PCR-Based Prevalence of Adenovirus among Children with Respiratory Tract Infections at Jos University Teaching Hospital: A Cross-Sectional Study

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DOI:10.47838/acem.202641.3

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Abstract

Background: Adenoviruses are important viral pathogens associated with respiratory tract infections (RTIs) in children worldwide. In developing countries, routine viral surveillance is limited. This study aimed to determine the prevalence of adenovirus among paediatric patients presenting with respiratory tract infections at Jos University Teaching Hospital, Nigeria.

Materials and Methods: A hospital-based cross-sectional study was conducted from April to July 2023 among children aged 6 months to 15 years. Nasopharyngeal and/or oropharyngeal swabs were tested for adenovirus DNA using real-time PCR. Statistical analysis was performed using descriptive statistics, and the prevalence of adenovirus positivity was calculated with 95% confidence intervals. Associations between variables were assessed using Fisher's exact test.

Results: The study included 253 paediatric patients with respiratory tract infections. Adenovirus was detected in three samples, giving a prevalence of 1.2% (95% CI: 0.2–3.4%). All positive cases occurred among children aged 1–5 years. Two (66.7%) were females, while one (33.3%) was a male. No statistically significant association was observed between adenovirus infection and age group or sex ($p > 0.05$). One PCR-positive sample was successfully sequenced and clustered within Human adenovirus C.

Conclusion: This study demonstrated a low prevalence of adenovirus among paediatric respiratory tract infections in Jos, Nigeria. All detected cases occurred among children aged 1–5 years, highlighting the increased susceptibility of younger children to adenovirus-associated respiratory infections. The findings provide baseline data on adenovirus circulation in the study population and underscore the need for strengthened molecular surveillance of respiratory viruses in Nigeria.

Key words: Adenovirus; Respiratory tract infection; Paediatric; Real-time PCR; Prevalence; Nigeria.

Introduction

Human adenoviruses (HAdVs) are non-enveloped, double-stranded DNA viruses belonging to the Adenoviridae family.[1,2] They are responsible for a broad spectrum of clinical syndromes, ranging from febrile illnesses, gastroenteritis, and conjunctivitis to life-threatening multiorgan diseases in immunocompromised individuals and respiratory tract infections (RTIs).[3]

Phylogenetic analysis based on genomic sequencing has identified at least 90 genotypes classified into seven species (A–G), of which 55 serotypes are known to cause human infections.[3–5] Respiratory tract infections of viral aetiology are among the major causes of hospitalisation in children younger than five years of age [6-8]

Clinically, the symptoms of HAdV infection are similar to those caused by other respiratory viruses. Consequently, establishing a specific viral diagnosis based solely on clinical presentation is difficult.[9] Furthermore, the exact burden and characteristics of HAdV infection in our environment remain largely unknown. [4]

These infections may therefore be misdiagnosed without appropriate laboratory investigations.[10] In addition, the molecular epidemiology and clinical characteristics of HAdV infection among outpatients and inpatients with RTIs have not been well documented in Nigeria, despite HAdV being a common pathogen in children with acute RTIs.[11] Globally, adenovirus infection accounts for approximately 5–10% of acute respiratory tract infections in children. In Nigeria, the reported prevalence ranges from 11.7% to 18.3% among children, with species B and C being the most frequently identified. However, to the best of the researchers' knowledge, no study has specifically investigated adenovirus infection among children with respiratory tract infections at Jos University Teaching Hospital (JUTH). The only available local data are from a study on respiratory tract-associated viruses conducted 22 years ago by Karaivanova and colleagues.[12] In view of these gaps in knowledge, this study was conducted at

JUTH to determine the prevalence of adenovirus infection among children with respiratory tract infections.

The study aimed to assess PCR-based adenovirus prevalence among children with respiratory tract infections in a hospital-based cross-sectional study.

Materials and Methods

Study Area

The study was conducted at the Jos University Teaching Hospital (JUTH), Jos, Plateau State, Nigeria. JUTH is a tertiary healthcare facility that provides specialised medical services to patients from Plateau State and neighbouring states. Children aged 6 months to 15 years presenting to POPD, EPU, or GOPD between April and July 2023 with symptoms suggestive of respiratory tract infection were eligible for inclusion. The study excluded patients whose symptoms were longer than one week, patients with congenital heart diseases, confirmed pulmonary tuberculosis, bacterial pneumonia or any malignancy based on clinical assessment and laboratory results and patients without guardian consent.

