ОГЛЯДИ ЛІТЕРАТУРИ

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INFLAMMATION-RELATED MORPHOLOGICAL ALTERATIONS IN THE MICROVASCULATURE

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Microvasculature is a complex structural and functional system that regulates blood supply of body organs, provides transcapillary exchange of fluid and supports the tissue homeostasis. The human microvasculature is represented by the system of small vessels: arterioles, capillaries, venules and arteriolovenular anastomoses. The vessels of this complex become flexible in the altered blood flow; they can deposit the blood corpuscles, get affected by spasms and pass plasma only, change their permeability for tissue fluid. Microvasculature is extremely sensitive to the insults of various factors. Microvascular dysfunction coexists or precedes the macrovascular diseases probably due to joint mechanisms of damage to vessels such as oxidative stress and inflammation. Disorders of microcirculation are one of the main components of inflammation. This article is aimed at the analysis of the scientific publications on the study of morphological alterations in the microvasculature in response to inflammation. The bibliosemantic method was used. The findings of current publications on the morphological alterations that occur in the microcirculation in response to inflammation have been investigated. The analysis has shown significant morphological alterations in the microvessels in response to the proinflammatory factors. Inflammatory processes are accompanied by the events of microvascular dysfunction, associated with hyperpermeability of capillaries, destruction of microvascular endothelial barrier, loss of antiadhesive function of endothelium, etc. In response to inflammation, the marked morphofunctional alterations in the microvasculature of the various organs are observed that are dependent on the time course of inflammation. Early onset is manifested mainly by the spasm of the vascular resistance and dilatation of the capacitance vessels. Disorders of blood rheological properties are manifested by stasis, sludge, microthrombosis.

Key words: microvasculature, inflammation, morphology.

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Microvasculature is a complex structural and functional system that regulates blood supply of body organs provides transcapillary exchange and supports the tissue homeostasis. The human microvasculature is represented by the system of small vessels: arterioles, capillaries, venules and arteriolovenular anastomoses. The vessels of this complex become flexible in the altered blood flow; they can deposit the blood corpuscles, get affected by spasm and pass plasma only, change their permeability for tissue fluid [1]. The renowned morphologist V.V. Kupriyanov and his co-authors, who have been studying blood microcirculation, came to the conclusion that arterioles, capillaries, arteriolovenular anastomoses and venules should be considered as a living substrate but not the passive, inert tubes, that adequately respond to the physical conditions and chemical agents [2]. Thus, the disorder of blood microcirculation is one of the main components or internal signs of inflammation.

Aim. This article is aimed at analysing the scientific publications on the morphological alterations in the microvasculature in response to inflammation.

The bibliosemantic method was used during the study. The data reported in current publications on the morphological alterations that occur in the microcirculatory vascular bed in response to inflammation have been analyzed.

According to current scientific views, microvasculature is an extremely sensitive to the insults of various factors, and responds to them by the developed specific or non-specific reactions. In particular, the acute stress affects the microvasculature of different internal organs, inducing the morphological alterations, indicating about its non-specificity [3].

Microvascular dysfunction leads to increased mortality from cardiovascular diseases and is considered to be a leading factor in the development and progression of cardiometabolic and renal pathology. Microvascular dysfunction coexists or precedes macrovascular diseases, presumably due to combined mechanisms of damage to vessels, e.g. oxidative stress and inflammation [4, 5].

Inflammation is considered as body's response to local lesion, formed over the process of evolution

and is characterized by alteration, disorder of microcirculation and proliferation, aimed at localization, destruction and elimination of damage agent, as well as the regeneration of affected tissues. Alteration, disorders of blood microcirculation (with exudation and emigration) and proliferation are the main components or internal signs of inflammation. Typically, in the foci of inflammation, five external (local) inflammatory manifestations are observed: redness (rubor), swelling (tumor), fever (calor), pain (dolor) and dysfunction. Inflammation causes a rapid reaction of the immune system with the entering of significant amount of immune cells into the foci of inflammation and increased level of cytokines by dozens and hundreds of times [6, 7].

