Section 6. Medical science


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Morphological aspects of poly-organic impact of radio frequency electromagnetic radiation in experiment

Abstract: The impact of radio frequency electromagnetic radiation (RFEMR) on morphological responses of some organs of experimental animals has been studied. The RFEMR effect was shown to manifest itself by pathological changes in the structure of the majority of organs and tissues with the critical impact of the micro-vascular bed impairment on not only morphological, metabolic but also many other homeostasis shifts that occurred.

Keywords: radio frequency electromagnetic radiation, morphological research, experimental animals.

Morphological changes in the body are the direct reflection of the processes which are responsible for compensatory and adaptive responses to the effect of environmental factors, as well as for detoxication of exo- and endotoxins, for regulation the immune response, homeostasis and other functions as well. Therefore, studying the morphological response at different levels of the of biological system organization being under the exposure to RFEMR is an important and urgent problem both from the point of view of understanding the mechanisms of the biological action and evaluation of its danger for human health [1,5–19; 2, 124–140].

Proceeding from the above-stated, the goal of the research was defined, i.e. to reveal the profile and dynamics of the morphological and functional changes in some organs and tissues (the brain, lungs, liver, stomach, large and small intestines, spleen, kidney, android glands) of rats in experiment with their exposure to RFEMR.

The experiment involved 72 white male rats weighing 220–280 g. All animals were randomly divided into four groups (20 animals in each group under study and 12 controls). The animals, have acclimatized for at least 1 week prior to any experimental procedures, were housed, monitored and exposed in accordance with the institutional animal ethics requirements. Food and water was provided ad libitum. Once acclimatized, groups of rats were placed into standard plastic cages with modified plastic lids, instead of metal lids, in order to avoid interference with the radiation. Animals of three groups under study were exposed to RFEMR at 1800 MHz frequency and energy stream density (ESD) of 50, 500 and 1000 mcW/cm², accordingly; group 4 (controls) was not exposed to RFEMR. SM-300 generator, BLWA 1719–20 capacity amplifier (20 watt), HL040 passive aerial (Germany) were used as the RFEMR source. The RFEMR set exposure was checked daily by measuring instruments of the energy stream density PZ — 18 (Russia) and NBM — 550 (Germany) with isotropic gauges. The experiment consisted of two stages: one month (the acute stage) and three months (the chronic stage) round-the-clock exposure (with a break for feeding). The laboratory animals (rats) were killed according to the IACUC recommendations (1) on the basis of PHS Policy. Morphological research was conducted by the standard methods of light microscopy with hematoxylin-eozin staining (h-e) [3, 36–37]. The following organs have been examined: the liver, brain, stomach, small and large intestines, spermary, heart.

Figure 1. The rat’s liver after 1-month exposure to 500 mcW/cm² (h-e, 10 x 16). Impairment of hepatocytes complexes, moderate hyperchromatism and degeneration (vacuiolization) of hepatocyte’s nucleus
The morphological changes arising in the liver tissue due to the RFEMR exposure were characterized by different severity level and developed both in hepatocytes and the vascular-stromal renal structures. The RFEMR exposure lasting 30 days in the dose of 50 mcW/cm² (Fig.1) did not lead to any lesions in the general liver architectonics. There were microcirculation impairments manifesting themselves in non-uniform plethora of the renal parenchyma of diffuse or mosaic character, emptying the portal part and sinusoids, impairment of hepatocyte complexes, moderate hyperchromia and degenerations of hepatocytes’ nucleus. However, we have not found out lymphoid-plasmocytic infiltration and fibrosis.

Even when the exposure was 500 mcW/cm², the impairment of the general architectonics of the liver manifested by dramatic reduction of relative volume of the microcirculation bed has already been observed. It is caused both by microcirculation disorders described above and a change in the hepatocyte size. More considerable changes were observed after RFEMR exposure at 1000 mcW/cm².

