European Journal of Internal Medicine 149 (2026) 106946

Contents lists available at ScienceDirect

European Journal of Internal Medicine

journal homepage: www.elsevier.com/locate/ejim

Review Article

Human infection with Andes hantavirus: an update for the general physician

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ARTICLE INFO

Keywords:
Outbreak
Viral infection
Hantavirus
Epidemiology
Prevention

ABSTRACT

Orthohantaviruses are zoonotic RNA-viruses transmitted to humans through inhalation of aerosols contaminated by infected rodent excreta. Among hantavirus species, Andes virus (ANDV) owns unique capacity for sustained human-to-human transmission, occurring via respiratory droplets and prolonged close contact with symptomatic individuals, with a median reproductive number exceeding 2 and an incubation period ranging from 9 to 40 days.

ANDV infection can present with a wide range of clinical manifestations. Of them, the most feared is Hantavirus cardiopulmonary syndrome (HCPS), a severe condition characterised by acute respiratory failure, haemodynamic instability, acute kidney injury, hepatic involvement, and dysregulated cytokine release, with high lethality. Disease severity correlates with the degree of neutrophilia, leukocytosis, lymphopenia, thrombocytopenia, and elevated lactate dehydrogenase. No approved antiviral therapy or vaccine currently exists; management remains entirely supportive, centred on oxygen supplementation, haemodynamic stabilisation, and renal replacement therapy when indicated. Case fatality rates reached 32% in a 2018–2019 outbreak, with death occurring a mean of 6.7 days from symptom onset.

The April 2026 outbreak aboard the MV Hondius cruise ship — involving passengers of 23 nationalities, with 8 cases (6 confirmed, 2 suspected), and 3 deaths reported as of 11 May 2026 (CFR 38%) — exemplifies how a zoonotic pathogen with limited human-to-human transmissibility can rapidly achieve global reach in the era of mass international travel, underscoring the urgent need for clinician awareness, prompt contact tracing, and internationally coordinated outbreak preparedness.

1. Current epidemiologic relevance of hantaviruses

Orthohantaviruses are RNA viruses belonging to the genus *Hantavirus* within the family *Bunyaviridae* and are enzootic viruses whose definitive hosts are wild rodents [1].

Occasionally, hantaviruses may spread to humans, usually through inhalation of aerosols contaminated by rodents' feces, urines or saliva, largely in the context of rural or gardening activities [2]. Removal of dust/litter contaminated by rodent excrements is a typical context where contagion occurs [3].

Depending on the rodent species and its geographical prevalence, different hantavirus-related clinical syndromes can be observed, including haemorrhagic fever with renal syndrome (HFRS), epidemic nephropathy ('*nephropathia epidemica*', NE) [4], and hantavirus cardio-pulmonary syndrome (HCPS), each associated with specific viral species and characterized by variable case fatality rates [5–7] (Fig. 1).

Being zoonotic in origin, hantaviruses are traditionally considered to associate with a very low risk of epidemic spread within humans. However, human-to-human transmission has been documented for Andes virus (ANDV) [8] circulating across south America (especially Argentina, Chile) [9] and living within the *Oligoryzomys longicaudatus* rodent host [7,10] (Fig. 2). Several limited outbreaks, mostly confined to the country of endemic spread, have been described over the last 3 decades [3,5,11].

A very recent small outbreak arising on the Dutch MV Hondius – a cruise ship devoted to extreme voyages to polar regions – is sparking fear

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https://doi.org/10.1016/j.ejim.2026.106946
Received 11 May 2026; Accepted 12 May 2026
Available online 26 May 2026

