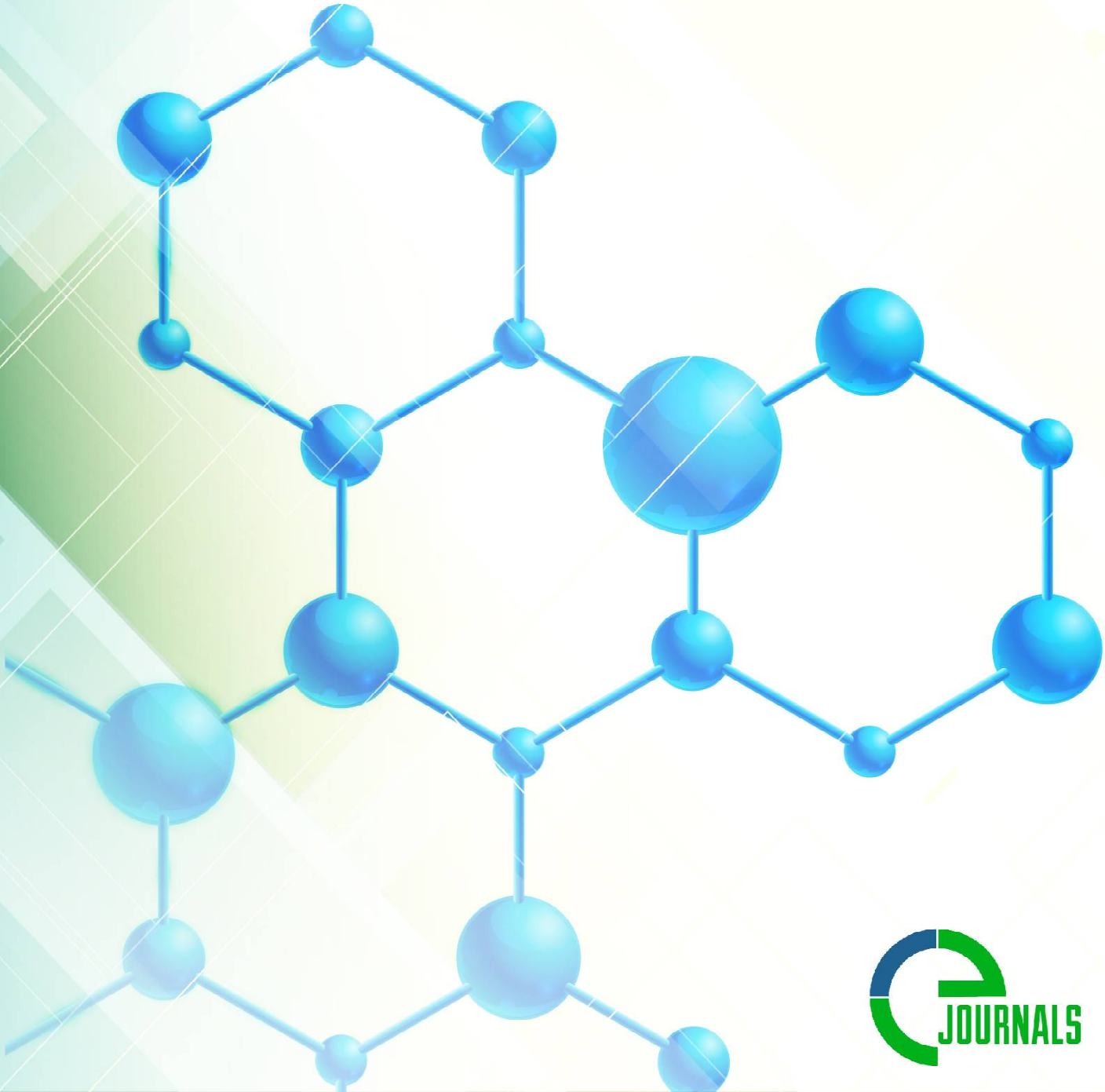
EUROPEAN JOURNAL OF

MOLECULAR MEDICINE



JOURNALS

European Journal of Molecular medicine

Volume 1, No.4, September 2021

Internet address: ttp://ejournals.id/index.php/EJMM/issue/archive
E-mail: info@ejournals.id
Published by ejournals PVT LTD
DOI prefix: 10.52325
Issued Bimonthly
Potsdamer Straße 170, 10784 Berlin, Germany

Requirements for the authors.

The manuscript authors must provide reliable results of the work done, as well as anobjective judgment on the significance of the study. The data underlying the work shouldbe presented accurately, without errors. The work should contain enough details andbibliographic references for possible reproduction. False or knowingly erroneous statementsare perceived as unethical behavior and unacceptable.

Authors should make sure that the original work is submitted and, if other authors'works or claims are used, provide appropriate bibliographic references or citations. Plagiarismcan exist in many forms - from representing someone else's work as copyright to copying orparaphrasing significant parts of another's work without attribution, as well as claimingone's rights to the results of another's research. Plagiarism in all forms constitutes unethicalacts and is unacceptable. Responsibility for plagiarism is entirely on the shoulders of theauthors.

Significant errors in published works. If the author detects significant errors or inaccuraciesin the publication, the author must inform the editor of the journal or the publisher aboutthis and interact with them in order to remove the publication as soon as possible or correcterrors. If the editor or publisher has received information from a third party that thepublication contains significant errors, the author must withdraw the work or correct theerrors as soon as possible.

