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РОЗДІЛ 1. Емерджентні хвороби та Єдине здоров'я
SECTION 1. Emergent Diseases and One Health

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Characteristics of the Epizootiology, Epidemiology, Diagnostics and Prevention of Crimean-Congo Hemorrhagic Fever

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Abstract. *Crimean-Congo hemorrhagic fever (CCHF) is a particularly dangerous zoonotic arboviral infection caused by the RNA-containing CCHF virus (CCHFV) of the genus Orthonairovirus in the family Nairoviridae. The disease is characterized by a wide geographic range, covering more than 50 countries in Africa, Asia, and Europe, including regions of Ukraine. The natural circulation of the pathogen is sustained by complex interspecies interactions, in which ixodid ticks of the genus Hyalomma serve as the primary vectors and long-term reservoirs, capable of transphasic and transovarial transmission of the virus. Additional amplifiers of the virus include domestic and wild mammals (cattle, sheep, goats, rodents, and hares), as well as ostriches, in which the infection is typically asymptomatic, while migratory birds contribute to the expansion of the range by transporting infected ticks. Routes of human infection include both vector-borne transmission via tick bites and contact transmission—through exposure to the blood, bodily fluids, or tissues of infected animals during their care or slaughter. In addition, there is a high risk of human-to-human transmission, particularly nosocomial transmission through direct contact with hemorrhagic secretions from patients in the acute phase of the disease. Additional routes of transmission include the alimentary route (through consumption of raw meat or unpasteurized milk) and the sexual route. The pathogenesis and clinical course of CCHF in humans are phasic in nature. Following the incubation period, a prehemorrhagic phase develops, characterized by severe systemic intoxication, which subsequently progresses to the hemorrhagic phase, accompanied by a petechial rash, ecchymoses, multiple internal and external hemorrhages, thrombocytopenia, and a risk of developing disseminated intravascular coagulation and multiple organ failure. The average case-fatality rate is 11.7%. Diagnosis of the disease is based on the use of molecular genetic methods (RT-PCR) during the early days of viremia, as well as serological tests (ELISA) to detect antigens and specific IgM and IgG antibodies. All procedures involving the pathogen require a high level of biosafety (BSL-3/BSL-4). The therapeutic strategy involves intensive pathogenetic and supportive therapy combined with early administration of antiviral drugs, particularly ribavirin. This article provides a detailed description of existing vaccine candidates. Given the lack of commercially available vaccines, specific preventive measures play a key role in preventing outbreaks: acaricidal treatment of animals, use of personal protective equipment, adherence to food hygiene, and strict implementation of infection control measures in healthcare facilities.*

Keywords: Crimean-Congo, *Bunyavirales*, polymerase chain reaction (PCR), ELISA, epizootic situation, diagnostics, risk assessment.

Crimean-Congo hemorrhagic fever (abbreviation: CCHF; synonyms: *Congo Fever, Central Asian Hemorrhagic Fever, Uzbekistan hemorrhagic fever, Hungribta (blood taking), Khunymuny (nose bleeding), Karakhalak (black death))* is a zoonotic arboviral infection that often progresses in animals without pronounced clinical signs but can pose a serious threat to humans.

In 1973, the CCHF virus (CCHFV) was identified as the causative agent of two diseases previously considered distinct: Congo fever (first identified in 1956) and Crimean fever (first identified in 1944–1945 among Soviet military personnel) (Čumakov, 1945; Hoogstraal, 1979; Whitehouse, 2004). It has now been established that this virus is endemic to a broad geographic region (more than 50 countries), including Africa, Asia, and Europe. The presence of the virus in these regions is

directly linked to the presence of the primary arthropod vector of CCHFV, the *Hyalomma* spp. ticks (Hoogstraal, 1979; Bente et al., 2013; Papa et al., 2015; CFSPH, 2019; Belobo, 2021), and more than 3 billion people are currently at risk of CCHF infection (Mears, 2022), which allows it to be characterized as the geographically most widespread tick-borne viral disease (Raabe, 2020). In Ukraine, at least 336 cases of this disease were reported between 1944 and 2025, but official statistics are incomplete, and the disease is often underdiagnosed. For example, hundreds of cases are reported annually in Turkey, dozens in Bulgaria, and isolated cases in Spain, though also practically every year (Fereidouni, 2025). In certain areas, the virus can circulate for years without being detected, although subclinical and mild clinical forms may be relatively common, and sporadic severe cases may remain undiagnosed etiologically. Depending on the available healthcare infrastructure and other factors, the case-fatality rate for CCHF can range from 5% to 80% during limited outbreaks (Schwarz, 1997; Leblebicioglu, 2016), averaging 11.7% (Belobo, 2021).

Aim of the study: To summarize and systematize current scientific data on the epizootiological and epidemiological characteristics, biological properties of the pathogen, the clinical course, laboratory diagnosis, as well as treatment and prevention strategies for Crimean-Congo hemorrhagic fever (CCHF) to improve the effectiveness of epidemiological surveillance and biosafety measures.

Etiology. *CCHFV* belongs to the genus *Orthonairovirus* of the family *Nairoviridae* and the order Bunyavirales. It is an enveloped virus with a single-stranded, segmented, negative-sense RNA and belongs to the *CCHF* serogroup, which also includes the *Tofla and Hazara* viruses. Various scientific sources recognize 6–7 major genetic clades of *CCHFV*. Some *CCHFV* strains may differ in virulence (Abudurexiti, 2019; CFSPH, 2019; Garrison, 2019).

CCHFV is inactivated by many disinfectants, including 1% hypochlorite, 70% alcohol, hydrogen peroxide, iodophors, glutaraldehyde, and formalin. It can also be inactivated by ultraviolet light or at a *pH < 6,* by heating at 56°C for 30 minutes or at 60°C for 15 minutes (Papa, 2015; Leblebicioglu, 2016; Blair, 2019; Public Health Agency of Canada, 2024).