Homeostatic microcirculatory response to infection can be harmful if it is hyperactive or deregulated [8]. For example, in chronic generalized periodontitis the state of regional hemodynamics and microcirculation in periodontal tissues is significantly impaired [9]. In the acute experimental sialoadenitis the response of microvasculature to the inflammatory process was determined during the entire period of the experiment [10]. Inflammation along with oxidative stress and autophagia are the leading factors of such microvascular complication of diabetes mellitus as diabetic retinopathy [11]. Inflammatory septic processes can be accompanied by a severe microvascular dysfunction, which is associated with hyperpermeability of capillaries, destruction of the microvascular endothelial barrier, arteriolar hypofunction of vasoconstrictors, lack of adrenergic sensitivity and tone of smooth muscle cells, which line arterioles; lack of antiadhesive function of endothelial surfaces; decreased density of the perfused capillaries [12-18].

The study of the impact of inflammatory process on the vascular walls of microvasculature of the fundic portion of rats' stomach in the acute experimental gastritis shows the significant changes in metric values of the lumen diameter of its elements. In this way, at the early stages of the experiment, arteriole responding to the spasm is a typical reaction of the resistance vessels on the alteration. The subsequent dilatation of venules is caused by the hypostatic events in the capacitance vessels and development of tissue hypoxia [19].

Moreover, our investigations show that disorders of blood microcirculation in rats are one of the main inflammatory manifestations in the acute experimental gastritis, induced by administration of λ-carrageenan, which is phlogogene by the impact mechanism. Spasm of arterioles in the stomachs of the experimental animals manifested by morphological changes on the 1st day of the experiment was observed even at the early stages of the experiment. Spastic phenomena (the nuclei of endothelial cells protruded into the lumen; the internal elastic membrane was visualized as the basophilic strip forming numerous high folds) were detected in the arterioles of the mucous and submucous membranes on the 2nd day of the experiment. The lumina of arterioles were densely filled with blood corpuscles. Numerous medium lymphocytes were detected in the perivascullar loose connective tissue. Following the 3rd day of the experiment arterioles were dilated in all membranes of the stomach. Blood corpuscles were detected in the lumina. The recovery of the morphological state of the arterioles was observed on the 21st day of the experiment. The vascular exchange responded to administration of λ-carrageenan by the dilatation from day 2 to day 7 of the experiment, caused, primarily, by the development of tissue hypoxia in the stomach wall due to spasm of the resistance vessels of the gastric wall. The wall of the capillaries was thinned, containing no haemocytes in their lumina. The surrounding interstitium showed morphological signs of hyperhydration. The recovery of the morphological state of the exchange vessels was observed on the 30th day of the experiment [20, 21].

The dependence between morphofunctional alterations of microvessels and the length of the inflammation course has also been proved by the findings of other researchers. Morphological analysis of restructuring of elements of microvasculature of red bone marrow in experimental aseptic peritoneal inflammation in rats showed that the state of arterioles, capillaries and venules was also in the direct dependence on the length of experimental aseptic inflammation [22].

The change in the diameter of the elements of the microvasculature of the epinephros, as well as synthetic activity of the adrenal glands, targeting to combat the inflammatory response, also demonstrate directed dependence on the length of the course of aseptic peritonitis. Active response of adrenal glands can be explained by the fact that they are essential component of neuroendocrine and stress-adaptive systems and are responsible for implementation and mobilization of the body defence during the exposure to various pathogenic factors. Microvasculature of the adrenal glands is the main segment, which, in addition to changes of the diameter and structure of the walls of its vessels, responds to inflammation by the leukocyte infiltration of the perivascular connective tissue [23].

The studies of the local reaction of the microvessels of the small and large intestine at the early stages of peritonitis (within 12 hours after the beginning of the experiment) show moderate dilatation of the venules in the small intestine mucosa. In the submucous and muscular membrane the morphometry confirms the fact of the spasm of the arterioles, reduced capillary blood flow and simultaneous stable dilatation of the venules. Within a day after the beginning of the experiment (the reactive stage of peritonitis) a significant increase in the average diameter of the venules, similarly to the previous time period of the experiment, and reduce of the average diameter of

the capillaries was detected in the mucous membrane of the small intestine. On the 2nd day of the experimental peritonitis (toxic stage) a paralytic dilatation of capillaries was observed in the mucous membrane of the small intestine. The average size of the venules was significantly greater than the similar value of the previous time periods of the experiment. Following the 3rd day of the experimental peritonitis (late stage) spasm of the arterioles in the wall of the small and large intestine became weaker. Paralytic dilatation of the capillaries of the villi and crypts was combined with dilatation of the venules, impaired rheological properties of the blood in the form of stasis, sludge, microthrombosis and changes in the permeability of vascular walls [24, 25].

