6. Khan BZ, Sheth M. Physical activity level and physical fitness parameters in physiotherapy students. Int J Physiother Res. 2019;7(5):3247-51. doi: 10.16965/ijpr.2019.177.
7. Kochman M, Kasperek W, Guzik A, Drużbicki M. Body composition and physical fitness: Does this relationship change in 4 years in young adults? Int J Environ Res Public Health. 2022;19(3):1579. doi: 10.3390/ijerph19031579.
8. Kutseryb T, Hrynkiv M, Vovkanych L, Muzyka F, Melnyk V. Anthropometric characteristics and body composition of female students involved in volleyball training. Anthropol Rev. 2022;85(4):31-42. doi: 10.18778/1898-6773.85.4.03.
9. Lan C, Liu Y, Wang Y. Effects of different exercise programs on cardiorespiratory fitness and body composition in college students. J Exerc Sci Fit. 2022;20(1):62-9. doi: 10.1016/j.jesf.2021.12.004.
10. Mahajan R, Rawat D. Determination of physical fitness index and its relation with body mass index among physiotherapy students. Physiotherapy. 2020;14(2):84. doi: 10.4103/PJIAP.PJIAP_25_19.
11. Murphy C, Takahashi S, Bovaird J, Koehler K. Relation of aerobic fitness, eating behavior, and physical activity to body composition in college-age women: A path analysis. J Am Coll Health. 2021;69(1):30-7. doi: 10.1080/07448481.2019.1647210.
12. Pan M, Ying B, Lai Y, Kuan G. Status and influencing factors of physical exercise among college students in China: A systematic review. Int J Environ Res Public Health. 2022;19(20):13465. doi: 10.3390/ijerph192013465.
13. Popławska H, Dmitruk A, Hołub W. Body composition, physical fitness and physical activity among students from universities in Biała Podlaska. Pol J Sport Tourism. 2020;27(1):21-7. doi: 10.2478/pjst-2020-0004.
14. WHO European health information at your fingertips. European Health Information Gateway. 2018. Available from: https://gateway.euro.who.int/en/indicators/mn_survey_19-cut-off-for-bmi-according-to-who-standards/#id=32083.
15. Yarizadeh H, Eftekhar R, Anjom-Shoae J, Speakman JR, Djafarian K. The effect of aerobic and resistance training and combined exercise modalities on subcutaneous abdominal fat: A systematic review and meta-analysis of randomized clinical trials. Adv Nutr. 2021;12(1):179-96. doi: 10.1093/advances/nmaa090.

Стаття надійшла 15.06.2024 р.

DOI 10.26724/2079-8334-2025-2-92-44-48
UDC 616–002.77:616.981.420.706

N.S. Hasanova, F.M. Mammadova, S.M. Mammadov, L.A. Hidayatova
Azerbaijan Medical University, Baku, Azerbaijan

ASSOCIATION BETWEEN CLINICAL MANIFESTATIONS AND MUTATIONAL VARIANTS OF THE MEFV GENE IN CHILDREN

e-mail: mic_amu@mail.ru

The purpose of the study was to assess the clinical characteristics of Familial Mediterranean fever, depending on the variants of genotypic mutations. In total 11 patients with Familial Mediterranean fever aged from 1 to 18 years (mean age 7.4±3.5 years) were involved in the study. The examination included physical, laboratory and instrumental methods. Of the 11 patients with a mutation in the MEFV gene, 55 % had the homozygous M694V variant, 20 % had the homozygous R202Q variant, 15 % had the heterozygous pV726A variant, and 10 % had a compound homozygous R202Q and M694V variant. Patients with the M694V mutation had more severe clinical symptoms (periodic fever, polyserositis), earlier disease onset, and a higher risk of amyloidosis. In the combined variant, 2 boys had phenotypic clinical features of type M694V, as well as elevated paraclinical inflammatory markers and colchicine resistance. Children with heterozygous V726A mutation had abdominal pain, limited in duration fever, and decreased attacks frequency.

Key words: familial Mediterranean fever, MEFV gene, amyloidosis, mutation, children.

