

BIOMEDICAL

Pathological Findings Observed in the Kidneys of Postnatal Male Rats Exposed to the 2100 MHz Electromagnetic Field

Recep Bedir,^a Levent Tumkaya,^b Tolga Mercantepe,^b and Adnan Yilmaz^c^aDepartment of Pathology, Recep Tayyip Erdogan University of Medical Faculty, Rize, Turkey^bDepartment of Histology and Embryology, Recep Tayyip Erdogan University of Medical Faculty, Rize, Turkey^cDepartment of Biochemistry, Recep Tayyip Erdogan University of Medical Faculty, Rize, Turkey

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Background. The widespread use by young people of modern communication devices such as mobile phones means that they are particularly exposed to electromagnetic fields (EMF) and other problems. However, few studies have researched the effects of long-term exposure to EMF in the kidney. We therefore investigated oxidative stress and apoptosis in long-term exposure to 2100 megahertz (MHz) in a rat model.

Materials and Methods. Twenty-four Sprague Dawley rats were divided into a control group ($n = 8$, no EMF exposure), a group exposed to 2100 MHz for 6 h for 30 d ($n = 8$), and a group exposed to 2100 MHz for 12 h for 30 d ($n = 8$). Immunohistochemical analysis was performed, using caspase-3 to evaluate apoptosis. Immediately after treatment, reduced glutathione (GSH), malondialdehyde (MDA) in kidney tissue and serum levels of various biochemical compounds were measured to detect oxidative stress.

Results. Deterioration was observed in the brush border in renal tubules of the EMF groups. The results of the immunohistochemical analysis revealed a greater number of positively stained renal tubular epithelial cells in the EMF groups as compared with that in the control group. In the EMF groups, renal MDA levels increased, and renal GSH levels decreased compared with those in the control group, as shown by a biochemical examination ($p = 0.00$ and $p = 0.00$, respectively).

Conclusion. The findings showed that exposure to 2100 MHz for 6 and 12 h induced oxidative stress-mediated acute renal injury, depending on the length of exposure and dosage. © 2018 IMSS. Published by Elsevier Inc.

Key Words: Apoptosis, Caspase-3, Electromagnetic field, Kidney, Oxidative stress.

Introduction

Despite the many benefits bestowed by communication devices, such as cell phones, exposure to the electromagnetic fields (EMFs) of these devices may pose potential health risks (1). As mobile phones are particularly popular among young people, they are exposed to EMF (2). Several studies have described the pathological effects of EMFs emissions from mobile phones (3–5). A number of studies have

described the potential causative roles of both natural and artificial sources of EMFs in health problems, depending on the biochemical, physiological, and morphological effects of these EMFs on living tissues, as well as the dose and duration of exposure (6,7). Depending on the mobile phone model and use characteristics, mobile phones emit a radiofrequency of 800–2,200 MHz (8,9). Third-generation mobile phones emit an EMF at a radio frequency of 2100 MHz (8). Studies have reported both reversible and irreversible pathological effects of exposure to a 900 MHz EMF on various organs (10–15). Although previous studies have focused on exposure to 2100 MHz EMF (16,17) none have examined the effect on the kidneys of exposure to 2100 MHz EMF. Healthy kidneys play numerous important roles. These include the removal of excess water,

Address reprint requests to: Tolga Mercantepe, Assistant Prof., Department of Histology and Embryology, Faculty of Medicine, Recep Tayyip Erdogan University, Islampasa Mahallesi, İki numaralı Şehitler Caddesi, No: 74, Rize, 53010, Turkey; Phone: (+90) 544 255 9555; FAX: (+90) 464 212 30 15; E-mail: tolgamercantepe@yahoo.com

maintenance of body homeostasis, regulation of chemical compounds in blood, elimination of waste products, secretion of hormones (renin, erythropoietin, vitamin-D, and prostaglandin), regulation of blood pressure, maintenance of bone health, and prevention of anemia (5). Although the mechanism involved in EMF-induced histopathological changes in tissues is unclear, studies reported that EMF exposure caused alterations in malondialdehyde (MDA) and glutathione (GSH) levels by triggering lipid peroxidation in association with free oxygen radicals (16,18). MDA is a product of unsaturated fatty acid breakdown and a marker of lipid peroxidation in tissue (Draper and Hadley 1990). Antioxidants, such as catalase, glutathione peroxidase, ceruloplasmin, GSH, and superoxide dismutase (SOD) protect cells against lipid peroxidation (19). A previous study reported that EMF exposure raised MDA levels in rats and reduced GSH levels (5). Reactive oxygen species (ROS) and other types of oxidative stress are known to cause apoptosis (20). Several studies also reported that a low EMF caused apoptosis (5,21). Caspases, particularly caspase-3 activation, play an important role in the terminal event prior to apoptosis (20).

