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Ferritin, IL-6 and human-beta-defensin-1 as prognostic markers of the course severity and treatment effectiveness of pulmonary tuberculosis

Current methods of investigation in TB patients, namely sputum microscopy, culture, and molecular genetic methods, although well-studied, have a number of disadvantages, such as low sensitivity, long time required to obtain results, or high cost. Because of this, the search for alternative diagnostic tools and methods for predicting the course and effectiveness of treatment in patients with tuberculosis becomes relevant. In this study, ferritin, interleukin-6 (IL-6), and human-beta-defensin-1 (HBD-1) were selected for comparison of prognostic performance.

Objective — to investigate the dynamics of ferritin, IL-6, and human-beta-defensin-1 levels against the background of the intensive phase of pulmonary tuberculosis therapy and to identify the most effective marker for predicting the effectiveness of treatment.

Materials and methods. 100 patients with pulmonary tuberculosis and 20 healthy individuals were included in the study. Examination of patients was carried out according to the current standards of providing medical care to tuberculosis patients. In addition, the patients' fasting blood ferritin, IL-6 and HBD-1 levels were determined at the beginning of treatment and after 60 days. Healthy individuals from the control group had a single determination of ferritin, IL-6 and HBD-1 blood levels on an empty stomach.

Results and discussion. At the beginning of treatment, the ferritin level was significantly lower (95.95 ± ± 8.68) ng/ml in patients who later effectively completed the intensive phase of anti-tuberculosis treatment than in patients with ineffective intensive phase of treatment (152.27 ± 8.85) ng/ml. The same trend persisted after 60 days: in the effective intensive phase — (123.87 ± 13.39) ng/ml, in the ineffective one — (239.76 ± 12.91) ng/ml, $p < 0.05$. In effective intensive phase of antituberculosis treatment, the level of IL-6 was significantly lower. Thus, at the beginning of treatment, it was (82.59 ± 6.89) pg/ml in patients with an effective intensive phase of treatment and (146.42 ± 8.04) pg/ml in patients with ineffective intensive treatment phase. After 60 days, it was (48.88 ± 4.19) pg/ml in patients with an effective intensive phase of treatment and (142.89 ± 9.11) pg/ml in patients with ineffective intensive treatment phase, $p < 0.05$. The level of HBD-1 was higher when the intensive phase of antituberculosis therapy was ineffective, as when measured at the beginning of treatment (effective intensive phase — (18.71 ± 3.31) pg/ml, ineffective intensive phase — (32.79 ± 8.31) pg/ml), as well as when measured after 60 days (effective intensive phase — (19.93 ± 3.58) pg/ml, ineffective intensive phase — (42.92 ± 12.99) pg/ml, $p < 0.05$).

Conclusions. Levels of ferritin, IL-6 and HBD-1 are significantly increased in tuberculosis patients compared to healthy individuals, which allows them to be considered as markers of tuberculosis inflammation. Higher concentrations of these markers, both at the beginning of treatment and after 60 doses, are predictors of failure of antituberculosis therapy. The strongest relationship between the studied markers and parameters of the severity of the tuberculosis process is observed in the study of HBD-1, which allows us to consider it as the most effective marker of the severity of the course among presented ones.

Keywords

Ferritin, IL-6, human-beta-defensin-1, tuberculosis, prognosis.

Tuberculosis remains an important global problem for a long time: about 10 million new cases of active tuberculosis are detected in the world and about 1.5 million people die from this disease annually [20]. This disease carries a large social burden, leading to the invalidation of patients not only due to local lesions, but also due to severe metabolic disorders [16, 17].

Current methods of investigation in TB patients, namely sputum microscopy, culture, and molecular genetic methods, although well-studied, have a number of disadvantages, such as low sensitivity, long time required to obtain results, or high cost. Because of this, the search for alternative diagnostic tools and methods for predicting the course and effectiveness of treatment in patients with tuberculosis becomes relevant [14, 15]. In this study, ferritin, interleukin-6 (IL-6), and human-beta-defensin-1 (HBD-1) were selected for comparison of prognostic performance.