Epizootiological and epidemiological data. *CCHFV* is the most geographically widespread tick-borne virus, with endemic regions including parts of Africa, Asia, and Europe (Bente, 2013; Messina, 2015; Raabe, 2020). It is estimated that 10,000 to 15,000 cases of CCHF are reported annually, predominantly in endemic countries, with only sporadic cases observed among travelers (as of 2016, 21 travel-associated cases had been reported) (Leblebicioglu et al., 2016; Raabe, 2020). According to a meta-analysis, the average seroprevalence of CCHFV among humans and animals was 4.7% and 24.6%, respectively, with an upward trend in recent years. This rate is 7.5 times higher among individuals at high risk of infection, including those exposed to tick bites, contact with CCHF patients, or occupations involving frequent contact with animals (Nasirian, 2019). According to ECDC data, as of the end of August 2026, four cases of CCHF (Spain) had been reported in the EU (ECDC, 2026).

CCHFV is transmitted primarily through the bites of infected ticks of the genus Hyalomma, although transmission through direct contact cannot be ruled out. In a wide range of animals, including cattle, cynomolgus macaques, donkeys, goats, ground squirrels, hares, horses, hedgehogs, ostriches, small rodents, and sheep, CCHFV infection can lead to viremia, which is short-lived, making it difficult to detect the virus since most animals are asymptomatic during this period. In most animals, infection is not accompanied by any clinical signs at all (Spengler, 2016; Garrison, 2019). CCHFV can be directly detected in Hyalomma ticks using tests for viral antigen or nucleic acid.

Ticks become infected through all known routes: horizontal transmission (feeding of larvae, nymphs, or adult ticks on an infected host with viremia); transphase (transstadium) transmission (the virus persists in the tick's body during its development and transition from one life stage to another); transvarial transmission; and transsexual (venereal) transmission (transmission of the virus from an infected male to an uninfected female during mating).

CCHFV has been isolated from domestic and wild mammals, specifically cattle, sheep, goats, buffalo, hares (e.g., the European hare, *Lepus europaeus*), African hedgehogs (*Erinaceus albiventris*), and mastomys (*Mastomys* spp.). Specific antibodies have been detected in many other species, such as horses, donkeys, camels, water buffalo, dogs, red foxes (*Vulpes vulpes*), wild dogs (*Lycaon pictus*), manuls (*Felis manul*), genets (*Genetta genetta*), several species of African ungulates (e.g., rhinoceroses – *Diceros bicornis*, *Ceratotherium simum*; giraffes – *Giraffa camelopardalis;* African buffalo – *Syncerus caffer*), hedgehogs (*Erinaceus europaeus*, *Hemiechinus auritus*), various rodents, and bats. Among seropositive species, susceptibility has been confirmed

through experimental infection of horses (horses, donkeys), European hares, rock hares (Lepus saxatilis), and some species of wild rodents.

Typical clinical signs have been observed only in experimental animal models, specifically in newborn rodents, certain immunodeficient mouse strains, and cynomolgus macaques (Macaca fascicularis) (Spengler, 2016; CFSPH, 2019).

Regarding the role of birds as a natural reservoir for CCHFV, ostriches are susceptible to the virus; they can develop mild viremia and serve as a source of infection for humans (particularly at slaughterhouses). Wild and migratory birds act as “vectors” for ticks of the genus Hyalomma, including infected ones, spreading them over thousands of kilometers between continents (Mostafavi, 2013; Mancuso, 2022).

In general, the functioning of the CCHF parasitic system can be schematically represented as shown in Fig. 1.