Previous studies investigated the effects of 900 MHz mobile phone radiation on the kidneys, which were low in duration (lasting a few minutes) (1,3,4). In reality, mobile phone radiation exposure is far longer, lasting hours (22,23). In addition, the widely used 3G frequently utilizes 2100 MHz (8). For these reasons, this study investigated pathological alterations including oxidative stress and apoptosis in the kidneys of rats exposed to 2100 MHz radiation for 6 and 12 h a day.

Materials and Methods

Ethical Approval

The study protocol was reviewed and approved by the Local Ethical Committee for Animal Experiments in University School of Medicine (Approval date: 24 April 2017; meeting number: 2017/18).

Animals and Experimental Procedures

Twenty-four 230–260 g Sprague Dawley rats (4–5 months old) were divided into a control group ($n = 8$), a group exposed to 2100 MHz EMF for 6 h ($n = 8$), and a group exposed to a 2100 MHz EMF for 12 h ($n = 8$). The rats in the EMF groups were exposed to 2100 MHz for 30 d during the first postnatal month, whereas the control group was not exposed to any EMF during the postnatal period. The animals were housed under optimal laboratory conditions throughout the experimental procedure (at $22 \pm 10^\circ\text{C}$ in a 12 h light:12 h dark cycle). Ad libitum access was given to water and standard pellet chow containing 21% protein (Purina) during EMF exposure.

The EMF groups were continually exposed to electromagnetic waves produced by an EMF generator (Anritsu MG3670B, Kanagawa, Japan) for 30 d, 6 and 12 h per day. The generator had a signal power of 3.47 mW (5.4 dBm) at 2100 MHz, which is similar to the level experienced in mobile phone talk mode. The whole body specific absorption rate (SAR) was calculated to be 0.024 W/kg (RadHaz SAR Equivalency Calculator Version 1.0, Richard Tell Associates, Inc., Mesquite, NV) (5). The signal was emitted through a 15 cm long rod antenna, which was placed 2 cm below the cage ($50 \times 30 \times 30$) in which the one animals were housed each cage (Figure 1). The antenna was placed in the middle and parallel to the cage's short axis (22). The principal objective of this model was to achieve continuous electromagnetic exposure for one month. Similar concerns have been addressed in previous research by means of designs such as a carousel set-up (5,23,24). In fact that, studies using them have very short exposure duration, not exceeding a few minutes. So, we decided to provide a comfortable, and also a small environment for the rats, and expose the environment to an EMF instead. The mainstay of our set-up is a dipole antenna placed along the cage's short axis, which is equally distant to the cage's each side, had irradiated the whole living space of the rat (18). The principal objective of this set-up was to ensure continuous electromagnetic exposure for 1 month.

Biochemical Assays

Preparation of homogenates. After rinsing, cold phosphate buffer was added to renal tissue specimens at a volume of 10 times that of the tissue. All the samples were homogenized in a homogenizer for 1 min. Homogenized tissues were then centrifuged at $+40^\circ\text{C}$ and 3000g for 15 min, and the supernatant portions were used for biochemical analyses.

Determination of MDA and GSH Levels

The Ellman method was employed to measure renal GSH levels (24). This is based on spectrophotometrical measurement of the color created by free sulfhydryl groups in liver

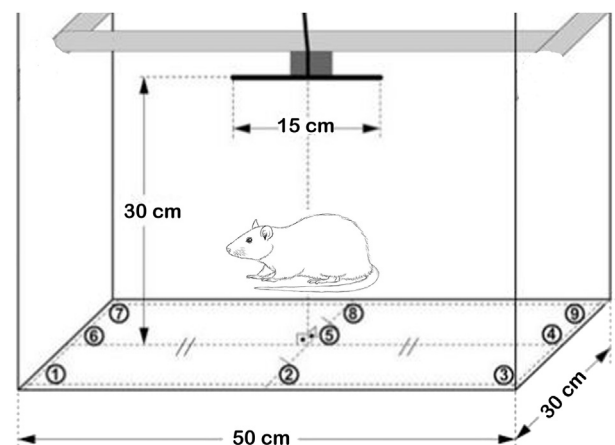


Figure 1. The size of the cage used to expose rats to a 2100 MHz EMF.

homogenate together with Elman reactive. MDA was measured using the method described by Draper and Hadley (25). MDA, the final product of lipid peroxidation, reacts with TBA and creates a pink complex that yields maximum absorbance at 532 nm.

Determination of Serum Urea, Creatine, Mg, Na, and Cl

Sera were obtained by centrifuging blood samples at 3000g for 10 min. The sera were assayed using an Architect C16000 auto-analyzer, and the levels of serum urea, creatine, Mg, Na, and Cl were determined (Architect c16000 Auto-analyzer, Abbott Diagnostics, Waltham, MA).

