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HOW TO PREVENT GENERALIZATION OF INFECTION ON PATIENTS WITH CHRONICALLY NON-HEALING ULCER

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Resume

According to the ICD-10 definition, chronic ulcers are wounds that do not heal within the usual healing period of this type of injury or location. Chronic ulcers most often develop in patients with a burdened morbid background in the form of diabetes mellitus complicated by angio- and/or europathy, decompensated form of venous insufficiency, in bedridden patients for a long period of severe patients. The urgency of the problem of treating a chronic ulcer or wound is due not only to its medical significance but also to its social and economic importance. Chronic wounds are a heavy burden for both patients and their family members. Due to the presence of pain, infection, loss of function in the affected area, as well as constant financial costs, not only does the quality of life decrease and the number of disabled people increases, but conditions are created for the generalization of infection, the development of surgical sepsis and the death of the patient. In this manuscript, we present a clinical case description of the features of the course of a chronic wound that was complicated by the generalization of the infection of the type of chronic sepsis.

Keywords

Long-term nonhealing wounds, complicated course of chronic inflammation, clinical case.

Introduction: Unresolved problems in the treatment of long-term nonhealing soft tissue wounds still account for the share of a negative impact on the economy in any country in the world, regardless of its level of development. In the literature, there is statistical information according to which more than a billion people around the world suffer from chronic soft tissue wounds [1].

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. For example, in a study by a group of specialists from the United States led by S.R. Nussbaum [2], it was revealed that

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almost 15% of Medicare holders (8.2 million) had at least one type of chronic wound.

Surgical infection was the most common category (4.0%), followed by diabetic infections (3.4%). Total Medicare cost estimates for all types of wounds ranged from $28.1 billion to $96.8 billion, including the cost of treating the infection, with the most expensive costs being for surgical wounds ($11.7, $13.1 and $38.3 billion), followed by diabetic foot ulcers ($6.2, $6.9 and $18.7 billion). It should be noted that the highest costs in the treatment of chronic wounds were outpatient settings ($9.9-$35.8 billion), followed only by the costs of patients who received treatment in an inpatient setting ($5.0-$24.3 billion). In order to identify the real scale of the impact of chronic wounds on health, we analyzed systematic literature published over the past 10–15 years in the most popular databases. The results showed that health-related quality of life was lowest among patients with physical abnormalities, including chronic wounds. The same number of patients was noted after limb amputation as a result of the progression of chronic wounds [3]. According to M. Olsson et al. [4] The burden of costs was mainly related to amputations in patients with concomitant type 2 diabetes mellitus, where the cost of hospitalization ranged from $12,851 to $16,267 for this patient population.

Patients with chronic wounds have a poor quality of life-related to overall health. Accordingly, the costs associated with the treatment of chronic wounds remain significant [5]. This dictates the need to develop and implement chronic wound management strategies aimed at improving health-related quality of life and effectively reducing costs for this group of patients. In this regard, clinical cases will be helpful for practitioners.

MATERIAL AND METHODS OF RESEARCH

The results of the clinical examination of 84 patients with chronic wounds who were treated and examined in the multidisciplinary clinic of the Tashkent Sate medical university are analyzed. All patients were represented by bedsores in 29 (34.5%) patients, ulcerative formations in patients with diabetic foot syndrome in 28 (33.3%) patients, and trophic ulcers due to complications of chronic venous insufficiency of the lower extremities in 27 (32.1%) patients. Male (68.7%) at average age 63.8 9.6 years The research methods were complex. Clinical methods of research included the collection of complaints, the identification of the history of the disease with the peculiarities of the course of the wound process throughout the entire period of its development. The obligatory stages of the examination of patients were the identification of both the etiological cause of chronic 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 chronic wounds was made by us on the basis of clinical and pathogenetic signs proposed by the conciliation conference.

Blood was inoculated in two vials with media to detect bacteremia and to study aerobic and anaerobic microorganisms. A microaerostat and dishes with 5% blood agar were placed in a thermostat and incubated at a temperature of +37 °C for 48–72 hours. Smears were stained by Gram. Colonies grown under aerobic and anaerobic conditions were compared according to their morphology and microscopy results. [8] and Gould [9]. The content of microorganisms in 1 ml of pathological material (exudate) was expressed in decimal logarithms of absolute numbers.