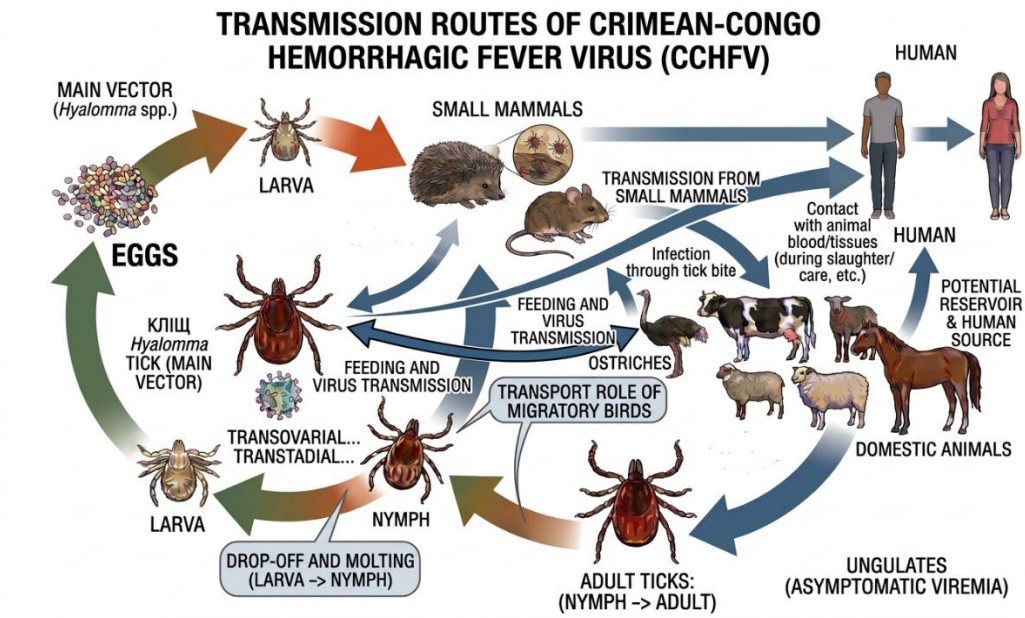


Fig. 1. Scheme of the functioning of a parasitic system CCHF

Geographic distribution. CCHFV is widespread in Africa, Asia, and countries in Southern and Eastern Europe, where ticks of the genus Hyalomma are present. Different clades and variants of the pathogen circulate in various regions. In Europe and Asia, its presence has been documented in northern Spain, Portugal, Hungary, Romania, Bulgaria, Ukraine, Kosovo, Albania, Macedonia, southern Russia, Mongolia, Kazakhstan, and Uzbekistan, although clinical cases of the disease in humans have not been reported in all of these countries. Although Hyalomma ticks are sometimes found in northern Europe and Asia likely after being carried by migratory birds these regions are considered unsuitable for the permanent establishment of Hyalomma species. Currently, the risks of CCHFV circulation in European countries are being extensively studied, including through the use of ecological niche modeling and other methods. The assessed risk was found to be low for most countries (Austria, Belgium, Germany, Luxembourg, the Netherlands, Slovenia, and Switzerland) and moderate for France and Italy, when considering only the consequences for animal health. When considering only the implications for public health, the risks were assessed as low for all countries, with the exception of Italy, where they were found to be moderate (Fanelli, 2021).

Mechanisms and routes of transmission of the pathogen. Humans are usually infected through tick bites or through contact with the blood and tissues of infected animals or humans. Ticks of the genus Hyalomma are the primary vectors of the CCHFV and are considered critical to its persistence in endemic areas. Hyalomma marginatum and Hyalomma asiaticum are important in Europe and Asia, respectively, but the virus is also found in other members of this genus, including some species that infect reptiles (e.g., *Hyalomma aegyptium*). In species of the genus *Hyalomma*, transovarial and transstadiary transmission, sexual transmission, and transmission during co-feeding (from infected ticks to uninfected ticks feeding simultaneously on an uninfected host) have been demonstrated. CCHFV can overwinter in unfed Hyalomma ticks, which can survive mild winters. The virus has also been detected in members of several species within the genera *Rhipicephalus, Boophilus, Dermacentor*, and Ixodes. It is unknown how many of these ticks are competent vectors, but some of them may transmit the virus locally. CCHFV has been reported

infrequently in other blood-feeding invertebrates, such as midges (*Culicoides* spp.) and soft ticks, but they are not believed to play any role in the epizootic and epidemiological processes of this infection. Experimental data indicate that the pathogen does not replicate in argasid ticks. Migratory birds may spread infected ticks between distant regions. CCHFV is present in blood, tissues, or crushed ticks can enter the body through contaminated mucous membranes or broken skin. Some cases were likely caused by the consumption of unpasteurized milk. Several cases of human infection have been reported in individuals who, shortly before the onset of illness, ate raw liver or consumed undercooked fresh meat. Human-to-human transmission may occur, particularly during close contact. Blood from bleeding patients is an important factor in virus transmission to family members and healthcare workers. Viral RNA has been detected in saliva and urine, as well as in swabs from the conjunctiva, nose, and rectum. At the same time, some studies suggest that skin contact with secretions and excretions that do not contain blood carries a relatively low risk of infection. In several cases, epidemiological investigations have indicated a possible sexual route of transmission: in particular, a case has been described in which a man presumably transmitted the virus to his wife during his recovery period. Airborne transmission of the virus has been reported infrequently primarily following laboratory accidents or medical procedures involving the generation of fine droplets or aerosols. During one unusual outbreak, cases were reported among several people who visited the room of a critically sick patient on a ventilator, even though they had no direct contact with him (Bente, 2013; Shayan, 2015; CFSPH, 2019; Blair, 2019; Raabe, 2020).

Intrauterine transmission of the virus in humans has been reported (Leblebicioglu, 2016; Ergönül, 2006). Whether the CCHFV can be transmitted through breast milk during the acute phase of the disease has not yet been proven, although viral RNA was not detected in the breast milk of two mothers who were breastfeeding prior to diagnosis, and their children did not become infected (Erbay, 2008). The possibility of milk contamination with blood (for example, during skin lesions) raises concerns, even if the virus is not directly shed into the milk. Consideration of this issue is most often based on an assessment of the risk of contact transmission from mother to child during lactation and the presence of viremia. The active virus typically circulates in patients' blood during the first 7–12 days after the onset of illness, after which it disappears as specific antibodies (IgM and IgG) are produced. However, viral RNA can sometimes be detected in the blood and bodily fluids (urine, saliva) for 2–4 weeks (in some cases up to a month) after recovery (Ergönül, 2006). Despite the possible presence of viral RNA during early convalescence, the risk of transmission to others decreases significantly as hemorrhagic manifestations (bleeding) subside and viral load decreases.

There is a risk of sexual transmission of the pathogen during convalescence or immediately after the patient is discharged from the hospital. In particular, there have been reported cases where men transmitted the virus to their female partners one week after the acute symptoms had resolved or after the completion of inpatient treatment. It has been shown that the reproductive tract is an "immunoprivileged zone" where the virus can localize and persist somewhat longer than in the general bloodstream. For CCHFV, the duration of shedding in semen is usually limited to the early convalescent period (several weeks after recovery); however, these risks persist even in mild forms of the disease (Sorvillo, 2026).

How animals become infected with CCHFV has not yet been fully studied; however, it can be assumed that ticks play a major role.

