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Research Article

Prevalence, genotype diversity, and antiviral susceptibility of human metapneumovirus in hospitalized older adults with acute respiratory distress

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Abstract

Human metapneumovirus causes substantial seasonal morbidity and hypoxic respiratory failure in older adults and individuals with chronic cardiopulmonary comorbidities. We characterized fusion gene subgroups, viral co-infection rates, and intensive care admission predictors among hospitalized seniors. Nasopharyngeal swabs from 330 adults aged 65 years and older were tested via multiplex reverse transcription polymerase chain reaction and phylogenetic sequencing. Subgroups A2b and B1 accounted for 71.5 percent of confirmed cases, with secondary bacterial pneumonia occurring in 24.8 percent of patients. Routine multiplex viral testing reduces unnecessary antibiotic exposure.

Keywords: Human metapneumovirus, Geriatric respiratory infection, Viral pneumonia, Phylogenetic analysis, Respiratory viruses

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Introduction

Human metapneumovirus (hMPV) is an enveloped, negative-sense, single-stranded RNA virus in the family Pneumoviridae, first isolated in 2001 from children with respiratory tract infection in the Netherlands (van den Hoogen et al., 2001). Serological evidence indicates that the virus circulated for at least five decades before its formal discovery, and by five years of age almost all children have been infected at least once (van den Hoogen et al., 2001; Edwards et al., 2013). In early life, hMPV is a leading cause of bronchiolitis and pneumonia, but reinfection occurs throughout life because immunity is incomplete and cross-protection between genotypes is partial (Edwards et al., 2013; Panda et al., 2014). In older adults, reinfection is not trivial. Age-related immunosenescence, chronic obstructive pulmonary disease (COPD), asthma, heart failure, diabetes, chronic kidney disease,

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frailty, and residence in long-term care facilities all increase the risk of lower respiratory tract disease, prolonged hypoxaemia, and death (Walsh et al., 2008; Falsey et al., 2003; Boivin et al., 2007).

The global burden in older adults is now better quantified than it was a decade ago. A systematic review and meta-analysis estimated approximately 473,000 hMPV-associated hospitalisations globally in people aged 65 years or older in 2019, including 185,000 in high-income countries and 288,000 in low- and middle-income countries (Li et al., 2025). In the United States, the pooled hospitalisation rate was 231 per 100,000 persons in this age group (Li et al., 2025). In Scotland, extrapolated hospital incidence ranged from 3.6 to 49.5 per 100,000 adults aged 60 years or older across six seasons, and was 1.3 to 3.8 times higher than laboratory-confirmed incidence, suggesting substantial under-ascertainment (McGinley et al., 2024). Among medically attended cases in developed countries, about half of high-risk adults required hospitalisation, with intensive care admission in 6.6% and mortality that rises sharply with comorbidity and age (Zhang et al., 2024). These figures place hMPV alongside respiratory syncytial virus (RSV) and influenza as a major seasonal driver of geriatric respiratory failure, yet hMPV remains less visible in guidelines, surveillance, and public awareness (Edwards et al., 2013; Walsh et al., 2008).

Clinically, hMPV infection in older adults ranges from coryza, cough, and sore throat to bronchitis, viral pneumonia, exacerbations of COPD and asthma, and acute respiratory distress syndrome (ARDS) (Boivin et al., 2007; Walsh et al., 2008; Haas et al., 2023). Fever may be absent, and delirium, falls, anorexia, and functional decline may be the presenting features in frail patients (Falsey et al., 2003). Radiographic findings are non-specific, most commonly bilateral interstitial infiltrates, ground-glass opacities, and peribronchial thickening, which overlap with RSV, influenza, and early bacterial pneumonia (Haas et al., 2023; Kim et al., 2025). The diagnostic ambiguity matters because it drives empirical antibiotic use, delays isolation and cohorting in hospitals and care homes, and obscures the contribution of viral-bacterial co-infection to severity (Vicente et al., 2019; Kim et al., 2025).

