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ORIGINAL ARTICLE

Epidemiology and Clinical Characteristics of Respiratory Syncytial Virus Infection among Children with Acute Respiratory Illness in Kathmandu, Nepal

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ABSTRACT

Introduction: Respiratory syncytial virus (RSV) is a major cause of acute lower respiratory tract infections in young children, particularly in low- and middle-income countries. Data on RSV epidemiology and clinical characteristics in Nepal remain limited.

Method: A hospital-based cross-sectional study was conducted from March to August 2025 among 122 children presenting with acute respiratory illness at a tertiary children's hospital in Kathmandu. Nasopharyngeal swabs were tested using real-time PCR for RSV detection and subtyping :RSV-A and RSV-B. Demographic and clinical data were analyzed using descriptive statistics and inferential tests, including Fisher's exact test and Mann–Whitney U test.

Result: RSV was detected in 19.7% (24/122) of cases—nearly one in five children—with a predominance among males (66.7%). RSV-A was the dominant subtype (75%), followed by RSV-B (25%). Infants constituted the majority of cases (54.2%). CT values demonstrated significantly higher viral loads in children under 12 months ($p = 0.02$), with a moderate positive correlation between age and Ct value ($r = 0.52$, $p = 0.01$), indicating decreasing viral load with increasing age. Although RSV-B showed relatively higher viral loads than RSV-A, the difference was not statistically significant ($p = 0.49$). Clinical features, including fever, cough, wheezing, and rhinorrhea, were comparable across subtypes, with no statistically significant differences ($p > 0.05$).

Conclusion: RSV was detected in nearly one-fifth of children with acute respiratory illness, predominantly affecting infants. RSV-A was the predominant subtype. Clinical features were similar between RSV-A and RSV-B infections. These findings highlight the substantial burden of RSV in young children in Nepal and underscore the need for enhanced surveillance and preventive strategies.

Key words: **Respiratory syncytial virus; Acute respiratory infection; Children; Nepal; RSV-A; RSV-B**

INTRODUCTION

Respiratory syncytial virus (RSV) is a leading cause of acute lower respiratory tract infections in infants and young children worldwide, contributing substantially to morbidity and mortality among children under five years of age, particularly in low- and middle-income countries[1]. Recent global estimates indicate that RSV is responsible for millions of infections annually, resulting in significant hospitalizations and deaths, with the highest burden observed in infants below one year of age[2]. Transmission occurs primarily through respiratory droplets, direct contact with infected individuals, or contaminated surfaces, facilitating rapid spread, especially in crowded settings[3]. RSV is an enveloped, negative-sense single-stranded RNA virus belonging to the family Pneumoviridae. It is classified into two major antigenic subtypes, RSV-A and RSV-B, based on genetic and antigenic variability[4]. Both subtypes co-circulate globally, with their predominance varying across seasons and geographic regions[5]. Although some studies suggest differences in virulence between RSV-A and RSV-B, the clinical significance of these differences remains unclear[5]. Clinically, RSV infection ranges from mild upper respiratory tract illness to severe bronchiolitis and pneumonia, particularly among infants, preterm neonates, and immunocompromised children[6]. In Nepal, acute respiratory infections remain a major cause of pediatric morbidity and mortality, accounting for a substantial proportion of outpatient visits and hospital admissions among children[7]. Previous hospital-based and surveillance studies from Nepal have identified RSV as an important viral pathogen in pediatric respiratory infections, with seasonal peaks typically observed during the winter months[8-9]. However, data on RSV subtype distribution and detailed clinical correlations remain limited, particularly in the context of routine molecular diagnostics. Therefore, this study aims to determine the prevalence of RSV infection among children presenting with acute respiratory illness in Kathmandu, Nepal, and to describe the associated clinical characteristics and subtype distribution. The findings are expected to enhance understanding of RSV epidemiology in Nepal and support evidence-based strategies for diagnosis, prevention, and future vaccine implementation.

