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Mathematical Assessment of the Risk of Developing Dysfunctions of Autonomic and Thyroid Statuses from the Point of View of Evidence-Based Medicine

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Abstract: At present, computational methods have been developed for diagnosing and predicting several somatic diseases, however, the prediction of diseases of bronchial asthma (BA), in particular BA with the autonomic and thyroid statuses disorders of (ATSD) has not been carried out using clinical and anamnestic data.

In our opinion, it is very important in the prevention of asthma to identify risk factors for asthma with ATSD by comparing various prognostic criteria. Using the method of normalization of intensive indicators (NII) by E. N. Shigan, based on the probabilistic Bayes method, prognostic matrices were developed based on the data of anamnesis, clinical symptoms.

Keywords: cardiointervalography, hyperfunction, broncho-pulmonary, pathogenetic, sympathetic origin.

Introduction

Studies carried out in diseases of the bronchopulmonary system and other diseases in the experiment and patients [9] showed that disturbances in the balance of ANS mediators are an important factor in the pathogenesis of respiratory pathology. The role of ANS in the development of clinical and pathogenetic manifestations of respiratory pathology in children was stated. At the same time, the presence of deep disorders of the ANS activity was established, depending on the period, severity, and characteristics of the course of the disease. In the initial period of recurrent processes in children, the prevalence of disorders of sympathetic origin was revealed, followed by parasympathetic effects [7].

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Recent studies have shown the effectiveness of cardiointervalography (CIG) in assessing autonomic status [5,10]. This is an accessible and promising method for objectively assessing the severity of children's conditions, reflecting the nature of the body's adaptive reactions. It is this method, due to its availability (it requires only a conventional electrocardiograph) and non-invasiveness, that has found wide application in pediatrics. CIG allows to indirectly assessment the role of humoral and central mechanisms of regulation of homeostasis, as well as the degree of stress of the adaptive mechanisms of the body [1, 5, 9, 13]. It was used in studies of the pathogenetic mechanisms of various non-infectious diseases of the gastrointestinal tract, vegetative-vascular dystonia, intestinal infections, chronic hepatitis, as well as respiratory pathology: frequent respiratory diseases, recurrent bronchitis, pneumonia, bronchial asthma, etc.

In recent years, the incidence of thyroid insufficiency has increased throughout the world. So, 5-10%. In the US population, overt hypothyroidism has been established, and among people 55 and older, it is found in 8-17% of Americans in different states. In Russia, a population survey for hypothyroidism has not yet been carried out, but the frequency of iodine deficiency goiter has been studied, it ranges from 26 to 65% in different regions. It is now recognized that iodine deficiency goiter is latent hypothyroidism [6].

The fact that the current socio-ecological situation has an extremely unfavorable effect on the state of the thyroid gland inspires serious concern [4]. According to E.N. Arsenyeva et al., Out of 117 children being treated not for a thyroid condition, half of the changes were noted, with 81% having hypofunction and 19% with hyperfunction of this organ [13].

There is an opinion that endocrine regulation in the pathogenesis of AD is due to the so-called "stress response" and the body's defense against the antigen [4,7,11]. A feature of childhood AD is the presence of dissociative disorders within the Neuro - Immune - endocrine complex. The most common example is the onset of AD in children who have undergone perinatal damage to the central nervous system. The frequency of intra- and perinatal pathology in children with BA is much higher than the average threshold and reaches 80%. Tissue hypoxia, which occurs during the pathological course of pregnancy and childbirth, leads to disruption of the synthesis of enzymes, high-energy compounds, disruption of energy metabolism [5]. The pathological course of labor can lead to damage to the brain stem structures that regulate the functions of the respiratory system, which contributes to the formation of bronchopulmonary pathology. This is confirmed by the fact that 60% of children with BA have EEG changes, indicating the involvement of the brain stem and hypothalamic structures in the pathological process, impairment of cortical-subcortical relationships [4, 5].

The influence of thyroid hormones on the formation of the respiratory system was revealed already at the stage of intrauterine development. It has been established that the inner embryonic layer forms both the respiratory organs and the main part of the thyroid gland. Its hormones affect the synthesis of β-receptors and the production of surfactants [8, 9]. Respiratory function changes are observed against the background of various changes in

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thyroid function. Thus, a low level of thyroid hormones can affect respiration through mechanisms such as a decrease in metabolic oxygen demand and the development of hypoventilation of the lungs, an increase in pressure in the left ventricle, remodeling of the respiratory muscles and lungs, an increase in hematocrit, changes in the level of neurotransmitters (serotonin, histamine) in areas brain involved in the control of respiration [13].