Histopathological Evaluation

Kidney tissues were fixed in a 10% formalin solution. Samples were subjected to routine pathological procedures, and sections 4–5 mm in thickness were obtained using a microtome (Leica, RM2525, Germany). The sections were subsequently stained with hematoxylin and eosin (Merck, Darmstadt, Germany) and counted 10 different areas randomly for each section under a photomicroscope (Olympus BX51, Olympus Corporation, Tokyo, Japan) by two histologists and one pathologist. The histologists and pathologist classifying the kidney tissue sections were also blinded to the treatment groups. During the microscopic examination, tissue sections were examined for the presence of deterioration of brush border structure in proximal tubules, vacuolization in the tubular epithelia, and debris accumulation in the lumen. Each of these three criteria was scored separately (26) (Table 1).

Immunohistochemistry (IHC) Procedures

Caspase-3 and apoptosis immunoreactivity status were detected using the streptavidin-biotin-peroxidase method. Kidney tissue samples were deparaffinized in xylene and rehydrated in ethanol series. Sections were incubated in 3% hydrogen peroxide in order to inhibit endogenous peroxidase activity. Antibody non-specific binding sites were blocked with normal bovine serum (1–10%). Primary Caspase-3 antibody (Gene Tex GTX110543) was diluted at a 1/50 and incubated for 60 min. Next, the slides were rinsed, and a biotinylated secondary antibody (Universal LSAB Kit-K0690, Dako, Denmark) was incubated for 30 min. Sections were then incubated with streptavidin-horseradish peroxidase (Universal LSAB Kit-K0690). Bound antibodies were identified by staining with 3,30 diaminobenzidine and washed in phosphate-buffered saline. After the hematoxylin counterstain, dehydration, clearing and mounting were performed (Bedir R, et al. 2015).

Semi-quantitative Analysis

Caspase-3 immunoreactivity status of the specimens with a three-point scoring system (Table 2). Two histologists and one pathologist, each blinded to the other's assessment,

Table 1. Tubular necrosis scoring method (Sung MJ, et al.)

Deterioration of brush border structure in proximal tubes	
Score	Percent (%)
0	No damage
1	≤10%
2	10–25%
3	26–75%
4	≥75%
Apical membranes blebbing	
0	No damage
1	≤10%
2	10–25%
3	26–75%
4	≥75%
Tubular epithelial cells losing each other or basal membrane junctions (debris accumulation in the lumen)	
0	No damage
1	≤10%
2	10–25%
3	26–75%
4	≥75%

assessed 30 different areas were counted randomly in each slide as mild (+), moderate (++) , severe (+++) or very severe (++++).

Quantitative Analysis

Renal corpuscle and proximal and distal tubule areas (μm^2) were measured using an Olympus DP2-BSW (Ver.2.1 to Ver.2.2, Build 6212, Olympus Corporation, Tokyo, Japan) software system. H&E stained slides were placed on the microscope tray, and their sectional boundaries were determined using this programme (27).

Statistical Analysis

Statistical analysis was performed using SPSS 18.0 (IBM Corporation, NY, USA). Parametric data was calculated as mean $\pm$ standard deviation. Differences between the groups were tested using one-way analysis of variance (ANOVA) followed by a Tukey HSD test (p values < 0.05 were regarded as significant). Nonparametric data analysis were calculated as median $\pm$ standard. Differences between the groups were tested using the Kruskal Wallis test (p values < 0.05 were regarded as significant).

Table 2. Grading of the Caspase-3 Staining Positivity Score

Grading of the Caspase-3 Staining Positivity Score	
0	None(less than 5%)
1	Mild (less than 25%)
2	Moderate (involvement 25–50%)
3	Severe (more than %50)

Table 3. Renal MDA and GSH levels in control and EMF groups (Mean $\pm$ Standard Deviation)

Groups	MDA (umol/g tissue)	GSH (umol/g tissue)
Control	0.22 $\pm$ 0.02	36.12 $\pm$ 0.73
EMF 6 h	0.25 $\pm$ 0.02 ^a	32.85 $\pm$ 3.57 ^a
EMF 12 h	0.26 $\pm$ 0.03 ^b	31.78 $\pm$ 0.70 ^b

h, hour; EMF, electromagnetic field.

^a $p < 0.05$ according to the control group.^b $p < 0.01$ according to the control group.

