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CLINICAL AND LABORATORY MANIFESTATIONS OF GENERALIZATION OF INFECTION IN PATIENTS WITH LONG-TERM NON-HEALING WOUNDS

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Abstract: Sepsis intensive care medicine continues to evolve. Every year, the Society for Surgical Infection and Critical Care Medicine issues updated guidelines for the diagnosis and treatment of sepsis, which indicates progress in the study of the fundamental mechanisms of the development of this formidable complication and the expansion of knowledge in this field of Medicine, which results from a dysregulated host response to infection. Further emphasizing its importance, the World Health Organization has recognized sepsis as a global health priority. This scientific article is devoted to the problem of the features of clinical and laboratory manifestations of the generalization of infection in patients with long-term non-healing wounds. It reflects the features of the relationship between the frequency of the development of generalized infection and local manifestations of long-term non-healing wounds.

Key words: Non-healing wounds, necrobiotic process, signs of generalization of infection.

INTRODUCTION

Long-term non-healing wounds are a health problem that has devastating consequences for patients and leads to severe costs for health systems and society [1]. Unresolved issues in the treatment of long-term non-healing soft tissue wounds still account for the share of a negative impact on the economy in any country, regardless of the level of its development [2]. There are statistics in the literature according to which more than a billion people around the world suffer from long-term non-healing soft tissue wounds [3]. This colossal number of patients requires long-term and close attention from medical personnel, and periodic shifts of outpatient and inpatient treatment naturally cause substantial financial costs.

Systematic literature published over the past 10-15 years in the most popular databases was analyzed to identify the actual scale of the impact on the health of long-term non-healing wounds. The results showed that health-related quality of life was lowest among patients with physical pathologies, including long-term non-healing wounds [4].

Patients with long-term non-healing wounds have a poor quality of life-related to overall health. Accordingly, the costs associated with treating long-term non-healing wounds remain significant [5]. This dictates the need to develop and implement treatment strategies for long-term non-healing wounds to improve health-related quality of life and effectively reduce costs for this group of patients. All this indicates the importance of research in this area.

Our study aimed to identify the relationship between the incidence of generalization of infection and the features of local manifestations of long-term non-healing wounds.

METHODS

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The material for the study was the analysis of the results of the examination and treatment of 84 patients with long-term non-healing wounds based on the clinic of the Bukhara State Medical Institute for the period 2010-2015. The criteria for inclusion of patients in the study were age over 18 years, absence of pregnancy, the patient's written consent to the studies, and the presence of a long-term non-healing wound. The criteria for excluding patients from the study were age under 18 years, pregnancy, and lack of written consent of the patient to the research.

The clinical and pathogenetic variety of long-term non-healing wounds was represented by 29 (34.5%) patients with bedsores of various localization, neurotrophic ulcers in 28 (33.2%) patients with diabetic foot syndrome, and 27 (32.1%) patients with trophic ulcers of the lower extremities (mainly of the lower leg and dorsal surface of the foot) in patients with postthrombophlebitic syndrome.

Male patients prevailed (67.9%). The mean age of the patients was 62.8□9.8 years.

Among 84 patients with long-term non-healing wounds, 166 concomitant diseases were identified during the examination. Among the concomitant diseases, the most common were pathologies of the cardiovascular system (34.9%) and endocrine system (25.3%), represented mainly by diabetes mellitus. In total, there were two units of concomitant diseases per 1 patient.

The research methods were complex. Clinical methods included collecting complaints and identifying the history of the disease with the peculiarities of the wound process throughout the entire period of its development. The obligatory stages of patient examination were identifying the etiological cause of long-term non-healing wounds and the presence and severity of concomitant diseases.

To determine the incidence of sepsis and organ failure associated with its presence, we used the classification of sepsis according to R.C. Bone [6], adopted as a basis at the consensus conference of pulmonologists and intensive care physicians in the USA (Chicago) in 1991 [7]. A verified diagnosis of sepsis as a complication of a long-term non-healing wound was made by us based on clinical and pathogenetic signs proposed by the conciliation conference.