Natural infection with CCHFV in animals is not accompanied by the development of clinical signs or fatalities. Seroprevalence among farm animals in endemic regions varies significantly: from low rates in certain areas to 50% or more in others, and in some natural foci, it reaches even higher levels. Fluctuations and changes in animal populations often precede outbreaks among humans. This is due to two main factors: the role of animals as effective amplifiers of the virus and the availability of a food source, which contributes to the growth and expansion of the range of tick vector populations (Papa, 2004; Blair, 2019; CFSPH, 2019).

Sporadic clinical cases or outbreaks occur regularly in some endemic regions. In other areas, they appear to arise unexpectedly after prolonged periods without apparent cases among humans. The latter can be explained by changes in animal and tick populations, a lack of adequate epidemiological surveillance, and the state of specific population immunity. Cases have been reported of people who recently moved to an endemic region falling ill (Leblebicioglu, 2016).

Based on the characteristics of the pathogen's mechanism and routes of transmission, occupational groups at increased risk of CCHFV infection include farmers, herders, veterinarians, slaughterhouse workers, laboratory staff, and healthcare workers, as well as individuals who are regularly exposed to ticks. The seasonal pattern of the disease is driven by fluctuations in tick

populations and increased human exposure to fresh blood and animal tissues (for example, during religious holidays such as Eid al-Adha).

Mortality. The case-fatality rate varies by region, ranging from 5% to 30% in most cases, although it can reach 80% during localized outbreaks. It is determined by a number of factors, including the availability and quality of medical care, the viral dose, the route of transmission, the presence of co-infections, and the genetic characteristics of the pathogen. Previously, most cases of CCHF were considered exclusively severe; however, serological studies indicate the presence of mild or subclinical forms in 2–30% of the population in endemic regions. In particular, in Turkey, where more than 11,811 cases of CCHF were reported between 2011 and 2024 with a case-fatality rate of only 4.7%, specific antibodies were detected in 10–15% of residents (Şahan, 2025), indicating that most cases are asymptomatic or mild. Similarly, up to 15% of the Greek population is seropositive, despite the minimal number of reported clinical cases, which is most likely due to the circulation of low-virulence variants of the virus. The case fatality rate among children, taking geographical data into account, will be discussed below.

Survival of the pathogen. CCHF can survive for a short time in the environment, especially in certain organic materials. The infectious virus was detected for up to 10 days, and sometimes longer, in blood stored at 4°C. Viral RNA was detected for up to 30 days in blood serum at 4°C, but the virus's infectivity was not assessed. It has also been reported that CCHFV remained infectious in blood serum for at least several days at an unspecified ambient temperature and was stable "under moist conditions" for 15 days at 4°C, 11 days at 20°C, and 7 hours at 37°C. When the sample was dried, the virus remained infectious for less than 24 hours (CFSPH, 2019).

Clinical signs and course of the disease. *Characteristics of CCHF in animals.* It is believed that when animals are infected with small amounts of the CCHFV, the infection is asymptomatic, or the clinical signs are quite mild. No classic clinical signs have been reported in naturally infected animals. Most experimentally infected animals (cattle, sheep, goats, horses, donkeys) and wild animals (e.g., hares, hedgehogs) also remained asymptomatic, although some individuals experienced a temporary, mild fever. In one experiment, two calves were lethargic and had a reduced appetite for several days. In one case, the virus was isolated during a fever in a cow amid a widespread outbreak of abortions among the herd. In one study, 4 out of 5 pregnant ewes gave birth to healthy lambs; one ewe experienced an abortion, although CCHFV was not isolated from the fetus.

Until recently, attempts to create animal models for studying CCHFV in humans had been unsuccessful, apart from newborn rodents, immunodeficient mice, and cynomolgus macaques (*Macaca fascicularis*). In the latter, a high mortality rate (up to 75%) was observed only following intravenous administration of a specific virus strain. Subcutaneous injections of virus-containing material were not fatal. In addition to nonspecific signs, such as loss of appetite, some animals developed oliguria, swelling of the muzzle and body, as well as various hemorrhagic signs. However, there is no evidence that this model can be extrapolated to naturally infected non-human primates, especially since other experimentally infected non-human primates, as well as cynomolgus macaques injected with a different strain of CCHFV, showed no symptoms of the disease or exhibited only a few mild signs, such as transient fever. One baboon developed itching and a rash on its limbs, accompanied by vasculitis and hemorrhages in those areas (Papa, 2004; Blair, 2019; CFSPH, 2019).

Characteristics of CCHF in humans. The incubation period in humans is 1–13 days (Table 1) (PNAS, 2024). Most cases develop clinical symptoms within a week.

CHFV in humans can range from asymptomatic to severe clinical forms. Clinical cases typically begin with a febrile, flu-like syndrome featuring symptoms such as headache, dizziness, photophobia, sore throat, neck pain, myalgia, and arthralgia. Fever can sometimes be very high. Gastrointestinal symptoms are also common, including nausea, vomiting, and diarrhea, as well as abdominal pain. Some patients develop a maculopapular rash or skin redness, and other symptoms, such as neurological signs (sudden mood changes, confusion, aggression) and cardiovascular changes (e.g., bradycardia, low blood pressure), are rarely reported. Occasionally, patients present with fever, epididymitis, and orchitis. Some individuals may experience a brief (24–48-hour) remission. Patients may either make a full recovery after the flu-like stage or develop a hemorrhagic syndrome of varying severity.