CHIEF EDITOR

EDITORIAL BOARD

EPIDEMIOLOGY OF PARVOVIRUS B19 INFECTION, CLINICAL MANIFESTATIONS AND PATHOGENETIC ASPECTS IN CHILDREN

Rasulova M.B., Kasimova R.I., Musabaev E.I.
Republican Scientific Research Institute
of Virology of the Republican Specialized
Scientific-Practical Medical Center of Epidemiology,
Microbiology of Infectious and Parasitic Diseases

Abstract.Parvovirus B19 is a common and highly contagious infectious disease, and the disease due to facial rash is referred to in some literatures as "infectious erythema", "the fifth disease". In children, the disease is mild and often does not require treatment. In patients with hemolytic pathology (hereditary spherocytosis, sickle cell anemia), immunodeficiency (congenital immunodeficiency), receiving immunosuppressive therapy, in HIV infection Parvovirus B19 causes aplastic crisis due to high erythrocyte aplasia, chronic aplastic crisis. B19 infection is considered dangerous for patients with hemolytic pathology.

Key words: Parvovirus B19, children, antibodies, clinical manifestations, pathogenesis

Parvovirus B19 is a common and highly contagious infectious disease, and the disease due to facial rash is referred to in some literature as Infectious Erythema, the fifth disease. [54.61]. In 1905, the Russian-French physician Leon Sheinis coined the term 5 disease [36,53,37]. Among children, rash is the fifth most common infectious disease, hence the name. Robert Villan was the first to call the disease "rubeola, sine catarrho" in 1799. In children, the disease is mild and often does not require treatment. In patients with hemolytic pathology (hereditary spherocytosis, sickle cell anemia), immunodeficiency (congenital immunodeficiency), receiving immunosuppressive therapy, in HIV infection (without ARVT) Parvovirus B19 causes aplastic crisis, chronic aplastic crisis due to high erythrocyte aplasia. B19 infection is considered dangerous for patients with hemolytic pathology [41]. To date, there have been few trials of B19 infection among HIV-infected children, and B19-associated anemia has been less common in HIV-infected patients receiving ARVT at the same time.

History of the discovery of the virus: In 1974, Cossart and his colleagues isolated the B19 virus in a HBV surface antigen test. The size of the electron microscope is 23 nm [3, 11]. The word "care" is derived from the Latin word "SMALL virus". Reminiscent of the parvovirus found in animals, this parvovirus is exclusively pathogenic to humans and was named parvovirus B19 in 1985 by the International Committee of Virus Taxonomy, also known as erythrovirus, and is considered to belong to the PARVOVIRIDAE family. [6]. Synonyms of the virus: Parvovirus B19, B19 virus, Erythrovirus B19 [7].

Taxonomy: A small virus that contains DNA, without a shell, consisting of 2 proteins and 1 stranded DNA molecule [14].The virus is resistant to lipid shell load and genome stability, the virion consists of 60 capsomers, the virus is highly resistant to organic solutions, chloroform, ether, alcohol, resistant to 60 minutes at 56 degrees and lipid solutions [23]. It is inactivated by formalin, boiling, and gamma radiation [35]. The main proteins of the virus: NS1 oxyl without basic structure and 2 structural VP1 and VP2 oxides [12, 17]. The basic structural protein VP2 accounts for 96% of the total protein, while VP1 accounts for 4% of the total protein [18]. The unstructured protein controls NS1 DNA-binding protein cell cytolysis, NS2 cell replication.

Cytopathology: Giant pronormablasts were first identified in the bone marrow of a

1948 transient aplastic crisis patient [51]. Giant pronormablasts are erythroid cells with a diameter of 25-32 microns, similar to cytoplasmic vacuolation, "dog's ear" (dog ears) (Fig. 1). [4, 10, 9, 19].

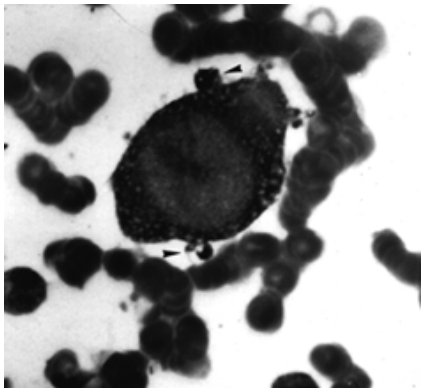


Figure 1 Giant pronormoblast. The arrows indicate the projection of the dog's ears.

Epidemiology: The B19 virus has spread to all countries of the world [49]

The last epidemic was in 1998. [39] The disease is observed sporadically throughout the year, the disease occurs in late winter, spring and early summer, an epidemic is observed every 3-5 years, characterized by cyclical fluctuations [27, 42].

Code- B08.3 Anthroponotic disease. There are currently 3 genotypes, the dominant being 1- genotype [47].

Distribution of IgG among the population: [45, 34, 46, 60]

Age	Indicator %
1-5	2-15
6-19	15-60
adults	60% and above

Research has been conducted by Kelly, H. A, and others to monitor age-related immunity to B19 infection in several hospitals in Victoria, Australia. Of the 824 serums in children aged 0-9 years, antibody to B19 increased by 28%, over the next 10 years by 51%, and by adults over 50 years of age by 78%. in the annual cycle 2 epidemics occur after 2 endemic years. B19 virus is more prevalent in countries with temperate climates, and collective immunity is high.