Iron is a trace element important for the metabolism of both the host and Mycobacterium tuberculosis (MTB). Pathogenic mycobacteria compete with the host for iron either by direct depletion of intracellular iron from the host cytoplasm, or by synthesis of siderophores and macromolecules, including transferrin, ferritin, or lactoferrin, which have a high affinity for capturing extracellular iron ions [5]. Anemia resulting from tuberculous inflammation is mainly caused by a problem with iron delivery, when red cell iron is not used sufficiently [19]. This condition is different from iron deficiency anemia, which is mostly caused by malnutrition. Although serum iron levels are low in both types of anemia, other components involved in iron homeostasis, including ferritin, are elevated in inflammation-related anemia. Due to the regularity of the increase in ferritin in tuberculosis lesions and its normalization with effective treatment, ferritin becomes a promising marker of the course and treatment effectiveness of tuberculosis process [12].

IL-6 acts as an independent pro-inflammatory cytokine, which is one of the first to be produced by monocytes, macrophages, and neutrophils during MTB infection. This interleukin has a wide range of effects on hepatocytes (stimulating the production of acute phase proteins), bone marrow cells, and also triggers a number of cytokine reactions [18]. This explains the perspective of using IL-6 as a marker of the course and effectiveness of tuberculosis treatment as its levels should respond sensitively to the occurrence of tuberculosis inflammation, the massiveness of the bacterial lesion and its reduction with effective treatment [9].

HBD-1 is the only beta-defensin that has a basic level of production, and is also produced directly under the influence of exogenous factors (pathogens) without endogenous mediators. It is a multifunctional modulator that exhibits innate antimicrobial activity and mediates an immune response, acting as a chemoattractant for $CD4^+$ T-helper and immature dendritic cells by binding to the chemokine receptor CCR6, as well as macrophages [6]. These features make the study of HBD-1 promising, first of all, as a marker of the severity of the course of the tuberculosis process and a possible predictor of the ineffectiveness of anti-tuberculosis chemotherapy [1].

Objective — to investigate the dynamics of ferritin, IL-6, and human-beta-defensin-1 levels against the background of the intensive phase of pulmonary tuberculosis therapy and to identify the most effective marker for predicting the effectiveness of treatment.

Materials and methods

100 patients with pulmonary tuberculosis and 20 healthy individuals were included in the study.

Examination of patients was carried out according to the current standards of providing medical care to tuberculosis patients. Mandatory diagnostic minimum included chest X-ray at the beginning of treatment and at the end of the intensive phase, sputum microscopy at the beginning of treatment and at the end of the intensive phase, sputum culture at the beginning of treatment.

To determine the severity of the clinical manifestations of the disease, we used a scoring system, in which each of the symptoms (cough, shortness of breath, weight loss, pain, malaise) was evaluated as 1 point. In addition, the patients' fasting blood ferritin, IL-6 and HBD-1 levels were determined at the beginning of treatment and after 60 days. Healthy individuals from the control group had a single determination of ferritin, IL-6 and HBD-1 blood levels on an empty stomach. To retrospectively determine the relationship between the level of the studied indicators and the effectiveness of antituberculosis treatment, patients were divided into groups depending on the effectiveness of the intensive phase of chemotherapy — effective (n = 62) and ineffective (n = 38). The effectiveness criteria were sputum conversion and the presence of positive clinical and X-ray dynamics, which allowed the patient to be transferred to the supportive phase of treatment.

Statistical data processing was carried out using Statistica 8.0 software environment using descriptive statistics, the Mann—Whitney test, and the Spearman correlation coefficient.

Results and discussion

When comparing the ferritin level between tuberculosis patients and practically healthy individuals,

a significantly higher ferritin level was found in tuberculosis patients ((106.57 ± 59.15) ng/ml (median — 96.74 ng/ml)) compared to the control group ((48.17 ± 31.26) ng/ml (median — 43.14 ng/ml)), $p < 0.05$ (Fig. 1).

Comparison of the ferritin level in patients depending on the effectiveness of the intensive phase of treatment showed a significant difference both at the beginning of treatment and after 60 doses. Thus, at the beginning of treatment, the ferritin level was significantly lower ((95.95 ± 8.68) ng/ml (median — 80.24 ng/ml)) in patients who later effectively completed the intensive phase of anti-tuberculosis treatment than in patients with ineffective intensive phase of treatment ((152.27 ± 8.85) ng/ml (median — 44.17 ng/ml)). The same trend persisted after 60 days: in the effective intensive phase — (123.87 ± 13.39) ng/ml (median — 112.24 ng/ml), in the ineffective one — (239.76 ± 12.91) ng/ml (median — 250.22 ng/ml), $p < 0.05$ (Fig. 2).

