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Original Article

Morphological assessment of structural reorganization of the testicular hemomicrocirculatory bed under conditions of diabetes mellitus and burn trauma

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Abstract

The study of structural changes in the testes under conditions of common pathologies, such as diabetes mellitus and burn disease, is extremely important due to their systemic impact on the vascular system and the high risk of developing multiple organ failure. The aim of this work was to conduct a morphometric assessment of the structural reorganization of the testicular hemomicrocirculatory bed in experimental diabetes mellitus, burn injury, and their combination. The study was conducted on 43 white male rats. The diameters of arterioles, capillaries, and venules, as well as the density of microvessels, were determined morphometrically. It was established that in all pathological conditions, there is a narrowing of the arterial link and an expansion of the venous link. The degree of pathological reorganization was more prominent in the left testis. The most pronounced changes were recorded in the combination of diabetes and burn: the diameter of the arterioles of the left testis decreased by 32.0%, precapillary arterioles by 36.0%, and the lumen of the hemocapillaries by 24.7%. At the same time, the diameter of the postcapillary venules increased by 44.2% and venules by 47.8%. The density of microvessels in the combined lesion decreased nearly twofold by 49.0%, indicating a critical deterioration of the blood supply and pronounced venous stasis. The combination of diabetes mellitus and burn injury leads to deep destructive restructuring of the microcirculatory bed of the testes.

Keywords: testis, microcirculation, morphometry, diabetes mellitus, burns, reproductive health

Introduction

In modern morphology, a pressing problem is the study of the structure of intact organs, tissues, and cells, as well as the patterns of structural changes when exposed to various endogenous and exogenous factors. This fully applies to the testes – the central organs of the male reproductive system – which are among the first to respond to environmental and internal stimuli. Further study of the structure of the testes in humans and experimental animals is important due to the widespread and increasing incidence of various types of diabetes mellitus (DM), which in the vast majority of cases lead to multiple organ failure [1, 2].

The last decades have been characterized by significant progress in the prevention, diagnosis, and treatment of various types of diabetes mellitus, which has significantly reduced mortality and disability related to macro- and microangiopathy in most developed regions. At the same time, it is worth noting that these achievements do not diminish the priority of studying this critical problem [3].

When studying biological objects, morphologists increasingly employ quantitative morphological methods

(morphometry). These methods allow for obtaining objective quantitative characteristics of structural reorganization in various physiological and pathological processes, as well as their logical interpretation [4, 5].

Clinicians and morphologists have begun to pay closer attention to the features of testicular structure remodeling in diabetes. A pathogenetic relationship is being traced between reproductive dysfunction and diabetes mellitus, both of which show an increasing trend [6, 7]. Researchers have demonstrated that in the presence of diabetes, reproductive outcomes deteriorate three to four times compared to the population without diabetes. It has been established that the pathogenetic mechanisms of diabetes in the development of erectile dysfunction carry greater significance and weight than traditional exogenous and endogenous factors [3].

Sexual function is a key element of a normal male lifestyle. Studies have demonstrated a correlation between erectile dysfunction (ED), age, and chronic diseases, with diabetes mellitus being the most prominent factor. ED is one of the most common chronic dysfunctions in men over 40 years of age, affecting millions worldwide. On average, the risk of developing ED is approximately 26 cases per 1,000 men annually. This risk increases with age and is exacerbated by diabetes mellitus, cardiovascular disease, arterial hypertension, urogenital surgery, adverse environmental factors, limited sexual education, and increased psycho-emotional stress. According to various authors and our own research, the prevalence of ED in patients with type 1 and type 2 diabetes ranges from 35% to 80%, making it the most frequent complication of diabetes in men [8, 9].

Hyperglycemia serves as the basis for pathophysiological changes in testicular structures, leading to impaired microcirculation and the development of morphofunctional alterations. Patients with diabetes mellitus frequently exhibit age-related sclerotic vascular lesions and impaired renal and liver function. A clear relationship has been traced between age-related decline in male sexual function and atherosclerosis, ischemic heart disease, arterial hypertension, prostatic hyperplasia, osteoporosis, and a tendency toward obesity. Furthermore, the incidence of gynecomastia increases with age. This is attributed to shifts in the plasma ratio of sex hormones (estrogens and androgens), which may result from liver or kidney failure, as well as the side effects of numerous medications necessary to maintain the overall health of these patients [10, 11].