At the Red Cross Mining Center in Fukuoka, Tsujimura, M. K., and others tested the mine for B19 using an immunodiffusion method submitted by donors between June 1991 and August 1994. During a 3-year trial period, 16 of 560,000 donors were diagnosed with the B19 virus. Interestingly, the majority of virus donors, 16 to 15, were found between February 1992 and January 1993, leading to an epidemic of infectious erythema in Fukuoka (December 1991 to August 1992). In March 1992, during the outbreak of infectious erythema, 4 cases were identified, during which the incidence of B19 was about 1/4000. Viremic patients ranged in age from 17-45 years, with the majority (11/

16) aged 31-39 years. Among donors, antibodies to B19 were also examined, with antibody levels of 40% at 16-30 years of age, a slow increase in middle age, and 92% of those over 61 years of age. In summary, the incidence of B19 is low in middle-aged and older people due to high levels of antibodies to B19. [60]

The distribution of anti-B19 IgG in England and Wales in 1988 by Cohen, B. J., and others was investigated by radioimmune analysis. The study examined 2,000 serums: 1,422 people, 374 children in clinics, and 300 women who came to the clinic. In children under 1 year of age, a decrease in maternal antibodies was observed, in 1-5 years of age anti-B19 IgG was 5-15%, in older children and adults this indicator was increased by 50-60%, and in adults over 70 years of age by 85%. [34].

Erythema or aplastic crisis during an epidemic is observed in the following young people: [28].

Age	Disease Observation %
under 5 years	10
5-15	70
15 and adults	70

Children under 1 year of age often do not get sick, due to transplacental immunity. According to a study conducted in Russia, Tikhonova found that children aged 4-10 years are the most affected, and 40-60% of children in schools and preschools are infected during the epidemic [59]. The disease often does not recur after the disease, but in immunodeficient people the disease may recur [26, 49]. In about 70-75% of people, IgG is detected in the deposit, the incidence of the disease is 15-30% [15, 40]. ENDERS. M, WEIDNER. A and others conducted studies on the serological distribution of B 19 among pregnant women and children in Germany between 1997 and 2004. 40517 pregnant women aged 17-45 years and 6060 children were tested for serum. This group is divided into 3:

A) 43/5924 - 0.7% IgM positive, 4097/5924 - 69.2% IgG positive were detected in 5924 pregnant women with gestational age and asymptomatic.

B) 665/15715 - 4.2% of IgM positive were detected in 15715 pregnant women with symptoms of the disease (rash, arthropathy or communication).

C) Out of 3186 children aged 0-18 years with symptoms of the disease (fever, arthropathy, lymphadenopathy, hematological abnormalities, respiratory symptoms), 66/541 - 12.2% IgG positive up to 10 years, 396/551 - 71.9% IgG in children over 12 years positively identified.

In 1997, 156/2480 - 6.3%, in 2003 28/1232 - 2.3% IgM positive. In this study, the prevalence of B19 IgG in Germany was 69.2%, compared to 50-55% in previous data. The prevalence of IgG was higher in 1999-2000 (70.5%) than in 1997-1998 (65.1%). After the 1997-1998 epidemic, the serological prevalence of B19 increased in 1999-2000. In Germany, screening for pregnant women for B19 is usually not performed, but B19 screening is performed if preschool staff, women working with children under 7, are diagnosed with pregnancy. Acute B19 infection is often asymptomatic in pregnant

women, but studies in recent years have shown complications. In summary, the distribution of IgG increases with age and with age. [40]

In Uzbekistan, Lokteva LM, Ibadullaeva NS and others studied the clinical and laboratory course of parvovirus infection among children. This is the first study on parvovirus B19 infection in Uzbekistan. The study examined 28 children from January to December 2019. The disease was most prevalent in preschool and primary school children, and was most prevalent in April and May. In this study, 84% of preschool children and adults were ill, with moderate to severe disease, rash, thrombocytopenia, and decreased hemoglobin.

Shipping way:

-Air-drops (respirator: through sputum, saliva, sneezing)

-vertical (from pregnant mother to child)

-parenteral (factor 8-9 concentrates when mining and its preparations are burned) [20].

Children with a diagnosis of hemophilia have been diagnosed with the virus when they receive a blood transfusion from a large number of donors, especially when 8-9 factors are burned [8].

Pathogenesis: The life cycle of B19 depends on the interaction of the cell receptor with the virus.

- The instigator
- Endocytosis

-Translocation of the genome to the cell nucleus

-DNA replication

-RNA transcription

-Protein transmission

-Assemble of virions

- Cell lysis (dissolution) and emergence of mature virions [57].

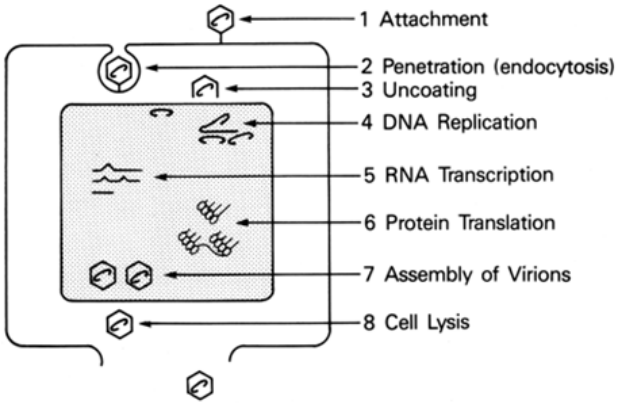


Figure 2. The life cycle of the B19.