A significantly higher level of ferritin was found in patients with destruction of pulmonary tissue ((115.91 ± 8.90) ng/ml (median — 125.68 ng/ml)) compared to patients without destruction ((88.68 ± ± 10.90) ng/ml (median — 79.42 ng/ml)), $p < 0.05$ (Fig. 3).

When comparing the level of IL-6, it was found to be significantly higher in tuberculosis patients ((96.22 ± 6.44) pg/ml (median — 104.20 pg/ml)) compared to practically healthy individuals ((54.17 ± 5.74) pg /ml (median — 55.25 pg/ml)), $p < 0.05$ (Fig. 4).

In effective intensive phase of antituberculosis treatment, the level of IL-6 was significantly lower. Thus, at the beginning of treatment, it was (82.59 ± ± 6.89) pg/ml (median — 78.20 pg/ml) in patients with an effective intensive phase of treatment and (146.42 ± 8.04) pg/ml (median — 140.40 pg/ml) in patients with ineffective intensive treatment phase. After 60 days, it was (48.88 ± 4.19) pg/ml (median — 45.90 pg/ml) in patients with an effective intensive phase of treatment and (142.89 ± ± 9.11) pg/ml (median — 146.38 pg/ml) in patients with ineffective intensive treatment phase, $p < 0.05$ (Fig. 5).

The level of HBD-1 was also significantly higher in patients with pulmonary tuberculosis ((22.21 ± ± 2.78) pg/ml (median — 9.17 pg/ml)) compared to healthy individuals ((8.97 ± 2.56) pg/ml (median — 3.37 pg/ml)), $p < 0.05$ (Fig. 6).

The level of HBD-1 was higher when the intensive phase of antituberculosis therapy was ineffective, as when measured at the beginning of treatment (effective intensive phase — (18.71 ± 3.31) pg/ml (median — 3.31 pg/ml)), ineffective intensive phase — (32.79 ± 8.31) pg/ml (median — 24.60 pg/ml), as well as when measured after 60 days (effective intensive phase — (19.93 ± 3.58) pg/ml (median —

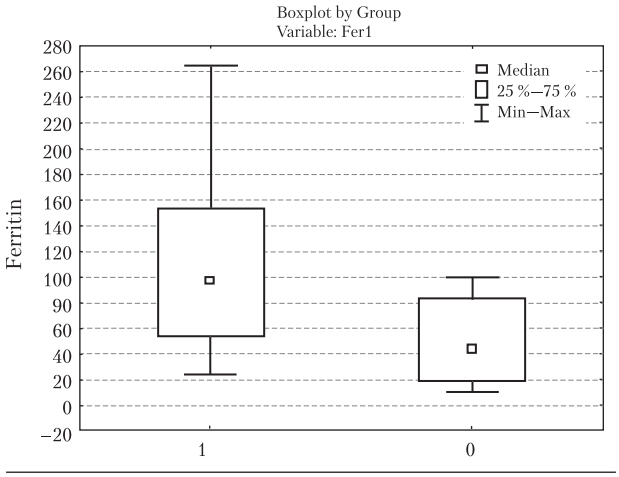

Fig. 1. **Comparison of ferritin levels between patients with tuberculosis (1) and healthy individuals (0)**

