. Microwave destruction, 24 h. Hematoxylin and eosin staining, $\times 40$ (*a, d*), scanning electron microscopy, $\times 1550$ (*b, e*), and orcein staining, $\times 80$ (*c, f*). *a*) thrombosis of abdominal aorta; *b*) plasma infiltration of AA walls; *c*) destruction of elastic membranes; *d*) thrombosis of vena cava inferior (VCI); *e*) plasma infiltration of VCI wall; *f*) preserved elastic membrane of VCI.

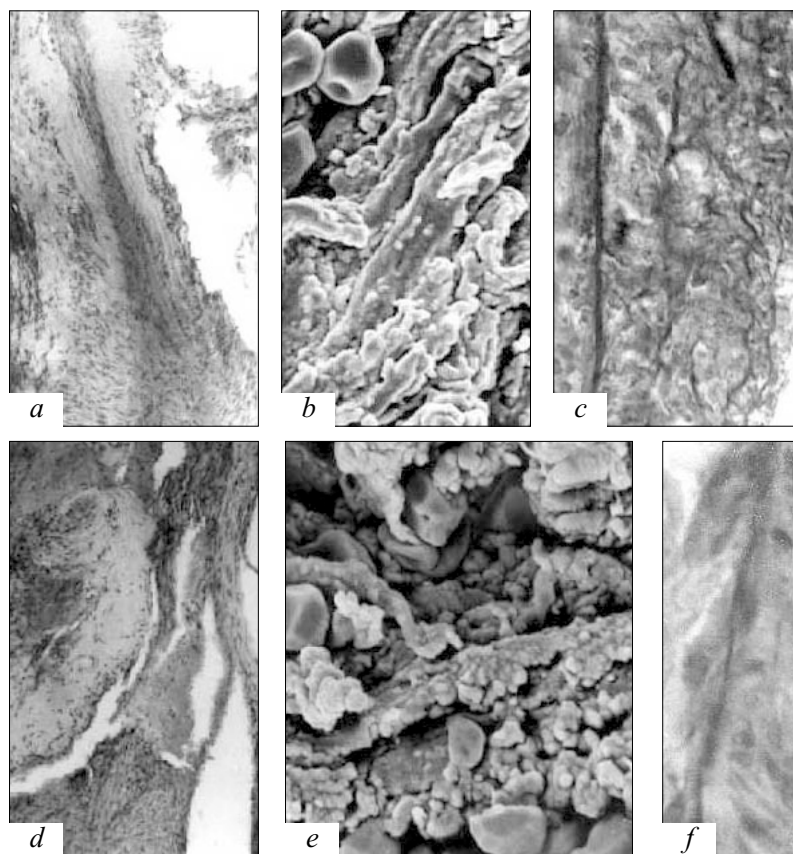


Fig. 2. Microwave destruction, day 7. Hematoxylin and eosin staining, $\times 40$ (*a*, *d*), scanning electron microscopy, $\times 1550$ (*b*, *e*), and orcein staining, $\times 80$ (*c*, *f*). *a*) collapsed abdominal aorta (AA) walls occluded with thrombi with signs of organization; *b*) collagen framework of AA wall with signs of plasma infiltration; *c*) disorganization of AA elastic membranes; *d*) thrombus with signs of organization in vena cava inferior (VCI); *e*) plasma infiltration of VCI wall; *f*) elastic membrane of VCI wall.

TABLE 1. Dynamics of Changes in Walls of Rat Abdominal Aorta and Vena Cava Inferior after Endovascular Microwave Destruction

Parameter	Term of experiment					
	Control	1 h	24 h	7 days	14 days	30 days
Vessel diameter, μ						
aorta	540-840			264-276	216-240	144-180
stenosis		180-240	180-240			
perifocally		480-600	480-600			
vein	384-770			264-300	216-240	144-180
stenosis		120-180	120-180			
perifocally		480-540	480-540			
Lumen*	Blood	Thrombosis	Thrombosis	Granulation tissue	Mature connective tissue	Fibrous tissue
Wall thickness, μ						
aorta	42-66	44-65	44-65	48-60	48-60	48-60
vein	12-24	12-24	12-24	36-48	36-48	36-48
Endothelium*	Clearly determined	Focal desquamation	Absent	Absent	Absent	Absent

Note. *Characteristics are given for aorta and vein.