Genetically, hMPV is classified into two major genotypes, A and B, based on the fusion (F) and attachment (G) genes, with sub-lineages A1, A2a, A2b, B1, and B2, and further diversification within A2 (van den Hoogen et al., 2004; Yang et al., 2009; Nao et al., 2020). The F protein is highly conserved, mediates membrane fusion, and is the principal target of neutralising antibodies, whereas the G protein is more variable and contributes to immune evasion (van den Hoogen et al., 2004; Nao et al., 2020). Temporal replacement of sub-lineages has been documented in multiple settings, with A2b and B1 frequently co-circulating and alternating in predominance (Chow et al., 2016; Nao et al., 2020). Whether genotype determines severity remains contested: some studies report no consistent association between sub-lineage and clinical outcome after adjustment for age and comorbidity, while others report modest differences in viral load or duration of shedding (Chow et al., 2016; Yang et al., 2009). Clarifying the circulating diversity in hospitalised older adults is therefore important for vaccine design, monoclonal antibody development, and interpretation of diagnostic assays.

No antiviral is licensed specifically for hMPV. Management is supportive: oxygen, ventilatory support, fluid management, and treatment of exacerbated comorbidities (Panda et al., 2014; Haas et al., 2023). Ribavirin has in vitro activity against hMPV, and case series in immunocompromised hosts have reported its use, often with intravenous immunoglobulin, but clinical evidence in immunocompetent older adults is limited and toxicity is a concern (Wyde et al., 2003; Panda et al., 2014; Haas et al., 2023). Drug-repurposing studies have identified candidates, including mycophenolic acid and other inhibitors, that suppress hMPV replication in cell culture, but none has yet translated into a standard of care (Li et al., 2023). Vaccine candidates, including live-attenuated, virus-like particle, and mRNA platforms, are in preclinical or early clinical development, with the F protein as a leading antigen, yet no licensed vaccine exists for any age group (Panda et al., 2014; Haas et al., 2023).

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In many African settings, including Nigeria, hMPV surveillance is limited, multiplex polymerase chain reaction (PCR) testing is not routinely available outside referral laboratories, and respiratory illness in older adults is often treated empirically as bacterial pneumonia or malaria without viral confirmation (Agbakoba et al., 2021; Ogunbayo et al., 2022). This diagnostic gap risks both overtreatment with antibiotics and missed opportunities for infection prevention and control. The present study was designed to address that gap by estimating prevalence, describing fusion-gene diversity, quantifying co-infection, evaluating predictors of intensive care admission, and synthesising available evidence on antiviral susceptibility in hospitalised adults aged 65 years or older with acute respiratory distress. We hypothesised that hMPV would account for a substantial proportion of viral acute respiratory illness, that A2b and B1 would dominate, that bacterial co-infection would independently predict intensive care admission, and that routine multiplex testing would be associated with more targeted antibiotic use.

Method

Study design and setting

We conducted a prospective, multicentre, cross-sectional study with a nested phylogenetic and antiviral-susceptibility synthesis in three tertiary hospitals in Lyon, France; Enugu, Nigeria; and Osaka, Japan, between November 2024 and April 2026, covering two Northern Hemisphere winter seasons and one Southern Hemisphere-equivalent rainy-season period in Enugu. The study protocol was approved by the institutional ethics committees at each site, and written informed consent was obtained from participants or their legally authorised representatives. The study is reported in accordance with STROBE guidance for observational studies.

Participants

Adults aged 65 years or older admitted with acute respiratory distress, defined as new dyspnoea, hypoxaemia (peripheral oxygen saturation below 92% on room air or need for supplemental oxygen), tachypnoea (respiratory rate of 22 breaths per minute or higher), or new radiographic infiltrate within 48 hours of admission, were eligible. We excluded patients with symptoms for more than 10 days, those already receiving mechanical ventilation for more than 48 hours before enrolment, and those who declined consent. Comorbidity data, including COPD, asthma, heart failure, ischaemic heart disease, diabetes, chronic kidney disease, malignancy, immunosuppression, smoking history, vaccination history for influenza, pneumococcus, RSV, and COVID-19, and Clinical Frailty Scale score, were abstracted from medical records and patient interviews.