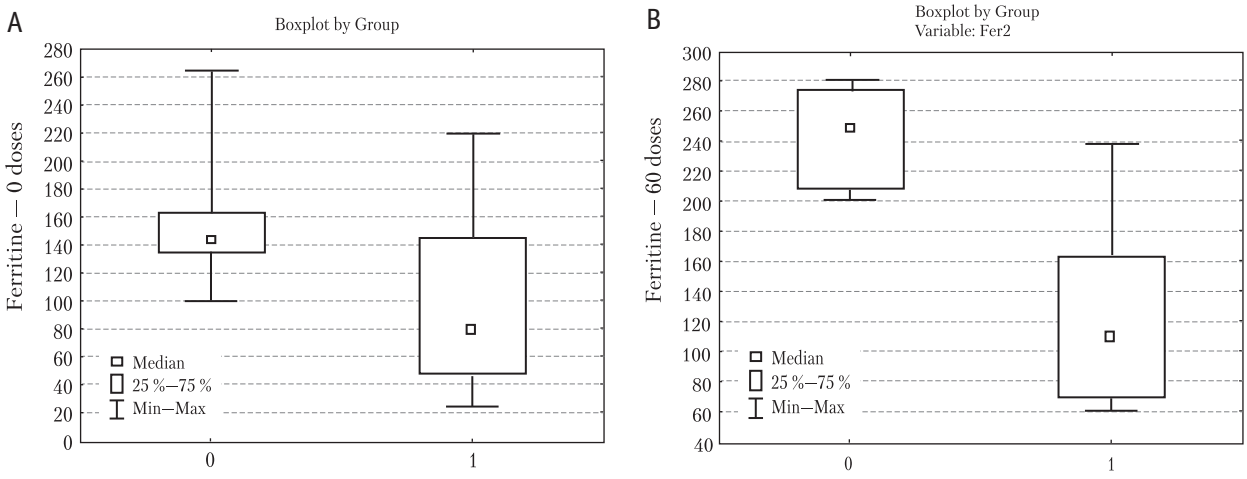

Fig. 2. **Comparison of ferritin level at the beginning of treatment (A) and after 60 days (B) in patients with ineffective (0) and effective (1) intensive phase of anti-tuberculosis treatment**

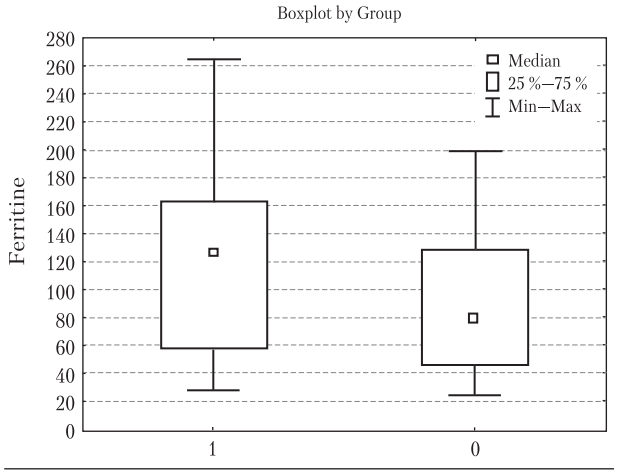

Fig. 3. **Comparison of ferritin level in patients with destruction of pulmonary tissue (1) and without destruction (0)**

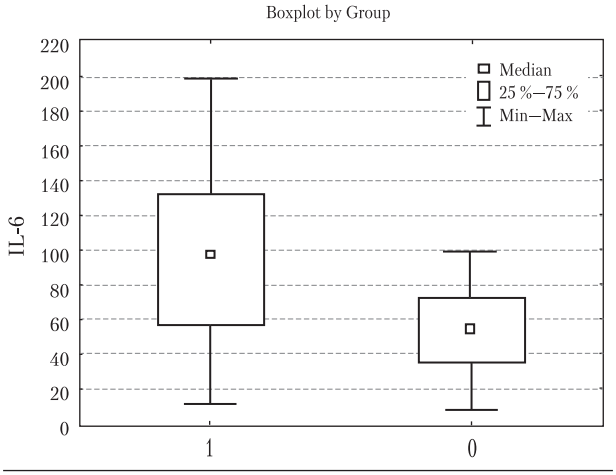

Fig. 4. **Comparison of the level of IL-6 in patients with tuberculosis (1) and healthy individuals (0)**

