



Particularities of renal change in burn disease

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Abstract

Acute renal failure is one of the major complications of burns and it is accompanied by a high mortality rate. Most renal failures occur either immediately after the injury or at a later period when sepsis develops.

Fifty four materials of autopsies of died patients being treated at Burn Department of Samarkand branch of RSCUMA during 2006-2014 years. We studied morphological changes of micro vessels, structural and functional diversion in renal cells in burn disease.

This study of kidneys made us possible to verity morphological substrate and pathogenesis of renal insufficiency in burn disease.

From the study we can distinguish typical features of morphological changes in kidneys in burn disease; damage to micro vessels and cellular elements.

Keywords: Burns, autopsies, microscope study of kidneys

Introduction

Acute kidney injury occurs in approximately one-quarter to one-third of patients with major burn injury [1-2].

Acute renal failure is one of the major complications of burns and it is accompanied by a high mortality rate. Most renal failures occur either immediately after the injury or at a later period when sepsis develops [3-6].

Sepsis, including burn sepsis, is one of the most complex problems of practical and theoretical medicine. It must first of all be considered as generalized infectious complication, developed on the background of burn trauma [7-9].

Late-onset renal failure is usually the consequence of sepsis and is often associated with other organ failure.

According to the data of certain researchers' renal insufficiency, complicating the course of burn disease is the direct cause of death in more than 30% of patients with burns [10-12].

We studied morphological changes of micro vessels, structural and functional diversion in renal cells in burn disease.

Material and methods

Fifty four material of autopsies of died patients (29 males and 23 females), being treated at Burn Department of Samarkand branch of RSCUMA during 2006-2014 years. In 52 patients the course of the disease was complicated by pieces of renal tissue 1,0 x 1,0 x 0,5cm, were fixed in 10% solution of neutral formalin. Histological study was performed on paraffin section of 5 mkm thickness, stained by hematoxilin and eosin. Kidney samples for electronmicroscopic study were fixed in 2, 5% solution of glutaraldegit. Ultrathin sections were contrasted with Plumb citras and studied in electronic microscope of "Philips".

Results of research

The research showed that distinctive changes of kidneys in burn sepsis revealed in histological preparations were caused by disturbances of microcirculations of various degree, manifested by sharp dilatation of blood capillaries lumen, aggregation of erythrocytes, micro

thrombus and plasma segmentation. The observed impairments and nephrocytes necrosis are the main morphological signs of renal insufficiency.

Marked destructive changes are noted both in cortical and medullary substance of kidneys. In electronic microscope study of cortical substance of kidneys in patients died of burn sepsis, first of all changes in the endothelium of blood capillaries are noted. Clots are often revealed in the lumen of these capillaries.

The process of endothelial cells becomes thin and partly destroyed. In places of basal membrane impairment there are lumens in the capillary wall. In basal part of endothelial cell of nephron proximal section there is destruction of cellular organelle. Mitochondria are swelled, with luminal matrix and destroyed crystals, they have uneven contour and are freely located in luminal cytoplasm. Membrane epithelial cells in the basal part, composing its banding, are completely lysed and remnants are attached to loosened basal layer. Collagenous fibres and fibroblast processes as well as changed capillaries are localized in intercellular space. Sharp decrease of ribosome number, lysis of endoplasmatic net membranes contribute to laminar effect of cytoplasm in which swelled mitochondria, changed nuclei and vacuoles are located most microvilli of brush border of cellular apical part are destroyed and moved away with its fragments into the nephron cavity. Thus cell wholeness is destroyed and openings are formed through which cellular contents is pouring out into the canaliculus cavity. In addition removing of microvilli and performing of erosion areas take place as a result of the process of normal filtration and microcirculation in kidneys is impaired.

In the process of research there were distinguished typical features of morphological changes in kidneys in burn disease: damage to microvessels and cellular elements.

Systemic damage to micro vessels is manifested by impairment of vascular wall wholeness, resulting in its necrosis, plasmatic impregnation and erythrocytic diapedesis. Similar changes prove the disturbance of permeability and functions of capillary endothelium. Thus, electronic microscope study of kidneys made us possible to verify morphological substrate and pathogenesis of renal insufficiency in burn disease. The observed destructive changes in kidneys, developing under the effect of bacteria, result in development of slagphenomenon, stasis, microvessels trombosis and hypoxia in future. Changes in cells of proximal and distal nepkron sections of renal cortical layer in the conditions of hypoxia are marked irregularly and are more often of irreversible character.

Almost complete basal banding and lysis of microvilli of brush border of cellular apical part of proximal canaliculi prevent normal reabsorption.

With this the degree of impairment of the main renal functions due to burn severity is an important prognostic sign of burn disease course. As a result of blood flow impairment and development of hypoxia, reversible and irreversible cellular impairment and development of necrosis areas in places of bacteria localization take place that makes the basis of renal insufficiency in burn disease complicated by sepsis.

In severe burn disease inflammatory processes in kidneys often result in lethal outcome. Their development may be caused by marked and extended disturbance of nonspecific barrier mechanisms function taking place in severe burn trauma damages, this moment being an important pathogenetic factor of early bacterial intoxication and sepsis development. One of the leading roles in the development of this process belongs to direct effect of bacteria and product of their vital activity to cellular structure.

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