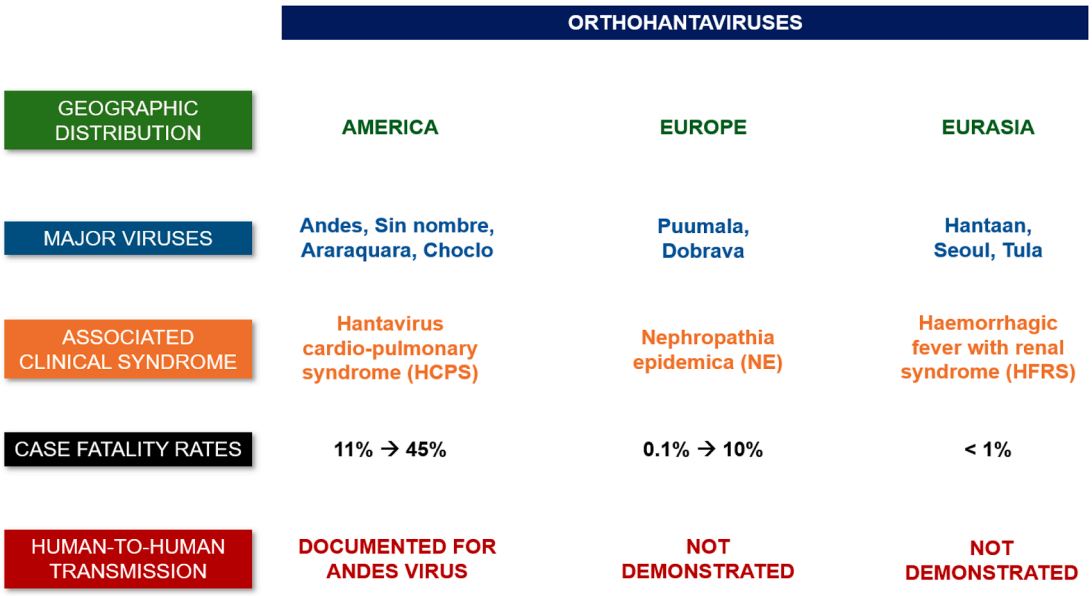

Fig. 1. Overview of the geographic distribution of orthohantaviruses and their associated clinical syndromes, case fatality rates, and evidence of human-to-human transmission. The figure summarizes the principal orthohantavirus species circulating in the Americas, Europe and Eurasia, and the corresponding human diseases—hantavirus cardio-pulmonary syndrome (HCPS), nephropathia epidemica (NE) and haemorrhagic fever with renal syndrome (HFRS). Reported case fatality rates vary by region and viral species, with documented human-to-human transmission limited to Andes virus.

Fig. 2. Illustration of a rodent species recognized as a natural reservoir of ortohantaviruses. Rodent hosts play a central role in ecology and transmission of hantaviruses to humans through exposure to contaminated excreta and aerosolized particles.

among citizens and public health authorities. Sustained by ANDV, this outbreak has the inherent features to become an infectious threat for several reasons: contacts on board within limited indoor spaces has definitely favoured spread; several passengers had already disembarked before the outbreak was detected, behaving as possible unintentional vehicles of the infection at their homes; some of these passengers have travelled by aircraft without any precautions; it is as yet unknown whether the virus subtype responsible for this outbreak has standard reproduction number and if the outbreak has ordinary attack and case fatality rates.

These issues suggest that a reappraisal of Andes hantavirus infection features is clearly warranted.

2. Andes hantavirus: epidemiology and transmission

ANDV is an enveloped, single-stranded, negative-sense RNA virus, whose segmented genome (3 parts, small, medium and large) encodes for nucleocapsid protein, two glycoproteins, and the RNA-dependent RNA polymerase [1,10] (Fig. 3). ANDV natural host is the long-tailed pygmy rice rat (*Oligoryzomys longicaudatus*) (Fig. 2) living primarily in the temperate forests, shrublands, and river margins of Chile and southwestern Argentina, justifying its South-American endemicity [7]. Infection is usually chronically persistent and mild or non-pathogenic in the definitive host, implying infected rodents disseminate the virus via saliva, urine, and faeces, throughout their lives [12–14]. Human infection is therefore rare outside of close animal / animal droppings contact,