Similar to the aforesaid, the changes in rats of group 1 were revealed after three months exposure at 50 mcW/cm². Unlike the previous group, dystrophic changes in hepatocytes were more expressed that can suggest activation of apoptosis processes at longer RFEMR exposure. When the RFEMR exposure during 3 months was greater, e.g. 500 and 1000 mcW/cm², we observed the further increase in the signs of liver architectonics impairment occurred due to critical microcirculation defects and accruing proteinosis. When examining the brain tissues of the rats of group 1 after one month exposure, no significant morphological changes were found, while at 500 mcW/cm² exposure, the neocortex edema in the form of bursting the deep layers’ tissue of the cortex was revealed. Changes in the brain tissue were even more considerable at 1000 mcW/cm² RFEMR.

The structural features of the RFEMR impact within 3 months (Fig. 2) on the brain tissue of the rats were characterized by rougher morphological changes looking like perineural and perivascular edema that has led to red blood cells sludge and formation of microthrombs, microcysts and microvesicles.

The morphological examination of the rats’ spermaries has revealed that the reproductive organ is the most sensitive to the RFEMR impact. According to our observation, the structural changes manifest themselves both at the tissue and cellular levels that could not but affect the regenerative, reproductive and endocrine functions of the male rats. In this aspect, the results of our research agree with U.G. Grigoriev’s opinion (1980) that “the sexual glands compose the group of the critical organs most sensitive to the exposure”.

The morphological changes in the rat spermary accrued under the effect of RFEMR with an increase in the exposure dose and time of the treatment from 1 till 3 months (Fig. 3). In spite of the fact that there are enough spermatids and spermatozooids in the lumen of seminiferous tubules, many of them had the impaired structure. The 1000 mcW/cm² RFEMR exposure within 3 months was morphologically characterized by the most negative impact on the rat spermary and demonstrated the complete loss of regenerative abilities of the spermatogenous epithelium.

When analyzing the morphological changes in the tissues of the gastrointestinal system in modeling “acute” and “chronic” types of RFEMR exposure, some characteristic structural regularities (Fig. 4, 5) have been also revealed.

The most expressed changes in the stomach were found in the mucous membrane, actually in the glandular layer and the gastric micro-villi, accruing in process of an increase in the RFEMR exposure. They were characterized by hypoplasia, hypotrophy and atrophy of mucus and glandular structures. Similar impairments were revealed in the tissues of the small and large intestines.
Figure 4. The rat's stomach. (1000 mcW/cm² RFEMR, 3 months) Deformation and partial destruction of the micro-villi of the gastric fundus fold. Signs of hypotrophy with atrophy sites (h-e., 10x16).

Figure 5. The rat’s small intestines. (1000 mcW/cm² RFEMR, 3 months), the thinned apical layers and the destroyed intestinal villi. Destruction with expressed hypotrophy and atrophy sites in the apical layers (h-e., 10x16).

In the cardiovascular system, there were also marked considerable morphological and functional changes aggravating in the process of an increase in the RFEMR dose and duration of its impact.

Micro-vascular impairment, in our opinion, is the critical factor in the expression of structural manifestations both in acute and chronic RFEMR impact on the organs and tissues.

Thus: The RFEMR impact is manifested by pathological changes in the structure of the majority of organs and tissues.

References:

Marfan syndrome and its genealogic characteristics

Abstract: Marfan syndrome is met in 62% of each generation of pedigree and it is called vertical transfer of the disease. Correlation of sick and healthy persons is about 1:1. Sick men and women have similar transmission rate of the disease to their children. In 26.1% of the patients alterations of the organ of vision was the single symptom of Marfan disease, besides genetic tests.

Keywords: Marfan syndrome, genealogic signs, optic manifestations, children.

Among all hereditary diseases of connective tissue the greatest interest for pediatricians and general practitioners is caused by Marfan syndrome as the life duration of these patients is limited by 30–40 years [3; 4]. That disease with autosomal-dominant type