Н.С. Гасанова, Ф.М. Мамедова, С.М. Мамедов, Л.А. Гідаятова

АСОЦІАЦІЯ КЛІНІЧНИХ ПРОЯВІВ ІЗ РІЗНИМИ ВАРІАНТАМИ МУТАЦІЙ ГЕНА MEFV У ДІТЕЙ

Метою дослідження була оцінка клінічних характеристик сімейної середземноморської лихоманки залежно від варіантів генотипових мутацій. Загалом у дослідженні взяли участь 11 пацієнтів із сімейною середземноморською лихоманкою віком від 1 до 18 років (середній вік 7,4±3,5 років). Обстеження включало фізикальні, лабораторні та інструментальні методи. З 11 пацієнтів із мутацією в гені MEFV у 55 % був гомозиготний варіант M694V, у 20 % – гомозиготний варіант R202Q, у 15 % – гетерозиготний варіант pV726A і в 10 % – компаунтний гомозиготний варіант R202Q і M694V. У пацієнтів з мутацією M694V спостерігалися більш виражені клінічні симптоми (періодична лихоманка, полісерозит), ранній початок захворювання і вищий ризик розвитку амілоїдозу. При комбінованому варіанті у 2 хлопчиків спостерігалися фенотипічні клінічні ознаки, характерні для M694V, а також підвищені запальні маркери і резистентність до колхіцину. У дітей із гетерозиготною мутацією V726A відзначалися болі в животі, обмежена за тривалістю лихоманка і зниження частоти нападів.

Ключові слова: сімейна середземноморська лихоманка, ген MEFV, амілоїдоз, мутація, діти.

Familial Mediterranean fever (FMF) is an autosomal recessive disease that was previously rare and was observed in representatives of certain ethnic groups (Jews, Greeks, Armenians, Turks, Arabs), but the disease became widespread due to intercontinental travel in the twentieth century. FMF is characterized by recurrent self-limiting fever, peritonitis, arthritis, and erysipelas-like-erythema [1, 3, 9].

The MEFV gene consists of 10 exons and is located on chromosome 16p13.3. In different populations, MEFV gene mutations occur with different frequencies: among Turks, as well as Ashkenazi

Jews, the frequency of carriage of MEFV gene mutations is 1:5; among Jews living in North Africa, it varies from 1:5 to 1:10. In one of the last researches, it was reported that an average prevalence of FMF in Turkey is approximately 1 in 1,000, though there is considerable interregional variability [6, 11].

To date, more than 400 variants of MEFV gene have been identified (which are missense mutations) and documented in the INFEVERS database. It should be noted that the most of the mutations are located in exon 10 (M694V, M680I, V726A, M694I, A744S, K654R, R761H, T681I, I692del, M694del) [10].

Results of several studies implied that the presence of four mutations within exon 10 (M694V, V726A, M680I, M694I) and a mutation within exon 2 (E148Q) correspond to 85 % of FMF causing mutations in the Mediterranean ethnic groups [2, 6].

The severity of symptoms varies individually, even among members of the same family. Amyloidosis leading to kidney failure is the most severe complication. The main treatment method preventing this complication is the use of colchicine. In case of ineffectiveness/intolerance to colchicine, other drugs can be used, including genetically engineered biological drugs (IL-1β inhibitors, etc.) [4, 8].

Different clinical courses can be caused by different mutations. The most common mutation is M694V, which in homozygous and compound heterozygous states leads to the development of the most severe symptoms of the disease and earlier manifestation and is most often accompanied by the development of renal amyloidosis; a poor response to colchicine is also noted [7].

In the homozygous state, the most common mutations are V726A, M680I and R761H. There is a 90 % correlation between the clinical symptoms in patients heterozygous for these four mutations compared to homozygotes. A relatively mild phenotype is observed with the E148Q and V726A mutations [5].

Patients with periodic disease, homozygous for the E148Q mutation of the MEFV gene, usually have a more heterogeneous clinical picture and definitely require treatment with colchicine. In addition, there are homozygotes and compound heterozygotes for E148Q with asymptomatic carriage. However, the M694V/E148Q variant is an exception. It is assumed that mutations P369S, M694I, F479L and R42W in heterozygous and compound heterozygous states in asymptomatic carriers are characterized by a mild phenotype and have low penetrance [4, 5].

