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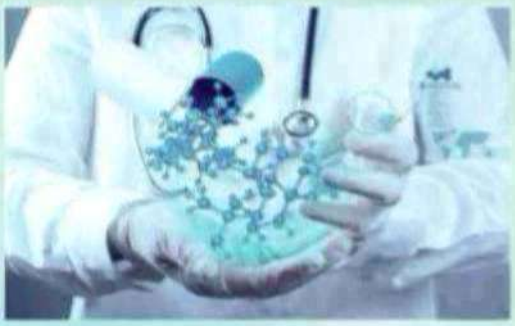

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METHODS FOR EARLY DETECTION OF THE DEVELOPMENT OF ADHESIVE DISEASE OF THE ABDOMINAL CAVITY

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✓ ***Resume***

The greatest diagnostic effectiveness is achieved through the comprehensive integration of immunological and molecular genetic indicators. The use of the PAS score significantly improves both the sensitivity and specificity of prognosis, making it the preferred tool for early risk stratification of abdominal adhesive disease.

Keywords: Abdominal cavity, adhesive disease, prognosis

СПОСОБЫ РАННЕГО ОПРЕДЕЛЕНИЯ РАЗВИТИЯ СПАЕЧНОЙ БОЛЕЗНИ БРЮШНОЙ ПОЛОСТИ

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✓ ***Резюме***

Наибольшая диагностическая эффективность достигается при комплексной интеграции иммунологических и молекулярно-генетических показателей. Использование шкалы «PAS» позволяет значительно повысить как чувствительность, так и специфичность прогноза, что делает ее предпочтительным инструментом ранней стратификации риска формирования спаечной болезни брюшной полости.

Ключевые слова: Брюшная полость, спаечная болезнь, прогнозирование

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✓ ***Rezyume***

Eng katta diagnostika samaradorligiga immunologik va molekulyar genetik ko'rsatkichlarning kompleks integratsiyasi orqali erishiladi. PAS ballidan foydalanish prognozning sezgirligini va o'ziga xosligini sezilarli darajada yaxshilaydi, bu esa uni qorin bo'shlig'i bitishma kasalligining erta xavf stratifikatsiyasi uchun afzal vositaga aylantiradi.

Kalit so'zlar: Qorin bo'shlig'i, kavshar kasalligi, bashoratlash

Relevance

Studying the mechanisms underlying the development of abdominal adhesive disease (AAD) has remained an important area of surgical research for decades. Classical concepts of AAD pathogenesis focused on impaired peritoneal healing. However, many aspects of the interaction between immune cells and the mesothelium during the development of fibrotic changes remain poorly understood, particularly at the level of molecular signaling pathways (1,3,5).

Particular attention has been paid to studying the role of the inflammatory response in the initiation of the adhesion process (AP) (2,4,6). However, it remains unclear which immunological mediators determine the transition from inflammation to the formation of persistent fibrous structures.

The problem of adhesion prevention using immunomodulation has been actively studied in recent years, but the results remain contradictory.

Diagnosing AAD before the development of intestinal obstruction is particularly challenging. Scientific studies indicate a lack of reliable noninvasive methods for diagnosing early adhesive intestinal obstruction, indicating insufficient understanding of early immunopathological markers (7, 8, 9, 10).

Immunological studies emphasize changes in the cytokine profile in the abdominal cavity after surgery (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 (AI) (7, 10) 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.

Thus, despite significant progress in the study of APAD, the development of effective methods for the diagnosis, prevention, and treatment of APAD and AI based on a thorough understanding of immunopathogenetic mechanisms remains an open question. This necessitates further fundamental and clinical research in this area.

Study objective: Develop methods for the early prediction of the development of adhesive intestinal obstruction.

Materials and methods

The study involved 265 patients with ASCI. The reference group consisted of 20 apparently healthy individuals with no history of abdominal surgery. The main observation cohort included 245 patients with DSBP, 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 relapse prediction and prevention.