Results

Biochemical Analysis

Renal MDA levels were significantly increased in the EMF groups (2100 MHz for 6 or 12 h) as compared with those in the control group, whereas renal GSH levels were significantly decreased in these groups as compared with those in the control group ($p < 0.05$ and $p < 0.01$, respectively) (Table 3). Serum urea and creatine levels were also significantly increased in the EMF groups as compared with those in the control group ($p < 0.05$ and $p < 0.01$, respectively). There was a statistically significant difference between sodium, magnesium, and chloride values in the 12 h EMF group versus those in the control group, whereas there was no significant difference in these parameters in the 6 h EMF group as compared with those in control group (Table 4).

Histopathological Analysis

In the control group, sections stained with hematoxylin-eosin, PAS, and Masson's trichrome exhibited a normal histological appearance (Figures 2A–4A). The histopathological analysis of kidney specimens from the 6 h EMF group revealed greater dilatation in Bowman's capsules and renal tubules as compared with specimens in the control group (Figure 2B). Loss of the brush border was detected in proximal tubules in the 6 h EMF group (Figure 3B). In this group, vacuolization-related deformation in tubular epithelia, in addition to debris accumulation in the lumen, was observed (Figures 2B and 3B). Furthermore, fibrosis was observed in intertubular areas in the 6 h EMF group (Figure 4B). Deformation due to vacuolization was also observed in glomerulus structures in the 6 h EMF group, as well as an increase in dilatation areas

(Figures 2B and 3B). Histopathological findings in the 12 h EMF group were similar to those in the 6 h EMF group, although brush border deterioration and debris accumulation were more severe (Figures 2C–4C).

Immunohistochemical Analysis

In terms of apoptosis, the results of the immunohistochemical analysis revealed a significant increase in caspase-3 expression in tubular epithelial cells in the EMF groups as compared with that in the control group (Figure 5B, Table 5). There was the statistically significant difference in the number of caspase-3 positive cells in the 6 h and 12 h EMF groups (Figure 5, Table 5).

Semi-quantitative Analysis

Tubular necrosis scores in the 6 h and 12 h EMF groups increased significantly as compared with those in the control group ($p < 0.05$) (Figure 2B, Table 6).

Quantitative Analysis

Bowman capsules, proximal and distal tubules area in the 6 h and 12 h EMF groups increased significantly as compared with those in the control group ($p < 0.05$) (Table 7).

Discussion

Previous studies have employed a similar exposure method design, such as a carousel set-up (5,28,29). Several recent studies reported that EMF emissions from mobile phones caused molecular and cellular damage in mature living human tissues (1,3,4). In studies of adult animals, researchers concluded that various frequencies and durations of EMF appeared to cause pathological changes, such as glomerular atrophy, pyknotic nuclei in the renal tubular epithelium, and cytoplasmic vacuolization (30,31). Bedir R, et al. (2015) reported that exposure to EMF emissions from mobile phones in the postnatal period appeared to cause mild histopathological damage in the kidney and that this may result in renal failure in the postnatal period. Koca O, et al. (1998) examined the effects of EMF emissions from mobile phones on renal tissues of rats in a light microscopy study. They observed glomerular damage, dilatation

Table 4. Serum Urea, Creatine, Mg, Na, Cl levels in control and EMF groups (Mean $\pm$ Standard Deviation)

Groups	Urea (mg/dL)	Creatine (mg/dL)	Magnesium (mg/dL)	Sodium (mmol/L)	Chlorine (mmol/L)
Control	38.14 $\pm$ 1.57	0.41 $\pm$ 0.01	2.20 $\pm$ 0.14	133.71 $\pm$ 1.50	94.57 $\pm$ 1.39
EMF 6 h	41.28 $\pm$ 1.11 ^a	0.43 $\pm$ 0.01 ^a	2.28 $\pm$ 0.06	136.14 $\pm$ 2.03	95.85 $\pm$ 1.21
EMF 12 h	42.71 $\pm$ 1.60 ^b	0.45 $\pm$ 0.02 ^b	2.40 $\pm$ 0.08 ^a	137.01 $\pm$ 2.08 ^a	97.00 $\pm$ 0.81 ^a

^a $p < 0.05$ according to the Control vs 6 h.^b $p < 0.01$ according to the Control vs 12 h.

