Hantavirus: Lethality, Transmission and What GPs Need to Know

Written by The Life Science Feed Editorial Team · Published 18 May 2026 · Edited by William Lopes

> Hantavirus sits in an uncomfortable clinical space: rare enough that most GPs will never see a confirmed case, lethal enough that missing one carries serious consequences. The immediate takeaway is this: hantavirus does not spread person-to-person in most species variants circulating outside South America, so mass public transmission events are not the threat. The threat is individual exposure to rodent reservoirs, delayed diagnosis, and the absence of any licensed antiviral treatment in most health systems.

KEY TAKEAWAYS

- The Pivot: Two distinct hantavirus syndromes exist with very different geographic distributions and fatality profiles. Hantavirus Pulmonary Syndrome (HPS), dominant in the Americas, carries case fatality rates of 30 to 40%. Haemorrhagic Fever with Renal Syndrome (HFRS), dominant in Europe and Asia, carries fatality rates generally below 5%, though Korean haemorrhagic fever caused by Hantaan virus reaches up to 15%.
- The Data: HPS case fatality rate: approximately 30 to 40%. HFRS case fatality rate: 0.1% to 15% depending on the causative species. No licensed antiviral therapy exists in most jurisdictions outside ribavirin use in HFRS, where evidence of benefit remains limited.
- The Action: Clinicians in endemic regions should include hantavirus in the differential for any patient presenting with unexplained fever, myalgia, and thrombocytopaenia, particularly those with recent rodent exposure. Early supportive care and ICU transfer planning are the primary levers available.

Hantaviruses are negative-sense, single-stranded RNA viruses belonging to the family Hantaviridae. They are transmitted to humans primarily through inhalation of aerosolised excreta (urine, faeces, saliva) from infected rodents.1 Each hantavirus species is tightly associated with a specific rodent reservoir host, and human infection is almost always a dead-end event with no onward transmission, with the notable exception of Andes virus (ANDV) in South America, which has documented capacity for human-to-human spread.1

The clinical landscape divides into two syndromes. Haemorrhagic Fever with Renal Syndrome (HFRS) is caused by Old World hantaviruses including Hantaan, Seoul, Puumala and Dobrava viruses, circulating across Europe, Asia and parts of the Middle East.2 Hantavirus Pulmonary Syndrome (HPS), also termed Hantavirus Cardiopulmonary Syndrome (HCPS), is caused by New World hantaviruses, most prominently Sin Nombre virus in North America and Andes virus in South America.2 Seoul virus is globally distributed via the domestic rat Rattus norvegicus and represents the species most plausible for imported cases appearing in non-endemic countries.1

The clinical picture and what makes these viruses dangerous

HFRS presents with a prodrome of abrupt fever, headache, myalgia and back pain, followed by haemorrhagic manifestations and acute kidney injury.2 The overall case fatality rate for HFRS ranges from less than 0.1% for Puumala virus infection (nephropathia epidemica) to 5 to 15% for Hantaan virus infection.2 Globally, an estimated 150,000 to 200,000 HFRS cases are reported annually, with the majority occurring in China.1

HPS is the more immediately lethal presentation. After a prodromal phase of 2 to 8 days resembling influenza, patients develop non-cardiogenic pulmonary oedema with rapid progression to respiratory failure.3 The case fatality rate for HPS caused by Sin Nombre virus sits at approximately 30 to 40% even with intensive care support.3 There is no licensed targeted antiviral therapy for HPS. Management is entirely supportive: mechanical ventilation, haemodynamic support, and in refractory cases, extracorporeal membrane oxygenation (ECMO), which case series suggest may improve survival in the most severe presentations, though controlled trial data are absent.3

For HFRS, intravenous ribavirin has been used in some settings, particularly in China and Korea, and early administration has been associated with reduced mortality in observational data.2 However, ribavirin is not universally approved for this indication, and the evidence base does not meet the threshold of randomised controlled trial validation in most regulatory jurisdictions. No vaccine is licensed outside of a bivalent inactivated vaccine available in China and South Korea for HFRS prevention, which has not received regulatory approval in Europe or North America.1

The incubation period across species ranges from 1 to 8 weeks, which complicates exposure history-taking considerably.1 Laboratory diagnosis relies on detection of hantavirus-specific IgM antibodies or PCR in the acute phase; the virus is rarely cultured in clinical settings given biosafety level 3 requirements.2 Thrombocytopaenia is a consistent early finding across both syndromes and, in the right geographic and exposure context, should prompt early serological testing rather than a wait-and-see approach.2

The pathogenesis of hantavirus infections involves widespread endothelial cell dysfunction, leading to increased vascular permeability. In HFRS, this manifests as capillary leakage in the kidneys, causing proteinuria, haematuria, and acute tubular necrosis. In HPS, the primary target is the pulmonary vasculature, resulting in severe non-cardiogenic pulmonary oedema and hypoxemia. The immune response plays a critical role, with an overzealous or dysregulated host immune response contributing to the severity of disease rather than solely viral cytopathic effects.3

For the general public, the practical risk is defined by behaviour: cleaning enclosed spaces with rodent infestation, agricultural work in endemic areas, camping or hiking in regions with known rodent reservoirs, and contact with pet rodents carrying Seoul virus.1 Wet cleaning of contaminated surfaces rather than dry sweeping, ventilating enclosed spaces before entry, and use of N95 respirators in high-exposure environments are the public health recommendations with the strongest mechanistic rationale, though direct RCT evidence for these interventions in humans does not exist.1 For broader perspectives on viral aetiology and effective management strategies pertinent to general practice, the Oxford Handbook of Infectious Diseases and Microbiology offers invaluable guidance.

> **CLINICAL IMPLICATIONS**
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> The 30 to 40% case fatality rate attached to HPS is the number that should recalibrate clinical instinct. Most GPs will reflexively consider influenza, COVID-19 or bacterial sepsis when confronted with acute fever, myalgia and rapid cardiopulmonary deterioration. Hantavirus earns a place in that differential specifically because the window for any meaningful intervention, namely aggressive early ICU transfer and haemodynamic support,

is narrow. A two-day delay attributed to a viral prodrome that looks generic is a delay with consequences that are difficult to reverse once pulmonary oedema establishes.

The absence of a licensed antiviral for HPS in 2024 is, frankly, an indictment of infectious disease research prioritisation. Hantavirus lacks the commercial profile to attract major pharmaceutical investment because transmission is not sustained and outbreak scale rarely reaches the threshold that triggers emergency funding. Ribavirin exists in the HFRS toolkit but its evidence base is observational and jurisdiction-dependent. The Chinese and South Korean inactivated vaccines for HFRS represent the only licensed preventive tools globally, and neither has cleared regulatory review in Europe or the United States. That gap is not a minor administrative oversight; it reflects a structural failure to treat geographically constrained, high-mortality diseases as development priorities until an outbreak forces the issue.

Patients who live in rural areas, work in agriculture, or have recently travelled to endemic regions in the Americas, Eastern Europe, or East Asia represent the population that carries actual risk. The communication challenge is precision: public anxiety about hantavirus following media coverage of isolated cases is disproportionate to the transmission biology, since person-to-person spread is essentially irrelevant outside of Andes virus exposure in South America. What is proportionate is targeted education about rodent exposure prevention directed at specific occupational and geographic risk groups, not blanket public alarm. GPs in non-endemic regions who receive patients returning from rural South America, rural China, or the Balkans with an undifferentiated febrile illness should treat the travel history as diagnostic information, not background noise.

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