Table 1

Clinical stages of CCHF and their characteristics

№	Period of infection process	Duration	Clinical and laboratory manifestations
1	Incubation	• **After a tick bite:** usually 1–3 days (up to 9 days) • **Transmission through infected blood:** usually 5–6 days (up to 13 days)	Asymptomatic period without clinical manifestations
2	Pre-hemorrhagic	On average, 2–4 days	• **Sudden, nonspecific symptoms:** fever, headache, muscle pain, dizziness, neck pain and stiffness, back pain, eye pain, sensitivity to light, and sore throat. • **Gastrointestinal disorders:** abdominal pain, nausea, vomiting, diarrhea. • **External signs:** hyperemia, conjunctivitis, jaundice. • **Neurological and other manifestations:** changes in mood (agitation), sensory perception, drowsiness, hepatomegaly, and splenomegaly.
3	Hemorrhagic	Short, usually 2–3 days	• **Hemorrhagic manifestations:** petechiae, widespread ecchymoses on the skin and mucous membranes, nosebleeds, bleeding gums, melena, hematemesis, hematuria, hemoptysis, bleeding at injection sites, from the vagina, uterus, and brain. • **Laboratory abnormalities:** thrombocytopenia, leukopenia, elevated levels of AST, ALT, LDH, CPK, and pro-inflammatory cytokines. • **Complications and risks:** hepatitis, acute renal, hepatic, and/or pulmonary failure, disseminated intravascular coagulation, multiple organ failure, shock. In fatal cases, death usually occurs during the second week.
4	Recovery (convalescence)	It begins approximately 9 days after the onset of the illness; full recovery can take up to 1 year	• **Symptoms of convalescence:** low blood pressure, tachycardia or bradycardia, polyneuropathy, breathing difficulties, dry mouth, vision and hearing problems, hair loss, memory loss, general weakness, headache, loss of appetite, and dizziness.

The hemorrhagic phase develops suddenly and usually lasts several days; however, it may last only one day or persist for more than a week. The first sign may be a petechial rash on the skin and mucous membranes. This may be followed by ecchymoses and bruises. Ecchymoses may be large; some of them cover most of a limb. Hematemesis, melena, nosebleeds, hematuria, hemoptysis, and bleeding (especially from venipuncture sites) are reported. Bleeding may also occur in other locations, including the eyes (subconjunctival and retinal hemorrhages) or the brain. In some cases, internal bleeding mimics acute appendicitis. Hepatomegaly and splenomegaly are common; hepatitis or jaundice may also be observed. Death is usually the result of hemorrhage, shock, or multiple organ failure. For survivors, the recovery period can be prolonged, especially in severe cases, and is characterized by marked general weakness, dry mouth, headaches, nausea, poor appetite, polyneuropathy, blurred vision, hearing and memory loss, temporary hair loss, and hepatorenal failure. The effects of CCHF on pregnancy have not been fully studied. Some pregnant women have had miscarriages, while others have given birth to healthy, uninfected children, especially when the course of the disease was relatively mild (Papa et al., 2004; CFSPH, 2019).

Regarding the characteristics of morbidity in children and adults, the main general differences are presented in Table 2.

Table 2

Comparison of the clinical course of CCHF in adults and children

Clinical and laboratory findings	Adult patients	Children (patients under 18 years of age)
Severity of the course	The course of the disease is most often severe or moderately severe; there is a high risk of multi-organ complications.	The course of the disease is predominantly mild or moderate; the incidence of severe complications is significantly lower.
Main symptoms	Severe myalgia, arthralgia, intense headache, and frequent gastrointestinal bleeding.	Fever, anorexia, nausea, vomiting, and abdominal pain are the predominant symptoms; neurological and hemorrhagic manifestations are less pronounced.
Laboratory changes	Severe thrombocytopenia, leukopenia, and a sharp increase in liver transaminase (AST, ALT) and creatinine levels.	Mild thrombocytopenia and leukopenia; laboratory markers of liver cytolysis are significantly lower than in adults.
Mortality rate	High; on average, it ranges from 10% to 30%, depending on the strain and the quality of intensive care.	Significantly lower, usually between 2 and 5 percent (or less).

At the same time, the clinical course of CCHF varies significantly not only by patient age but also by geographic region, due to differences in viral genotypes, characteristics of medical care, and other factors. For example, in Turkey, a classic pattern is observed—the disease is significantly milder in children than in adults, with a low overall case-fatality rate (Table 3) (Hoogstraal, 1979; Bente, 2013; Blair, 2019; CFSPH, 2019). In Iran, by contrast, a high proportion of severe cases was reported among children, with a case-fatality rate of up to 26%. In the former USSR, no statistically significant difference in disease severity was found between children and adults. As for current trends, in recent years, thanks to improved screening, mild and asymptomatic cases are being reported more frequently, although some of them remain etiologically undiagnosed (Blair, 2019; CFSPH, 2019).

Table 3

Comparative Analysis of Geographic Differences in the Course of CCHF in Children

Country / Research	Characteristics of the Clinical Course in Children	Child mortality
Turkey	Milder course compared to adults, minimal hemorrhagic manifestations	Low (mostly < 5%)
Iran	A high proportion of severe cases, marked multi-organ dysfunction	Up to 26%
Former USSR	The same severity in children and adults	It depended on many factors
Global Trends	Increase in the proportion of resolved, mild, and asymptomatic cases	It varies depending on the region and the virulence of the virus

Diagnostics. Various laboratory methods can be used to diagnose CCHF, and the biosafety level (BSL) requirements for conducting diagnostic tests vary depending on the method used and the country (depending on the risk level assigned to CCHFV in that country). In some countries, notably France, Germany, Italy, Sweden, Switzerland, and the United Kingdom, BSL-4 precautions are recommended for diagnostic testing for CCHFV, while other countries recommend BSL-3 or

BSL-2 precautions (Fernandez-Garcia, 2014; Weidmann, 2016). In Ukraine, CCHFV is classified as a Containment level 4 pathogen.