Overall, the study design combined experimental and clinical components, ensuring a comprehensive assessment of the problem and enabling the development of a continuous chain from laboratory pathogenesis studies to practical testing and implementation in clinical surgery.

Results and discussion

Identification of key immunological, biochemical, and molecular genetic markers provided the basis for developing methods for the early prediction of DSBP. At this stage, the goal was to integrate the selected indicators into a unified system that allows for a quantitative assessment of the risk of dense adhesion formation. An important element is the determination of diagnostic thresholds for each marker, as well as testing the effectiveness of their combined use by calculating sensitivity, specificity, and predictive value. This strategy enables the transition from fundamental pathogenesis analysis to practical diagnostic tools applicable in clinical surgery.

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 peritoneal adhesion syndrome (PAS). This scale is designed to quantitatively assess the individual likelihood of developing peritoneal adhesion syndrome (PAS) and peritoneal adhesion syndrome (PAS) in patients following abdominal surgery.

The PAS prognostic scale is based on the results of a comprehensive analysis of 11 indicators reflecting key links in the pathogenesis of peritoneal adhesion syndrome (PAS). 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 interval risk stratification is constructed in three levels: a score of ≤5 points indicates no risk of coarse adhesion formation; values in the range of 6-24 points correspond to a low probability requiring dynamic observation; a score of ≥25 points is considered a high-risk zone for the formation of dense adhesions and SBAP.

Each of the included parameters correlates with a specific link in pathogenesis: an IL-6/IL-10 imbalance reflects the stability of the inflammatory response; an increase in TGF-β1/Tgfb1 and Col1a1 indicates myofibroblast activation and increased collagen I synthesis; an increase in VEGF characterizes the severity of angiogenesis. The "fibrinogen - PAI-1/Serpine1 - tPA" cluster reflects dysregulation of fibrinolysis and records the preservation of the fibrin matrix; increased Timp1 expression indicates connective tissue stabilization due to the suppression of matrix metalloproteinases.

Thus, the integrated PAS score reflects not only the totality of quantitative changes but also the pathogenetic burden of individual components of the process. Unlike traditional assessment systems, this scale provides strict numerical stratification and eliminates subjectivity in interpretation, which improves the reproducibility and clinical applicability of the method.

The scale's software implementation does not require specialized equipment or licensed software, and the calculation algorithm can be implemented in an interactive spreadsheet, a Microsoft Excel macro, or a simple web interface. The user enters the values of the analyzed parameters, after which the system automatically assigns each of them a corresponding number of points and calculates the final score, which is then interpreted according to risk levels.

A total score of less than or equal to 5 points indicates that patients are at no risk of developing SBFS. Within this range, cytokine profile, fibrinolytic factors, and gene expression indicators are within physiological limits, reflecting a balance between inflammatory and anti-inflammatory responses, the preservation of fibrinolytic mechanisms, and the absence of signs of activated fibrogenesis. In this category of patients, the formation of persistent fibrous adhesions in the postoperative period appears unlikely.

A range of scores from 6 to 24 points corresponds to a low risk of developing SBFS. In these cases, moderate deviations in individual parameters (IL-6, TGF-β1, VEGF, PAI-1, Col1a1, etc.) are observed, which may indicate a tendency toward activation of fibrinolytic imbalance and fibrotic cascades. However, compensatory mechanisms (IL-10, tPA, and a maintained collagen I/III balance) still partially restrain the process. These patients are likely to develop loose adhesions, characterized by an immature collagen matrix, limited vascularization, and a certain degree of reversibility.