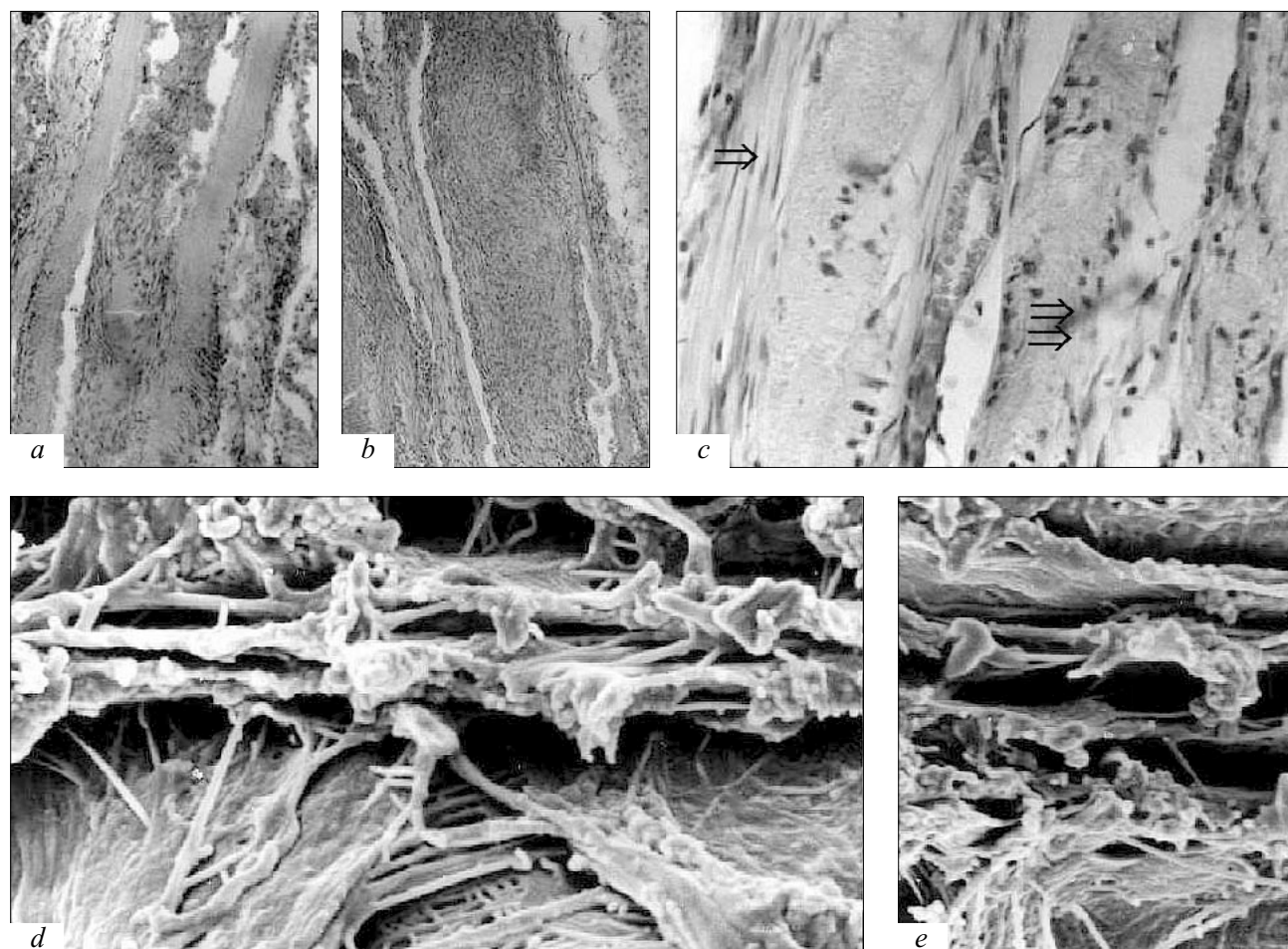


Fig. 3. Microwave destruction, days 14 (a, b) and 30 (c-e). Necrosis of abdominal aorta (a) and vena cava inferior (b) walls, thrombus organization in vessels. Thinning of aorta walls, sclerosis of the venous wall, fragments d and e correspond to areas indicated on c by one and two arrows, respectively.

walls were presented by continuous bundles of collagen and elastic fibers infiltrated with macrophages and fibroblasts oriented along these fibers. VCI lumen contained fine connective tissue, venous walls showed signs of sclerosis. The endothelium was absent.

On day 30, AA and VCI were sharply narrowed and filled with thin bundles of fibrous tissue containing capillaries. AA walls were atrophic, thinned, and contained fine collagen fibers separated by enlarged interfibrillar space. Some elastic structures, mainly the inner elastic membrane, were seen. VCI walls were sclerotic probably due to reaction of paravascular tissues. The adventitia in AA and VCI still contained oriented collagen bundles and the inner elastic membranes. Fibrous tissue occluding the vascular lumens was oriented along the vessel axis. In the exposed area macrophages and fibroblasts were located between collagen fibers, which probably indicated cicatrization of the damaged vessels.

Thus, endovascular microwave destruction caused irreversible changes in AA and VCI walls in rats. This

procedure induced thrombosis and necrosis of cells in the vascular walls, but preserved fibrous framework. Thrombi excluded the vessels from circulation, which led to atrophy and sclerosis of the aortic and venous walls and obliteration of AA and VCI lumens with connective tissue. A characteristic feature of AA and VCI damage after microwave destruction is preservation of the initial fibrous skeleton in the damaged area. It cannot be excluded that it serves as a framework for migration of infiltrating cells and connective tissue bundles during organization of the venous walls and obliteration of AA and VCI lumens. It is possible that this fibrous skeleton plays a role of a framework for directed infiltrate migration and development of fibrous tissue.

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