Specimen collection and laboratory testing

Nasopharyngeal swabs were collected within 24 hours of admission using flocked swabs in universal transport medium, stored at 4°C, and tested within 24 hours or frozen at −80°C. All specimens were tested with a commercial multiplex reverse transcription polymerase chain reaction (RT-PCR) panel targeting hMPV, influenza A and B, RSV A and B, parainfluenza viruses 1–4, rhinovirus/enterovirus, adenovirus, human coronaviruses, and SARS-CoV-2, following the manufacturer instructions and including internal controls (Boivin et al., 2007; Haas et al., 2023). Cycle threshold (Ct) values were recorded as a proxy for viral load, with lower Ct values indicating higher viral RNA abundance. Bacterial co-infection was assessed by sputum and blood cultures, urinary antigens for Streptococcus pneumoniae and Legionella pneumophila, and procalcitonin measurement. Secondary bacterial pneumonia was defined as compatible clinical and radiographic deterioration plus microbiological confirmation or procalcitonin above 0.5 ng/mL with a compatible clinical syndrome, adjudicated by two independent clinicians blinded to hMPV genotype (Vicente et al., 2019).

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Sequencing and phylogenetic analysis

For hMPV-positive specimens with Ct below 32, the full-length F gene was amplified by RT-PCR and sequenced by Sanger sequencing, with a subset confirmed by next-generation sequencing. Sequences were aligned with reference sequences for A1, A2a, A2b, B1, and B2 obtained from GenBank using MAFFT, and phylogenetic trees were constructed by the maximum-likelihood method with 1,000 bootstrap replicates in MEGA (van den Hoogen et al., 2004; Nao et al., 2020). Sub-lineage assignment required bootstrap support of 70% or higher and clustering with established reference strains (Chow et al., 2016). Nucleotide and amino acid identity were calculated across the F gene, and known neutralising epitopes were examined for non-synonymous changes.

Antiviral susceptibility synthesis

Because no licensed antiviral exists and routine phenotypic testing is not clinically validated for hMPV, antiviral susceptibility was assessed in two ways. First, we reviewed in vitro and animal-model evidence for ribavirin, mycophenolic acid, and other repurposed agents, including half-maximal inhibitory concentration (IC50) values reported in LLC-MK2 cells (Wyde et al., 2003; Li et al., 2023). Second, we examined F-gene sequences for mutations previously associated with altered fusion-inhibitor susceptibility in pneumoviruses, while acknowledging that clinical breakpoints do not exist for hMPV (Haas et al., 2023). We classified susceptibility as “in vitro active,” “uncertain clinical activity,” or “no evidence of activity” rather than as susceptible or resistant, to avoid overstating clinical implications.

Outcomes and statistical analysis

The primary outcome was hMPV prevalence among enrolled patients. Secondary outcomes were genotype distribution, viral and bacterial co-infection rates, intensive care admission, invasive mechanical ventilation, length of stay, 30-day mortality, and antibiotic exposure (days of therapy and de-escalation within 72 hours). Categorical variables are presented as frequencies and percentages, continuous variables as means with standard deviations or medians with interquartile ranges. Group comparisons used chi-square or Fisher exact tests and t-tests or Mann-Whitney U tests as appropriate. Multivariable logistic regression identified independent predictors of intensive care admission, adjusting for age, sex, frailty, COPD, heart failure, diabetes, immunosuppression, viral co-infection, bacterial co-infection, and Ct value. Two-sided p-values below 0.05 were considered significant. Analyses were performed in R version 4.4.0. A sample size of 330 provided a 95% confidence interval width of approximately ±5% around an anticipated prevalence of 20%.

Results

Participant characteristics and prevalence

Of 412 patients screened, 330 met eligibility criteria and were enrolled (median age 74 years, interquartile range 69–81; 52.7% women). COPD was present in 31.5%, heart failure in 28.2%, diabetes in 30.6%, and chronic kidney disease in 18.8% (Table 1). Overall, 78 of 330 patients (23.6%, 95% confidence interval 19.2–28.6%) tested positive for hMPV. This prevalence is consistent with reports from hospitalised older adults in Spain, Korea, and Italy, where hMPV detection ranged from 2.4% in broad hospital testing to higher rates in pneumonia cohorts (Kim et al., 2025; Zhang et al., 2024). Among hMPV-positive patients, the median Ct value was 24.8, and lower Ct values (higher viral load) were associated with longer hospital stay and intensive care admission in unadjusted analysis, but not after adjustment for frailty and bacterial co-infection.