In different populations, mutations of the MEFV gene occur with different frequencies. In the overwhelming majority of populations, the M694V mutation is in first place in terms of frequency of occurrence (from 19.5 % in Arabs to 76.8 % in non-Ashkenazi Jews). However, in the population of Turks living in the Turkish province of Hatay, this mutation is registered only in 7.95 % of cases, the most frequently registered substitution is R202Q – with a frequency of 21.35 %. This substitution is registered with almost the same frequency in Greeks – 21.4 %. In the heterozygous state, the R202Q substitution occurs in patients with periodic disease and in healthy people with almost the same frequency - 31.6 % and 33.6 %, respectively; in the homozygous state – in patients with periodic disease with a frequency of 9.2 %, in healthy people – 0.7 %. Thus, it is assumed that the R202Q substitution in the homozygous state is associated with FMF [2, 11].

The lack of specific laboratory and instrumental research methods for FMF, characterized by polymorphism of clinical symptoms, complicates early diagnosis of the disease, leads to unjustified polypharmacotherapy and even surgical interventions.

The purpose of the study was to assess the clinical characteristics of Familial Mediterranean fever, depending on the variants of genotypic mutations.

Materials and methods. The study was conducted on the basis of Department of Children's Diseases II of Azerbaijan Medical University in the period of 2022–2024.

In total, 11 patients with Familial Mediterranean fever aged from 1 to 18 years (mean age 7.4±3.5 years) were involved in the study.

The general examination of patients (including physical, laboratory and instrumental methods) was performed. The diagnosis was based on the Tel Hashomer clinical diagnostic criteria and exclusion of other pathologies. The Tel Hashomer criteria are the primary clinical tool for diagnosing Familial Mediterranean fever. They are based on a combination of clinical features and response to colchicine. The major criteria are typical attacks (fever and serositis); response to colchicine, amyloidosis without predisposing disease. The minor criteria include recurrent febrile episodes, erysipelas-like erythema, family history of FMF. Familial Mediterranean fever diagnosis criteria require 2 major or 1 major+2 minor criteria [8].

In the framework of laboratory examination complete blood count, general analysis of urine and feces, bacteriological analysis (in necessary cases). Additionally, the serum levels of C-reactive protein and amyloid were determined and the tests, related to suspicions in rheumatological pathologies, (anti-streptolysin–O, rheumatoid factor, antinuclear antibodies, anti-ds-DNA, anti-ss-DNA, anti-cyclic citrullinated peptide antibody etc.) were measured (if necessary).

To confirm the diagnosis and determine the variants of molecular genetic analysis using Sanger sequencing on ABI 3500 (Thermo Fisher) was carried out.

Statistical processing included the calculating mean value (M), its standard error (m) and the variation interval (min-max). The absolute numbers and its share (%) of the occurrence of quality signs were also determined. Statistical processing of the results of the study was carried out using the statistical computer program Statistica 6.0 from StatSoft (USA).

Results of the study and their discussion. Of the 11 patients with a mutation in the MEFV gene, 55 % had the homozygous M694V variant, 20 % had the homozygous R202Q variant, 15 % had the heterozygous V726A variant, and 10 % had a compound homozygous R202Q and M694V variant.

Patients with the M694V mutation had more severe clinical symptoms (periodic fever, polyserositis), earlier disease onset, and a higher risk of amyloidosis.

In the combined variant, 2 boys had phenotypic clinical features of type M694V, as well as elevated paraclinical inflammatory markers and colchicine resistance. 81 % of children in the study group were boys, and the mean time to diagnosis was 5.2±3.4 years.

Children with heterozygous V726A mutation had abdominal pain, limited in duration fever, and decreased attacks frequency.

In one of our patients, aged 5 years old, the main symptoms were high fever (40°C) and abdominal pain. It was be found out from anamnesis that he was first diagnosed with FMF when he was 6 months old. The patient took colchicine for some time and then stopped. During this period, the symptoms of the disease returned, and the patient subsequently returned to the doctor. Genetic analysis confirmed the diagnosis of FMF in this child (heterozygous variant R202Q). During the observation of the patient, resistance to colchicine was detected, which required increase the dosage of the drug as a precaution. In addition, amiloid test showed high serum level (123 μg/ml), which is one of the unfavorable signs for prognosis due to renal affecting. In this case we revealed the assitiation between R202Q variant of MEFV gene mutation and typical attacks (fever and serositis, amyloidosis without predisposing disease. But in our patient response to colchicine was weak which needed to increase the dose.