B19 infection has a high tropism to the erythroid cell. Erythroid antigen R or Globozid R, which belongs to the category of erythroids, is a specific antigen and is a receptor for B19, individuals without R antigen (200 000: 1) are considered resistant to B19 infection [5, 57].

In Bethesda, on the northwestern edge of Washington, D.C., Brown et al., In the Hematology Department of the National Institute of Heart, Lung, and Mining, 2 control groups were formed from 17 sites. 11 and 0/6) no R antigen was detected in erythrocytes. Conclusion R-antigen parvovirus B19 is a cell receptor, and the R-antigen

load population in erythrocytes is considered resistant to this infection [5]. The R antigen is a human mining group system based on the A4GALT gene on chromosome 22. The R antigen was discovered in 1927 by Karl Landsteiner and Philip Levin.

Immune response:

The course of the disease depends on the age, hematological and immunological status of the organism.

B19 infection occurs in 2 phases:

1 phase. Viremia begins 1 week after ingestion through the nose, hyperthermia, weakness, myalgia, nausea, runny nose.

2 phase. Rash, arthralgia are observed. Ig M is released after 10-12 days and is stored for up to 3 months [45]. After 2-3 weeks, Ig G appears, IgA can also be detected, which protects the infection from nasopharyngeal loading is local immunity. [24]. Ig G can be stored for life or the disease can be relapsed depending on the immune system. People with IgG usually do not relapse, and relapse is rare [13].

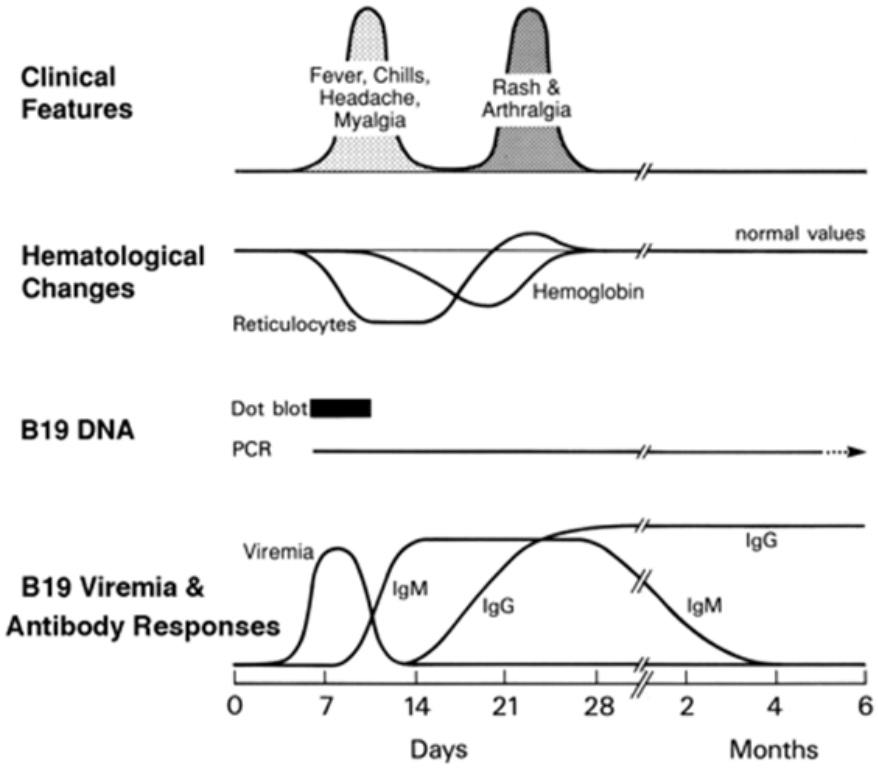


Figure 3

Immunological, virological and clinical course of B19 infection. [2,21]

During viremia reticulocytopenia is observed, recovered in 7-10 days, hemoglobin decreases, known neutropenia, lymphopenia, thrombocytopenia are observed.

Clinic: The incubation period is 6-11 days, with an average of 20 days.

There are 5 main types:

1.Infectious erythema (mainly in children)

2.Arthropathy (in adults)

3.Transient aplastic crisis (in patients with hemolytic pathology)

4.Aplastic anemia

5.Congenital infections (fetal death, miscarriage, fetal damage are observed until the 20th week of pregnancy).

In the prodromal period, 15-30% of children have subfebrile fever, weakness, headache, myalgia, catarrhal symptoms, nausea, vomiting, diarrhea, rash occurs 20-22 days after the virus enters the body, and after the rash the patient is considered uninfected [56]. The rash first appears on the face as a light pale red color, then lasts for 1-4 days and spreads to the cheeks, ash, body, buttocks, the rash is mainly in the areas of the wrinkles, the rash is expected from the skin, dermatologists also call "gloves-sock syndrome" [22].The rash does not occur on the nose, lip triangle, forehead and mouth. The rash persists for 10 days to 3 weeks, the center of the rash is inflamed, and there is no scarring after the rash. Rash 2 - fox increases with temperature, sunlight, stress, heat, cold [50]. In the patient, shrinkage is observed in 70%, especially in the heel area of the foot, the patient is considered contagious until fever and rash.