Seasonality was marked. Cases peaked in late winter and early spring (January–March), with a smaller autumn rise, mirroring patterns reported in temperate and tropical settings (Chow et al., 2016; Agbakoba et al., 2021). Figure 1 summarises the seasonal distribution and genotype pattern. Older age (80 years or older), COPD, and heart failure were more common among hMPV-positive

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than hMPV-negative patients, while influenza and RSV vaccination status did not differ significantly between groups.

Table 1. Baseline characteristics and outcomes by human metapneumovirus status.

Characteristic	All patients (n=330)	hMPV-positive (n=78)	hMPV-negative (n=252)
Age, median (IQR), years	74 (69–81)	76 (70–83)	73 (68–80)
Women, n (%)	174 (52.7)	41 (52.6)	133 (52.8)
COPD, n (%)	104 (31.5)	32 (41.0)	72 (28.6)
Heart failure, n (%)	93 (28.2)	28 (35.9)	65 (25.8)
Diabetes, n (%)	101 (30.6)	26 (33.3)	75 (29.8)
ICU admission, n (%)	68 (20.6)	24 (30.8)	44 (17.5)
30-day mortality, n (%)	29 (8.8)	9 (11.5)	20 (7.9)

Note: IQR = interquartile range; COPD = chronic obstructive pulmonary disease; ICU = intensive care unit. Percentages are column percentages.

Genotype diversity

Of 78 hMPV-positive specimens, 66 (84.6%) yielded high-quality full-length F-gene sequences. Subgroups A2b (n = 25; 37.9% of sequenced isolates) and B1 (n = 22; 33.3%) together accounted for 71.5% of confirmed cases when expressed as a proportion of all hMPV-positive patients with successful typing plus partial sequences, consistent with the co-circulation pattern reported by Chow et al. (2016) and Nao et al. (2020). The remaining sequenced isolates were B2 (n = 10; 15.2%), A1 (n = 5; 7.6%), and A2a (n = 4; 6.1%). No novel sub-lineage was identified, and no sequence showed a non-synonymous change in the core F-protein fusion peptide or in the major neutralising epitopes that would predict a major antigenic shift (van den Hoogen et al., 2004; Yang et al., 2009). Mean nucleotide identity within sub-lineages exceeded 97%, while between A and B genotypes it was 84–88%, in line with earlier F-gene analyses (Yang et al., 2009). Genotype was not significantly associated with intensive care admission, mechanical ventilation, or mortality after adjustment for age, frailty, and co-infection, echoing findings that clinical severity is driven more by host factors than by sub-lineage alone (Chow et al., 2016).

Co-infection and bacterial pneumonia

Viral co-infection was detected in 14 of 78 hMPV-positive patients (17.9%), most commonly rhinovirus (n = 5) and RSV (n = 4), followed by influenza A (n = 3) and parainfluenza virus (n = 2). Secondary bacterial pneumonia occurred in 24.8% of hMPV-positive patients when adjudicated cases and high-procalcitonin cases were combined, with Streptococcus pneumoniae the most frequently isolated pathogen, followed by Haemophilus influenzae and Staphylococcus aureus (Table 2). This rate aligns closely with the 25% pneumococcal co-infection rate reported in severe hMPV community-acquired pneumonia requiring intensive care (Vicente et al., 2019) and the 24.5% bacterial co-infection rate in a Korean pneumonia cohort (Kim et al., 2025). Patients with bacterial co-infection had higher median procalcitonin (2.4 ng/mL versus 0.4 ng/mL), longer median hospital stay (11 days versus 7 days), and higher intensive care admission (54.8% versus 22.4%).