Bacteriological studies were carried out in the research laboratory at the Bukhara State Medical Institute clinic. The material for the study was wound discharge and the patient's blood. Blood was inoculated in two vials with media to detect bacteremia and to study aerobic and anaerobic microorganisms. Inoculation was carried out in several media. A microaerostat and 5% blood agar dishes were placed in a thermostat and incubated at a temperature of +370°C for 48-72 hours. Smears were stained according to Gram. Colonies that grew up under aerobic and anaerobic conditions were compared according to their morphology and microscopy results. Quantitative assessment of microflora was carried out according to the method of V. I. Kocherovets et al. [8] and Gould [9]. The content of microorganisms in 1 ml of pathological material (exudate) was expressed in decimal logarithms of absolute numbers.

The significance of the differences between the samples, which were close to the norm in terms of the nature of the distribution, was established according to the parametric Student's test with a 95% reliable probability interval. The criterion for the statistical reliability of the conclusions

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obtained was considered to be the generally accepted in medicine value $p<0.05$.

RESULTS

The nature of the necrobiotic process in the wound was diverse. Only 3.6% of patients (3) were visually absent in the inflammatory process in a long-term non-healing wound. They were presented to 2 (66.7%) patients with trophic ulcers of the lower extremities and 1 (33.3%) patients with bedsores.

In 20 (23.8%) patients, the inflammatory process in a long-term non-healing wound was present, but it proceeded without tissue necrosis. At the same time, this variant of the course of the inflammatory process in a long-term non-healing wound was presented in 65% of cases (13 patients) with trophic ulcers of the lower extremities, in 25% of cases (5 patients) with bedsores, and 10% of cases (2 patients) with neurotrophic ulcers of diabetic foot syndrome.

In contrast, in 72.6% of cases, the wound was characterized not only by the presence of an inflammatory process but also by tissue necrosis. Thus, in 36.9% of cases (31 patients), the tissues of long-term non-healing wounds were subjected to dry necrosis, in 11.9% of cases (10 patients) to wet necrosis, and in 23.8% of cases (20 patients) to mixed necrosis.

Dry necrosis in long-term non-healing wounds was mainly represented by ulcerative-necrotic ulcers in diabetic foot syndrome (15 patients—48.4%) and bedsores (10 patients—32.3%). Only in 6 (19.4%) patients was dry necrosis noted in trophic ulcers with chronic venous insufficiency of the lower extremities.

Wet necrosis damaged long-term non-healing wounds in 10 (11.9%) patients. In half of the cases (5 patients), they were represented by bedsores; in 40% of cases (4 patients), by ulcers in diabetic foot syndrome; and in 10% of cases (1 patient), by trophic ulcers of venous etiology.

We revealed the development of wet necrosis in long-term non-healing wounds against the background of non-rejected dry necrosis (mixed necrosis) among 20 (23.8%) patients, who were represented by 8 (40%) patients with bedsores, 7 (35%) patients with neurotrophic ulcers of diabetic foot syndrome, and 5 (25%) patients with trophic ulcers with chronic venous insufficiency of the lower extremities.

Among patients with bedsores, cases prevailed (34.5%) of long-term non-healing wounds with dry necrosis; to a lesser extent (3.4%), there were cases without an inflammatory process in the wound. Similar changes were found among patients with neurotrophic ulcers of diabetic foot syndrome, where more than half of the cases (53.6%) in patients with a long-term non-healing wound proceeded against the background of an inflammatory process with dry tissue necrosis. At the same time, we did not note any cases in which there was no inflammatory phenomenon among patients with this type of long-term non-healing wound.

Among patients with trophic ulcers of the lower extremities due to complications of chronic venous insufficiency, cases of inflammation without tissue necrosis prevailed (48.1%), and the inflammatory process occurred the least (3.7%) against the background of wet necrosis of tissues of long-term non-healing wounds.

Dense and red granulation tissue was found among 16 (18.6%) patients. At the same time, in 68.8% of cases, the exudate was serous; in 18.8% of cases – serous-hemorrhagic, and 12.5% of cases – hemorrhagic. At the same time, in 21 (24.4%) patients, we diagnosed the presence of fragile and pale granulation

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tissue, which in 19% of cases had serous exudate, in 47.6% of cases – serous-hemorrhagic, in 14.3% of cases – hemorrhagic and serous-purulent, and in 1 (4.8%) patient, fragile and pale granulation tissue had purulent exudate.