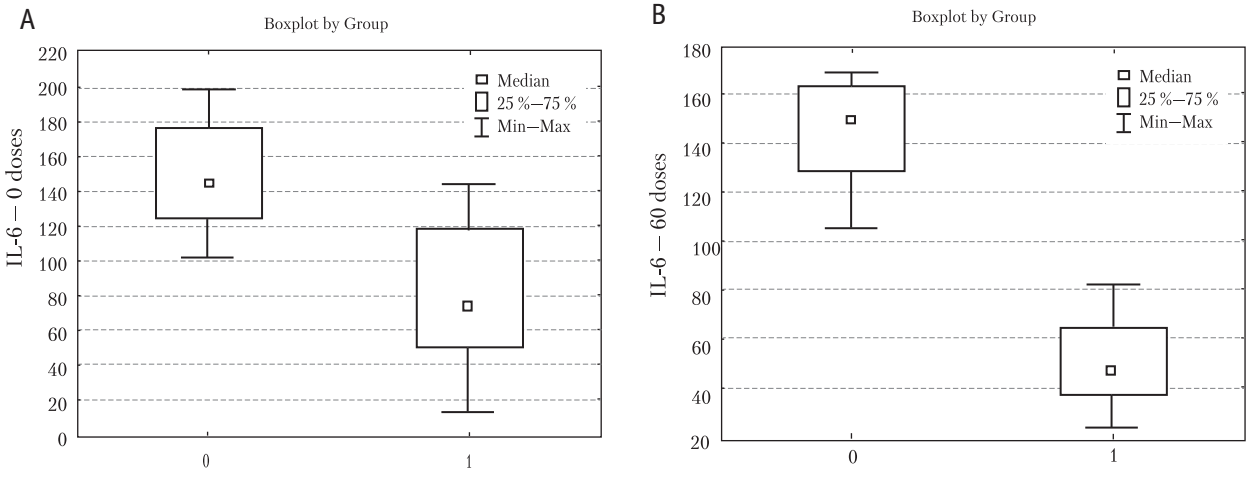

Fig. 5. **Comparison of the level of IL-6 at the beginning of treatment (A) and after 60 days (B) in patients with ineffective (0) and effective (1) intensive phase of anti-tuberculosis treatment**

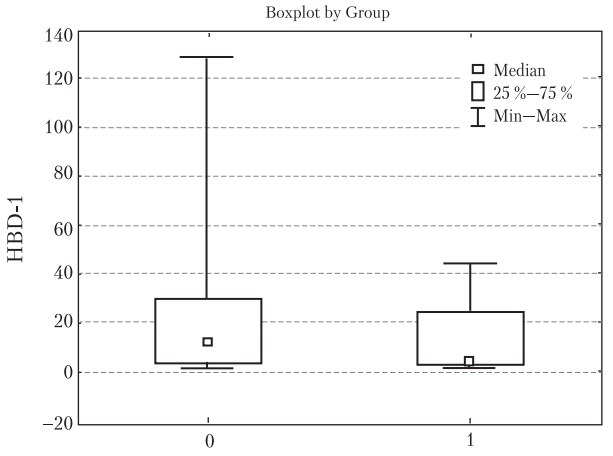

Fig. 6. **Comparison of the level of HBD-1 in patients with tuberculosis (1) and healthy individuals (0)**

7.17 pg/ml)), ineffective intensive phase — (42.92 ± 12.99) pg/ml (median — 28.50 pg/ml), $p < 0.05$ (Fig. 7).

The level of HBD-1 was also significantly higher in patients with pulmonary tissue destruction ((26.88 ± 3.83) pg/ml (median — 11.41 pg/ml)) compared to patients without pulmonary tissue destruction ((14.27 ± 3.39) pg/ml (median — 4.64 pg/ml)), $p < 0.05$ (Fig. 8).

When studying the relationships between the studied indicators and severity of the tuberculosis process, it was found that HBD-1 showed positive, significant ($p < 0.05$) correlations with the volume of lung tissue damage (+0.33), clinical manifestations, assessed by a point scale (+0.52), the massiveness of bacterial excretion detected by microscopy (+0.48) and culture (+0.33), as well as a negative correlation with body mass index (BMI) (–0.32). IL-6 showed a significant ($p < 0.05$) negative correlation with BMI (–0.39). Ferritin showed significant ($p < 0.05$) positive correlations with clinical manifestations assessed by a scoring scale (+0.30), the volume of lung tissue damage (+0.25) and the