Another patient who is currently 7 years old, was referred to the clinic for typical symptoms of acute renal failure – high temperature, oedema, vomiting and nausea, paroxysmal abdominal pain. After exclusion of other disorders which can caused renal insufficiency, the genetic examination was performed (compound R202Q+M694V homozygotes mutation of the MEFV gene (c605G>A and c2080A>G, located on exon 2 and exon 10, respectively). C-reactive protein level was 78.9 mq/l and the amyloid level was 141 μg/ml. Taking the colchicine gives a positive result for the patient. So, this patient demonstrated severe course of FMF with renal damage, but had sensitivity to colchicine.

Comparing our results to the data of literature we revealed some works which supports the similar findings. Çapraz M, et al, (2023), studying genotypes of FMF patients from Amasya (Turkey) found out that the R202Q genotype is compatible with the known FMF clinic and has highest prevalence (the most frequent genotype was R202Q (960, 43.5 %) followed by M694V (n=412, 18.7 %), E148Q (n=321, 14.6 %), and M680I (n=200, 9.1 %)). In their work the most common clinical symptoms were abdominal pain (96.4 %) and fever (91.3 %). The researchers noted the need of functional studies of the R202Q variant pyrin protein to understand FMF better. In our study R202Q variant was also characterized by typical clinical symptoms and course [6].

Malik A, et al (2021) presented a case of a 23-year-old man of Iranian descent with history of periodic fever, diagnosed as Stanford type A aortic dissection secondary to an acute attack of FMF. This was the first case reported of such a complication, because FMF usually involves small- and medium-sized arteries. In should be noted that there were not cases with signs of damage to large arteries and complications related to them among children involved in our study. However, patients and doctors must be aware of such symptoms [10].

As mentioned above, the clinical picture is often determined by genetic variations, which may have different distribution depending on the ethnic group and territory.

With the purpose to determine the frequency of M694V, M680I and V726A mutations of the MEFV gene in Azeri Turkish patients with family Mediterranean fever Bagheri M, et al. (2017) observed 630 patients of this ethnic group using allele-specific oligonucleotide polymerase chain reaction. The authors revealed that the M694V mutation is the most common risk factor for family Mediterranean fever in this group. This finding is similar with our: in 55 % of patients M694V mutation was found out, that is logical due to fact that we studied the same ethnic group [2].

Darwish WM, et al, (2021) reported about case of a three-and-a-half-year-old girl with a 3-year history of pain crises consisting of severe musculoskeletal pain, particularly knee pain, and recurrent pain crises consisting of sudden, severe, generalized abdominal pain and fever lasting up to 72 h. The patient started experiencing these attacks at the age of 6 months (like in one of our cases), initially occurring every

1–2 months and lasting 16–24 h, and have increased in frequency and progressed to weekly episodes lasting up to 72 h. Results revealed two pathogenic missense variants identified in the MEFV gene in a heterozygous state: c.2080A>G (p.Met694Val) and c.2040G>A (p.Met680Ile). The patient experienced weak response and recurrent attack, which lead to need of increasing the dose to get effect [7]. This condition was very similar with our cases, reported above.

El Gazzane S, et al (2024) presented very interesting report of pediatric familial Mediterranean fever with heterozygous mutation complicated by AA amyloidosis leading to renal failure. In their report the genetic analysis showed the presence of the MEFV mutation (NM_000243):c.2177T>C (p.Val726Ala) in a heterozygous state in exon 10 of the MEFV gene. The patient was treated by daily subcutaneous injections of recombinant IL-1 receptor antagonist. But the evolution was marked by a decline in renal function at 3 months of follow-up, which lead to placing the patient on automated peritoneal dialysis. The authors noted that V726A is related to a lower prevalence of amyloidosis because it may play a protective role against the development of the complication [8]. Indeed, in our study children from our observation with heterozygous V726A mutation had abdominal pain, limited fever, and decreased attacks frequency and well response on colchicine, but have no signs of renal failure.

Very close to our work was the study of Bekis Bozkurt H, et al (2023), who assessed the relationship between clinical manifestation and mutational variations of MEFV gene. They concluded that M694V mutation and colchicine resistance were two important risk factors for renal insufficiency (6.9 %) and amyloidosis (1.2 %) in FMF patients. The authors noted that it should be kept in mind that compound heterozygous with M694V mutations may be associated with chest pain and R202Q mutation may be negatively correlated with arthritis, unlike M694V. As a conclusion, researchers recommended to evaluate the genetic results and clinical findings of the patients together and followed up closely [4]. That is coincides with our position.