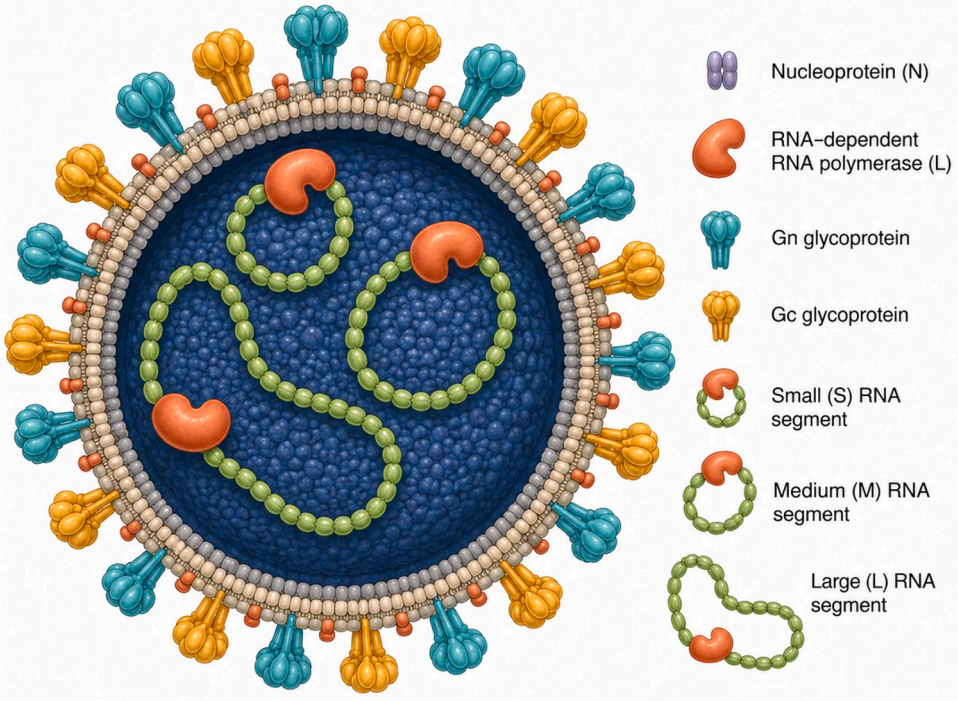

Fig. 3. Schematic representation of the hantavirus virion structure. The enveloped particle contains three negative-sense single-stranded RNA segments (small, medium, and large) associated with nucleoprotein (N) and the RNA-dependent RNA polymerase (L). The viral envelope is studded with Gn and Gc glycoprotein complexes involved in host-cell attachment and membrane fusion.

and follows either rodent bites or inhalation of dust contaminated by dried rodent dirt (infectious aerosols) [3,7,12].

Human-to-human transmission of ANDV has been consistently described [11,15]. Epidemiologic analyses and genomic comparisons allowed to identify inhalation of droplets or aerosolized virions as the routes of person-to-person transmission. This is deemed possible only after prolonged and close contact with symptomatic persons [9,11,15]. Other hantavirus species demonstrated vertical transmission during pregnancy [16], while ANDV has been responsible for transmission via breast milk [17] and kissing [9], and efficient transmission during sexual intercourse has also been reported. Sharing bedroom at the time of viral prodrome (see below) or shortly thereafter seems to be an efficient source of contagion [7,9,10].

In the context of one of the largest described outbreaks [11], the median reproductive number (i.e. the number of secondary cases caused by an infected person during his/her infectious period) was >2 (up to 6 initially) and the mean (±SD) serial interval (i.e. the time from symptom onset in a primary case to symptom onset in an epidemiologically-linked secondary case) was approximately 23±7 days. Patients with a higher viral load and showing signs of liver injury were more likely than other patients to spread infection. ANDV incubation appears to range from 9 to 40 days [11].