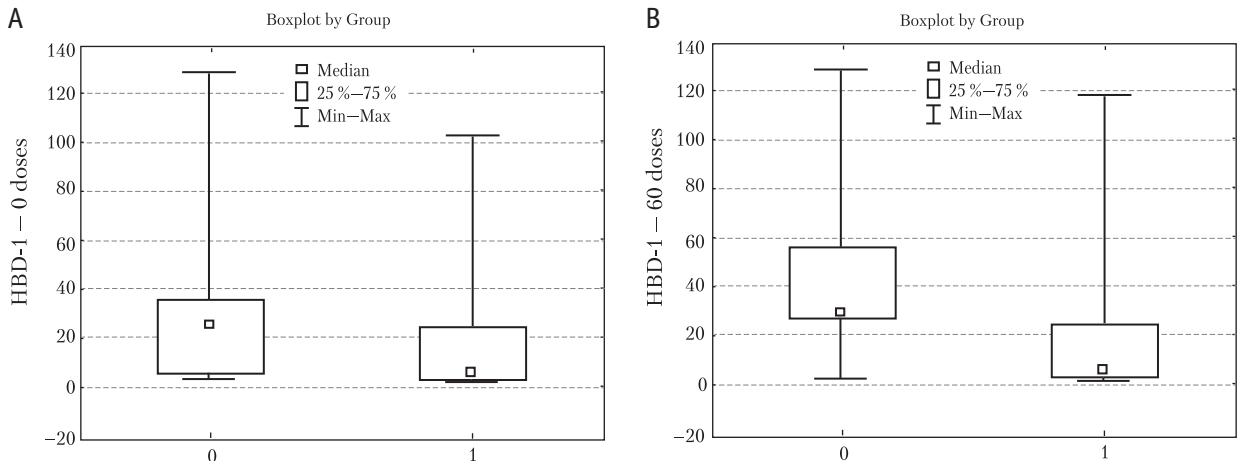

Fig. 7. **Comparison of the level of HBD-1 at the beginning of treatment (A) and after 60 days (B) in patients with ineffective (0) and effective (1) intensive phase of anti-tuberculosis treatment**

massiveness of bacterial excretion revealed by the culture (+0,23), as well as a negative correlation with BMI (—0.37). Significant ($p < 0.05$) positive correlations between the investigated parameters were found: HBD-1 — ferritin (+0.22), HBD-1 — IL-6 (+0.31), ferritin — IL-6 (+0.28) (Fig. 9).

The obtained data made it possible to detect significantly higher levels of ferritin, IL-6 and HBD-1 in tuberculosis patients before the start of treatment compared to healthy individuals, which once again confirms the fact that these pro-inflammatory markers are included in the anti-tuberculosis treatment even at the beginning of the disease immune response, which leads to an increase in their concentration in the blood of patients [3, 6, 10].

All 3 studied markers showed higher levels at the beginning of treatment and after 60 days in patients in whom the intensive phase of anti-tuberculosis treatment was ineffective. The studied biomarkers are reagents of the acute phase of inflammation and their production is stimulated by the direct effect of mycobacteria on the cells of the immune system, therefore, with a high bacterial load and its slow decrease or even the absence of positive dynamics, which later turns to ineffectiveness of the intensive phase of treatment. Ferritin, IL-6 and HBD-1 react first, which allows predicting the likely ineffectiveness of anti-tuberculosis treatment long before the results of microbiological diagnostics [7, 9, 11]. With effective anti-tuberculosis treatment, the levels of all studied indicators decrease, which makes it possible to use them as quick markers of the effectiveness of anti-tuberculosis therapy. Also, increased levels of HBD-1 and ferritin were observed in the destruction of lung tissue by the tuberculosis process.

When studying correlations between the studied indicators and clinical parameters, the greatest

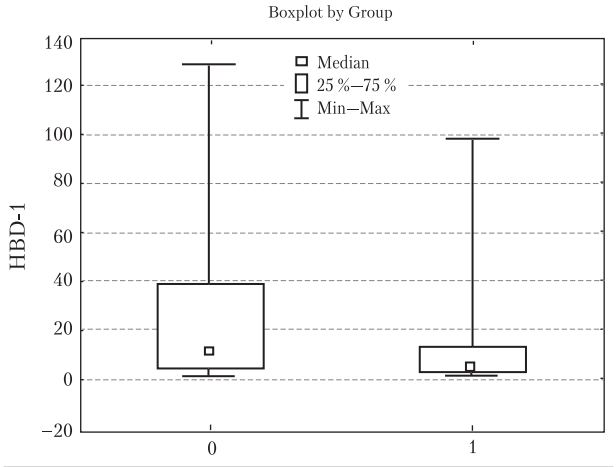

Fig. 8. **Comparison of the level of HBD-1 in patients with destruction (1) and without destruction (0) of pulmonary tissue**

strength and number of correlations was observed in HBD-1, which makes it the most important prognostic factor among the studied ones. Thus, there were positive relationships with the massiveness of bacterial excretion, the volume of lung tissue damage, and the severity of clinical symptoms, assessed on a point scale, and a negative relationship with BMI. Therefore, more severe the lesions and worse the patient's somatic condition leads to the higher the level of HBD-1.