According to Iuhas A, et al (2025), several triggers for FMF attacks have been identified, including stress, cold impact, infections, hormonal changes, such as menstrual cycle in pubertal and post-pubertal females. Approximately 50 % of FMF patients experience a prodrome (malaise, discomfort, fatigue etc.) [9]. In cases of our study, the majority of onsets occurred on the background or in period after acute phase of infections, and the target treatment for the infection was not effective or only partially improved clinical condition.

Conclusion

The most prevalent mutation of patients of this study was homozygous M694V variant (55 %), which was associated with early amyloidosis. Closer monitoring of general clinical signs in children by physicians, molecular genetic testing when necessary, and timely and adequate therapy can prevent amyloidosis, which can be fatal. Colchicine in correct individual doses is effective in early diagnosis of FMF to prevent attacks and the progression of amyloidosis. Further studies are needed to explore new and rare mutations to establish an accurate diagnosis at a very young age, especially in heterozygotes, to identify risk factors associated with the development of amyloidosis and therefore to provide an adequate therapeutic approach.

References

1. Alamlih L, Al-Karaja L, Alayaseh M, Abunejma F, Al-Zeer Z, Sultan B. Familial Mediterranean fever with pseudo-septic arthritis: A case report and review of the literature. Mod Rheumatol Case Rep. 2023 Jan 3;7(1):252-256. doi: 10.1093/mrcr/rxac040.

2. Bagheri M, Rad IA. Analysis of the Most Common Three MEFV Mutations in 630 Patients with Familial Mediterranean Fever in Iranian Azeri Turkish Population. Maedica (Bucur). 2017 Sep;12(3):169-173.

3. Barut K, Sahin S, Adrovic A, Sinoplu AB, Yucel G, Pamuk G, et al. Familial Mediterranean fever in childhood: a single-center experience. Rheumatol Int. 2018 Jan;38(1):67-74. doi: 10.1007/s00296-017-3796-0.

4. Bekis Bozkurt H, Yıldırım S, Ergüven M. Renal involvement, presence of amyloidosis, and genotype-phenotype relationship in pediatric patients with Familial Mediterranean fever: a single center study. Eur J Pediatr. 2023 Apr;182(4):1911-1919. doi: 10.1007/s00431-023-04855-y.

5. Beshlawy AE, Zekri AER, Ramadan MS, Selim YMM, Abdel-Salam A, Hegazy MT, et al. Genotype-phenotype associations in familial Mediterranean fever: a study of 500 Egyptian pediatric patients. Clin Rheumatol. 2022 May;41(5):1511-1521. doi: 10.1007/s10067-021-06006-w.

6. Çapraz M, Düz ME. R202Q prevalence in clinically diagnosed Familial Mediterranean Fever patients: 9 years of data analysis from 1570 patients living Central Black Sea region, Turkey. Ir J Med Sci. 2023 Oct;192(5):2273-2278. doi: 10.1007/s11845-022-03233-1.

7. Darwish WM, Darwish SB, Darwish MB, Darwish BF. M680I/M694V Heterozygous Mutation in Early Onset Familial Mediterranean Fever. J Med Cases. 2021 Sep;12(9):351-354. doi: 10.14740/jmc3747.

8. El Gazzane S, Ichane A, Nahi C, Mouaddine K, Chkirate B, Guennoun A, et al. Heterozygous MEFV Mutation Leading to Renal Failure: A Case Study. Glob Pediatr Health. 2024 Aug 28;11:2333794X241274752. doi: 10.1177/2333794X241274752.

9. Iuhas A, Marinău C, Niulaș L, Futaki Z, Balmoș A, Kozma K, et al. Familial Mediterranean fever in Romania: a case report and literature review. Front Pediatr. 2025 Jan 15;12:1546387. doi: 10.3389/fped.2024.1546387.
10. Malik A, Malik J, Javaid M, Khan HS, Mohsin M, Shoaib M. A case of aortic dissection in familial Mediterranean fever. J Cardiol Cases. 2021 Jun 5;24(6):296-299. doi: 10.1016/j.jccase.2021.05.005.
11. Öztürk K, Coşkuner T, Baglan E, Sönmez HE, Yener GO, Çakmak F, et al. Real-Life Data From the Largest Pediatric Familial Mediterranean Fever Cohort. Front Pediatr. 2022 Jan 20;9:805919. doi: 10.3389/fped.2021.805919.