Diagnosis in animals. Laboratory tests for CCHFV are not typically used for direct clinical diagnosis in animals due to the asymptomatic nature of the infection. Serological methods primarily serve an epidemiological purpose in assessing seroprevalence in endemic regions to evaluate the risk of transmission to humans. The primary method used for this purpose is the enzyme-linked immunosorbent assay (ELISA). In addition, complement fixation tests (CFT) and immunofluorescence assays (IFA) are used. Viremia in animals is confirmed by virus isolation and reverse-transcription polymerase chain reaction (RT-PCR). The duration of viremia is approximately one week in farm animals and 2 to 15 days in small laboratory or wild mammals (Vanhomwegen et al., 2012; Papa et al., 2015; Leblebicioglu, 2016; Blair, 2019; CFSPH, 2019).

Diagnosis in humans. Verification of a diagnosis in humans is based on the detection of the pathogen, its components in biological samples, or antibodies (Table 4).

Table 4

Characteristics of CCHF Diagnostic Methods

Research methodology	What it detects / Purpose	Optimal timeframes and key features	The material under study
RT-PCR and isothermal amplification (LAMP)	Viral RNA	From the very first days of the illness; RNA levels rise as the disease progresses	Blood, plasma, biopsy specimens
Virus isolation (cell culture)	Live virus (on SW-13, Vero, BHK-21, CER, and LLC-MK2 cell lines)	Most effective during the first 5–6 days of the illness	Blood, biological fluids, tissues
Antigen-based ELISA	Viral antigen proteins	Early stages; become inaccessible after antibodies appear	Blood (serum)
Serological methods (ELISA, RIF)	Specific antibodies (IgM, IgG)	They appear 7–9 days after the onset of symptoms; low titers are observed in fatal cases	Paired blood sera
Immunohistochemistry (IHC)	Viral antigens in parenchymal organs	Postmortem or retrospective diagnosis	The patient's tissues following an autopsy
Bioassay on laboratory animals	Live virus (in newborn or immunodeficient mice)	High sensitivity, but rarely used due to biosafety risks	Blood, organ suspensions
Limited/outdated methods (CFT, HIT, neutralization reaction)	Antibodies or the virus	They are rarely used due to low sensitivity (CFT/HIT) or the high risks associated with working with live viruses	Blood serum

The main methods include virus isolation, detection of viral RNA using RT-PCR, and immunological detection of antigens in blood or tissue samples. Urine, saliva, and other secretions may also contain viral nucleic acids; however, their diagnostic value has not yet been sufficiently studied. During pathological examination (autopsy), CCHFV is successfully detected in parenchymal organs and tissues, particularly in the liver, spleen, lungs, bone marrow, kidneys, and brain (Shepherd, 1986; Vanhomwegen, 2012; CFSPH, 2019).

The diagnosis of CCHF is based on a combination of RT-PCR and serological methods, and biosafety measures when handling biological samples from patients are subject to regular review (Weidmann, 2016). Due to the high variability of CCHFV strains, many RT-PCR tests detect only specific local variants of the virus. At the same time, there are universal test systems capable of detecting most known variants of the virus, including the highly divergent AP92 strain. Other nucleic acid detection methods include micro- and macrochip-based assays, as well as loop-mediated

isothermal amplification (LAMP). As the disease progresses clinically (especially prior to a fatal outcome), the concentration of viral RNA typically increases significantly.

Isolation of the live virus is most effective during the first week of the illness (primarily in the first 5–6 days); however, in severely ill patients, high viral titers may persist for longer. To isolate CCHFV, the following cell culture lines are used: SW-13, Vero, CER, LLC-MK2, and BHK-21. The identified viral isolates are characterized using RIF or RT-PCR. The virus neutralization test is rarely used due to strict biosafety requirements when working with live pathogens.

The method of intracerebral infection of newborn or immunodeficient mice is highly sensitive (surpassing cell cultures); however, in modern clinical practice, it is used only in the absence of better alternatives due to biosafety risks. ELISA is effective for detecting viral antigens in the blood during early stages, but not after the appearance of specific antibodies. Immunohistochemical (IHC) studies are used for retrospective or postmortem diagnosis using biopsy or autopsy specimens collected during an autopsy. As for methods for detecting antibodies, the main ones are ELISA and the indirect immunofluorescence assay (IFA). Outdated methods (the complement fixation test (CFT) and the hemagglutination inhibition test (HIT)) are no longer recommended due to their low sensitivity. Specific antibodies are usually detectable 7–9 days after the onset of clinical symptoms (sometimes earlier). In patients who die, antibody titers are often low or absent, as death occurs before a full-blown humoral response can develop. Qualitative or quantitative IgM testing and paired serum testing are performed to detect an increase in IgG titers (Vanhomwegen, 2012; CFSPH, 2019; Raabe, 2020).

Treatment, prevention, and control measures. Measures to combat CCHF can be divided into therapeutic, veterinary, and epidemiological measures; personal protection and hygiene; anti-epidemic measures in medical and preventive care facilities; and specific prevention (Table 5).

The cornerstone of patient management for CCHF is pathogenetic and symptomatic therapy. Severely ill patients require admission to an intensive care unit. Specific drug treatments include ribavirin and hyperimmune serum. Although ribavirin is widely used, its clinical efficacy remains a subject of debate. Like any other specific treatment, it is most effective when administered as soon as possible after the onset of symptoms (ideally within the first 48 hours) (Johnson, 2018). It is also used for post-exposure prophylaxis in individuals at high risk of severe disease. Hyperimmune serum is used in some countries for treatment and post-exposure prophylaxis (particularly in Bulgaria), although clinical data on its efficacy are limited (Papa, 2004, 2015; Leblebicioglu, 2012, 2016). Currently, the specific activity of favipiravir against CCHFV (both alone and in combination with ribavirin) is being actively studied; it has shown promising results in experimental animal studies, but clinical trials involving humans have not yet been conducted (CFSPH, 2019).