The positive relationships between the level of ferritin, clinical manifestations of tuberculosis, the massiveness of bacterial excretion and the volume of lung tissue damage can be explained not only by the increase in the synthesis of ferritin, as an acute-phase protein, by the host organism, but also by MTB-associated hyperferritinemia, since with a high bacterial load, MT transform free iron in macrophages and the intercellular space into ferritin to avoid the toxic effects of free iron (oxidative stress

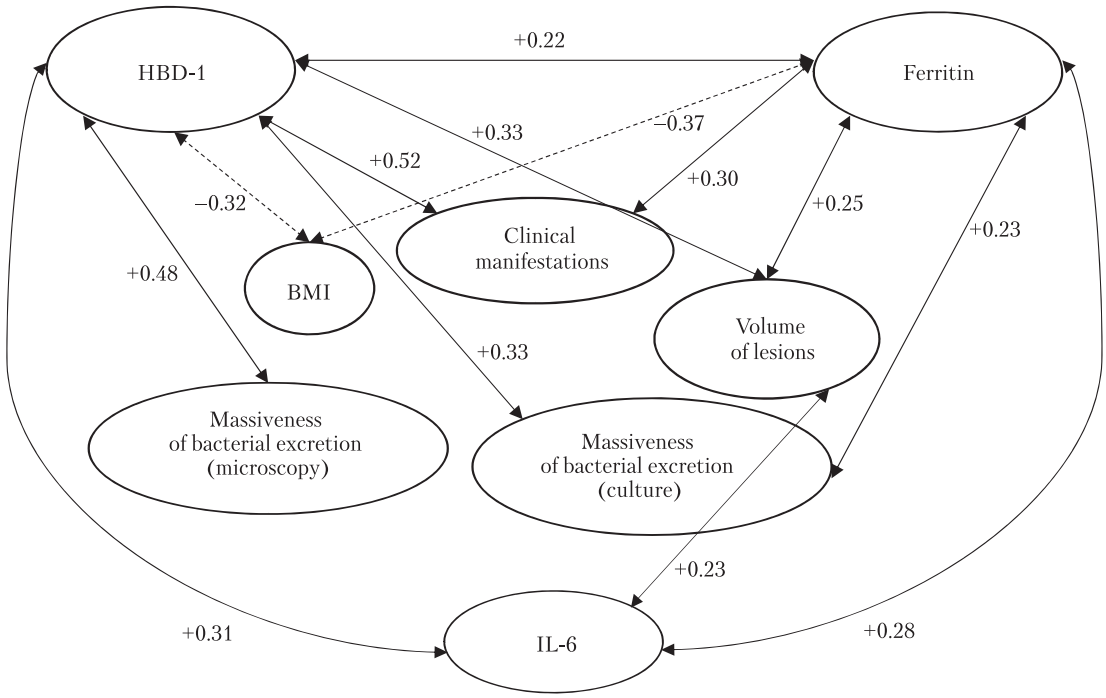

Fig. 9. **Relationships between HBD-1, IL-6, ferritin and severity of tuberculosis lesions**

due to the catalysis of the formation of active oxygen radicals) [1]. In addition, with excessive inflammation, the host organism itself can deposit free iron in the form of ferritin to prevent the Fenton reaction, which leads to the formation of free radicals and increased inflammation with the destruction of the host organism's own cells [8].

A positive relationship was found between the level of IL-6 and the volume of lung tissue damage, since the larger is the area of specific inflammation, the more immune cells are involved in the anti-tuberculosis immune response and the higher is the production of pro-inflammatory peptides, including IL-6 [2].

The positive correlations between HBD-1, ferritin and IL-6 were also observed. The production of these peptides by the cells of the host organism begins immediately under the direct influence of MTB, but later they stimulate the synthesis of each other and other cytokines, triggering a cascade of inflammatory reactions [4].