METHODS

Study Design and Setting

A hospital-based cross-sectional study was conducted at a government-run tertiary care children's hospital in Kathmandu, Nepal. Sample collection was carried out from March 2025 to August 2025 among patients attending Kanti Children's Hospital. Nasopharyngeal swab samples were collected under aseptic conditions. Collected samples were transported to the Central Diagnostic Laboratory, an ISO 15189-accredited and Government of Nepal-certified

Category A laboratory, where they were stored at −80 °C until further processing. Molecular analyses were performed at the Central Diagnostic Laboratory and the Central Department of Microbiology, Tribhuvan University, Kirtipur.

Study Population

Children presenting with symptoms of acute respiratory illness, including cough, fever, wheezing, or difficulty breathing, were enrolled. A total of 122 children were included in the study.

Data Collection

Demographic information (age, gender), clinical features (cough, fever, wheezing), and exposure history (contact with respiratory illness) were recorded using a structured questionnaire.

Laboratory Analysis

RNA Extraction[10]

Nasopharyngeal swab specimens collected in viral transport medium (VTM) were subjected to viral RNA extraction using a silica column-based extraction method (Biochroma-compatible viral RNA extraction kit), following the manufacturer's instructions. Briefly, 200–300 µL of the clinical specimen was mixed with lysis buffer containing chaotropic salts to ensure viral inactivation and nucleic acid release. An internal control (IC) provided with the PCR kit was added to the lysis step to monitor extraction efficiency and potential PCR inhibition.

Following lysis, ethanol was added to facilitate nucleic acid binding, and the mixture was transferred to a spin column. The bound RNA was washed sequentially with wash buffers to remove impurities and potential PCR inhibitors. A final high-speed centrifugation step was performed to eliminate residual ethanol. Viral RNA was then eluted in 60 µL of RNase-free elution buffer and either used immediately for downstream analysis or stored at −80°C until further processing.

Real-Time RT-PCR for RSV Detection and Subtyping[11]

Detection and differentiation of respiratory syncytial virus (RSV) subtypes A and B were performed using the RealStar RSV RT-PCR Kit 3.0 (Altona Diagnostics, Hamburg, Germany), according to the manufacturer's instructions.

PCR reactions were prepared by combining 5 µL of Master A and 15 µL of Master B to obtain a total master mix volume of 20 µL per reaction. For each sample, 10 µL of extracted RNA was added to the master mix, resulting in a final reaction volume of 30 µL. When not added during extraction, the internal control was included in the PCR reaction mix as recommended.

Amplification was carried out on a real-time PCR platform under the following cycling conditions: reverse transcription at 50 °C for 10–20 minutes, initial denaturation at 95 °C for 2–3 minutes, followed by 45 cycles of denaturation at 95 °C for 10–15 seconds and annealing/extension at 58–60 °C for 30–60 seconds, during which fluorescence data were acquired.

Quality Control and Interpretation of Results

Each PCR run included a positive control, negative control, and internal control to ensure assay validity. The assay detects RSV-A and RSV-B using subtype-specific fluorescent probes in separate detection channels, along with an internal control channel.

A sample was considered positive when amplification was observed within the specified cycle threshold (Ct) range (Ct ≤ 40), provided that the internal control showed valid amplification. Samples with no RSV amplification but a valid internal control signal were considered negative. Failure of internal control amplification indicated possible PCR inhibition or extraction failure, and such samples were retested.

Data Handling

Cycle threshold (Ct) values were recorded for all positive samples. Ct values were used as a proxy for viral load in subsequent statistical analyses.

Statistical Analysis

Data were analyzed using descriptive statistics. Frequencies and percentages were calculated for categorical variables. Clinical characteristics between RSV-A and RSV-B infections were compared using Fisher's exact test. A p-value of less than 0.05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the institutional review committee of Kanti Children's hospital and Institutional Review Board of Nepal Health Research Council. Informed consent was obtained from parents or guardians of all participants.