Local clinical methods of wound examination were based on the assessment of the nature of the necrobiotic process in the wound. The presence/absence of a local inflammatory process and the type of tissue necrosis (dry, wet or mixed) were visually assessed.

The type of tissue in the chronic wound bed was determined, which could be in the form of dense and red granulation, brittle and pale granulation, fibrous film or tissue, as well as in the form of eschar formation.

The nature of wound exudate (serous, hemorrhagic, purulent), its colour (colourless, pink to red, white, creamy and green), consistency (transparent, watery, bloody, and thick) and the smell of exudate (present/not present) were assessed.

RESULTS AND DISCUSSION

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

In cases where the surface of a long-term nonhealing 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 odour.

The study of microbial wound contamination in patients with long-term nonhealing 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 nonhealing wounds was equal to 106-107 CFU/ml (on average 6.5±0.08 lg 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 lg CFU/ml), B. Fragilis (17.0±0.4 lg CFU/ml), F. nucleatum (10.0±0.2 lg CFU/ml), Peptostreptococcus (9.0±0.31 lg CFU/ml), Peptococcus (8.0±0.2 lg CFU/ml) and Eubacterium (3.0±0.1 lg CFU/ml) were seeded to a greater extent. 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) they did not have any general signs indicating generalization of the infection at all. Among them, the main part were 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 turned out to be 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 aetiology turned out to be 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 also 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 noted 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).

Systemic inflammatory response syndrome in the form of two clinical and laboratory signs was diagnosed among 11 (13.1%) patients. They were evenly distributed (5 patients each) between patients with neuropathic ulcers due to diabetic foot syndrome and trophic ulcers of post-thrombophlebitic syndrome (45.5% each, respectively). Among patients with bedsores, there were only 1 clinical cases (9.1%).

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, patients with 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, respectively). The same variation was found in relation to hyperthermia/hypothermia (50% each, respectively).

As for patients with bedsores, it should be noted that 1 patient had 2 clinical and laboratory signs of a systemic inflammatory response syndrome in the form of leukocytosis and tachycardia.

We identified three clinical and laboratory signs of systemic inflammatory response syndrome among 14 (16.7%) patients with long-term nonhealing wounds. More than half (64.3%) of them were patients with bedsores. In 28.6% of cases (4 patients) these were patients with neutrotrophic 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, leukocytosis/leukopenia among patients with long- term nonhealing wounds was noted in 72.7% of cases among patients with bedsores. Such a situation was also noted in relation to 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 nonhealing wounds, we identified four clinical and laboratory signs of systemic inflammatory response syndrome, which were among patients with bedsores (2 patients) and diabetic foot syndrome (1 patient). Thus, the analysis of the distribution of patients depending on the number of clinical and laboratory signs of the systemic inflammatory response syndrome, 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

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organ. An example is the following clinical case: Patient S.S., born in 1973, applied to the nephrology department of the multidisciplinary clinic of the Tashkent Medical Academy after a long period of examination and treatment in other hospitals with a diagnosis of chronic pyelonephritis in the acute stage.

The main complaints were pain in the lumbar region, frequent urination, periodic hyperthermia, palpitations, and pronounced general weakness.

The patient has been suffering from long-healing ulcers of the right tibia for 2 years, which appeared after thrombophlebitis of the deep veins of the lower extremities (figure). He received treatment on an outpatient and inpatient basis.

Over the past month, the patient has developed swelling of the lower extremities and free fluid in the abdominal and pleural cavities, weight loss.

Ultrasound showed diffuse changes in the kidneys, moderate splenomegaly, and hydracalcinosis.