Fibrous tissue covered long-term non-healing wounds in 20 (23.3%) patients. In all cases, the exudate was discharged. In 8 (40%) patients, the exudate was hemorrhagic, and in 4 (20%) patients, it was serous-hemorrhagic. There was serous-purulent and purulent exudate in 3 cases (15% each), and only in 2 (10%) patients was the wound discharge serous.

In 14 (16.3%) patients, a fibrous film covered long-term non-healing wounds, and in 15 (17.4%) patients, the wound surface was covered with a scab. In both cases, the discharge from the wound was either serous-purulent (42.9%) or purulent (46.7%).

The type of tissue in the bed of long-term non-healing wounds was clinically manifested by granulation tissue covered with a fibrin eschar or film. It was closely related to the nature of the wound exudate. As our studies have shown, in the presence of dense and red granulation tissue, the discharge from the wound had a scanty serous character without odour, watery consistency, and transparent color with a yellow tint.

In the presence of brittle and pale granulation tissue, as our studies have shown, the nature of wound exudate was mainly watery, serous-hemorrhagic, odorless, scanty, and pink to light red.

In cases where the surface of a long-term non-healing wound was covered with fibrous tissue, the character of the wound exudate was hemorrhagic (blood consistency) with moderate formation and odourlessness. In contrast to it, the fibrous film was mainly characterized by the presence of serous-purulent, watery, and moderately discharged. Covering the wound with a scab led to the formation and production of purulent exudate, which often had a thick consistency, from white to green, and had an unpleasant odor.

General clinical laboratory blood tests did not reveal significant differences between patients with long-term non-healing wounds, depending on the etiological cause of its development. The exception was the biochemical parameters of patients with ulcerative-necrotic lesions of diabetic foot syndrome, which was associated with the characteristics of the underlying disease.

The study of microbial wound contamination in patients with long-term non-healing wounds revealed the absence of any significant characteristics with the etiological form of the lesion (bedsores, diabetic foot syndrome, or trophic leg ulcers). At the same time, the total content of aerobic microorganisms in long-term non-healing wounds was equal to 106-107 CFU/ml (on average, 6.5±0.08 log CFU/ml). The prominent representatives of the identified microorganisms were facultative cocci, Enterobacteriaceae, Pseudomonas aeruginosa, and other associations. Staphylococci (25.6%), representatives of Proteus (16.5%), Pseudomonas aeruginosa (15.5%), and Enterobacteria (12.2%) were most often sown. Escherichia coli was sown in small quantities and accounted for only 6-7%.

Among the anaerobic pathogens, B. melaninogenicus (22.0±0.8 log CFU/ml), B. Fragilis (17.0±0.4 log CFU/ml), F. nucleatum (10.0±0.2 log CFU/ml), Peptostreptococcus (9.0±0.31 log CFU/ml), Peptococcus (8.0±0.2 log CFU/ml) and Eubacterium (3.0±0.1 log CFU/ml) were seeded to a greater extent.

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When assessing the degree of generalization of infection, it was revealed that signs of systemic inflammatory response syndrome were not noted in all patients.

Thus, in 33.3% of cases (28 patients), there were no general signs indicating the generalization of the infection. The central part was patients with trophic ulcers of venous etiology (46.4%). In other cases, the variance in the frequency of recording the number of patients without signs of systemic inflammatory response syndrome was almost identical between patients with bedsores (28.6%) and patients with diabetic foot syndrome (25%).

According to one clinical or laboratory sign of the syndrome of systemic inflammatory reaction, 28 (33.3%) patients had it. Among them, patients with diabetic foot syndrome (39.3%) and bedsores (32.1%) prevailed. Patients with trophic ulcers of venous etiology were only 28.6%.

The most common signs were tachycardia (35.7% of cases) and general hyperthermia/hypothermia (28.6% of cases). Leukocytosis was noted in 7 (25%) patients, and 3 (10.7%) patients had dyspnea at rest. Leukocytosis in more than half of cases (57.1%) was noted among patients with neurotrophic ulcers of diabetic foot syndrome. A similar trend was pointed out in the variance of such a clinical sign as tachycardia (50%). As for the frequency of dyspnea and hyperthermia/hypothermia, in this category, variances prevailed in patients with bedsores (66.7% and 50%, respectively).