Materials and Methods

To compile the prognostic table, comparable indicators of the predicted phenomenon were obtained according to the gradations of the most important factors. The significance of factors and their gradations was determined by using the relative risk indicator (R). This indicator is the ratio of the maximum intensity indicator (c) to the minimum (d) within each factor ($R = c / d$).

If the factor does not affect, then it is equal to one. The higher R, the greater the significance of the factor for the occurrence of this type of pathology.

The essence of the method lies in the fact that instead of the usual intensive indicators, NII is used, which can be calculated by the formula: $N = r / M$, where: N is the normalized intensive indicator (NII), r is the intensive ATSD indicator per one hundred surveyed, M is the “normalizing index".

In this case, the average frequency of ATSD according to the data of the entire study (per 100 subjects) is taken as a normalizing value.

For example, in sick children, the incidence of asthma is 37.5, and the incidence of ATSD was 55.6. The same indicator among all those surveyed was 48.1. This value was taken as a "normalizing" indicator (M). Substituting the corresponding values into the above formula, we obtained the following normalized intensive indices: in BA patients, $NII1 = 37.5 / 48.1 = 0.78$ and $NII2 = 55.6 / 48.1 = 1.12$ in patients with ATSD. Relative risk index (R) = $1.12 / 0.78 = 1.44$.

NIIs for all other risk factors were calculated similarly. The obtained NIIs are the initial standard with which it is possible to give an integrated assessment of the risk of ATSD occurrence both for a separate factor and for their complex.

As you know, factors have different strengths of influence on the development of ATSD. Therefore, we took into account the value of the relative risk indicator for each factor. Knowing the indicator of the relative risk (R) of the onset of the disease and the normalized intensive indicator (N), it is possible to determine the strength of the influence on the occurrence of ATSD for each factor, i.e. predictive coefficient (X).

This value is defined as follows: $X = R \cdot N$, where X is an integrated indicator of risk from the strength of the influence of an individual factor (predictive coefficient); N - NII ATSD; R is an indicator of relative risk.

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If we take into account that in our example the indicator of relative risk (R) was 1.44, NII1 - 0.78, NII2 - 1.12, then the integrated indicator of the power of influence of each factor, i.e. Predictive coefficient was:

1.44 · 0.78 = 1.12, the minimum value;

1.44 1.12 = 1.61, the maximum value.

Below is a prognostic matrix based on the history and clinical symptoms (Table 1).

The predictive matrix includes all the factors selected for forecasting with their gradation and values of the integrated risk indicator from the strength of the influence of an individual factor (X), the relative risk indicator for each factor (R), and their sum for a complex of factors (RN), as well as the normalizing value - average an indicator of the frequency of ATSD according to the data of the entire study (N).

Table 1

Predictive matrix for a comprehensive risk assessment RPS occurrence

Risk factors	Gradation of factors	%	NII	R	X		
						min	max
Chr. tonsillitis	yes	55,6	1,03	1,07	1,10	0,93	1,07
	no	37,5	0,97		0,93		
Aller. diathesis	yes	64,8	1,47	3,11	4,57	1,47	4,57
	no	20,8	0,47		1,47		
Adenomas	yes	37,0	1,45	2,96	4,31	1,45	4,31
	no	12,5	0,49		1,45		
Atopic. dermatitis	yes	66,7	1,39	2,46	3,42	1,39	3,42
	no	27,1	0,56		1,39		
Anemia	yes	46,3	1,21	1,59	1,92	1,21	1,92
	no	29,2	0,76		1,21		
Obesity	yes	37,0	1,51	3,56	5,37	1,51	5,37
	no	10,4	0,43		1,51		
NRED	yes	25,9	1,14	1,38	1,59	1,14	1,59
	no	18,8	0,83		1,14		
Thymomeglia	yes	14,8	1,26	1,78	2,24	1,26	2,24
	no	8,3	0,71		1,26		
Allergic. rhinitis	yes	64,8	1,44	2,83	4,06	1,44	4,06
	no	22,9	0,51		1,77		
Aller. rhinosinusitis	yes	53,7	1,40	2,58	3,62	1,40	3,62
	no	20,8	0,54		1,40		
	yes	7,4	1,26	1,78	2,24	1,26	2,24