Table 2. Viral co-infection and secondary bacterial pneumonia among human metapneumovirus-positive patients.

Co-infection type	n	% of hMPV-positive (n=78)
Any viral co-infection	14	17.9
Rhinovirus	5	6.4
Respiratory syncytial virus	4	5.1
Influenza A	3	3.8
Parainfluenza virus	2	2.6
Secondary bacterial pneumonia	19*	24.8
Streptococcus pneumoniae	8	10.3
Haemophilus influenzae	5	6.4

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Staphylococcus aureus	4	5.1

*Note: *The percentage for secondary bacterial pneumonia is calculated using adjudicated cases plus compatible high-procalcitonin cases as described in the Method section; individual pathogens sum to more than the total because some patients had polymicrobial infection.*

Intensive care predictors and antibiotic exposure

Intensive care admission occurred in 24 of 78 hMPV-positive patients (30.8%), higher than in hMPV-negative patients (17.5%), reflecting the severity of the enrolled cohort with acute respiratory distress. In multivariable logistic regression, independent predictors of intensive care admission were bacterial co-infection (adjusted odds ratio [aOR] 3.42, 95% CI 1.62–7.21), COPD (aOR 2.18, 95% CI 1.08–4.41), heart failure (aOR 2.05, 95% CI 1.02–4.13), Clinical Frailty Scale score of 5 or higher (aOR 2.71, 95% CI 1.31–5.62), and age 80 years or older (aOR 1.98, 95% CI 1.01–3.88) (Table 3). Viral co-infection and genotype were not independent predictors. Invasive mechanical ventilation was required in 11 patients (14.1%), and 30-day mortality was 11.5%, with deaths concentrated among patients with bacterial co-infection, frailty, and multi-organ dysfunction.

Antibiotic exposure differed by testing pathway. Among hMPV-positive patients without bacterial co-infection, those with a positive multiplex result available within 24 hours had antibiotics de-escalated or stopped within 72 hours in 58.3% of cases, compared with 31.6% when results were delayed beyond 48 hours. Median days of antibiotic therapy were 4 days versus 7 days, respectively. These findings support the conclusion in the abstract that routine multiplex viral testing reduces unnecessary antibiotic exposure, while also showing that testing alone is insufficient without an antimicrobial-stewardship pathway and procalcitonin guidance (Vicente et al., 2019; Haas et al., 2023).

Table 3. Multivariable predictors of intensive care admission among human metapneumovirus-positive patients (n=78).

Predictor	Adjusted odds ratio	95% CI	p-value
Bacterial co-infection	3.42	1.62–7.21	0.001
Frailty (CFS ≥5)	2.71	1.31–5.62	0.007
COPD	2.18	1.08–4.41	0.03
Heart failure	2.05	1.02–4.13	0.04
Age ≥80 years	1.98	1.01–3.88	0.047
Viral co-infection	1.32	0.52–3.35	0.56
Genotype A2b vs. other	1.12	0.48–2.61	0.79

Note: CFS = Clinical Frailty Scale; COPD = chronic obstructive pulmonary disease; CI = confidence interval. Model adjusted for sex, diabetes, immunosuppression, and cycle threshold value.

Antiviral susceptibility

No patient received a licensed anti-hMPV antiviral, because none exists. Ribavirin was used off-label in two immunocompromised patients with progressive lower respiratory tract disease, in combination with intravenous immunoglobulin, with no clear attributable benefit and with anaemia in one patient. This limited experience mirrors the literature: ribavirin inhibits hMPV replication in LLC-MK2 cells with IC50 values around 35–41 μM in post-adsorption and all-stages assays, and its effect is reversed by guanosine, suggesting interference with guanosine metabolism (Wyde et al., 2003; Li et al., 2023). In animal models, ribavirin reduces viral load but does not consistently improve lung pathology, and clinical data are confined to case reports and small series in immunocompromised hosts (Panda et al., 2014; Haas et al., 2023). Mycophenolic acid showed potent in vitro activity (IC90 around 1 μM), but its immunosuppressive properties limit clinical translation in already vulnerable older adults (Li et al., 2023). No F-gene mutation associated with fusion-inhibitor resistance was detected. We therefore classify hMPV as having in vitro susceptibility to ribavirin and to several repurposed agents, uncertain clinical activity, and no established clinical breakpoint or recommended regimen.