A total score of 25 points or more is considered a high risk for the development of dense adhesions. This group exhibits pronounced immune and molecular genetic abnormalities, including significant increases in IL-6, TGF-β1, VEGF, fibrinogen, PAI-1, and expression of Col1a1, Tgfb1, Timp1, and Serpine1, along with decreased IL-10 and tPA activity. The combination of these changes indicates persistent activation of the inflammatory-fibrous circuit, suppression of fibrinolysis, and intensive synthesis of mature collagen I, which pathogenetically determines the transition to the formation of dense, difficult-to-resolve adhesions with a high probability of clinically significant SBBP and, consequently, acute inflammatory bowel syndrome.

Thus, the "PAS" scale not only allows for patient stratification by risk but also reflects the prevalence of certain pathogenesis factors, ensuring its pathogenetic validity and clinical diagnostic value.

To illustrate the practical application of the PAS prognostic scale, clinical examples of calculating the total score in patients of various risk groups are provided.

This category of patients is most likely to develop loose adhesions with incomplete connective tissue maturity and partial reversibility. Clinical observation confirmed moderate severity of SBBP without signs of acute cerebral palsy.

To improve the accuracy and reproducibility of the prognostic assessment in the main group of patients, an automated data interpretation system based on artificial intelligence technologies was used. This approach minimized researcher bias and ensured a standardized analysis of a combination of immunological, biochemical, and molecular genetic parameters.

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 scores 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 accounted for 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 enabled comparative analysis not only of total scores but also of the contribution of individual indicators to risk in a given patient. Thus, the system enabled the identification of "leading" determinants (for example, the dominant contribution of Col1a1, Serpine1, or PAI-1) and the identification of individual pathogenesis patterns.

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 the PAS scale interpretation and laid the foundation for further digitalization of the method and its integration into clinical decision support systems.

A comparative analysis of the diagnostic efficacy of various prognostic scale variants revealed that the use of immunological indicators alone provided a sensitivity of 72.5% and a specificity of 68.3%. The positive predictive value was 74.2%, while the negative predictive value was 66.1%. The area under the receiver operating characteristic curve (AUC) was 0.742 (95% CI 0.681-0.795), corresponding to moderate discriminatory power. Using a model based solely on molecular genetic markers demonstrated comparable results: sensitivity of 70.1%, specificity of 71.4%, positive predictive value of 73.5%, negative predictive value of 68.9%, and AUC of 0.736 (95% CI 0.672–0.789).

Thus, using both immunological and genetic parameters alone was characterized by limited predictive accuracy.

The highest results were obtained by integrating the indicators into the comprehensive PAS scale. This option provided 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 ROC curve was 0.861 (95% CI 0.812–0.902), which corresponds to high predictive power and significantly exceeds the values of partial models.

Overall, the analysis showed that the greatest diagnostic effectiveness is achieved with the comprehensive integration of immunological and molecular genetic indicators. The use of the PAS score significantly improves both the sensitivity and specificity of prognosis, making it the preferred tool for early risk stratification of SBPS 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 characteristics of the immune response.

Molecular genetic markers (Col1a1, Tgfb1, Timp1, Serpine1) reflect the processes of connective tissue remodeling, extracellular matrix stabilization, and suppression of fibrinolysis. Accordingly, their expression creates a more stable profile, determining the transition from reversible phases of inflammation to the formation of mature fibrotic structures. Using only one set of parameters in isolation limits predictive capabilities, as the immunological panel predominantly records transient changes, while the genetic panel reflects more structural changes, which 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.

Thus, combining immunological and molecular genetic parameters provides a holistic reflection of the key mechanisms of SP and explains the high predictive accuracy demonstrated in the ROC analysis.

Conclusions

1. Combining immunological and molecular genetic parameters provides a holistic reflection of the key mechanisms of SP and explains the high predictive accuracy demonstrated in the ROC analysis.

2. Using only one set of parameters in isolation limits predictive capabilities, as the immunological panel predominantly records transient changes, while the genetic panel reflects more structural changes, which are less sensitive to the initial phase of the process. The integration of these data within the PAS scale allows for the simultaneous consideration of both dynamic and stable links in pathogenesis, which increases the sensitivity and specificity of the model.

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