Seasonality may be observed as a consequence of rapid growth of *Oligoryzomys longicaudatus* flocks [18]: this can be fuelled by blooming and seeding of bamboo species, translating into increased spread of ANDV during spring and summer [7,19]. Rodent population cycles are a key predictor of hantavirus epidemic periodicity in humans [18]; mathematical modelling confirms that these seasonal dynamics are a major driver of outbreak occurrence [19].

3. Pathophysiology and clinical presentation

Respiratory infection is a direct consequence of airborne viral spread and associates with ANDV dissemination [11]. In addition to the lungs, ANDV efficiently infects endothelial cells, primarily those of the pulmonary capillary [5]. Endothelial cell infection translates into increased vascular permeability responsible for most disease manifestations [20], and is largely due to virus-induced functional changes rather than direct viral-induced cell injury. Current understanding suggests that endothelial dysfunction could be triggered by binding of the virus to cell receptors modulating permeability, platelet activation and immune-inflammatory responses [5,21]. Endothelial cell involvement encompasses vascular beds of the heart, liver, kidney and peripheral circulation, causing the typical systemic compromise of hantavirus syndromes [5]. Consistently, epidemiological data indicate that hantavirus infection is associated with an increased risk of acute myocardial infarction and stroke, likely reflecting systemic endothelial injury and prothrombotic activation [22].

Table 1
Severity of respiratory involvement in Andes hantavirus disease.

GRADE I	PRODROMAL SYMPTOMS WITHOUT ANY CLINICALLY RELEVANT RESPIRATORY COMPROMISE
GRADE II	Mild to moderate respiratory failure without hemodynamic compromise
GRADE III	Severe respiratory failure with hemodynamic compromise
GRADE IV	Severe respiratory insufficiency and hemodynamic compromise, refractory to treatment (usually associated with fatal outcome)

ANDV can cause a range of clinical manifestations spanning from asymptomatic or mild febrile illness to severe forms of HCPS [2,12]. Cardiopulmonary symptoms are often preceded by a 2-to-7-day prodromal phase, characterized by fever in almost all cases, weakness in two-thirds, headache or myalgia in more than half, and gastrointestinal manifestations (in as many as 55% of cases, and including nausea, vomiting, diarrhoea and abdominal pain). Lumbar pain and ocular symptoms are also common. Few hours or days after the onset, acute respiratory distress often ensues, showing highly variable severity [2,11].

In the context of the most recent and large human outbreak [11], respiratory failure occurred in 76% of cases. Some patients showed