Arthropathy: In 1985, the association of arthropathy with B19 infection was confirmed [52,62]. The results of a study by White, D. G., A. D. Woolf, P. P., and others showed that B19 infection was detected in 19/153 who were diagnosed with synovitis. In these patients, sudden onset of symmetrical peripheral polyarthropathy was moderate to severe, the dynamics improved within 2 weeks, but in 17 patients the symptoms lasted for more than 2 months, and in 3 patients for more than 4 years. B19 infection occurs in adults without rash, mainly with arthritis, and in children mainly with rash [62].

Reid D. M. et al have studied the association of disease with arthritis when parvovirus-associated infectious erythema is present in the Grampion region. Pain in the joints was observed in 42 of the patients who had recently contracted parvovirus. Of these, 17 patients were admitted to the clinic and 13 were surveyed on the basis of questionnaires, and the remaining 12 patients did not follow the full clinic. Rash was observed in 3 children and 13/27 adults. Arthritis was more common in adults than in children, mostly in women. Arthritis was characterized by symmetrical, concave small joint damage. Symptoms of the disease lasted up to 4 weeks, in 1 adult patient up to 6 months. In 3 children, joint damage was symmetrical and in 1 case lasted up to 7 months [52]. In children, arthralgia is observed in 10% and occurs in the background of rash or in the form of polyarthritis or arthralgia after rash [16]. Joints are affected symmetrically, edema, pain, local hyperthermia are observed, it should be noted that in Parvovirus infection joint damage is safe [55].

Hepatitis: a rare form [44, 58, 63]. It resembles acute hepatitis, the liver size is moderately elevated, moderately Alt, Ast rises, signs of intoxication are observed, and liver enzymes normalize within 3 weeks [31, 25].

HIV infection: In the first study, 1 in 50 patients was not diagnosed with B19 viremia [29]. In HIV patients, B19 was detected in 30 to 5 (17%) cases when the hematocrit was less than 24 and in 13 to 4 (31%) cases when the hematocrit was less than 20. It is believed that the most common cause of severe anemia in HIV patients may be the B19 virus [1]. In children, persistent anemia associated with B19 infection occurs when SD4 is less than 100. Transmission of B19 infection in HIV infection: antigen neutralization due to low humoral immunity, prolonged viremia, chronic disease, anemia, erythrocyte aplasia. If the SD4 is equal to or less than 100, there is still a deficiency after treatment, the course of ARVT is known and unknown.

Myocarditis: Several cases of myocarditis and perimiocarditis have been identified among children [32, 38, 30]. The condition of 24 children infected with B19 in Paris in 1992 was studied by Borreda, D., S. Palomera, and others. From January 1, 1987 to December 31, 1990, children with B19 parvovirus infection were examined, and patients were tested for B19 IgM during laboratory diagnostic burns. Of these, 4 patients had hematological symptoms, 1 patient had transient bone marrow aplasia, 1 patient had autoimmune hemolytic anemiawith betathalassemia, and 2 patients had peripheral

thrombocytopenia. Skin lesions were observed in 8 patients, rash in 6, nodular erythema in 1, Gianotti-Krosti syndrome in 1 patient, and no infectious erythema in any patient. In 7 cases with joint damage, 3 patients showed unknown symptoms of viral diseases, of which a 5-month-old child developed severe acute myocarditis, 1 patient with hepatitis A had acute liver failure. This study confirms the broad spectrum of clinical manifestations of B19 infection, resulting in the question of whether B19 infection may be involved in the genesis of chronic juvenile arthritis. [32].

Prevention: testing of children with emancipation for B19 [33]; testing donors for B19 prior to bone marrow transplantation [43]. The vaccine has not yet been developed. Production of 2 recombinant vaccines (MEDI-491 and VAI-VP 705) continues, with the vaccine capsule consisting of VP1 and VP2.

Conclusion

1.Initiate testing of donors for PV B19 in order to prevent hemocontact transfusion transmission of the virus.

2.Examination of pregnant women (especially the contingent working with children by profession) in the first and second trimesters for PV B19.

3.It is necessary to expand research on the course of the disease, the epidemiological situation, laboratory tests, especially in pregnant women and children.

4.Organization of lectures on the disease among general practitioners, obstetricians, gynecologists, dermatologists, pediatricians, laboratory physicians.

In Uzbekistan, very little research has been conducted on the analysis of antibodies against B19 among children.

References.

1.Абковиц, Дж. Л., Браун К. Э., Вуд Р. У., Ковач Н. Л., Грин С. В. и Янг Н. С. 1997 . Клиническая значимость парвовируса B19 как причины анемии у пациентов с инфекцией вируса иммунодефицита человека. J. Infect. Дис. 176 : 269 - 273.CrossRefPubMedWebofScienceGoogleScholar

2.Андерсон, М. Дж., П. Г. Хиггинс, Л. Р. Дэвис, Дж. С. Уиллман, С. Е. Джонс, И. М. Кидд, Дж. Р. Паттисон и Д. А. Тиррелл. 1985 . Экспериментальная парвовирусная инфекция у человека. J. Infect. Дис. 152 : 257 -265.