In the overall structure of traumatic injuries, thermal injury ranks third and leads to the development of both toxemia and circulatory disorders in the male genitals [12, 13]. Thermal burns are among the most common traumatic injuries globally, with approximately fifty million people sustaining burns annually worldwide. Burn disease often results in severe disability for the victims.

According to statistics, the average age of patients in burn units is 24 years, with an average burn size of 19% of the total body surface area. Most recorded cases of burn injuries are caused by negligence, while the remaining cases are associated with smoking and alcohol consumption. The face and hands are the most common areas of injury, respiratory tract lesions occur frequently, whereas eye burns are the least common. Men, especially those of a young age, are more prone to burn injuries. Two-thirds of all burns are caused by contact with hot or corrosive substances, and only a quarter of all cases result from fire and flame [14].

It has been established that cutaneous burn injury (exceeding 15% of the total body surface area in adults) triggers the development of burn disease. Its primary components include a generalized catabolic reaction at the injury site and within all internal organs, systemic inflammatory and apoptotic responses, endogenous intoxication, and multiple organ failure – all of which have been documented in experimental and clinical studies. Burn disease is characterized by specific stages, burn shock (days 1–3), burn septicotoxemia (lasting until skin restoration), and convalescence (from skin restoration until the full recovery of organ and system functions). Burn disease involves a complex constellation of clinical, morphological, biochemical, and metabolic disorders [15].

The initiating factors of these disorders are toxins and various biologically active substances derived from the destruction of the body's own tissues at the burn site (the histotoxic theory of burn disease pathogenesis). These decomposition products enter the bloodstream, leading to autointoxication and circulatory-toxic hypoxia in organs and tissues not initially affected by the thermal injury. These secondarily affected tissues then become additional sources of toxins, further exacerbating and spreading endogenous intoxication [13].

A significant role in the development of burn-induced endogenous intoxication is played by various substances, including proteolytic enzymes and medium-molecular-weight proteins, which are products of the proteolytic cleavage of blood plasma and tissue peptides. Other contributors include microbial toxins, cytokines, prostaglandins, and immune reaction mediators. The rapid progression of endogenous intoxication

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leads to other characteristic manifestations of burn disease, such as hypermetabolic syndrome, systemic inflammatory and apoptotic responses, and multiple organ dysfunction syndrome [16].

The clinical challenges associated with the progression of burn disease, diabetes mellitus, and their complications stem from the lack of a comprehensive theoretical foundation. Specifically, the structural and functional mechanisms underlying the development of both diseases and their associated complications have not been fully elucidated. The relevance of this problem is further underscored by the increasing number of experimental studies in this field. It is noteworthy that the structural changes in the testicular hemomicrocirculatory bed under various types of diabetes mellitus and burn disease remain insufficiently studied.

The aim of this study was to perform a morphometric analysis of the structural reorganization of the testicular hemomicrocirculatory bed under conditions of diabetes mellitus, burn injury, and their combination.

Material and methods

The study was conducted on the testes of 43 white male rats, divided into four groups. The first group (n=7) consisted of intact animals, the second group (n=12) included rats with experimental streptozotocin-induced diabetes, the third group (n=12) comprised experimental animals with burn injury and the fourth group (n=12) consisted of rats with both experimental streptozotocin-induced diabetes and burn injury (one animal died during the study). Experimental diabetes mellitus was induced by a single intraperitoneal injection of streptozotocin at a dose of 50 mg/kg, previously dissolved in 0.1 M citrate buffer (pH 4.5). The development of hyperglycemia in the second and fourth groups was confirmed by blood glucose levels, which averaged (14.24±0.79) mmol/L, compared to (4.03±0.4) mmol/L in the control group. Blood glucose concentration was determined by the enzymatic colorimetric method (GOD-PAP) using a BS-3000M semi-automatic analyzer (Sinnowa, China) and biochemical kits from Diagnosticum Inc. (Hungary). Burn injury was inflicted under thiopental anesthesia using two copper plates heated to 100°C. The plates were applied to the depilated skin of the back and lateral surfaces for 12 seconds. All animals were euthanized by exsanguination under thiopental anesthesia 28 days after the start of the experiment.