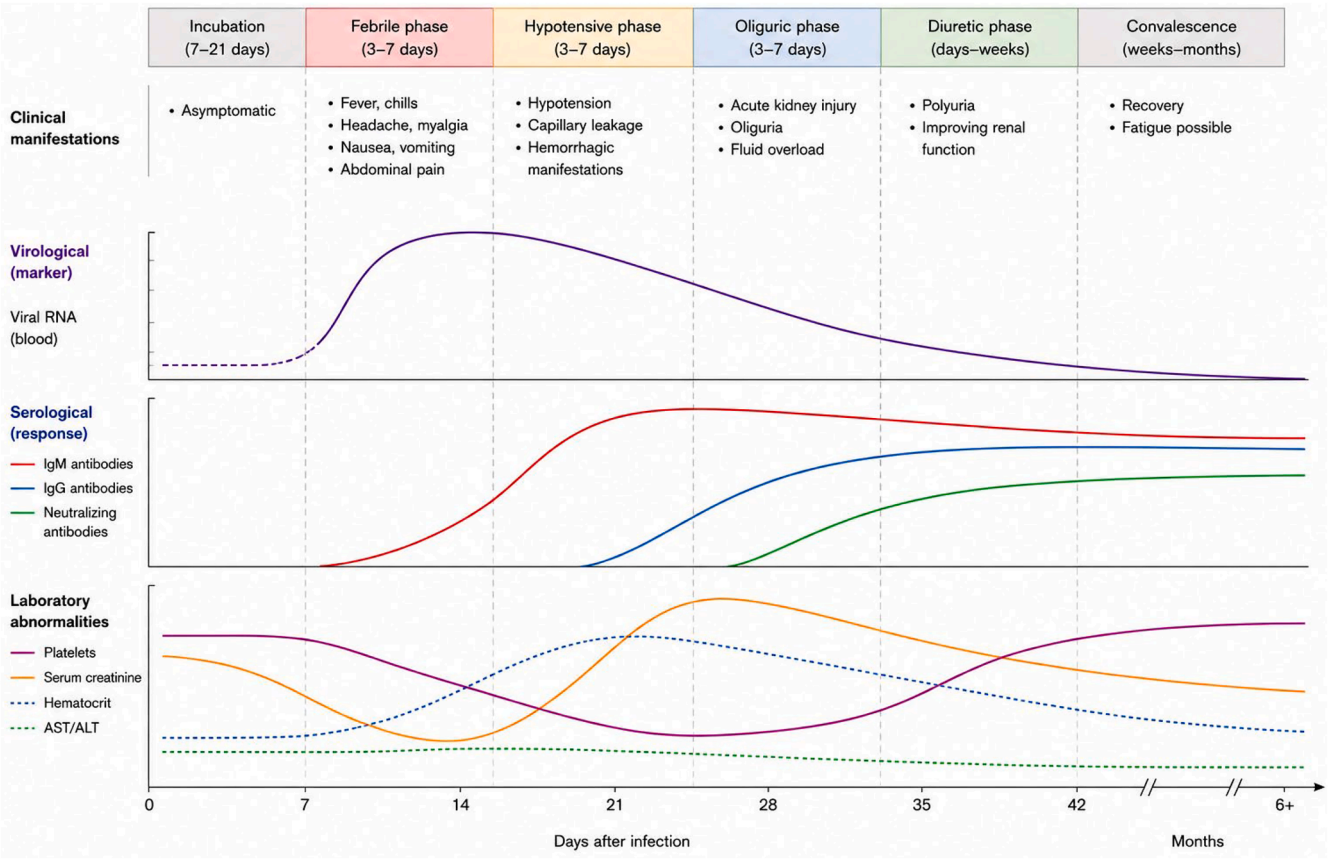

Fig. 4. Clinical, virological, serological, and laboratory timeline of hantavirus infection. Viral RNA levels peak during the febrile and hypotensive phases and progressively decline thereafter, whereas IgM antibodies appear early and are followed by sustained IgG and neutralizing antibody responses. Representative laboratory alterations include thrombocytopenia, increased serum creatinine, hemoconcentration, and mild elevations of liver enzymes during the acute phases of disease.
AST, aspartate aminotransferase; ALT, alanine aminotransferase.

relatively mild hypoxaemia requiring supplemental oxygen, whereas more severely ill subjects quickly developed progressive pulmonary oedema with hypotension and severe hypoxia, leading to haemodynamic compromise and need for intubation and intensive care support (Table 1) [11] A substantial proportion of these severely ill cases eventually died due to mixed vasogenic and cardioplegic shock in the context of severe inflammatory response. Capillary leak syndrome may be severe enough to also cause hypovolemia, contributing to shock [5,7]

Cardiopulmonary compromise is usually heralded by cough, shortness of breath, palpitations and severe malaise due to hypotension, associated with signs of thrombocytopenia, including petechiae and a purpuric rash. HCPS either leads to death within 24–48 h or quickly resolves [2].

Fig. 4 summarizes clinical and biochemical course of ANDV infection.