RESULTS

During the study period, a total of 122 children with respiratory illness were enrolled, including 70 males and 52 females. Among them, 24 (19.7%) tested positive for respiratory syncytial virus (RSV). Of the RSV-positive cases, 16 (66.7%) were male and 8 (33.3%) were female. RSV subtype A accounted for 75.0% (n = 18) of infections, while RSV subtype B constituted 25.0% (n = 6) (Table 1).

Table 1. Distribution of RSV positivity among enrolled children (n = 122)

Variable	Number (n)	Percent(%)
Total children enrolled	122	100
RSV-positive children	24	19.7
RSV-negative children	98	80.3
Male RSV-positive	16	66.7
Female RSV-positive	8	33.3
RSV A-positive	18	75.0
RSV B-positive	6	25.0

Association of Gender with RSV Positivity and RSV Subtypes

among Enrolled Patients

There was no statistically significant association between gender and RSV positivity (Fisher's exact test, p = 0.34). The odds of RSV positivity were higher among males compared to females, but this was not statistically significant (odds ratio [OR] = 1.63; 95% CI: 0.63–4.20).

Similarly, the distribution of RSV subtypes (A and B) between males and females did not show a statistically significant difference (Fisher's exact test, p = 0.10). However, males had higher odds of being infected with RSV A compared to females (OR = 7.00; 95% CI: 0.92–53.1), though this association was not statistically significant (Table2).

Table 2. Association between Gender and RSV Infection Status, Including RSV Subtypes, among Enrolled Patients (n = 122)

Gender	Total(%)	RSV +ve(%)	RSV -ve (%)	RSV A (%)	RSV B (%)
Male	70	16 (22.9)	54 (77.1)	14 (87.5)	2 (12.5)
Female	52	8 (15.4)	44 (84.6)	4 (50.0)	4 (50.0)
Total	122	24 (19.7)	98 (80.3)	18 (75.0)	6 (25.0)
P-value		0.34		0.10	

RSV distribution age category wise

Among the 24 RSV-positive children, the majority were infants (54.2%), followed by children aged 1–2 years (20.8%). Male children constituted two-thirds of cases (66.7%), indicating a male predominance among RSV infections.

Table 3. RSV distribution age category wise

Age category	Definition	Number (%)
Newborns	≤28 days	0 (0.0)
Infants	28 days – ≤1 year	13 (54.2)
1–2 years	1 year – ≤2 years	5 (20.8)
2–5 years	2 years – ≤5 years	4 (16.7)
>5 years	> 5 years	2 (8.3)
Total		24 (100.0)

Association of RSV A and B subtype with age category and gender

There was no statistically significant association was observed between RSV subtype and age category and gender. Both RSV-A and RSV-B infections were predominantly seen in infants (<12 months), accounting for 55.6% and 66.7% of cases, respectively, followed by lower proportions in older age groups, with no cases of RSV-B detected in children older than 5 years. The distribution across age categories did not differ significantly between the two subtypes (p = 0.76). Similarly, although a higher proportion of males was observed among RSV-A cases (72.2%), RSV-B infections showed an equal distribution between males and females (50% each), and this difference was not statistically significant (p = 0.61). Overall, RSV-A and RSV-B demonstrated similar demographic patterns in terms of age and gender distribution in the study population(Table 4).

Table 4. Association of RSV A and B subtype with age category and gender

Variable	RSV A (n=18)	RSV B (n=6)	p-value
Age category			0.76
Infant (<12 months)	10 (55.6%)	4 (66.7%)	
1–2 years	3 (16.7%)	1 (16.7%)	
2–5 years	4 (22.2%)	1 (16.7%)	
>5 years	1 (5.6%)	0 (0.0%)	
Gender			0.61
Male	13 (72.2%)	3 (50.0%)	
Female	5 (27.8%)	3 (50.0%)	

Descriptive and comparative statistics of Ct values and age for RSV A and RSV B cases

Descriptive and comparative statistics of Ct values and age for RSV A and RSV B cases are summarized in Table 5. Although RSV B cases showed a lower median Ct value compared to RSV A, the difference was not statistically significant (Mann–Whitney U = 65.0, p = 0.49), with a small effect size (rrb = –0.20).