The testicular hemomicrocirculatory bed was studied by vascular casting with an India ink-gelatin mixture, injected through the abdominal aorta. Three to four hours after the injection, the testes were collected and fixed in a 10% neutral buffered formalin solution for two weeks. Sections (30–40 μm thick) were prepared using a freezing microtome, dehydrated in ethanol, cleared in methyl salicylate, and mounted in polystyrene. The resulting specimens were examined under an MBD-15 binocular microscope at various magnifications. Additionally, histological sections were stained with hematoxylin and eosin (H&E) from testicular tissue samples previously filled with the India ink-gelatin mixture [17]. Morphometric analysis included measuring the diameters of arterioles (DA), precapillary arterioles (DPA), hemocapillaries (DH), postcapillary venules (DPV), and venules (DV), as well as determining microvessel density (MVD) per 1 mm^2 in the tissues of the left (LT) and right (RT) testes. Quantitative data were processed statistically. Statistical analysis was performed using the STATISTICA software package at the Department of System Statistical Research of the I. Horbachevsky Ternopil National Medical University. Significant differences between groups were determined using Student's t-test [18].

Experimental procedures and euthanasia were conducted in compliance with the "General Ethical Principles of Animal Experimentation" adopted by the First National Congress on Bioethics (Kyiv, 2001), and in accordance with the "European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes" (Strasbourg, 1986). The study also adhered to the Law of Ukraine "On the Protection of Animals from Cruelty" (dated February 21, 2006).

Results

A morphometric study of the hemomicrocirculatory bed of the left and right testes was conducted in laboratory adult white male rats under the specified experimental conditions. The results obtained for the left testis are presented in Table 1.

A comprehensive analysis of the data presented in Table 1 revealed a pronounced structural reorganization of the testicular hemomicrocirculatory bed, as confirmed by significant changes in the studied morphometric parameters. Under simulated streptozotocin-induced diabetes, the arteriolar diameter of the left testis decreased from (18.20±0.30) μm to (14.88±0.24) μm 18.2% ($p<0.05$). Following burn injury and combined damage (diabetes and burn injury), this morphometric

Table 1: Morphometric characteristics of the hemomicrocirculatory bed of the left testis in experimental animals (M±m).

Parameter	Animal group			
	Group 1	Group 2	Group 3	Group 4
DA, μm	18.20±0.30	14.88±0.24*	13.91±0.18**	12.37±0.12***
DPA, μm	10.82±0.12	8.95±0.12***	8.14±0.20***	6.93±0.09***
DH, μm	6.12±0.09	5.60±0.09**	5.17±0.09***	4.61±0.12***
DPV, μm	12.58±0.15	15.83±0.15*	16.37±0.11**	18.14±0.08***
DV, μm	26.57±0.30	33.52±0.30**	36.42±0.2**	39.28±0.18***
MVD, n/mm²	3843.3±28.2	2794.5±21.3***	2360.5±18.3***	1958.9±15.7***

Note: * – p<0.05; ** – p<0.01; *** – p<0.001 compared to the intact group.

indicator decreased by 23.6% (p<0.01) and 32.0% (p<0.001), respectively. The diameter of the precapillary arterioles in the left testis followed a similar pattern.

The diameter of precapillary arterioles in the intact group was (10.82±0.12) μm, whereas in groups 2, 3, and 4, it was (8.95±0.12) μm, (8.14±0.20) μm, and (6.93±0.09) μm, respectively. These morphometric parameters showed statistically significant differences (p<0.001) across all groups. Specifically, this indicator was 17.3% lower in group 2 compared to the control, and 24.8% and 36.0% lower in groups 3 and 4, respectively. These findings indicate a reduction in the vascular throughput of the arterioles in the left testis and a subsequent deterioration of its blood supply.