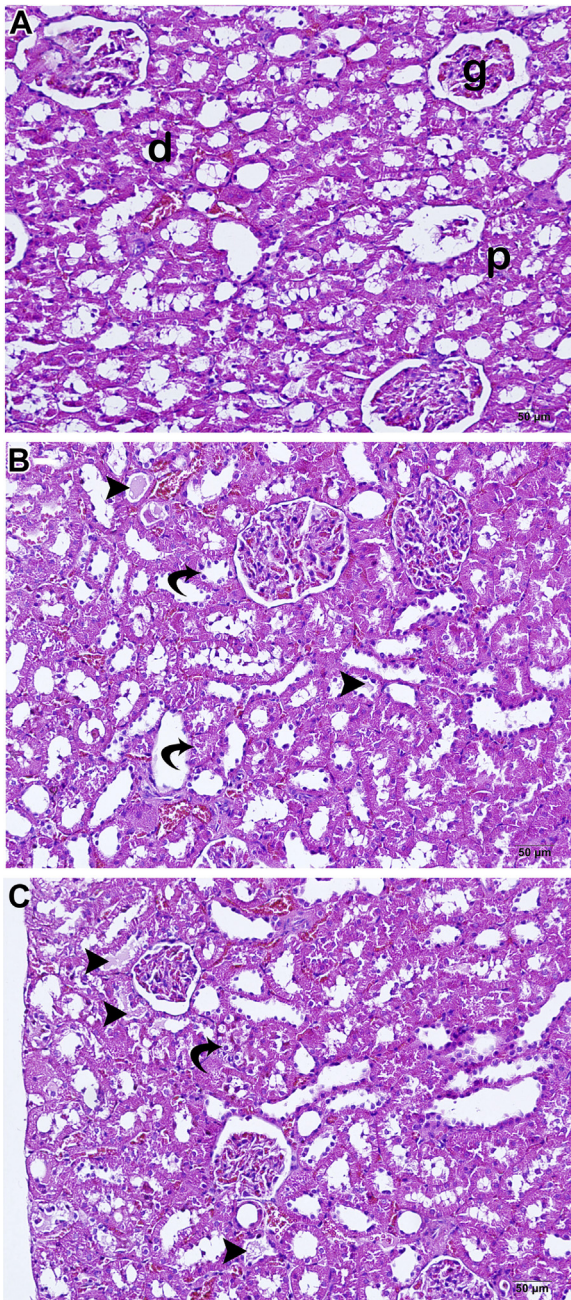


Figure 2. Light microscopic image of the renal tissue. H&E staining. Proximal tubule (p). Distal tubule (d). Glomerulus (g). A(x400): Normal glomerulus (g), and proximal (p), and distal tubules (d) were observed in cross-sections from the control group. B(x200): Cross-sections from the 6 h EMF group revealed debris accumulations (arrowhead) in the tubular lumens and vacuolization (curved arrow) in epithelial cells. C(x200): Cross-sections from the 12 h EMF group revealed debris accumulations (arrowhead) in the tubular lumens and vacuolization (curved arrow) in epithelial cells.

in Bowman's capsule, the formation of large holes between renal tubules, tubular damage, perivascular edema, and inflammatory cellular infiltration. Türedi S, et al. (2015) used light microscopy to examine renal tissues of rats exposed to