Table 5. Descriptive and comparative analysis of Ct values and age among RSV A and RSV B–positive cases (n = 24)

Variable	Statistic	RSV A (n = 18)	RSV B (n = 6)	U	p	Effect size (rrb)
Ct value	Mean ± SD	28.33 ± 6.71	25.95 ± 5.00	—	—	—
	Median (IQR)	27.69 (12.49)	24.67 (4.38)	65.0	0.49	–0.20
	Range	17.58–38.36	21.41–35.10	—	—	—
Age	Median	7 months	10 months	—	—	—
	Range	34days–6 years	4months–3.5 years	—	—	—

Association of age with Ct values

The median Ct value in children <12 months was 24.6, compared to 30.3 in children ≥12 months, demonstrating a significantly higher viral load in younger children (p = 0.02).

Table 6: Association of age with Ct values

Age Group	Median Ct (IQR)	Test	p-value
<12 months	24.6 (~21–35)	Mann–Whitney U	0.02
≥12 months	30.3 (~26–36)		

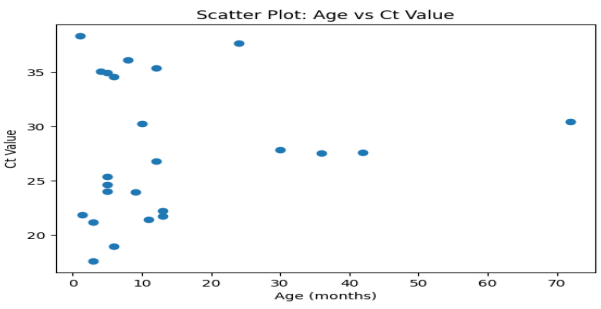

Figure 1: Scatter plot showing the relationship between age (months) and Ct value among RSV-positive cases

A moderate positive correlation was observed between age and Ct value (r = 0.52, p = 0.01), indicating that viral load decreases with increasing age.

Clinical Symptoms Comparison in between RSV A and RSV B positive patients

Clinical presentations were similar between RSV A and RSV B infections. Fever, cough, wheezing, and rhinorrhea were the most common symptoms observed in both groups, with no significant differences (p > 0.05).

Table 7. Clinical Symptoms Comparison in between RSV A and RSV B positive patients

Symptom	RSV A (n=18) (%)	RSV B(n=6)(%)	p-value
Fever	14 (77.8)	3 (50.0)	0.18
Cough	17 (94.4)	6 (100)	0.60
Wheezing	12 (66.7)	5 (83.3)	0.42
Rhinorrhea (running nose)	13 (72.2)	5 (83.3)	0.56
Difficulty breathing	6 (33.3)	2 (33.3)	1.00
Tachypnea	5 (27.8)	2 (33.3)	0.78
Poor feeding	3 (16.7)	1 (16.7)	1.00

DISCUSSION

In this study, the prevalence of RSV infection among children with respiratory illness was 19.7%, highlighting RSV as a significant contributor to pediatric respiratory infections in Kathmandu. This finding is comparable to reports from South Asian settings, including India and Bangladesh, where RSV positivity rates among hospitalized children range from 15% to 30%[12-13]. Similar prevalence has also been reported in Pakistan and China[14-15], supporting the substantial burden of RSV across Asia. Variations in prevalence across studies may be attributed to differences in study populations, seasonal timing, healthcare-seeking behavior, and diagnostic methods.