Thrombocytopenia is associated with acute kidney injury [23,24]; in severe cases, coagulation abnormalities may progress to disseminated intravascular coagulation, which further worsens prognosis and organ damage [25]. Renal swelling due to severe organ inflammation is accompanied by interstitial haemorrhage, microvascular inflammation, and peritubular capillaritis, with a syndromic presentation consistent with acute tubular necrosis [24,26]. Hematuria, proteinuria – sometimes in the nephrotic range – and a drop in glomerular filtration rate occur initially [24,27], and can require renal replacement therapy. Resolution associates with hypertension, polyuria and hyponatremia [23]. In addition to thrombocytopenia and rising creatinine, increased lactic dehydrogenase levels, transaminases, immunoblast counts and hematocrit can be seen at haemato-chemistry [7,11]. Liver involvement with organ swelling and rise in liver injury markers is also often observed, and was almost universal in the recent Argentina outbreak [5, 11].

Leukocytosis, neutrophilia, lymphopenia and thrombocytopenia are observed in most patients. Haematological changes have prognostic implications, as disease severity associates with higher prevalence and severity of neutrophilia (OR 8.8; 95% CI. 2.5–72), leukocytosis (OR 3.7; 95% CI. 1.7–10), lymphopenia (OR 3.0; 95% CI 1.2–10), thrombocytopenia (OR, 1.9; 95% CI. 1.3–3.3) and elevated LDH (OR, 1.3; 95% CI. 1.1–1.5) [11]. This pairs with cytokine changes, with significant upregulation of interferon-γ, interleukin (IL)-4, IL-6, IL-16, and tumour necrosis factor (TNF)-α, and downregulation of TNF-β, IL-9, and platelet-derived growth factor [7]. Overall, ANDV-associated HCPS may progress to a dysregulated cytokine release syndrome or “cytokine storm” [11,24,28].

In the 2018–2019 outbreak, the overall case fatality rate was 32% (11 of 34 patients). Death occurred rather fast, with a mean time from symptom onset to decease of 6.7 days (interquartile range, 4 to 7) [11]. Based on existing (limited) knowledge, the severity of HCPS depends on the patient's immune response pattern and genetic factors and, possibly, viral strain pathogenicity [2].

4. Etiological diagnosis

The clinical suspicion of orthohantavirus infection is based on the epidemiological context and is often very hard to raise outside of endemic areas [9,24]. Laboratory confirmation usually comes from a positive serology, where both IgM and IgG are found to be positive [7,