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Laryngo-tracheitis	no	4,2	0,71		1,26		
Winter-spring	yes	16,7	1,21	1,60	1,94	1,21	1,94
	no	10,4	0,76		1,21		
Autumn-winter	yes	22,2	1,19	1,52	1,82	1,19	1,82
	no	14,6	0,78		1,19		
Free thyroxine	>27,2	85,2	1,70	5,75	9,80	1,70	9,80
	<27,2	14,8	0,30		1,70		
Thyroxine	<91,0	70,4	1,41	2,38	3,34	1,41	3,34
	>91,0	29,6	0,59		1,41		
Triiodothyronine	<1,60	77,8	1,56	3,50	5,44	1,56	5,44
	>1,60	22,2	0,44		1,56		
TSH	<3,80	85,2	1,70	5,75	9,80	1,70	9,80
	>3,80	14,8	0,30		1,70		

In addition to the prognostic table, we determined the possible range of risk values for the complex of factors taken. The determination of the possible risk range was carried out as follows.

In the prognostic table, we find the minimum values of the prognostic coefficient (X) for each factor and sum them up. This value is the initial value of the risk of this pathology.

So, for example, in table 1 for an integrated assessment of the risk of ATSD, the minimum values of the prognostic indices (X) for all factors were as follows:

0.93 + 1.42 + 1.45 + 1.39 + 1.21 + 1.51 + 1.14 + 1.26 + 1.44 + 1.40 + 1.26 + 1.21 + 1, 19 + 1.70 + 1.41 + 1.56 + 1.70 = 23.23

In this case, the minimum initial risk value is 23.23.

Then, similarly, we find the sum of the maximum values of the prognostic indices for each factor.

1.07 + 4.57 + 4.31 + 3.42 + 1.92 + 5.37 + 1.59 + 2.24 + 4.06 + 3.62 + 2.24 + 1.94 + 1, 82+ 9.80 + 3.34 + 5.44 + 9.80 = 66.55

In this case, the risk range is 23.23–66.55.

Hence it follows that the higher the value of the standard integrated indicator of the risk of ATSD occurrence as a result of the influence of the complex of the studied factors, the higher the likelihood of the risk of developing ATSD in a given person and the more grounds for allocating him to the group of unfavorable prognosis.

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In this regard, we have identified a possible risk range (23.23-66.55), as well as sub-ranges. In practice, it is better to divide the entire risk range into three intervals: weak (7.74-22.18), medium (22.19-44.36), and high (44.37-66.55) likelihood of risk of developing ATSD.

Table 2

Values of sub-ranges and groups of individual risk predictions emergence of ATSD

Sub-range	Sub-range size	Risk group
Weak probability	7,74-22,18	Favorable forecast
Average probability	22,19-44,36	Attention
High probability	44,37-66,55	Poor prognosis

Thus, the threshold values of the final prognostic coefficients and the risk groups for the onset of pathology were determined (Table 2).

To illustrate, let us give an example of determining the individual risk of developing ATSD with different nature of the forecast.

Individual prediction can be used in hospitals for treatment while obtaining a reasonable prognosis of the course of the disease and anticipating possible complications. Also, with the help of individual prediction, it is possible to develop a targeted scheme for the prevention of patients with broncho-pulmonary diseases.

Conclusion

Thus, changes in the hormonal system in BA patients are in a state of defensive adaptation with rapid depletion of these processes in severe BA, which is accompanied by hormonal imbalance.

The clinical manifestations of thyroid hypofunction are diverse, many of them are nonspecific, which is associated with certain difficulties in its timely recognition. In this regard, we have attempted to identify specific complaints of BA patients regarding hypofunction of the thyroid gland.

Acknowledgment

Thus, the majority of BA patients during the period of exacerbation of the disease are characterized by activation of the sympathetic division of the ANS, tension, and in severe cases, depletion of the compensatory capabilities of the autonomic nervous system. The data obtained from the analysis of heart rate variability can be used not only for the individual selection and control of bronchodilator therapy but also for the timely appointment of vegetative-correcting and cardiovascular agents.

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