Conclusions

Levels of ferritin, interleukine-6 and human-beta-defensin-1 are significantly increased in tuberculosis patients compared to healthy individuals, which allows them to be considered as markers of tuberculosis inflammation. Higher concentrations of these markers, both at the beginning of treatment and after 60 doses, are predictors of failure of antituberculosis therapy. The strongest relationship between the studied markers and parameters of the severity of the tuberculosis process is observed in the study of human-beta-defensin-1, which allows us to consider it as the most effective marker of the severity of the course among presented ones.

There is no conflict of interest.
Participation of the authors: concept and design of the study — O.S. Shevchenko, L.D. Todoriko; collection of material — I.A. Ovcharenko; processing of material — O.M. Svets; writing the text — O.O. Pohorielova; text editing — S.L. Matvyeyeva.

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Феритин, ІЛ-6 і β-дефензин-1 як прогностичні маркери тяжкості перебігу та ефективності лікування туберкульозу легень

Сучасні методи дослідження у хворих на туберкульоз (мікроскопія мокротиння, культуральні дослідження та молекулярно-генетичні методи) хоча є добре дослідженими, мають низку недоліків, зокрема низьку чутливість, тривалий час, необхідний для отримання результатів, високу вартість. Через це пошук альтернативних діагностичних засобів і методів прогнозування перебігу та ефективності лікування у хворих на туберкульоз є актуальним.

Мета роботи — дослідити динаміку рівня феритину, інтерлейкіну-6 (ІЛ-6), β-дефензину-1 на тлі інтенсивної фази терапії туберкульозу легень та виділити найкращий маркер прогнозування ефективності лікування.

Матеріали та методи. У дослідження було залучено 100 пацієнтів із туберкульозом легень та 20 практично здорових осіб. Обстеження пацієнтів проводили згідно з чинними стандартами надання медичної допомоги хворим на туберкульоз. Додатково визначали концентрацію феритину, ІЛ-6 та β-дефензину-1 у крові натще на початку лікування і через 60 днів. Практично здоровим особам із групи контролю проводили одноразове визначення рівня феритину, ІЛ-6 та β-дефензину-1 у крові натще.

Результати та обговорення. На початку лікування у пацієнтів, які згодом ефективно завершили інтенсивну фазу протитуберкульозного лікування, рівень феритину був статистично значущо нижчим (95,95 ± 8,68) нг/мл, ніж у пацієнтів, у яких інтенсивна фаза протитуберкульозного лікування була неефективною (152,27 ± 8,85) нг/мл. Таку саму тенденцію спостерігали через 60 днів: за ефективної інтенсивної фази лікування — (123,87 ± 13,39) нг/мл, за неефективної — (239,76 ± 12,91) нг/мл ($p < 0,05$). За ефективної інтенсивної фази протитуберкульозного лікування рівень ІЛ-6 був статистично значущо нижчим. На початку лікування він становив (82,59 ± 6,89) пг/мл у пацієнтів з ефективною інтенсивною фазою лікування та (146,42 ± 8,04) пг/мл — у пацієнтів з неефективною, через 60 днів — відповідно (48,88 ± 4,19) та (142,89 ± 9,11) пг/мл ($p < 0,05$). Вміст β-дефензину-1 був вищим за неефективної інтенсивної фази протитуберкульозної терапії як на початку лікування (ефективна інтенсивна фаза — (18,71 ± 3,31) пг/мл, неефективна інтенсивна фаза — (32,79 ± 8,31) пг/мл), так і через 60 днів (відповідно (19,93 ± 3,58) і (42,92 ± 12,99) пг/мл, $p < 0,05$).

Висновки. Рівень феритину, ІЛ-6 та β-дефензину-1 статистично значущо підвищується у хворих на туберкульоз порівняно зі здоровими особами, що дає підставу вважати їх маркерами туберкульозного запалення. Вищі концентрації цих маркерів як на початку лікування, так і через 60 днів є предикторами невдачі протитуберкульозної терапії. Найсильніший взаємозв'язок між досліджуваними маркерами і параметрами тяжкості туберкульозного процесу зареєстровано для β-дефензину-1, що дає підставу вважати його найефективнішим маркером тяжкості перебігу.

Ключові слова: феритин, ІЛ-6, β-дефензин-1, туберкульоз, прогнозування.

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