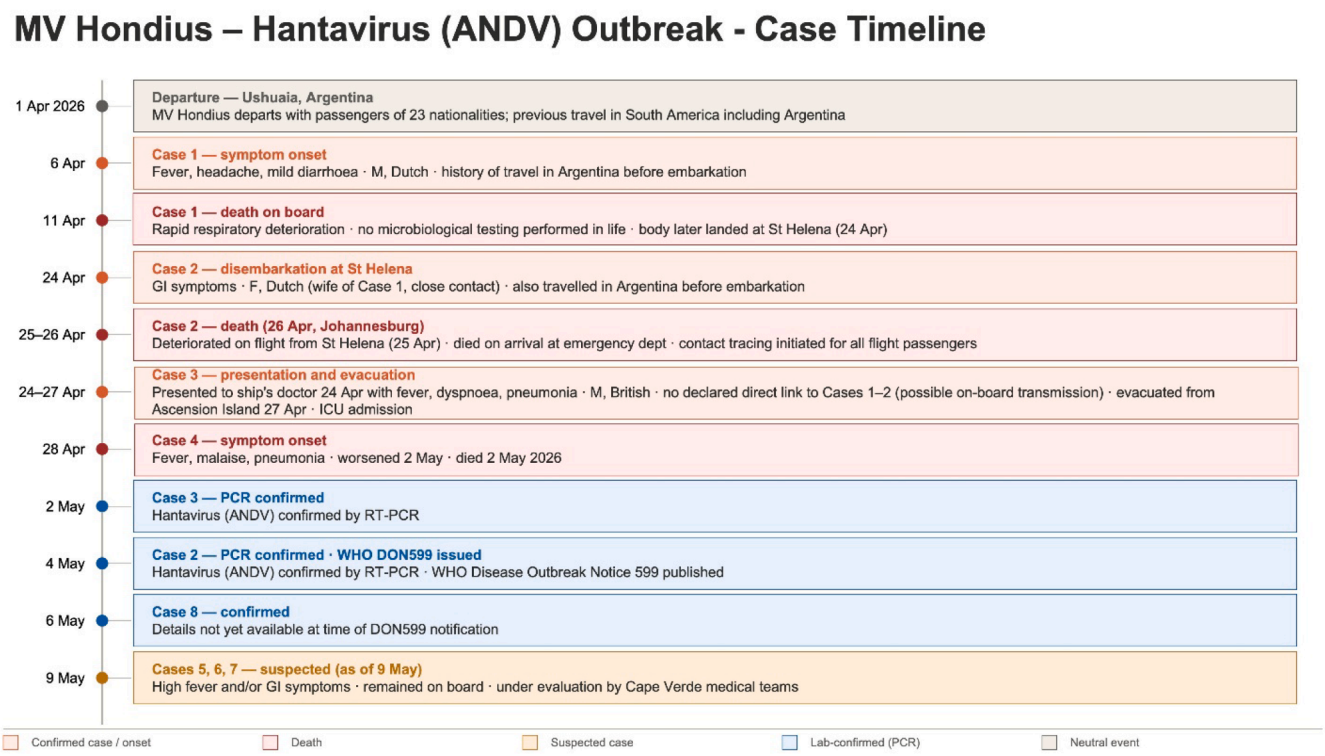

Fig. 5. Epidemiological timeline of confirmed and suspected hantavirus (Andes virus, ANDV) cases associated with the MV Hondius outbreak, April–May 2026. Each event is plotted chronologically along the vertical axis. Colour coding denotes case classification: confirmed cases with symptom onset (coral), fatal outcomes (red), suspected cases (amber), and laboratory-confirmed cases by RT-PCR (blue). Cases 1 and 2 (Dutch couple) shared a common exposure history in Argentina prior to embarkation, consistent with a primary zoonotic source. Case 3 (British male) had no declared epidemiological link to Cases 1–2, suggesting secondary human-to-human transmission on board. Cases 4–8 present onset dates temporally compatible with an incubation period of 2–4.
M, male; F, female; GI, gastrointestinal, ICU, intensive care unit; RT-PCR, reverse transcription polymerase chain reaction.

23] [Fig. 4]. Later in the course of the disease, IgM titres wane with rise of IgGs [29,30]. Multiple commercially available assays show similar diagnostic performance, including enzyme immunoassays, immunofluorescence assays, immunoblot assay and immunochromatography assay [7,31]. Serological cross-reactivity is common but does not significantly affect diagnostic accuracy (considered to be >95%) outside of endemic areas, where very limited seroprevalence exists [31,32].

Molecular diagnostic confirmation can be sought, when needed, by detection of viral RNA through RT-PCR, that is positive on whole blood and plasma very early in the disease course, with an average viral load of 4 to 5E+07 copies/mL [11,20,29]. Viremia is however short-lived and RT-PCR can become negative after few weeks from onset [10,29].

5. Supportive care and treatment

The first and most important management strategy is infected patient isolation. Considering the time of incubation, a real 'quarantine', i.e. isolation for up to 40 days, appears wise in close contacts of infected patients [9,11].

Treatment hinges on management of oxygen, fluids and electrolytes, and is therefore largely symptomatic or supportive. Patients with mild respiratory complications can be treated with oxygen supplementation [11,33]. Those who progress to advanced HCPS, with hemodynamic and/or severe respiratory compromise, should be admitted to the intensive care unit [7,33]. Circulatory support can be delivered by cautious use of inotropic drugs together with attentive management of fluids. Vasopressors may be needed in cases of overt shock [9,23].