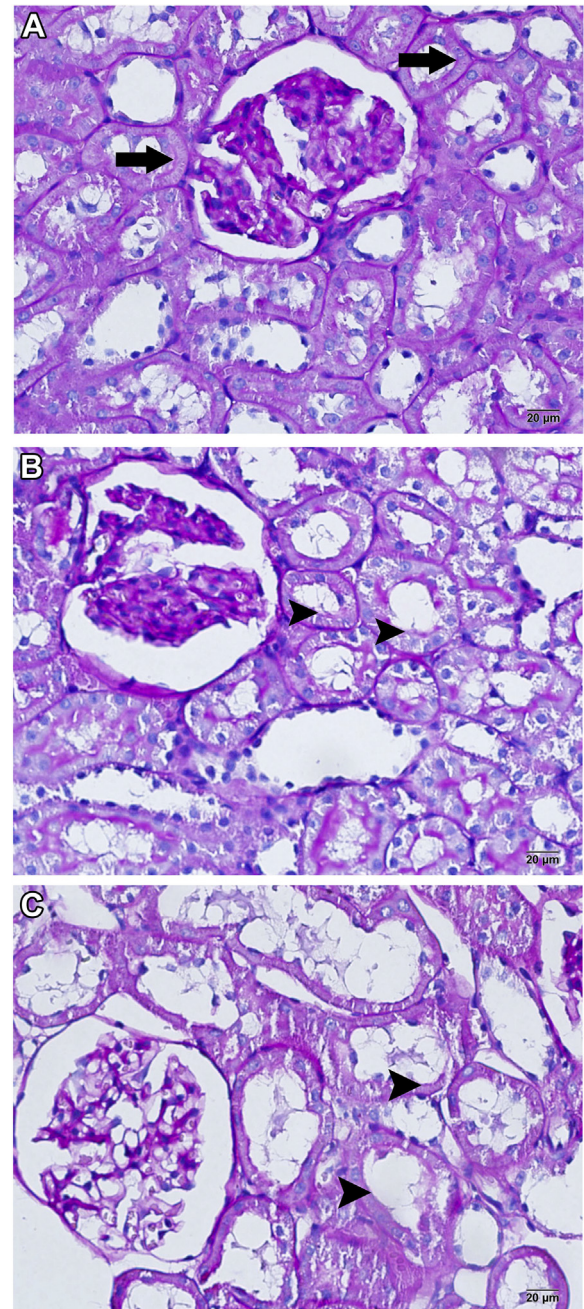


Figure 3. Light microscopic image of the renal tissue. PAS staining. A(x400): Cross-sections from the control group showing proximal tubules with normal brush borders (arrow) (median TNS score: 1.00 ± 0.67). B(x400): Cross-sections from the 6 h EMF group showing atypical proximal tubules with loss of brush border structure (arrowhead) (median TNS score: 5.00 ± 1.33). C(x400): Cross-sections from the 12 h EMF group showing atypical proximal tubules with loss of brush border structure (arrowhead) (median TNS score: 7.00 ± 0.98).

a 900 MHz EMF in the postnatal period and reported severe glomerular degeneration, diffuse tubular dilatation, and vacuolization in tubular epithelial cells, as well as a low level of edema. Kuybulu AE, et al. (2016) observed tubular damage in rat kidney following prolonged exposure to EMF

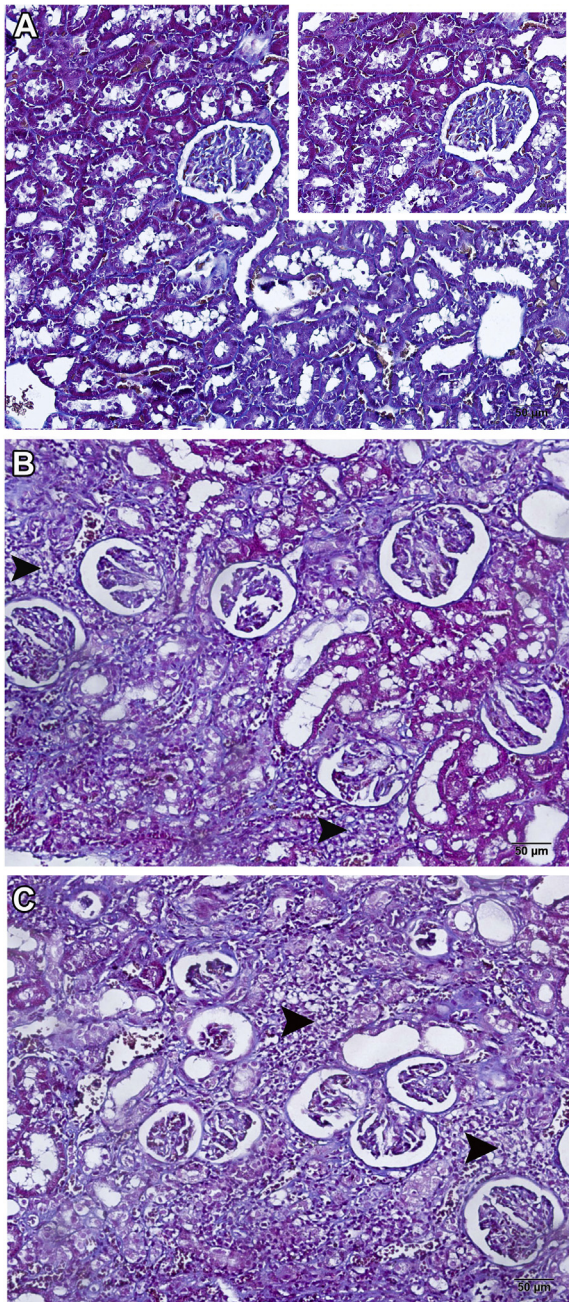


Figure 4. Light microscopic image of the renal tissue. Masson's trichrome staining. A (x400): Cross-sections from the control group showing a normal kidney structure B(x200): Cross-sections from the 6 h EMF group showing a slight increase fibrosis in the interstitial area (arrowhead). C(x200): Cross-sections from the 12 h EMF group showing an increase fibrosis in the interstitial area (arrowhead).

emissions from 2.45 GHz wireless devices in the prenatal and postnatal periods and reported that this may cause chronic renal failure. Tubular damage in kidneys plays an important role in the development of acute renal failure (26). Glomerular damage is an important marker of the severity of kidney damage (32). In the present study, the findings of light microscopy were compatible with those