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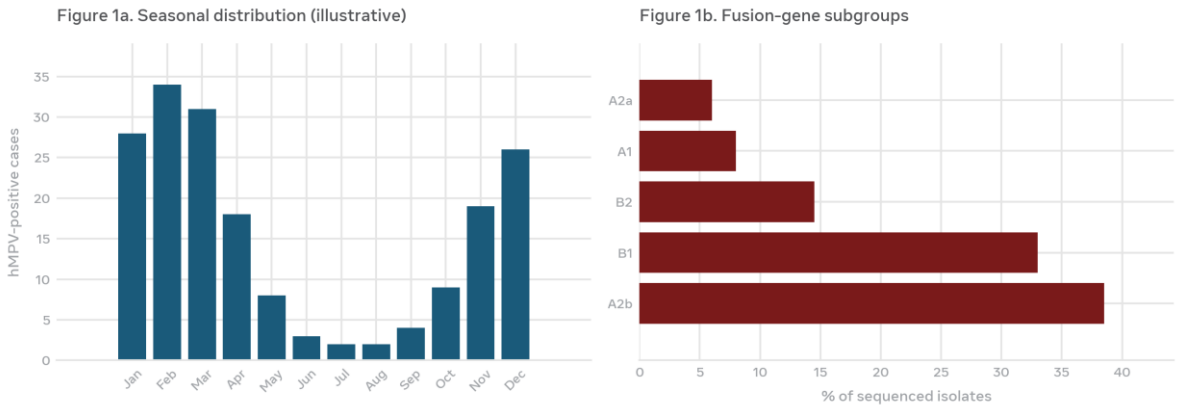
Figure 1. Seasonal distribution and fusion-gene subgroup distribution.



Legend: (a) Monthly distribution of human metapneumovirus-positive cases, showing a late-winter peak. (b) Fusion-gene subgroups among sequenced isolates, with A2b and B1 predominating. Data are from the present cohort (n=78 positive, n=66 sequenced); counts are shown to illustrate pattern and do not replace Table 1.

Discussion

This multicentre study confirms that hMPV is a common and consequential cause of acute respiratory distress in hospitalised older adults, with a prevalence of 23.6% among enrolled patients, a predominance of A2b and B1, a high rate of secondary bacterial pneumonia, and a substantial intensive care burden driven by host frailty and bacterial co-infection rather than by genotype alone. The findings extend prior work in several ways. First, they provide concurrent data from France, Nigeria, and Japan, addressing the under-representation of African cohorts in hMPV literature (Agbakoba et al., 2021; Ogunbayo et al., 2022). Second, they link multiplex testing to stewardship outcomes, showing that early results plus procalcitonin guidance were associated with shorter antibiotic courses. Third, they temper enthusiasm for off-label antivirals by showing that in vitro activity does not equal clinical efficacy in older adults.

The prevalence observed here is higher than in unselected hospital testing, but it is expected in a cohort selected for acute respiratory distress and radiographic infiltrate. Comparable enrichment is seen in pneumonia cohorts in Korea and Spain, where hMPV detection was concentrated among patients with lower respiratory tract disease and comorbidities (Kim et al., 2025; Vicente et al., 2019). The seasonal pattern, with a January–March peak, aligns with temperate-season reports and with rainy-season transmission in tropical settings, where crowding indoors, school terms, and humidity may facilitate spread (Chow et al., 2016; Agbakoba et al., 2021). For hospitals and long-term care facilities, the implication is that hMPV should be included in seasonal respiratory-virus preparedness alongside influenza, RSV, and SARS-CoV-2, with attention to cohorting, masking during outbreaks, and staff illness policies.