3.Бернс, К.И. 1996 . Parvoviridae : вирусы и их репликация, с. 2173 - 2197. В BN Fields, DM Книпе, PM Хоули, RM Chanock, JL Мельника, TP Monath, Б. Ройзман и SE Straus (ред.), Поля вирусологии . Липпинкотт-Рэйвен, Филадельфия, Пенсильвания.Google Scholar.

4.Браун, К. Э., Дж. Мори, Б. Дж. Коэн и А. М. Филд. 1991 . Размножение парвовируса B19 invitro в первичной культуре печени плода. J. Gen. Virol. 72 : 741 -745.CrossRefPubMedWebofScienceGoogleScholar.

5.Браун, К. Э., Дж. Р. Хиббс, Г. Галлинелла, С. М. Андерсон, Э. Д. Леман, П. Маккарти и Н. С. Янг. 1994 . Устойчивость к инфекции парвовируса B19 из-за отсутствия рецептора вируса (антигена эритроцита P). N. Engl. J. Med. 330 : 1192 - 1196.CrossRefPubMedWebofScienceGoogleScholar).

6.Зигл, Г., Р. К. Бейтс, К. И. Бернс, Б. Дж. Картер, Д. К. Келли, Э. Курстак и П. Таттерсолл. 1985 . Характеристики и систематика Parvoviridae. Intervirology 23 : 61 -73.CrossRefPubMedWeb of ScienceGoogle Scholar).

7.Кан Дж. С., Кесебир Д., Котмор С. Ф. и др. (Июль 2008 г.). "Сероэпидемиологиябокавируса человека, определенная с использованием рекомбинантных вирусоподобных частиц" . J. Infect. Дис . 198 (1): 41-50. DOI : 10.1086 / 588674 . PMID 18491974 .

8.Климович Н.В., Матвеев В.А., Романова О.Н., Черновецкий М.А. Эпидемиологическая характеристика парвовирусной инфекции у детей с гематологическими заболеваниями. Охранаматеринстваидетства. 2011; 1: 42 - 46.

9.Кодури, PR 1998 . Новая цитоморфология гигантских проэритробластов парвовирусной инфекции B19. Являюсь. J. Hematol. 58 : 95 -99. CrossRefPubMedGoogle Scholar.

10.КолЕ.О., М. Дж. АшериП.А. Бертон. 1988 . Внутриутробное инфицирование парвовирусом человека B19: исследование с помощью световой и электронной микроскопии. J. Med Virol. 24 : 55 -66.PubMedWeb of ScienceGoogle Scholar.

11.Коссарт, Ю. Э., А. М. Филд, Б. Кант, Д. Уиддоус. 1975 . Парвовирусоподобные частицы в сыворотке крови человека. Ланцет I : 72 -73.Google Scholar

12.Котмор, С.Ф., В. К. Маккай, Л. Дж. Андерсон, С. Р. Астелл и П. Таттерсолл. 1986 . Идентификация основных структурных и неструктурных белков, кодируемых парвовирусом человека B19, и картирование их генов путем прокариотической экспрессии геномных фрагментов. J. Virol. 60 : 548 -557.

13.Леманн Х.В., фон Ланденберг П., Модров С. (2003). "Парвовирусная инфекция B19 и аутоиммунное заболевание". AutoimmunPед . 2 (4): 218-223. DOI : 10.1016 / S1568-9972 (03) 00014-4 . PMID 12848949).

14.Ло И, Цю Дж. Парвовирус человека B19: механистический обзор инфекции и репликации ДНК. Будущий Virol. 2015; 10 (2): 155-167. DOI: 10.2217 / fvl.14.103.

15.ЛушноваИ.В. ПарвовирусnаяB19 инфекция. Педиатр. 2010; 1: 115 - 118

16.Ноктон, Дж. Дж., Л. К. Миллер, Л. Б. ТакериДж. Г. Шаллер. 1993 . Артрит, связанный с парвовирусом человека B19, у детей. J. Pediatr. 122 : 186 -

190.CrossRefPubMedWebofScienceGoogleScholar

17.Одзава К., Дж. Аюб, Ю.С. Хао, Г. Курцман, Т. Шимада и Н. Янг. 1987 . Новая карта транскрипции патогенного парвовируса B19 (человека). J. Virol. 61 : 2395 - 2406.

18.Одзава К. и Н. Янг. 1987 . Характеристика капсидных и некапсидных белков парвовируса B19, размножающихся в культурах клеток костного мозга эритроида человека. J. Virol. 61 : 2627 -2630.

19.Одзава К., Курцман Г. и Янг Н. 1987 . Продуктивное инфицирование парвовирусомB19 эритроидных клеток костного мозга человека invitro. Blood 70 : 384 -391.

20.ПаттисонJR, Пат G (1996). БаронС. идр. (ред.). Парвовирусы. В: Медицинская микробиология Баррона (4-е изд.). Univ Техасского медицинского отделения. ISBN 978-0-9631172-1-2).

21.Пату, Г., Д. Пиллэй, С. Мьинт и Дж. Паттисон. 1993 . Характеристика анализа вложенной полимеразной цепной реакции для обнаружения парвовируса B19. J. Clin. Microbiol. 31 : 540 -546.

