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Morphological Characteristics of Testicles under Radiation

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Resume: There are practically few works in the literature in which morphological changes of testicles under radiation radiation would be presented. Pathomorphological study of the state of the seminal tubules revealed morphological signs of spermatogenesis disorders, mainly in the form of delayed development of the seminal tubules in - hypospermatogenesis of I, II and III degrees, a spermatogenesis (sclerosis of the seminal tubules, Sertoli cell syndrome).

Key words: testicular radiation, hypospermatogenesis, aspermatogenesis.

With a moderate decrease in the renal reserve, the course is usually asymptomatic. Even patients with mild or moderate renal insufficiency may not have symptoms of increased levels of urea nitrogen in the blood (AMC) and creatinine. Nocturia is often observed, especially due to the inability to concentrate urine. Apathy, fatigue, lack of appetite and decreased clarity of thinking are often the earliest manifestations of uremia.(1)

With more severe kidney disease (for example, the estimated glomerular filtration rate [EGFR] < 15 ml/min/1.73 m2), neuromuscular symptoms may appear, including pronounced muscle twitching, peripheral sensory and motor neuropathy, muscle cramps, hyperreflexia, restless legs syndrome and seizures (usually as a result of hypertensive or metabolic encephalopathy).(2)

Anorexia, nausea, vomiting, weight loss, stomatitis and an unpleasant taste in the mouth are very common. The skin tone may turn yellow-brown. Sometimes crystals of urea with sweat are released on the surface of the skin, forming uremic frost. Itching can cause serious inconvenience. Nutritional deficiency leading to generalized tissue loss is a distinctive feature of chronic uremia.

As a rule, the presence of chronic kidney disease (CKD) is suspected for the first time with an increase in serum creatinine levels. The first step is to determine whether the kidney failure is acute, chronic or acute, turned into chronic (for example, an acute disease that further disrupts kidney function in a patient with CKD-see the table of Differences between acute kidney injury and Chronic Kidney Disease [Distinguishing Acute Kidney Injury From Chronic Kidney Disease]). The cause of renal failure has also been determined. Sometimes determining the duration of kidney failure helps to determine the cause; sometimes it is easier to determine the cause than the duration, and finding out the cause helps to determine the duration.

The examination includes a urine analysis with a microscopy of urinary sediment, an assessment of the level of electrolytes, urea nitrogen, creatinine, phosphates, calcium and a general blood test. Sometimes special serological tests are needed to determine the cause. In the differential diagnosis of acute renal failure and CRF, anamnesis of increased creatinine or abnormalities in urine analysis is most useful. Changes in urine analysis depend on the underlying disease of PN, but large (> 3 leukocytes in diameter) and especially waxy cylinders usually prevail in any severe renal insufficiency.(5)

Ultrasound of the kidneys usually helps to identify obstructive uropathy and differentiate acute renal failure from CRF based on the size of the kidneys. With the exception of some diseases (see the table The main causes of Chronic Kidney Disease [1], CRF is accompanied by shrinking and a decrease in

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kidney size (usually up to < 10 cm in length) with a thin hyperechoic cortical layer. It is especially difficult to establish an accurate diagnosis in the terminal stage of chronic kidney disease. The final diagnostic tool is a kidney biopsy, but it is not recommended to perform it if reduced sclerosed kidneys are detected on ultrasound; the high risk of the procedure outweighs the low diagnostic significance.(6)

The stages of CKD determine the severity of the disease. The classification of CKD includes 5 stages.

Stage 1: Normal GFR (≥90 ml/min /1.73 m2) in combination with persistent albuminuria or a known pathology of the kidney structure or hereditary pathology.

Stage 2: GFR 60-89 ml/min/1.73 m2.

Stage 3a: 45-59 ml/min/1.73 m2

Stage 3b: 30-44 ml/min/1.73 m2

Stage 4: GFR 15-29 ml/min/1.73 m2

Stage 5: GFR <15 ml/min/1.73 m2

GFR (in ml/min/1.73 m2) in CKD can be assessed using the creatinine formula developed within the framework of the program "Cooperation in the field of Epidemiology of Chronic Kidney Diseases" (CKD-EPI) (1): 141 × (serum creatinine) -1.209× 0.993 age. The result is multiplied by 1.018 if the patient is female, and by 1.159 if the patient is African American. For Afro-American women, the result is multiplied by 1.018 × 1.159 (1.1799) Alternatively, GFR can be determined using a timed (most often 24 hours) urinary creatinine clearance using creatinine levels measured in serum and urine; this formula tends to give a GFR value 10-20% higher. It is used when the evaluation of serum creatinine may not be very reliable (for example, in sedentary patients, very obese or very thin). Serum cystatin C is an alternative marker of endogenous GFR used as a confirmatory test in people with extrarenal factors affecting serum creatinine levels (for example, extremely high or low muscle mass, consumption of exogenous creatine, amputations or neuromuscular diseases, as well as diets high in protein or exclusively based on plants). GFR is calculated by the formula: CKD-EPI cystatin C equation (7).