In the acute enteritis, the resistance vessels of the small intestine microvasculature was characterized by the dramatic decrease in the values of the average diameters of arterioles with maximum on the 2nd – 3rd days and increase in the maximum rate on day 15. Their restoration to the values of the intact group was detected only on day 45 of the experiment. In the exchange vessels the dynamics of changes in the mean values was similar to the arterioles. In contrast to the resistance and exchange vessels, the acute enteritis causes the enlargement of the diameters of the capacitance vessels with maximum rate on day 15 – 25 with restoration to the values of the intact group up to day 45 of the experiment [26].

The results of another report showed that in the jejunum, the simulation of the acute aseptic inflammation of the peritoneum first led to the narrowing of the diameter of arterioles and capillaries, significantly marked on day 2 – 3 of the experiment ($p<0.05$), followed up by the significant dilatation on day 14 of the experiment ($p<0.05$). The diameter of the venules was significantly enlarged on day 14 of the experiment ($p<0.05$). In the acute aseptic inflammation of the peritoneum along with transplantation of the cryopreserved placenta, the diameter of the arterioles and capillaries first became narrower (significantly on day 2; $p<0.05$) and then enlarged (on day 3-10; $p<0.05$) [27]. Experimental administration of placental tissue also causes reactive changes in the microvasculature of the rats' red bone marrow, manifested by the significant enlargement of the average diameters of the vascular lumen, especially at the stages of the experiment [28]. In the rat's spleen microvasculature affected by experimental acute aseptic inflammation of the peritoneum, administration of cryopreserved placenta caused marked morphofunctional changes. In the acute aseptic peritonitis, a significant response of small blood vessels was detected over all the period of the experiment. Single administration of cryopreserved placenta was accompanied by the significant changes in the diameters of the arterioles and capillaries within 2-7 days of the experiment; restoration of the diameters of microvessels to the values of the control group usually began on day 5-7; on day 14 no significant difference from the metric parameters of the control group was noted [29].

The studies of our colleagues have also confirmed that experimental aseptic inflammation, induced by intraperitoneal administration of 5 mg λ-carrageenan leads to microcirculation disorders in the venules and capillaries of rats' testicles on day 1 and day 2 of the experiment. Consequently, plethora and stasis occur and lumen of the vessels is overfilled with blood corpuscles. Static phenomena in vessels lead to the oedema of vascular wall [30].

Thus, publications of the modern researchers report about the significant morphological alterations that occur in the microvessels in response to the impact of proinflammatory factors. Inflammatory processes are accompanied by the events of microvascular dysfunction, related to the increased permeability of capillaries, destruction of microvascular endothelial barrier, loss of antiadhesive function of the endothelium, etc. Findings of the majority of experimental studies we have analyzed show that at the early stages of inflammation the arterioles respond by spasm, which is a typical reaction of the resistance vessels to alterations. Subsequent dilatation of venules is caused by the stasis in the capacitance vessels and development of tissue hypoxia. Disturbances in the blood rheological properties are manifested by the stasis, sludge, microthrombosis. Morphofunctional alterations in the microvasculature depend on the length of inflammation process.

Conclusion

1. In response to experimental inflammation, the marked morphofunctional alterations are detected in the microvasculature of various organs.
2. At the early stages of inflammation they are manifested mainly by the spasm of resistance vessels and dilatation of the capacitance vessels. Disturbances in the blood rheological properties are manifested by the stasis, sludge, microthrombosis.
3. Morphofunctional alterations in the microvasculature are dependent on the length of inflammatory course.

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Реферат

МОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ МИКРОЦИРКУЛЯТОРНОГО СОСУДИСТОГО РУСЛА НА ФОНЕ ВОСПАЛЕНИЯ: ОБЗОР ЛИТЕРАТУРЫ

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Ключевые слова: микроциркуляторное русло, воспаление, морфология.