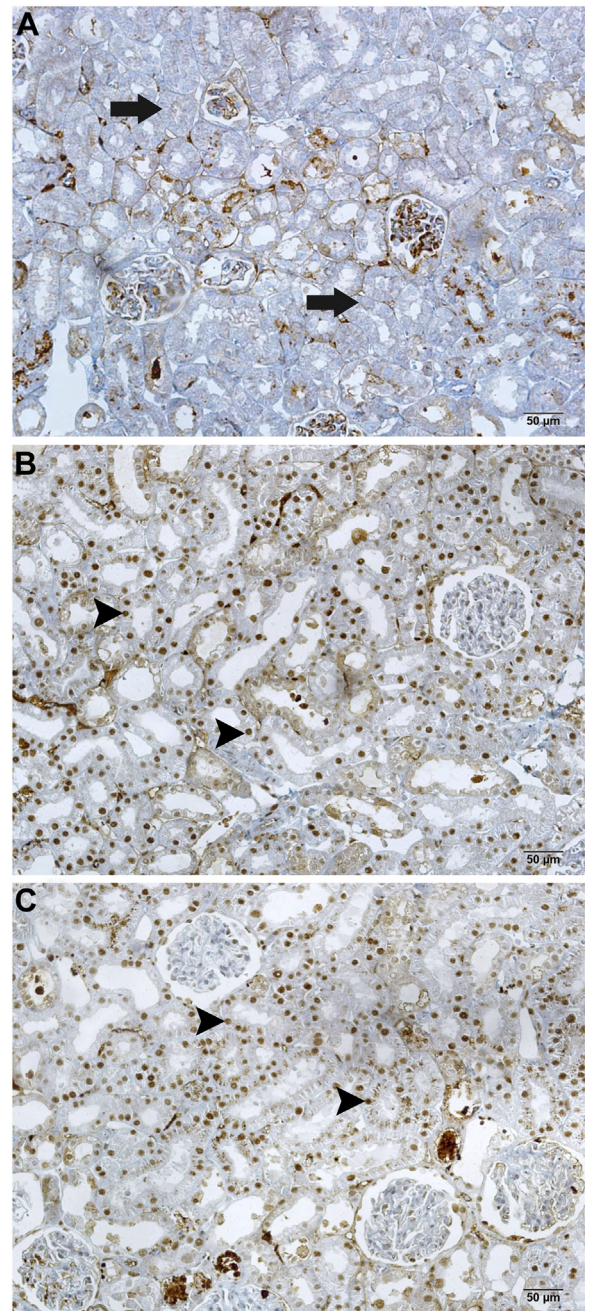


Figure 5. Light microscopic image of staining with Caspase-3. A (x200): Cross-sections from the control group showing normal proximal and distal tubular structures (Caspase-3 positivity score median: 0.00 ± 0.41) B(x400): Cross-sections from the 6 h EMF group showing apoptotic cells among the tubular epithelial cells (arrow) (Caspase-3 positivity score median: 2.00 ± 0.41). C(x400): Cross-sections from the 12 h EMF group showing apoptotic cells among the tubular epithelial cells (arrow) (Caspase-3 positivity score median: 3.00 ± 0.41).

of other previous studies. Furthermore, contrary to other studies, we determined that EMF increases tubular necrosis proportional to the duration of exposure.

Ozguner MF, et al. (2005) reported the formation of free radicals in association with mobile phone exposure and

Table 5. Caspase-3 positivity scores (Median $\pm$ Standard Deviation).

Control	0.00 $\pm$ 0.41
EMF 6 h	2.00 $\pm$ 0.41 ^a
EMF12 h	3.00 $\pm$ 0.41 ^a

EMF, electromagnetic field.

Kruskal Wallis -Tamhane's T2 test.

^a*p* <0.05 vs. to control group.

described adverse effects of these free radicals on kidneys in a rat study (33). Ragy MM, (2015) examined the effect of 900 MHz EMF emissions from mobile phones on the brain, liver, and renal tissues of male albino rats. In the study, EMF exposure resulted in a significant increase in MDA levels and a significant decrease in total antioxidant capacity values in renal tissues. Ragy MM, (2015) concluded that EMF emissions from mobile phones may cause oxidative stress and biochemical stress in renal tissues of albino rats. Ulubay M, et al. (34) reported decreased total renal volume and numbers of glomeruli in rats exposed to a 900 MHz EMF in the prenatal period.

Oxidative stress is defined as deterioration of the oxidative balance as a result of an increase in reactive oxygen species (ROS), such as hydroxyl radicals, superoxide radicals, and hydrogen peroxide, during cellular metabolism and an absence of antioxidants detoxifying these ROS (2,6,16,30). An EMF causes oxidative stress by altering antioxidant defense mechanisms in tissues, and the resulting increase in free radicals affects biological systems (35). There is evidence that EMF emissions impair pathways that neutralize superoxide anions and hydroxyl radicals. For example, SOD activity and GSH levels, both oxidative stress markers, decreased in kidneys exposed to a 60 MHz EMF for 2 h daily (6). Öztürk A, et al. (31) reported a significant increase in MDA levels, indicating oxidative stress, and a decrease in levels of GSH, which plays a role in the antioxidant defense mechanism, in renal and testicular tissues of rats exposed to a 50 MHz EMF (31). In another study, a low-dose EMF increased oxidative stress and led to toxicity in tissues by weakening the antioxidant defense system (36). Bodera P, et al. (37) determined that the risk of oxidative stress in brain, liver, blood, and kidney tissues in rats exposed to a 1800 MHz EMF increased, depending on the duration and amount of exposure. Türedi S, et al. (38) reported that exposure to 900 MHz for 1 h daily caused various pathological