There are no licensed preventive vaccines against CCHF. Historically, an inactivated mouse brain vaccine was used in the former Soviet Union and Bulgaria; however, data on its efficacy are limited (Papa, 2004, 2015). Nevertheless, research aimed at developing vaccines against this infection is actively underway. Various modern approaches are being used for this purpose. In 2019, the WHO published a preliminary draft roadmap for research and development aimed at combating CCHF, which prioritized the development and implementation of the most essential medical measures in countries affected by this infection. The specific objectives, milestones, and timeframes of the roadmap have been updated and expanded to reflect scientific progress, including the identification of monoclonal antibodies with therapeutic potential and the advancement of four vaccine candidates to Phase I clinical trials. The characteristics of the vaccine candidates, experimental vaccines, and the vaccine used in Bulgaria, as mentioned above, are presented below (Table 6). Among the vaccine candidates, it is worth noting the vector-based vaccines, for which clinical trials have already begun. One of these vaccines (ChAdOx2 CCHF) was developed by the University of Oxford using technology similar to that of the Oxford/AstraZeneca COVID-19 vaccine, in which an adenovirus is used as the vector (Saunders, 2023; University of Oxford, 2024). Another vector-based vaccine is based on the attenuated (weakened) Modified Vaccinia Ankara (MVA) virus, which has lost its ability to replicate in mammalian cells. A gene encoding the CCHFV surface glycoprotein has been inserted into the MVA genome (Dowall, 2016). DNA, mRNA, and subunit vaccines are currently in the experimental or preclinical trial stages (Garrison, 2017; Hawman, 2021; Appelberget, 2022).

Table 5

Overview of Measures to Combat CCHF

Areas of Focus and Activities	Measures and strategies	Features and execution modes
Veterinary and epidemiological measures	• Control of vectors. • Quarantine measures in animal husbandry. • Processing of meat products.	• Use of acaricides to eliminate ticks on animals. • Keeping ostriches tick-free for 14 days prior to slaughter (and a 30-day quarantine prior to export). • Aging meat at a temperature of 4–8 °C for 24 hours for post-slaughter acidification (virus inactivation). Prohibition on the consumption of raw meat and unpasteurized milk.
Personal protection and hygiene	• Protection against tick bites. • Protection during contact with animals.	• Use repellents and wear protective clothing (pants tucked into boots). Perform regular self-examinations and carefully remove ticks with protected hands. • Use gloves and protective clothing when handling blood or tissues from farm animals. Maintain the surrounding environment (mow the grass).
Medical care and intensive care	• Pathogenetic therapy. • Specific medications\	• Supportive care for critically ill patients in intensive care units. • Ribavirin (most effective within the first 48 hours). • Hyperimmune serum (in certain countries, such as Bulgaria). • Experimental treatments: favipiravir (tested only on animals).
Epidemic prevention measures in healthcare facilities	• Barrier precautions. • Protection against aerosols.	• Use of standard barrier precautions, N95 respirators (or equivalent), and eye protection during procedures that generate aerosols. • Placement of patients in separate, well-ventilated rooms or isolation suites. • Adherence to protocols for the safe burial of the deceased.
Specific prevention	• Post-exposure prophylaxis. • Vaccination.	• Post-exposure administration of ribavirin or hyperimmune serum to individuals at high risk of infection. • There are no widely available commercial vaccines (historically, an inactivated mouse brain vaccine was used in the USSR and Bulgaria, but data on its use are limited).

Among the main obstacles to completing vaccine development, the following economic factors should be noted: a lack of interest on the part of the commercial pharmaceutical industry due to low market potential compared to high development costs (Dowall, 2016); the difficulties in organizing Phase III clinical trials due to the sporadic nature of outbreaks; the limited availability and complexity of animal models (the need for specialized immunodeficient mice or monkeys), as well as strict requirements for working with the virus in BSL-4 laboratories, which significantly impact preclinical trials (Garrison, 2019); the high heterogeneity of the CCHFV genome and the difficulty of inducing cross-immunity that would provide equal protection against virus variants from different geographic regions (Suschak, 2021).

Table 6

Characteristics of the vaccine used against CCHF and vaccine candidates

Name / Type of content creation platform	Vaccine / vaccine candidates	Mechanism of action and vector	Advantages	Key issues and challenges	Implementation/Testing Status	Literary source
Inactivated (1st generation)	Mouse-brain vaccine	An inactivated virus derived from the brain tissue of newborn mice	The only locally administered vaccine (in use in Bulgaria since 1953)	Outdated technology; low immunogenicity; risk of side effects; not approved by the FDA or EMA	Local license (Bulgaria)	Papa A., 2011
Vector (MVA)	MVA-CCHF	A modified, attenuated Ankara vaccine virus with the CCHFV glycoprotein genome	High safety profile; stimulates robust T-cell and humoral immunity	Requires confirmation of protection against various CCHFV genotypes	Phase I clinical trials	Dowall, 2016
Viral (ChAdOx)	ChAdOx2 CCHF	Chimpanzee adenovirus vector carrying the CCHFV glycoprotein genome	A strong immune response after a single dose; no neutralizing antibodies against the vector in humans	The risk of reduced efficacy with repeated boosters using the same vector	Preclinical / Phase I clinical trials	Saunders, 2023; University of Oxford, 2024
DNA vaccines	pVAX-CCHFV	Plasmid DNA encoding the glycoprotein precursor and the CCHFV nucleoprotein	High temperature stability; simple and cost-effective scalable production	Immunogenicity in humans is unknown; a vaccination series is required	Efficacy was demonstrated in a cynomolgus macaque model	Garrison, 2017, Hawman, 2021
mRNA vaccines	mRNA-LNP CCHF	Messenger RNA in lipid nanoparticles encoding a vaccine antigen	Speed of development; high immunogenicity and adaptability to viral mutations	The need for strict adherence to the cold chain	Preclinical studies	Appelberg, 2022
Subunit	Recombinant Gn/Gc	Purified recombinant viral antigen proteins combined with an adjuvant	Maximum safety (no live or vector-borne material)	Requires the mandatory use of adjuvants and the administration of several booster doses	Preclinical studies	Saunders, 2023