The hemocapillary lumen of the left testis in streptozotocin-induced diabetes also decreased by 8.5% with a high degree of statistical significance (p<0.01). Similarly, this indicator was 15.5% lower than the control value in burn injury (p<0.001) and 24.7% lower in the case of combined damage (diabetes and burn injury) (p<0.001).

Quantitative morphological analysis established that the venous vessels of the testicular hemomicrocirculatory bed expanded under simulated streptozotocin-induced diabetes. Specifically, under these experimental conditions, the diameter of the postcapillary venules of the left testis significantly increased by 25.8%, and that of the venules by 26.2%. Furthermore, the microvessel density per unit area of the studied organ's tissue decreased significantly by nearly 27.3% (p<0.001). These changes indicate both a deterioration in arterial blood supply and impaired venous drainage.

Venous vessels of the testicular hemomicrocirculatory bed also exhibited expansion in the groups with burn injury and combined lesions (diabetes and burn injury). Specifically, the diameter of the postcapillary venules in the left testis increased by 30.1% (p<0.01) in the burn injury group and by 44.2% (p<0.001) in the combined lesion group. Similarly, the diameter of the venules increased by 37.1% (p<0.01) and 47.8% (p<0.001), respectively. Microvessel density per unit area of the studied tissue significantly decreased by 38.6% (p<0.001) in the third group and by 49.0% (p<0.001) in the fourth group. These findings indicate a progressive deterioration of arterial blood supply and a significant impairment of venous drainage.

The structural reorganization of the microvessels in the right testis under simulated experimental conditions followed a pattern similar to that described above (Table 2). Notably, morphometric analysis revealed that the degree of remodeling in the hemomicrocirculatory bed differed between the left and right testes. In streptozotocin-induced diabetes, the arteriolar diameter of the right testis significantly decreased from (18.22±0.30) μm to (15.35±0.27) μm 15.75%, (p<0.01). Under conditions of burn injury and combined damage (diabetes and burn injury), this morphometric parameter decreased by 22.1% (p<0.001) and 29.6% (p<0.001), respectively.

The diameter of the precapillary arterioles in the intact right testis was (10.85±0.12) μm, while in groups 2, 3, and 4, it was (9.05±0.09) μm, (8.37±0.09) μm, and (7.15±0.03) μm, respectively. These morphometric parameters showed statistically significant differences (p<0.001) across all groups. Analysis of group 2 revealed that this quantitative morphological indicator was 16.6% lower than the control, while in groups 3 and 4, it decreased by 22.9% and 34.1%, respectively. These findings indicate a reduction in the vascular capacity of the arterioles in the right testis and a subsequent deterioration of its blood supply.

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Table 2: Morphometric characteristics of the hemomicrocirculatory bed of the right testis in experimental animals (M±m).

Parameter	Animal group			
	Group 1	Group 2	Group 3	Group 4
DA, µm	18.22±0.30	15.35±0.27**	14.20±0.12***	12.83±0.09***
DPA, µm	10.85±0.12	9.05±0.09***	8.37±0.09***	7.15±0.03***
DH, µm	6.12±0.09	5.67±0.15*	5.25±0.18**	4.88±0.12**
DPV, µm	12.54±0.15	15.72±0.12*	16.05±0.15**	17.95±0.15***
DV, µm	26.53±0.30	33.56±0.27**	35.91±0.30**	39.04±0.30**
MVD, n/mm²	3836.8±30.3	2946.9±23.1***	2352.3±25.2***	2102.3±25.2***

Note: * – p<0.05; ** – p<0.01; *** – p<0.001 compared to the intact group.

The hemocapillary lumen of the right testis in streptozotocin-induced diabetes decreased by 7.35% (p<0.05). Similarly, this indicator was 14.2% lower than the control value in the burn injury group (p<0.01) and 20.3% lower in the combined damage group (diabetes and burn injury) (p<0.001).

The efferent vessels of the hemomicrocirculatory bed (postcapillary venules and venules) expanded under the simulated experimental conditions. Specifically, in animals with streptozotocin-induced diabetes, the diameter of the postcapillary venules in the right testis significantly increased by 25.4%, while the diameter of the venules increased by 26.5%. Furthermore, the microvessel density per unit area of the studied organ's tissue significantly decreased by nearly 23.2% (p<0.001). These structural alterations indicate both a deterioration in arterial blood supply and impaired venous drainage.