22.Сантоха С, Ньето Гонсалеса G, Сантос-Бриз А, Гутьеррес Zufiaurre Mde L, L Cerroni, Kutzner H, L. Requena (декабрь 2011 г.). "Иммуногистохимическое обнаружение парвовируса B19 в папулопурпурическойсиндроме" перчатки и носки ": прямые доказательства вовлечения вирусного эндотелия. Отчет о трех случаях и обзор литературы ". Американский журнал дерматопатологии . 33 (8):790-5. DOI : 10.1097/ DAD.0b013e318221bc41 . PMID 22024574 . S2CID 41097601) .

23.Шварц, Т.Ф., С. Серке, А. фон Брунн, Б. Готтентрагер, Д. Хун, Ф. Дейнхардт и М. Роггендорф. 1992 . Термостабильность парвовируса B19: кинетика инактивации. Zentbl. Бактериол. 277 : 219 -223.Google Scholar.

24.Эрдман, Д.Д., М.Дж. Ашер, Ч. Цоу, Е.О. Кол, Г.В. Гэри, С.Каджигая, Н.С. Янг и Л.Дж. Андерсон. 1991 . Антитела IgG, IgA и IgM, специфичные к парвовирусу B19 человека, и ДНК в образцах сыворотки от людей с инфекционной эритемой. J. Med. Virol. 35 : 110 -115.CrossRefPubMedGoogle Scholar)

25.Abe K., Kiuchi T., Tanaka K. Edamoto Y, Aiba N, Sata T. Characterization of erythrovirus B19 genomes isolated in liver tissues from patients with fulminant hepatitis and biliary atresia who underwent liver transplantation. Int. J. Med. Sci. 2007; 4(2): 105 - 109.

26.Anderson L.J. et al. Risk of infection following exposures to human parvovirus B19. BehringInst.Mitt.1990; 85: 60-63.

27.AndersonL.J., GillespieS.M., TorokT.J., HurwitzE.S., TsouC. J., GaryG.W.. Risk of infection following exposures to human parvovirus B19.Behring Inst. Mitt.1990; 85: 60 - 63.

28.Anderson, L. J. 1987. Role of parvovirus B19 in human disease. Pediatr. Infect. Dis. J.6:711-718.CrossRefPubMedWeb of ScienceGoogle Scholar

29.Anderson et al., Letter, Ann. Intern. Med. 102: 275, 1985.

30.Beghetti et al., Letter, Eur. J. Pediatr. 159: 135-136, 2000; Saint-Martin et al., Letter, J. Pediatr. . 116: 1007-1008, 1990)

31.Bernuau J., Durand F., Valla D. Parvovirus B19 infection and ful-minant hepatitis. Lancet. 1999; 353 (9154): 754 - 755.

32.Borreda, D., S. Palomera, B. Gilbert, A. Lienhardt, and L. de Lumley. 1992. 24 cases of human parvovirus B19 infection in children. Ann. Pediatr. (Paris)39:543-549. (In French.). Google Scholar

33.Brown, K. E., N. S. Young, B. M. Alving, and L. H. Barbosa. 2001. Parvovirus B19: implications for transfusion medicine. Summary of a workshop. Transfusion41:130-

135.CrossRefPubMedGoogle Scholar

34.Cohen, B. J., and M. M. Buckley. 1988. The prevalence of antibody to human parvovirus B19 in England and Wales. J. Med. Microbiol.25:151-153. CrossRefPubMedWeb of ScienceGoogle Scholar

35.Cohen and Brown, Letter, J. Infect. 24: 113-114, 1992.

36.David M. Morens. Fifth Disease: Still Hazy After All These Years

37.Dictionary of Virology

38.Enders, G., J. Dotsch, J. Bauer, W. Nutzenadel, H. Hengel, D. Haffner, G. Schalasta, K. Searle, and K. E. Brown. 1998. Life-threatening parvovirus B19-associated myocarditis and cardiac transplantation as possible therapy: two case reports. Clin. Infect. Dis.26:355-358.CrossRefPubMedWeb of ScienceGoogle Scholar

39.ENDERSM; WEIDNERA; ENDERSG (2006). "Современные эпидемиологические аспекты инфицирования парвовирусом человека B19 во время беременности и детства в западной части Германии" . Эпидемиология и инфекция . 135 (4): 563-569. DOI : 10.1017 / S095026880600731X . PMC 2870617 . PMID 17064457 .

40.Enders, M. Weidner A., Enders G. Current epidemiological aspects of human parvovirus B19 infection during pregnancy and childhood in the western part of Germany. Epidemiol. Infect. 2007; 135: 563 - 569.

41.Fjaerli, HO; Vogt, H .; Бруу, А.Л. (1991). "Парвовирус человека B19 как причина апластического криза при наследственном сфероцитозе". Tidsskriftдля den Norske Laegeforening . 111 (22): 2735-2737. PMID 1658972 .

42.Gillespie S.M., Cartter M.L., Asch S., Rokos J.B., Tsou C. J., Gary G. Occupational risk of human parvovirus B19 infection forschool and day-care personnel during an outbreak of erythema infectiosum. J. Am. Med. Assoc. 1990; 263 (15): 2061 - 2065.