A male predominance among RSV-positive cases (66.7%) was observed, which is consistent with findings reported from Nepal and other South Asian countries[16-17]. Although the association between gender and RSV positivity was not statistically significant in this study, the observed trend may be explained by biological factors such as smaller airway caliber and sex-related differences in immune responses in male infants[18]. Similarly, no significant association was found between gender and RSV subtype distribution, which is in agreement with studies conducted in East Asian settings such as China and Japan[19-20].

RSV subtype A was the predominant strain (75.0%) in this study, consistent with reports from multiple regions, including Asia and Europe[21-23]. The predominance of RSV-A may be related to its greater genetic variability and potential for immune evasion; however, alternating dominance between RSV-A and RSV-B has been widely documented across different epidemic seasons[23].

Age-wise analysis demonstrated that the majority of RSV infections occurred in infants (<1 year), consistent with findings from studies conducted in Bangladesh, Pakistan, and other low- and middle-income countries[24-25]. Increased susceptibility in younger children may be explained by immature immune systems, smaller airway anatomy, and limited prior exposure to RSV.

The analysis of Ct values showed that younger children (<12 months) had significantly lower Ct values, indicating higher viral loads compared to older children. Similar observations have been reported in studies from China and Japan[26-27]. The moderate positive correlation between age and Ct value further supports that viral load decreases with increasing age, possibly reflecting the gradual development of partial immunity following repeated exposure. Although RSV-B cases showed lower median Ct values than RSV-A, this difference was not statistically significant, consistent with previous studies that have reported no clear subtype-specific differences in viral load[28].

Clinical manifestations were comparable between RSV-A and RSV-B infections, with fever, cough, wheezing, and rhinorrhea being the most frequently observed symptoms. The absence of significant differences in clinical presentation between subtypes aligns with findings from studies conducted in both Asia and Europe[22,29] suggesting that RSV subtype alone may not be a reliable predictor of disease severity.

Beyond South Asia, similar RSV epidemiological patterns have been reported globally. Studies from multiple countries in Africa have demonstrated RSV prevalence ranging from approximately 10–20% among children with acute respiratory infections, indicating a substantial burden in low-resource settings comparable to South Asia[30].

CONCLUSION

RSV contributes substantially to pediatric respiratory illness in Nepal, disproportionately affecting infants who demonstrate higher viral loads. The predominance of RSV-A and the absence of meaningful differences in clinical profiles between subtypes support a unified clinical management approach. Integration of molecular diagnostics such as RT-PCR into routine practice, along with strengthened RSV surveillance and prioritization of high-risk groups, can improve early detection and support timely clinical decision-making. These measures are also essential for guiding future immunization strategies. Overall, these findings provide locally generated evidence that bridges laboratory diagnostics with public health practice and supports health system strengthening in Nepal. This study has limitations that should be considered when interpreting the findings. This study was conducted at a single tertiary care center in Kathmandu with a relatively small sample size of RSV-positive cases, which may limit the generalizability of the results to the broader pediatric population in Nepal.

The cross-sectional design restricts the ability to establish temporal relationships between viral load, disease severity, and clinical outcomes. In this study, only RSV A and RSV B subtyping was performed, and further molecular characterization at the genotype level was not included, which may have provided additional epidemiological insights. In addition, detailed assessment of disease severity markers such as oxygen requirement, duration of hospitalization, and clinical scoring systems was not incorporated, which could have strengthened the clinical correlation analysis.

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AUTHOR CONTRIBUTIONS

Ram Krishna Shrestha designed the study, conducted laboratory work, analyzed data, and drafted the manuscript. Sanjeet Kumar Shrestha assisted in clinical data collection and manuscript review., Bishnu Prasad Upadhyay contributed to methodology and data interpretation., Prakash Ghimire assisted in data management, and literature review., Megha Raj Banjara supervised the study, contributed to study design and data interpretation, and critically revised the manuscript, and approved the final version for publication. All authors approved the final manuscript.

CONFLICT OF INTEREST

The authors declare no competing interests

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