The diameters of the postcapillary venules in the right testis of animals with burn injury and combined lesions increased by 28.0% (p<0.01) and 43.1% (p<0.001), respectively, while the venule diameters increased by 35.4% (p<0.01) and 47.2% (p<0.01). Microvessel density per unit area of the studied organ significantly decreased by 38.7% (p<0.001) in group 3 and by 45.2% (p<0.001) in group 4. These findings indicate a deterioration in arterial blood supply and a significant impairment of venous drainage.

The presented morphometric parameters demonstrate that the remodeling of the hemomicrocirculatory bed was most pronounced in the combined pathology (diabetes mellitus and burn injury). Furthermore, these changes predominated in the left testis, which may be attributed to the anatomical peculiarities of the venous outflow from this organ.

Discussion

In the testicular circulatory system, the primary role belongs to the hemomicrocirculatory bed, which comprises resistive, exchange, capacitive, and distributive elements. According to most researchers, transcapillary exchange and blood cell emigration occur within the exchange vessels (hemocapillaries) and partially at the beginning of the postcapillary venules, also known as high endothelial venules. In tissues with intensive metabolism, hemocapillaries are highly numerous. Notably, hemocapillaries function intermittently, being alternately recruited or excluded from active circulation [19]. Despite numerous scientific works devoted to the structural and functional changes in the testicular hemomicrocirculatory bed, the alterations in their angioarchitectonics under various pathological processes specifically the conditions modeled in this study remain far from fully understood.

Diabetes mellitus remains one of the most pressing medical and socio-economic health challenges worldwide. The primary target of diabetic damage is the vascular wall. Throughout the progression of diabetes, antioxidant defense mechanisms are compromised, leading to profound oxidative stress. Diabetes induces alterations in antioxidant enzyme activity across various tissues, resulting from protein glycation, oxidative stress, and imbalances in trace element levels. The central pathogenic factor is hyperglycemia, which through the activation of the polyol (sorbitol) pathway, protein kinase C, and advanced glycation end-products leads to the development of oxidative stress, reduced nitric oxide bioavailability, and activation of the pro-inflammatory NF-kB pathway.

Increased synthesis of extracellular matrix proteins by vascular wall cells accelerates the development of atherosclerosis and macroangiopathy while simultaneously damaging the hemomicrocirculatory bed. Furthermore, vessel repair and neovascularization depend on the recruitment of circulating endothelial progenitor cells from the bone marrow in response to ischemia. Given the complex, multi-level interaction between cells and regulatory cytokines, the study of angiogenesis remains highly relevant, particularly under hyperglycemic conditions [20, 21].

The hallmark of angiogenesis is the sprouting of new vessels from pre-existing ones. During local ischemia, endothelial cells (ECs) are activated by hypoxia-induced signaling, primarily through the secretion of vascular endothelial growth factor (VEGF), whose receptors are selectively expressed on ECs. The interaction between VEGF and its receptors triggers protease expression in ECs, which degrades intercellular junctions and the basement membrane, allowing ECs to proliferate and migrate into ischemic tissue along a chemoattractant gradient [22, 23].

The alterations in the hemomicrocirculatory bed identified in our study serve as morphological markers for an unfavorable prognosis regarding structural transformations in the testes during experimental diabetes. The severity of these microvascular changes was most pronounced in the left testis under the combined influence of diabetes mellitus and burn injury.

A defining characteristic of burn injury is that primary extensive skin damage triggers the development of secondary pathologies across nearly all organ systems, manifesting as complications of burn disease. The clinical outcome depends on the body's adaptive capacity and the ability of various systems to compensate for resulting homeostatic disruptions. Several factors aggravate the severe course of burn disease, including massive fluid evaporation through damaged skin, increased intrafascial pressure, elevated hydrostatic pressure coupled with decreased plasma oncotic pressure, erosive-ulcerative bleeding, and gastrointestinal paresis.