43.Heegaard, E. D., and P. B. Laub. 2000. Parvovirus B19 transmitted by bone marrow. Br. J. Haematol.111:659-661.CrossRefPubMedGoogle Scholar

44.Hillingso, J. G., I. P. Jensen, and L. Tom-Petersen. 1998. Parvovirus B19 and acute hepatitis in adults. Lancet351:955-956.CrossRefPubMedWeb of ScienceGoogle Scholar

45.1nderson, L. J., C. Tsou, R. A. Parker, T. L. Chorba, H. Wulff, P. Tattersall, and P. P. Mortimer. 1986. Detection of antibodies and antigens of human parvovirus B19 by enzyme-linked immunosorbent assay. J. Clin. Microbiol.24:522-526. Abstract/FREE Full TextGoogle Scholar

46.Kelly, H. A., D. Siebert, R. Hammond, J. Leydon, P. Kiely, and W. Maskill. 2000. The age-specific prevalence of human parvovirus immunity in Victoria, Australia compared with other parts of the world. Epidemiol. Infect.124:449-457.CrossRefPubMedGoogle Scholar

47.Molenaar-де Бакер МВт, Лукашов В.В., ван Binnendijk RS, Boot HJ, Zaaijer HL (2012). "Глобальное сосуществование двух эволюционных линий парвовируса B19 1a, различающихся синонимичными позициями по всему геному" . PLOS ONE . 7 (8): e43206. Bibcode : 2012PLoSO ... 743206M . DOI : 10.1371 journal.pone.0043206 . PMC 3418230 . PMID 22912828 .

48.Morens D. M. Fifth disease: Still hazy after all these years. (англ.) // JAMA. - 1982. - 6 August (vol. 248, no. 5). - P. 553-554. - doi:10.1001/jama.1982.03330050035026. - PMID 7097899.

49.Mossong, J., Hens N., Friederichs V., Davidkin I., Broman M., Litwincka B. et al. Parvovirus B19 infection in the European countries: seroepidemiology, force of infection, and maternal risk of infection. Epidemiol.Infect. 2008; 136 (8): 1059 - 1068

50.Naides, SJ 1999 . Заражение парвовирусом B19. Curr. Заразить. Дис. Rep. 1 : 273

-278.PubMedGoogle Scholar.

51.Owren, PA 1948 . Врожденная гемолитическая желтуха: патогенез гемолитического криза. Кровь 3 : 231 -248.

52.Reid, D. M., T. M. Reid, T. Brown, J. A. Rennie, and C. J. Eastmond. 1985. Human parvovirus-associated arthritis: a clinical and laboratory description. Lanceti:422-425.CrossRefGoogle Scholar

53.Robert R. Briney. Primary Cutaneous Actinomycosis

54.Sabella C, Гольдфарб J (октябрь 1999 года). "Парвовирусные инфекции B19" . Я семейный врач . 60 (5): 1455-60. PMID 10524489 .

55.Savaresi I., Atypical manifestations of congenital parvovirus B19 infection. Eur. J. Pediatr. 2008; 167 (12): 1463 - 1466.

56.Servey JT, Reamy BV, Hodge J (февраль 2007 г.). "Клинические проявления парвовирусной инфекции B19" . Я семейный врач . 75 (3): 373-376. PMID 17304869 . Проверено 6 ноября 2009 года) .

57.Slavov S. N., Kashima S., Pinto A. C., Covas D. T. Human parvovirus B19: general considerations and impact on patients with sickle-cell disease and thalassemia and on blood transfusions (англ.) // FEMS Immunol Med Microbiol. - 2011. - Vol. 62, no. 3. - P. 247-262. - doi:10.1111/j.1574-695X.2011.00819.x. - PMID 21585562.

58.Sokal, E. M., M. Melchior, C. Cornu, A. T. Vandenbroucke, J. P. Buts, B. J. Cohen, and G. Burtonboy. 1998. Acute parvovirus B19 infection associated with fulminant hepatitis of favourable prognosis in young children. Lancet352:1739-1741.CrossRefPubMedWeb of ScienceGoogle Scholar

59.Tikhonova N.T. Evaluation of the spread of parvovirus infection in Moscow. Information Letter of the Health Committee of Moscow. Moscow; 2004; (11): 12 (in Russian)

60.Tsujimura, M., K. Matsushita, H. Shiraki, H. Sato, K. Okochi, and Y. Maeda. 1995. Human parvovirus B19 infection in blood donors. Vox Sang.69:206-212.CrossRefPubMedGoogle Scholar

61.Vafaie J, Шварц P. (2004). "Парвовирусные инфекции B19". IntJDermatol . 43 (10): 747-749. DOI : 10.1111/ j.1365-4632.2004.02413.x . PMID 15485533 .

62.White, D. G., A. D. Woolf, P. P. Mortimer, B. J. Cohen, D. R. Blake, and P. A. Bacon. 1985. Human parvovirus arthropathy. Lanceti:419-421.Google Scholar

63.Yoto, Y., T. Kudoh, K. Haseyama, N. Suzuki, and S. Chiba. 1996. Human parvovirus B19 infection associated with acute hepatitis. Lancet347:868-869.CrossRefPubMedWeb of ScienceGoogle Scholar Drago et al., Letter , Br. J. Dermatol. 141: 160-161, 1999).