Table 6. Tubular Necrosis Score (TNS) Analysis (Median $\pm$ Standard Deviation)

Control	1.00 $\pm$ 0.67
EMF 6h	5.00 $\pm$ 1.33 ^a
EMF12h	7.00 $\pm$ 0.98 ^b

Median $\pm$ Standard Deviation.

Kruskal Wallis -Tamhane's T2 test.

^a*p* <0.05 vs. to control group.^b*p* <0.05 vs. to EMF6h group.**Table 7.** Quantitative measurement (μm^2) data (Mean $\pm$ Standard Deviation)

Groups	Proximal tubules area (μm^2)	Distal tubules area (μm^2)	Bowman capsules area (μm^2)
Control	1521.64 $\pm$ 485.03	1905.28 $\pm$ 426.55	13357.52 $\pm$ 2218.61
EMF 6h	2364.974 $\pm$ 261.61 ^a	2452.20 $\pm$ 638.75 ^a	15302.37 $\pm$ 2959.82 ^a
EMF 12h	2305.41 $\pm$ 794.93 ^a	2749.72 $\pm$ 356.83 ^a	20830.17 $\pm$ 4277.71 ^a

Tukey HSD test.

^a*p* >0.05 vs. to control group.

changes in kidney and bladder tissues and increased oxidative stress in rats aged 22–59 d. In another study of male rats aged 13–21 d exposed to a 900 MHz EMF during the prenatal period, Odacı E, et al. (5) reported a decrease in SOD and catalase activities at in 21 d old rats. Kuybulu AE, et al. (39) reported that kidneys of rats exposed to prolonged EMF emissions from a 2.45 GHz wireless device during the prenatal and postnatal periods were adversely affected by oxidative stress. In the present study, MDA levels significantly increased, whereas those of GSH significantly decreased as compared with those in the control group.

Ragy MM, (40) reported that EMF exposure led to changes in biochemical parameters due to the induction of oxidative stress. Although Salem et al. reported that EMF exposure had no effect on serum creatinine and serum urea, another study found that EMF exposure increased serum creatinine and serum urea levels. In contrast, Moussa SA, et al. (41) reported that EMF increased serum creatinine and serum urea levels. Similarly, in the present study, both serum creatinine and urea values increased (Table 4). Additionally, Bedir R, et al. reported that a 900 MHz EMF resulted in no changes in serum sodium, magnesium, or chloride levels. In contrast, in the present study, although there was no change in sodium levels in the 6 h EMF group, sodium, magnesium, and chloride levels increased in the 12 h EMF group (Table 4). The discord in the findings of the two studies may be due to the lower frequency in the previous study (900 MHz) as compared with that (2100 MHz) in the current study. Similarly, Ozlem N, et al. (42) reported that tissue damage levels increased in line with the EMF frequency. In the present study, although there were no changes in sodium, magnesium, or chloride levels in the 6 h EMF group, the levels of these parameters increased in the 12 h EMF group in line with the duration and dose of EMF exposure. Similarly, Özorak A, et al. (43) reported that cellular damage in biological systems developed in line with the power intensity of EMF frequency and the duration of exposure.

Tubular cells are the most sensitive part of the kidney to oxidative stress (5,16,26,30,34,37). In cases of acute renal failure, apoptotic cell numbers rapidly increase, and DNA degradation also increases (27,28,32). Sehitoglu I, et al.

(44) reported a significant increase in caspase-3 activity in testicular tissues of fetal rats exposed to a 900 MHz EMF for 12 h over 20 d as compared with that in a control group. Bedir R, et al. examined renal tissues of rats exposed to a 900 MHz EMF during the first 20 prenatal days. They reported diffuse positive staining with caspase-3 in the cytoplasm of proximal and distal tubular epithelial cells as compared with that in a control group (28). In the present study, more severe caspase-3 positive staining occurred in tubular epithelial cells of the rats exposed to the EMF.

Conclusion

This study showed that 2100 MHz EMF caused pathological changes in the kidney and increased oxidative stress. Further research is required concerning the effect of mobile phone radiation on intracellular calcium levels and molecules involved in oxidative damage in the kidney.

Acknowledgments

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