The progression of burn disease depends on several factors: the severity of the injury, the patient's age, the timing of treatment initiation, and the presence of comorbidities. In turn, burn severity is determined by the total surface area of the wounds and the depth of tissue damage [13]. Thermal injury causes instantaneous total or partial destruction of the skin and underlying tissues, occurring both directly from the thermal agent and secondarily due to subsequent ischemic processes.

The triggering mechanism for these pathological changes involves morphofunctional disorders within the burn wound itself, where three distinct zones are formed: the zone of primary (coagulation) necrosis, the zone of ischemia (stasis), and the zone of hyperemia (reactive edema) [14]. The zone of coagulation necrosis is characterized by irreversible morphological changes and a complete lack of circulation. Bordering this is the zone of ischemia (or paranecrosis), characterized by a marked slowdown of blood flow leading to stasis. While tissues in this zone initially remain viable, vascular damage, microcirculatory disturbances, endothelial injury, and thrombosis eventually exacerbate tissue ischemia. The complete cessation of blood flow (stasis) results in the expansion of primary necrosis, clinically manifesting as the deepening of the burn wound and the development of endogenous intoxication associated with circulatory-toxic hypoxia.

Circulatory-toxic hypoxia is characterized by two interrelated components that affect organ cells following a cutaneous burn injury. The first is bioenergetic hypoxia, associated with the impaired function of cellular bioenergetic organelles (mitochondria). The second is metabolic hypoxia, which involves a limited capacity for effective oxygen delivery to the cells.

Disordered fluctuations in the mitochondrial partial pressure of oxygen lead to the incomplete oxidation of cytochrome C oxidase. This inhibition disrupts the electron and proton flows within the respiratory chain, causing a sharp decline in ATP levels alongside a simultaneous increase in ADP and AMP. These changes trigger the characteristic metabolic hallmarks of burn-induced endotoxis: accelerated anaerobic glycolysis, depleted glycogen stores, and the development of metabolic acidosis [24, 25].

The subsequent reduction in cellular ATP and the resulting distortion of energy-dependent ion transport leads to the dysregulation of transmembrane influx and efflux. This inevitably disrupts cell-cell and cell-extracellular matrix interactions, ultimately resulting in irreversible structural destruction and cell death.

A pivotal role in membrane damage and the disorganization of intercellular interactions following burn injury [24] is played by the cascade activation of free radical oxidation and associated lipid peroxidation. The resulting by-products degrade high-molecular-weight compounds, thereby disrupting the structural mechanisms essential for cellular viability. A rapid catabolic response, characterized by the breakdown of complex organic compounds and widespread cellular destruction, triggers a compensatory anabolic reaction

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aimed at cellular regeneration. Collectively, this state manifests as hypermetabolism. Hypermetabolic syndrome in burn disease contributes significantly to the progression of multiple organ dysfunction syndrome, which gradually but steadily affects all internal organs. For each affected organ, this process typically follows three sequential stages: organ dysfunction, organ insufficiency, and finally, organ failure [21].

The conducted studies and the obtained results demonstrate that under conditions of diabetes mellitus and burn injury, the arterial (arterioles, precapillary arterioles) and exchange (hemocapillary) links of the hemomicrocirculatory bed undergo narrowing, while the venous component (postcapillary venules and venules) undergoes expansion. The severity of these microvascular alterations was most pronounced in the left testis under the combined influence of diabetes mellitus and burn injury.

Conclusion

The obtained data indicate that under conditions of diabetes mellitus and burn injury, pronounced hemodynamic and structural alterations occur within the testicular hemomicrocirculatory bed. These changes significantly disrupt blood flow, impair trophic support, and play a critical role in the pathomorphogenesis of testicular lesions. The most severe remodeling of microvascular elements was observed in the combined pathology (diabetes mellitus and burn injury), leading to extensive structural reorganization of the testes. This process is characterized by significant destructive changes in the ultrastructures of blood capillaries, Leydig cells, the walls of the convoluted seminiferous tubules, Sertoli cells (sustentocytes), and spermatogenic epithelium. These pathomorphological alterations predominated in the left testis in the combined damage group, which is attributed to the specific anatomical features of the venous outflow from this organ.

Conflict of interest

The authors declare no conflict of interest.

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