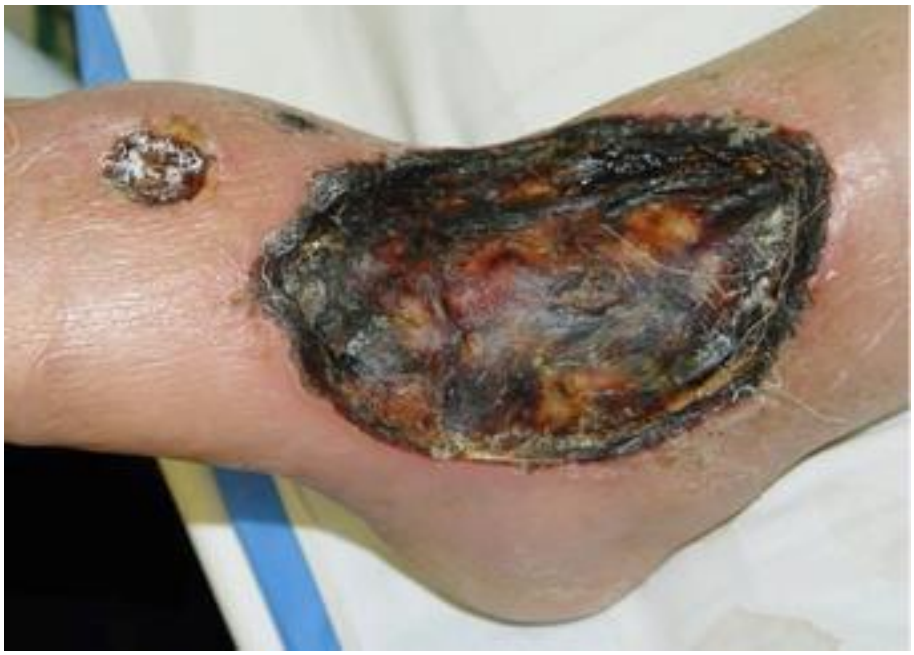

Figure. Trophic ulcers of the lower extremities in the patient presented in the description of this clinical example

On the radiography of the chest cavity organ, left-sided exudative pleurisy was noted. Over the past two weeks, the patient's cervical and submandibular lymph nodes have enlarged. Blood tests revealed hypoproteinemia (39 g/l), dysproteinemia, an increase in alkaline phosphatase up to 143 U/l, urea up to 18.3 mmol/l, creatinine up to 82 μmol/l, leukocytosis up to 16x109/l, thrombocytosis - 488x109/l, erythrocytes 4.7x1012/l, haemoglobin - 78g/l.

Bacteriological culture of the wound made it possible to identify Staphylococcus aureus in titer 109, and urine culture revealed Staphylococcus aureus in titer 107.

The results of hemaculture are negative. The diagnosis was established: post-thrombophlebitic syndrome complicated by trophic ulcers of the right tibia. Chronisepsis with kidney damage. Pyelonephritis. The nature of the necrobiotic process in chronic wounds can be diverse [10]. Thus, according to our data, in 23.8% of patients, the inflammatory process in a long- term nonhealing wound was present, but it proceeded without the presence of tissue necrosis. At the same time, this variant of the course of the inflammatory process in a long-term nonhealing wound was presented in 65% of patients with trophic ulcers of the lower extremities, in 25% of cases with bedsores, and in 10% of cases 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, the tissues of long-term nonhealing wounds were subjected to dry necrosis; in 11.9% of cases – wet necrosis and in 23.8% of cases – mixed necrosis. Patients with dry necrosis in long-term nonhealing wounds can mainly be represented by cases of ulcerative-necrotic ulcers in diabetic foot syndrome [11]. According to the literature, damage to long-term nonhealing wounds by wet necrosis is noted among 10–11.9% of patients. They can often be represented by bedsores [12]. The development of wet necrosis in long-term nonhealing wounds against the background of non- rejected dry necrosis, i.e. mixed necrosis, can occur both among patients with bedsores and among patients with neurotrophic ulcers of diabetic foot syndrome [13]. As our studies have shown, among patients with trophic ulcers of the lower extremities due to complications of chronic venous insufficiency, cases of the course of the inflammatory process without tissue necrosis prevailed (48.1%), and the inflammatory process occurred least of all (3.7%) against the background of wet necrosis of tissues of long-term nonhealing 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 in 12.5% of cases – hemorrhagic in nature. At the same time, in 21 (24.4%) patients, we diagnosed the presence of fragile and pale granulation 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 nonhealing 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. In 3 cases (15% each), there was serous-purulent and purulent exudate, and only in 2 (10%) patients, the wound discharge was severe. A fibrous film covered long-term nonhealing wounds in 14 (16.3%) patients, 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%) in nature.