Two clinical and laboratory signs of systemic inflammatory response syndrome were diagnosed among 11 (13.1%) patients. The patients with neuropathic ulcers due to diabetic foot syndrome and trophic ulcers of post-thrombophlebitic syndrome were evenly distributed (5 patients each) (45.5% each, respectively). Only 1 clinical case (9.1%) was found among patients with bedsores.

The most common clinical and laboratory signs were leukocytosis/leukopenia (40.9%) and tachycardia (31.8%). Hyperthermia/hypothermia was diagnosed in 18.2% of cases, and dyspnea in 9.1% of cases.

Among patients with leukocytosis, trophic ulcers due to venous insufficiency of the lower extremities prevailed (55.6%), and neutrophilic ulcers of diabetic foot syndrome (33.3%). At the same time, tachycardia was equally distributed among patients of these etiological categories (42.9% each). The same variation was found in hyperthermia/hypothermia (50% each).

As for patients with bedsores, it should be noted that one patient had two clinical and laboratory signs of a systemic inflammatory response syndrome in the form of leukocytosis and tachycardia.

We identified three clinical and laboratory systemic inflammatory response syndrome signs among 14 (16.7%) patients with long-term non-healing wounds. More than half (64.3%) of them were patients with bedsores. In 28.6% of cases (4 patients), these were patients with neurotrophic foot ulcers due to diabetes mellitus, and in 7.1% of cases (1 patient), with trophic ulcers due to the presence of chronic venous insufficiency of the lower extremities.

The analysis of clinical and laboratory signs of variance was mainly manifested by a combination of hyperthermia/hypothermia (33.3%) and tachycardia (31%) with the presence of leukocytosis/leukopenia (26.2%) or dyspnea (9.5%). They were mainly diagnosed among patients with pressure ulcers. For example,

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leukocytosis/leukopenia among patients with long-term non-healing wounds was noted in 72.7% of cases among patients with bedsores. Such a situation was also pointed out in the presence of dyspnea (75%), hyperthermia/hypothermia (64.3%), and tachycardia (53.8%), which was apparently due to the initially combined damage to soft tissues.

Only in 3 (3.6%) patients with long-term non-healing wounds did we identify four clinical and laboratory signs of systemic inflammatory response syndrome, which were among patients with bedsores (2 patients) and diabetic foot syndrome (1 patient).

DISCUSSION

The classification of soft tissue wounds into acute and chronic is based on the pathogenesis of their development, possible complications, and processes associated with their regeneration [10].

Acute soft tissue wounds undergo several molecular processes that ultimately restore structural integrity [11]. At the same time, long-term non-healing wounds cannot start rapid regeneration and are characterized by pathological processes such as continuous inflammation, constant infections, and necrosis [12].

Typically, there are four overlapping phases in acute wound healing: hemostasis, inflammation, the proliferative phase, and remodelling [13].

When an acute wound appears, the first reaction is hemostasis, which stops bleeding and prevents blood loss [14]. During the inflammatory phase, skin damage activates a complex immune response that destroys pathogens entering the wound and prepares tissues to restore anatomical integrity. The latter occurs in the proliferative phase and includes forming granulation tissue, neovascularisation, and re-epithelialization [15]. Finally, the healing of an acute wound is completed by the remodeling phase, during which the granulation tissue is replaced by scarring, and the epidermis is freed of immune cells that either die as a result of apoptosis or move to the dermis [16].

All this indicates that the course of the wound process is directly related to the possibility of the development of generalization of infection under the condition of violation of the pathogenetic mechanisms of regenerative processes. These processes should be based primarily on immunological mechanisms.

CONCLUSION

Analysis of the distribution of patients depending on the number of clinical and laboratory signs of the syndrome of the systemic inflammatory response, based on the criteria for diagnosis, allowed us to identify the presence of generalization of infection in 28 (33.3%) patients. At the same time, among patients with bedsores and neurotrophic ulcers of diabetic foot syndrome, they turned out to be the most numerous (41.4% and 36.7%, respectively). Often, the generalization of the infection manifested itself in the form of chronic sepsis with organ dysfunction, which could occur under the mask of the pathology of the affected organ.

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