The role of albumin infusions to preserve circulating volume in severe capillary leak syndrome remains unclear [2,24]. Continuous renal replacement therapy may be needed to treat uraemia [23,33]. Respiratory support should initially be given noninvasively, reserving invasive mechanical ventilation for the most severe cases [7,11]. Sometimes, shock ensues extremely rapidly and death occurs before any support can be deployed [7,10].

Very limited and anecdotical experience exists on the use of icatibant acetate, a bradykinin receptor antagonist, in hantavirus disease (although not for ANDV-HCPS) [26,33,34]. Alleged icatibant therapeutic effects would stem from the fact that hantavirus-infected endothelial cells are characterised by hyper-activation of kinin-kallikrein system, resulting in increased liberation of bradykinin, a major inducer of vascular permeability and a driver of capillary leak syndrome [35]. Further studies are needed to explore this hypothesis.

6. Preventive measures

Contact tracing and active case finding of persons infected with or exposed to ANDV is clearly warranted [9,11]. Geographic distribution of hub centres capable of performing diagnostic tests is also needed [11]. Ordinary infection control measures, including isolation, use of personal protection equipments (such as FFP2/3 face mask to attend cases) and standard disinfection procedures have to be implemented [9,11,12]. No effective vaccine against orthohantavirus infections are currently available [5].

7. Public health relevance of the current MV Hondius ANDV outbreak

The MV Hondius outbreak represents a public health event of exceptional complexity: departing Ushuaia, Argentina, on 1 April 2026, the Dutch-flagged vessel carried passengers of 23 nationalities across remote South Atlantic regions, with cases dispersing across multiple continents before the outbreak was detected [36,37]. As of 11 May

2026, eight cases had been reported — six confirmed and two suspected — including three deaths [37,38]; the clinical and epidemiological characteristics of each case are summarized in Fig. 5. The temporal distribution of cases, with onset spanning from 6 to 28 April, is consistent with a 'superspreader event', with secondary cases arising after an incubation period of 2–4 weeks [37,38]. The confined indoor environment of the ship, the long incubation period of ANDV, and the rapid international dispersal of exposed passengers before outbreak detection represent the key drivers of public health concern [37–39]. Nevertheless, WHO, ECDC and CDC concur in assessing the overall global risk as low, given that ANDV transmission requires prolonged close contact with symptomatic individuals and that pre-symptomatic spread has not been demonstrated [37–41]. The MV Hondius event serves as a stark reminder that zoonotic pathogens with even limited human-to-human transmissibility can rapidly acquire global reach in an era of mass international travel, underscoring the need for clinician awareness, robust surveillance, and internationally coordinated outbreak preparedness.

8. Executive summary

Andes virus (ANDV) is the only hantavirus capable of sustained human-to-human transmission, via respiratory droplets, prolonged close contact, and sexual intercourse, with a median reproductive number exceeding 2 and an incubation period of up to 40 days.

ANDV can be responsible for severe systemic presentation like hantavirus cardiopulmonary syndrome (HCPS), a life-threatening illness characterised by acute respiratory failure, haemodynamic instability, acute kidney injury, liver involvement, and dysregulated cytokine release, carrying a case fatality rate of up to 32% in the absence of any approved antiviral therapy or vaccine.

The April 2026 MV Hondius outbreak, involving passengers of 23 nationalities with eight cases and three deaths, illustrates how a zoonotic pathogen with even limited human-to-human transmissibility can rapidly achieve global reach, underscoring the need for clinician awareness and internationally coordinated outbreak preparedness.

Declaration of competing interest

The authors declare the following financial interests / personal relationships which may be considered as potential competing interests:

EDM received institutional funding, personal speaker fees or advisory board membership honoraria from Pfizer, Angelini, Advanz pharma, Infectopharm, Shionogi, Menarini, Mundipharma, Tillots pharma, Trx, outside of this work and in the last 3 years.

The other authors report no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

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