Гемомикроциркуляторное русло является сложной структурно-функциональной системой, которая регулирует кровенаполнение органов, обеспечивает транскапиллярный обмен и поддерживает тканевой гомеостаз. Гемомикроциркуляторное русло у человека представлено системой мелких сосудов: артериолами, капиллярами, венулами и артериоло-венулярными анастомозами. Сосуды этого комплекса чувствительны к изменениям кровотока; они могут депонировать форменные элементы крови или быть спазмированными и пропускать только плазму, изменять свою проницаемость для тканевой жидкости. Гемомикроциркуляторная система чрезвычайно чувствительна к воздействию различных факторов. Микроциркуляторная дисфункция сопровождает или предшествует общему расстройству кровообращения, вероятно, из-за таких механизмов поражения сосудов как окислительный стресс и воспаление. Нарушение гемомикроциркуляции является одним из основных компонентов воспаления. В статье проведен анализ научных публикаций, посвященных изучению морфологических изменений в гемомикроциркуляторном русле, которые возникают в ответ на воспаление. Авторы использовали библиосемантический метод, ими проанализированы выводы из современных публикаций, в которых

освещались морфологические изменения, возникающие в гемомикроциркуляторном кровотоке в ответ на воспаление. Проведенный анализ показал значительные морфологические изменения в сосудах гемомикроциркуляторного русла в ответ на воздействие провоспалительных факторов. Воспалительные процессы сопровождаются проявлениями гемомикроциркуляторной дисфункции: гиперпроницаемостью капилляров, разрушением эндотелиального барьера, потерей антиадгезивной функции эндотелия и др. В ответ на воспаление отмечаются морфофункциональные расстройства в гемомикроциркуляторном русле различных органов, проявления которых зависят от времени течения воспаления. В начальном периоде преимущественно отмечается спазмирование сосудов сопротивления и расширение емкостных сосудов. Нарушение реологических свойств крови выражается явлениями стаза, сладжирования и микротромбообразования.

Реферат

МОРФОЛОГІЧНІ ЗМІН МІКРОЦИРКУЛЯТОРНОГО СУДИННОГО РУСЛА НА ТЛІ ЗАПАЛЕННЯ: ОГЛЯД ЛІТЕРАТУРИ

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Ключові слова: мікроциркуляторне русло, запалення, морфологія.

Гемомікроциркуляторне русло є складною структурно-функціональною системою, яка регулює кровонаповнення органів, забезпечує транскапілярний обмін і підтримує тканинний гомеостаз. Гемомікроциркуляторне русло у людини представлене системою дрібних судин: артеріолами, капілярами, венулами та артеріоло-венулярними анастомозами. Судини цього комплексу чутливі до змін кровотоку; вони можуть депонувати формені елементи крові або бути спазмованими і пропускати лише плазму, змінювати свою проникність для тканинної рідини. Гемомікроциркуляторна система надзвичайно чутлива до впливу різних факторів. Мікроциркуляторна дисфункція супроводжує або передує загальному розладу кровообігу, ймовірно, через такі механізми ураження судин як окисний стрес та запалення. Розлад гемомікроциркуляції є одним із основних компонентів запалення. У статті проведено аналіз наукових публікацій, присвячених вивченню морфологічних змін у гемомікроциркуляторному руслі, які виникають у відповідь на запалення. Було використано бібліосемантичний метод; проаналізовано висновки сучасних публікацій, у яких висвітлювалися морфологічні зміни, що виникають у гемомікроциркуляторному кровотоці у відповідь на запалення. Проведений аналіз показав значні морфологічні зміни в судинах гемомікроциркуляторного русла у відповідь на вплив прозапальних факторів. Запальні процеси супроводжуються проявами гемомікроциркуляторної дисфункції: гіперпроникністю капілярів, руйнуванням ендотеліального бар'єру, втратою антиадгезивної функції ендотелію тощо. У відповідь на запалення відмічаються морфофункціональні розлади в гемомікроциркуляторному руслі різних органів, прояви яких залежать від часу перебігу запалення. У початковому періоді переважно відмічається спазм опірних судин та розширення ємносних. Порушення реологічних властивостей крові проявляються явищами стазу, сладжування та мікротромбоутворення.