The CKD-EPI formula is more accurate than the formulas for changing the diet for renal insufficiency (MDRD), Cockcroft and Gault, especially for patients with close to normal GFR. The CKD-EPI equation gives fewer false positive results indicating chronic kidney disease and evaluates the possible outcome better than other formulas.

Radiation in lethal doses causes profound changes in the testicles. In the first days after exposure to X-rays at a minimally lethal dose [1,3].There is a sharp fullness of capillaries and small blood vessels, swelling of the interstitial tissue, death of spermatogenic cells. Quantitative determination of the incidence of certain types of spermatogenic cells shows that spermatogenesis of type B is the most radio-sensitive. The maximum death of such cells occurs within 24 hours after irradiation at LD50. Spermatogenesis of type A is more stable [6,8]. However, type II spermatogenesis involves a population of reserve stem cells, which rarely divide in the adult body, and renewing cells, which are constantly in a state of division. The first type of cells undergoes degenerative changes only when irradiated at doses of at least 1000 R, and the second - after irradiation at a dose of 100 R. Like all intensively dividing cells, the last type of spermatogonaise dies in the interphase or at the very beginning of prophase [4,5]. The radio sensitivity of germ cells increases as they mature, so spermatozoa are affected more than spermatogonez [2,4]. At the same time, the spermatozoa and spermatozoa of the irradiated organism do not lose their ability to fertilize. In the first days after irradiation, no changes are detected in Sertoli and Leydig cells [13]. By the end of the 1st week of

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radiation sickness, only a few altered spermatocytes, spermatids, a large number of multinucleated and bizarre forms of spermatogenic cells, as well as hyperplastic Leydig cells are found in the tubules [13,16,17]. However, the reduction of cells in the seminal tubules is mainly due not to necrobiotic and dystrophic changes in them, but to the cessation of division [8,9,10]. radiation damage to the kidneys. It is significant that in the recovery period of radiation sickness there is no complete morphological regeneration of the seminal tubules. Even a month after irradiation at a minimally lethal dose, there are still dormant tubules containing only follicular cells [1,3]. In the long term after irradiation, significantly less than usual spermatogenesis of all types and spermatocytes of the 1st order are also detected [6,7,8]. The same changes were described in the testicles of people who died as a result of atomic bombings of Japanese cities, and in large animals irradiated during experimental explosions of atomic devices [13,16].

The purpose of the study. To study the morphological changes of the testicles during radiation radiation.

Material and methods of research

An experimental study was conducted on the testicles of 51 white rats (Table 1). The material was divided into 2 groups.

Table 1 Distribution of experimental material

Number of animals		Total
control	in the experiment, the effect of radiation radiation	
25	26	51

As a result of the study, the following morphological changes were found

Degree of lesions						Total
Hypospermatogenesis			Retention of seminal tubules	Spermatogenesis		
1 degree	2 degree	3 degree		Sertoli Cell Syndrome	Sclerosis Seminal tubules	
3	7	8	3	2	3	26

Pathomorphological study of the state of the seminal tubules revealed morphological signs of spermatogenesis disorders, mainly 10% in the form of delayed development of the seminal tubules, 70% hypospermatogenesis of I, II and III degrees, 20% aspermatogenesis (sclerosis of the seminal tubules, Sertoli cell syndrome). The revealed structural changes of the seminal tubules fit into the picture repeatedly described in secretory infertility.

The results of the study can be used in the activities of a general practitioner and urologists, endocrinologists. The obtained data can be included in the program of histology, pathophysiology, urology and pediatrics in the sections "Structure and development of the organs of the male reproductive system", "Microscopic structure of the organs of the male reproductive system", "Influence of environmental factors on the reproductive function of the body ", "Etiology and pathogenesis of male infertility".

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