Since infected farm and wild animals are primarily asymptomatic carriers of the disease, prevention efforts focus on minimizing the risk of transmission to humans and preventing the pathogen from being introduced into areas where there is a risk of its establishment. This includes vector control (the use of acaricides is the primary method for reducing tick populations); quarantine measures in animal husbandry (Papa, 2015; Leblebicioglu, 2016; Blair, 2019; CFSPH, 2019), and proper processing of meat products. It is essential to avoid consuming unpasteurized milk and raw meat, as well as to follow hygiene guidelines during food preparation.

Personal protective measures include the use of repellents (particularly those containing DEET (N,N-diethyl-meta-toluamide)) and wearing protective clothing (long sleeves, pants tucked into boots) while in areas where ticks may be present; regular self-examination of the skin and clothing; ticks should be removed without direct contact with bare hands; use of personal protective equipment (rubber gloves, protective clothing) when coming into contact with the blood, tissues, or bodily fluids of farm animals. The area surrounding the residence must be maintained on an ongoing basis (mowing tall grass, removing shrubs, applying insecticides). In healthcare facilities, compliance with infection control requirements is essential, and in diagnostic laboratories, strict biosafety and biosecurity protocols must be followed.

Regarding infection control, the WHO has now developed relevant guidelines which, among other things, state that a single case of CCHF is already considered an outbreak of this infection (WHO, 2024).

Conclusions.

1. Crimean-Congo hemorrhagic fever is one of the most dangerous arboviral infections with a wide geographic range, sustained by the circulation of Hyalomma spp. ticks and a natural reservoir consisting primarily of mammals.

2. The main threat to humans comes from tick bites, as well as direct contact with the blood of infected animals and people, which poses a high risk of occupational diseases and nosocomial transmission.

3.Effective control of CCHF requires a comprehensive approach: early molecular-genetic and serological diagnosis, timely initiation of pathogenetic and specific treatment (ribavirin), as well as strict adherence to biosafety measures in veterinary medicine and the healthcare system.

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Особливості епізоотології, епідеміології, діагностики та профілактики Крим-Конго геморагічної гарячки

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Резюме. *Крим-Конго геморагічна гарячка (CCHF) є особливо небезпечною зоонозною арбовірусну інфекцію, спричиненою РНК-вміщуючим вірусом CCHF (CCHFV) роду Orthonairovirus родини Nairoviridae. Хвороба характеризується широким географічним ареалом, що охоплює понад 50 країн Африки, Азії та Європи, включаючи й регіони України. Природна циркуляція збудника забезпечується складними міжвидовими зв'язками, де основними векторами та довготривалими резервуарами виступають іксодові кліщі роду Hyalomma, здатні до трансфазової та трансоваріальної передачі вірусу. Додатковими ампліфікаторами вірусу є свійські й дикі ссавці (велика та дрібна рогата худоба, гризуни, зайці), а також страуси, у яких інфекційний процес зазвичай протікає безсимптомно, тоді як перелітні птахи сприяють розширенню ареалу, транспортуючи інфікованих кліщів. Шляхи зараження людини охоплюють як трансмісивну передачу через укуси кліщів, так і контактний шлях – під час взаємодії з кров'ю, біологічними рідинами чи тканинами інфікованих тварин у процесі їх догляду або забою. Крім того, існує високий ризик передачі вірусу від людини до людини, зокрема внутрішньолікарняної при безпосередньому контакті з геморагічними виділеннями пацієнтів у гострій фазі захворювання. Допоміжними шляхами зараження вважаються аліментарний (через споживання сирого м'яса або непастеризованого молока) та статевий. Патогенез та клінічний перебіг CCHF у людей мають фазовий характер. Після інкубаційного періоду розвивається передгеморагічна фаза з вираженим загальноінтоксикаційним синдромом, що згодом переходить у геморагічну фазу, яка супроводжується петехіальним висипом, екхімозами, множинними внутрішніми й зовнішніми кровотечами, тромбоцитопенією та ризиком розвитку синдрому дисемінованого внутрішньосудинного згортання і поліорганної недостатності. Середній рівень летальності становить 11,7%. Діагностика захворювання ґрунтується на застосуванні молекулярно-генетичних методів (RT-PCR) у перші дні віремії, а також серологічних досліджень (ІФА) для детекції антигенів і специфічних антитіл класів IgM та IgG. Усі маніпуляції зі збудником вимагають високого рівня біобезпеки (BSL-3/BSL-4). Терапевтична стратегія передбачає інтенсивну патогенетичну та підтримувальну терапію в поєднанні з раннім застосуванням противірусних препаратів, зокрема рибавірину. У статті наведена детальна характеристика існуючих вакцин-кандидатів. Зважаючи на відсутність комерційних вакцин, ключову роль у запобіганні спалахам відіграє специфічна профілактика: акарицидна обробка тварин, використання індивідуальних засобів захисту, дотримання харчової гігієни та суворе впровадження заходів інфекційного контролю в закладах охорони здоров'я.*

Ключові слова: Крим-Конго, Bunyavirales, полімеразна ланцюгова реакція (ПЛР), імуноферментний аналіз (ІФА), епізоотична ситуація, діагностика, оцінка ризиків.

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