The type of tissue in the bed of long-term nonhealing wounds was clinically manifested by the presence of granulation tissue covered with a fibrin eschar or film and was closely related to the nature of wound exudate [14].

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 odor, watery consistency, transparent color with a yellow tint.

CONCLUSION

Analysis of the distribution of patients depending on the number of clinical and laboratory signs of the systemic inflammatory response syndrome, 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). As this clinical example has shown, a long- term disease, which was accompanied by a non-specific clinical picture, required the mandatory exclusion of the bacteriological factor in the development of this type of complication. In this case, the sluggish inflammatory process of a long-term nonhealing wound led to kidney damage in the form of sluggish urological diseases. In fact, we were dealing with chronic sepsis, which manifested itself not only with signs of systemic inflammatory response syndrome but also with organ dysfunction.

Ethics approval and consent to participate - All patients gave written informed consent to participate in the study.

REFERENCES:

1. Garraud O., Hozzein W.N., Badr G. Wound healing: time to look for intelligent, «natural» immunological approaches? // BMC Immunol. - 2023;18(Suppl 1):23.

2. An Economic Evaluation of the Impact, Cost, and Medicare Policy Implications of Chronic Nonhealing Wounds. / S.R. Nussbaum, M.J. Carter, C.E. Fife, et al. // Value Health. 2018;21(1):27-32.

3. Analysis of the stimulating effect of platelet-rich autoplasma and lymphatic stimulating technologies in the treatment of patients with trophic ulcers of venous aetiology / Yu. Sociology. Philosophy. Applied research. – 2023. – № 6. – P. 102-107.

4. The humanistic and economic burden of chronic wounds: A systematic review. / M. Olsson, K. Järbrink, U. Divakar, et al. // Wound Repair Regen. 2019;27(1):114-125.

5. Bogomolov M. S. Venous trophic ulcers – conservative treatment and its monitoring with the help of modern technologies // VII Congress of Surgeons of Siberia: Collection of Scientific and Practical Works, Krasnoyarsk, November 13–14, 2019 / Responsible Editor T.G. Ruksha. – Krasnoyarsk: Verso, 2019. – P. 251–260.

6. Bone R. Sir Isaac Newton, sepsis, SIRS and CARS // Crit. Care Med. 1996, vol. 24, #7 – P.1125-1127.

7. Draft resolution of the conciliation conference "Sepsis – definition of the concept, issues of terminology", Moscow, 13.10.98 // Surgery, 1999, No10-P.8-9.

8. Kocherovets V.I., Petrakov A.A., Ponomareva T.R., Mikhailova V.S. Metodicheskie rekomendatsii po mikrobiologicheskoy diagnostike zabolevanii pozvedennykh nesporevoruzhnymi anaerobnymi bacteriami [Methodological recommendations for microbiological diagnostics of diseases caused by non-spore-forming anaerobic bacteria]. - 1986. – 49 p.

9. Gould J.C. Quantity and quality in the diagnosis of urinary tract infections // Brit. J. URL. – London, 1965. – Vol. 37. – P. 7-12.

10. Ameri A.H., Moradi Tuchayi S., Zaalberg A. IL- 33/regulatory T cell axis triggers the development of a tumour-promoting immune environment in chronic inflammation. // Proc. Natl. Acad. Sci. USA- 2019; 116:2646–2651.

11. An angiogenic switch in macrophages involving synergy between Toll-like receptors 2, 4, 7, and 9 and adenosine A(2A) receptors. / G. Pinhal-Enfield, M. Ramanathan, G. Hasko, et al. // Am. J. Pathol. 2023;163(2):711–21.

12. An economic evaluation of the impact, cost, and medicare policy implications of chronic nonhealing wounds. / S.R. Nussbaum, M.J. Carter, C.E. Fife, et al. // Value Health.2018;21:27–32.

13. ATP modulates acute inflammation in vivo through dual oxidase 1-derived H2O2 production and NF- kappaB activation. / S. de Oliveira, A. Lopez-Munoz, S. Candel, et al. // J. Immunol. -2014;192(12):5710– 9.

14. Baggiolini M., Loetscher P., Moser B. Interleukin-8 and the chemokine family. // Int J Immunopharmacol. 1995;17(2):103–8.