A METHOD FOR DETERMINING THE DEVELOPMENT OF ADHESIVE DISEASE OF THE ABDOMINAL CAVITY

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Background. Immunological studies emphasize changes in the cytokine profile in the abdominal cavity after surgical interventions (2,5), but the question of how to specifically modulate this process without compromising tissue regeneration remains unresolved.

Existing clinical guidelines for the treatment of adhesive intestinal obstruction (AIO) (1,3,4) are based primarily on empirical approaches. The lack of individualized strategies that take into account the patient's immunological status highlights the insufficient development of prevention and treatment methods.

The objective of the study was to develop methods for the early prediction of the development of abdominal adhesive disease.

Materials and Methods. The study included 265 patients with APAD. The reference group consisted of 20 apparently healthy individuals without previous abdominal surgery. The main observation cohort included 245 patients with APAD, including a control group (137 patients) treated according to the traditional regimen, and a study group (128 patients) who were treated with a developed diagnostic and treatment algorithm for predicting and preventing recurrence.

Results and Discussion. Given the high clinical and prognostic significance of the identified determinants, we developed an original prognostic scale, the PAS (Peritoneal Adhesion Score), designed to quantitatively assess the individual risk of developing APAD. This scale is designed to quantitatively assess the individual likelihood of developing APAD and APAD in patients after abdominal surgery.

The PAS prognostic scale is based on a comprehensive analysis of 11 indicators reflecting key links in the pathogenesis of the development and progression of spondylitis. It includes four cytokines and growth factors (IL-6, TGF-β1, VEGF, IL-10), three indicators of the coagulation and fibrinolysis system (fibrinogen, PAI-1, tPA), and four molecular genetic markers of connective tissue remodeling (Col1a1, Tgfb1, Timp1, Serpine1). Each indicator has fixed risk gradations and is assessed at 0, 1.5, or 3 points depending on the degree of deviation from the norm. Thus, the final score ranges from 0 to 33. The algorithm was based on the results of a multivariate logistic analysis, which was used to generate a weighting matrix for each marker included in the PAS scale. The system automatically assigned points to each parameter, summarized the final results, and generated a conclusion classifying the patient into one of three risk zones (no risk, low risk, or high risk). The algorithm also took into account variation in values within series, allowing for more flexible interpretation of borderline cases and the identification of subgroups with unstable profiles. The use of artificial intelligence provided several advantages, including reduced analysis time, elimination of arithmetic errors, standardization of calculations, and the ability to accumulate a database for subsequent automated optimization of threshold values. This approach increased the reliability of PAS scale interpretation and laid the foundation for further digitalization of the method and its integration into clinical decision support systems. The highest results were achieved by

integrating the indicators into the comprehensive PAS scale. This approach yielded a sensitivity of 86.8% and a specificity of 84.5%. The positive predictive value reached 88.1%, while the negative predictive value was 83.7%. The area under the receiver operating characteristic (ROC) curve was 0.861 (95% CI 0.812–0.902), indicating high predictive power and significantly exceeding the values of partial models. Overall, the analysis demonstrated that the greatest diagnostic efficacy is achieved with the comprehensive integration of immunological and molecular genetic parameters. The use of the PAS scale significantly increases both the sensitivity and specificity of prognosis, making it the preferred tool for early risk stratification of SBBP development. It should be noted that the identified advantage of the integrated model has a pathogenetic basis, as immunological parameters (IL-6, TGF-β1, VEGF, IL-10) better reflect the early stages of the inflammatory response, angiogenesis, and cytokine imbalance. Such changes are characterized by pronounced dynamism and can vary depending on the degree of tissue trauma and individual immune response characteristics.

Thus, combining immunological and molecular genetic parameters provides a comprehensive understanding of the key mechanisms of SP.

Conclusions:

1. Combining immunological and molecular genetic parameters provides a comprehensive understanding of the key mechanisms of SP and explains the high predictive accuracy demonstrated by the ROC analysis.

2. Using only one set of parameters in isolation limits predictive capabilities, as the immunological panel primarily captures transient changes, while the genetic panel reflects structural changes to a greater extent, but these are less sensitive to the initial phase of the process. Integrating these data within the PAS scale allows for the simultaneous consideration of both dynamic and

stable components of pathogenesis, thereby increasing the sensitivity and specificity of the model.

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