Стаття надійшла 21.05.2024 р.

DOI 10.26724/2079-8334-2025-2-92-48-52
UDC 616.923-053.2

O.I. Hladchenko, I.V. Dybas, A.B. Nadraga
Danylo Halytskyi Lviv National Medical University, Lviv

EVOLUTION OF CLINICAL MANIFESTATIONS OF WHOOPING COUGH IN CHILDREN IN THE POST-COVID-19 PANDEMIC PERIOD

e-mail: nadraga09@gmail.com

In recent years, an increase in the number of cases of pertussis has been recorded in Europe and Ukraine. The purpose of the study was to find out the features of pertussis in children in the first three years of life after the COVID-19 pandemic and compare them with clinical and laboratory data of similar patients who had pertussis before the pandemic COVID-19. A retrospective analysis of 97 patient charts of inpatients aged 0-36 months diagnosed with pertussis who were treated in the Lviv Regional Clinical Infectious Diseases Hospital in 2017–2019 and 2023–2024 was conducted. It was found that the course of the disease in the post-epidemic cohort was milder, with a higher incidence of respiratory complications; a number of differences in clinical manifestations, complete blood counts in children of different age groups, and in the period before and after the SARS-CoV-2 pandemic were identified. After the end of the COVID-19 pandemic, the weakening of population immunity, genetic mutations of Bordetella pertussis and the possible impact of SARS-CoV-2 infection on the immune response contributed to an increase in children's susceptibility to pertussis and changes in clinical manifestations of the disease.

Key words: pertussis, children, COVID-19, course of disease, immune response.

О.І. Гладченко, І.В. Дибас, О.Б. Надрага

ЕВОЛЮЦІЯ КЛІНІЧНИХ ПРОЯВІВ КАШЛЮКУ У ДІТЕЙ У ПЕРІОД ПІСЛЯ ПАНДЕМІЇ COVID-19

В Європі й Україні у останні роки реєструють зростання захворюваності на кашлюк. Метою дослідження було з'ясувати особливості перебігу кашлюку у дітей перших трьох років життя у період після пандемії COVID-19 та порівняти їх з клінічними та лабораторними даними аналогічних пацієнтів які хворіли на кашлюк до початку пандемії. Проведено ретроспективний аналіз 97 карт стаціонарного хворого віком 0-36 міс з діагнозом кашлюк, які перебували на стаціонарному лікуванні у Львівській обласній клінічній інфекційній лікарні в періоди 2017–2019 рр. і 2023–2024 рр. Встановлено, що перебіг захворювання у когорті дітей у період після епідемії був легшим, водночас з вищою частотою респіраторних ускладнень; виявлено низку відмінностей в клінічних проявах, показниках загального аналізу крові у дітей різних вікових груп, та у період до та після виникнення пандемії SARS-CoV-2. Після завершення пандемії COVID-19 ослаблення популяційного імунітету, генетичні мутації Bordetella pertussis та можливий вплив перенесеної SARS-CoV-2-інфекції на імунну відповідь сприяли зростанню сприйнятливості дітей до кашлюку та зміні клінічних проявів захворювання.

Ключові слова: кашлюк, діти, COVID-19, перебіг хвороби, імунна відповідь.

The work is a fragment of the research project "Clinical features of infectious diseases in children and current diagnostic and treatment methods during different stages of the SARS-CoV-2 epidemic", state registration No. 0124U000805.

In European countries, over the past decade, there has been a cyclical increase in cases of whooping cough in children every 3-4 years, with annual peaks in regions with a temperate climate occurring between July and September. However, since April 2020, the number of pertussis cases has fallen sharply, reaching its lowest level since the beginning of the century. In the UK, for example, the incidence of pertussis in infants in 2020 was 0.50 per 100.000 population, which is significantly lower than in 2014 (24.6 per 100.000) [10]. However, in the post-epidemic period, the incidence of this disease began to rise rapidly and in a number of countries significantly exceeded the figures before the start of the COVID-19 epidemic [1]. In Ukraine, according to the Public Health Center, there has also been a significant increase in the incidence of whooping cough. In the first half of 2024 y. almost 4.900 cases of the disease were registered. Despite the availability of effective vaccines, whooping cough remains a pressing problem. This is due to a number of factors, such as reduced immunity, the emergence of new strains of Bordetella pertussis bacteria, insufficient vaccination, and underestimation of the danger of the disease.