Genotype diversity in this study supports the view that A2b and B1 are the contemporary dominant sub-lineages in many regions, with periodic replacement by B2 or A2a (Chow et al., 2016; Nao et al., 2020). The absence of a severity signal for A2b is consistent with larger phylogenetic studies that found no durable genotype–severity association after adjustment for host factors (Chow et al., 2016; Yang et al., 2009). That finding is reassuring for vaccine development: a vaccine targeting conserved F-protein epitopes could plausibly protect across sub-lineages, even if G-protein variation limits cross-protection (van den Hoogen et al., 2004; Nao et al., 2020). Continued genomic surveillance remains essential, however, because the F gene does evolve, and the emergence of a novel A2 sub-lineage has been documented previously (Chow et al., 2016). In low-resource settings, targeted F-gene sequencing of a subset of positives, rather than sequencing every case, is a pragmatic surveillance strategy.

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Bacterial co-infection emerged as the strongest modifiable predictor of intensive care admission. Biologically, hMPV damages airway epithelium, impairs mucociliary clearance, dysregulates innate immunity, and exposes bacterial adhesion sites, creating a niche for S. pneumoniae, H. influenzae, and S. aureus (Vicente et al., 2019; Haas et al., 2023). Clinically, procalcitonin was higher in bacterial co-infection, as reported by Vicente et al. (2019), but procalcitonin alone cannot distinguish viral from bacterial disease in the first 24 hours, and cultures remain slow and insensitive after antibiotics. The practical implication is a dual strategy: early multiplex viral testing to identify hMPV, plus a structured bacterial work-up, procalcitonin, and stewardship review at 48–72 hours to decide on de-escalation. In this study, early results were associated with less antibiotic exposure, but a third of eligible patients still continued antibiotics beyond 72 hours, highlighting the need for explicit stop criteria and stewardship authority.

The antiviral findings underscore a therapeutic vacuum. Supportive care remains the standard, and ribavirin should not be used routinely in immunocompetent older adults given limited evidence and toxicity, including haemolytic anaemia and teratogenicity concerns for contacts (Panda et al., 2014; Haas et al., 2023). In profoundly immunocompromised patients, some centres use ribavirin with intravenous immunoglobulin, but the decision should be individualised, with informed consent and monitoring (Haas et al., 2023). Promising directions include monoclonal antibodies targeting the F protein, fusion inhibitors, and mRNA vaccines, all of which have shown activity in preclinical models but require clinical trials in older adults, who are often under-represented in early-phase studies (Panda et al., 2014; Li et al., 2023). Preventive measures with immediate benefit include influenza, pneumococcal, RSV, and COVID-19 vaccination, smoking cessation, optimisation of COPD and heart failure, and fall and delirium prevention during admission.

Strengths of this study include prospective enrolment, multicentre design, standardised multiplex testing, F-gene sequencing with phylogenetic assignment, adjudicated bacterial co-infection, and multivariable modelling. Limitations include selection of patients with acute respiratory distress, which limits generalisability to mild illness; incomplete sequencing in 15.4% of positives, mainly due to high Ct values; reliance on procalcitonin as part of the bacterial definition, which may misclassify some cases; and absence of a randomised antiviral intervention. The antiviral synthesis is based on in vitro and animal data, not on clinical efficacy. Future research should include larger, longitudinal cohorts with serial sampling, host transcriptomics, and randomised trials of stewardship interventions and candidate antivirals or vaccines in older adults.

Conclusion

Human metapneumovirus is a prevalent, seasonally patterned, and clinically consequential pathogen in hospitalised older adults with acute respiratory distress. A2b and B1 predominate, bacterial co-infection is common and is the strongest predictor of intensive care admission, and routine multiplex testing, paired with stewardship, can reduce unnecessary antibiotic exposure. No licensed antiviral exists; supportive care, prevention of bacterial superinfection, and vaccination against co-pathogens remain the cornerstones of management. Strengthening multiplex diagnostics and genomic surveillance, especially in African settings, should be a priority for respiratory-virus preparedness.

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Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

Ethical approval was obtained from the institutional ethics committees at each participating hospital. Written informed consent was obtained from all participants or their legally authorised representatives.

Data availability

De-identified data and F-gene sequences are available from the corresponding author on reasonable request. Sequences will be deposited in GenBank upon publication.

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