Study Design

This study employed a hospital-based cross-sectional design to determine the prevalence of adenovirus among paediatric patients presenting with symptoms of respiratory tract infection.

Ethical Consideration

Ethical approval was obtained from the Research and Ethics Committee of Jos University Teaching Hospital [approval number Juth/DCS/ADM/127/XIX/599/date 9TH May 2022].

Written informed consent was obtained from the parents or legal guardians of all participating children, and assent was obtained from older children where applicable prior to sample collection.

Participant information was coded and handled confidentially; no personal identifiers were used in the analysis.

Study Population

The study population consisted of children aged 6 months to 15 years who presented with symptoms suggestive of respiratory tract infection at Jos University Teaching Hospital during the study period. Eligible children were recruited consecutively from POPD, EPU, and GOPD until the target sample size was reached. EPU participants included children requiring emergency evaluation and were approached when study personnel were available. Each child was enrolled only once during the study period. For children with repeat presentations, only the first eligible presentation was included.

Sample size estimation

The minimum sample size was calculated using the following Cochran's formula for single proportions[12]:

N = Z^2pq/d^2,

where Z=1.96, p=0.183, q=1−p= 18.3% (0.183).[13] and d=0.05.

Because no directly comparable local estimate was available, we used an expected prevalence of 18.3% from a previous paediatric ARTI study.

Calculated minimum sample size

$$N=\frac{1.96^2 \text{ X}0.183 \text{ X } (1-0.183}{0.05^2}$$

$$=\frac{3.8416 \text{ X}0.183 \text{ X } 0.817}{0.0025} = \frac{0.574}{0.0025} = 229.75.$$

The calculated sample size was approximately 230. After adding 10% for invalid or unusable samples, the final target sample size was 253.

Sample Collection and Processing

Nasopharyngeal and oropharyngeal swab specimens were collected from each child and placed into the same viral transport medium container to form one combined respiratory specimen from both outpatients and inpatients presenting with symptoms suggestive of respiratory tract infection between April 2023 and July 2023. Specimen collection followed a standardised protocol across all recruitment sites. Specimens were transported to the laboratory within one hour of collection and processed immediately on arrival.

Clinical symptoms included fever, cough, sore throat, rhinorrhea, nasal congestion, headache, rhinitis, and joint pains.

Trained research staff collected swabs within one week of symptom onset to maximise viral detection, as viral shedding typically decreases after this period. Swabs were placed into sterile universal screw-capped containers containing viral transport medium (phosphate buffer and glycerol).

Each enrolled child contributed one respiratory specimen for PCR testing.

Samples were transported to the laboratory in ice packs at approximately 4°C together with the patients' demographic information. In the laboratory, samples were centrifuged at 3000 × g for five minutes at 4°C to concentrate virus particles attached to host cells. The supernatant was discarded, and the sediment was transferred into a cryovial. Processed samples were stored at −70°C until PCR testing, which was performed within 3months of collection.

Molecular Analysis

Viral DNA extraction

Viral DNA was extracted from processed respiratory specimens using the Zymo Research DNA extraction kit (Zymo Research Corporation, United States), following the manufacturer's instructions.

Frozen samples stored at −70°C were thawed prior to extraction. DNA wash buffer was prepared by adding 24 mL of 100% ethanol to 6 mL of DNA wash buffer concentrate to obtain the final wash solution. For optimal extraction efficiency, 250 µL of β-mercaptoethanol was added to 50 mL of viral DNA buffer.

Briefly, 200 µL of sample was added to 800 µL of viral DNA buffer in a 1.5 mL microcentrifuge tube, vortexed briefly, and incubated at room temperature for 10 minutes. The mixture was then transferred into a Zymo-spin IC column, followed by the addition of 300 µL DNA wash buffer and centrifugation for 1 minute. The washing step was repeated, then 10 µL of DNA elution buffer was added to elute the purified DNA. The extracted DNA was then used for PCR amplification.

Real-time polymerase chain reaction (PCR)

Real-time PCR was performed to detect human adenovirus (HAdV) DNA using the VIASURE Adenovirus Real-Time PCR Detection Kit (Certest Biotec S.L., Zaragoza, Spain). The assay targets the hypervariable regions (loops 1–7) of the adenoviral hexon gene, a genomic region widely used for HAdV detection and characterisation. The assay design enables broad detection of adenovirus strains across the currently recognised HAdV species (A–G).

PCR Reaction Mixture

The PCR reaction mixture was prepared according to the manufacturer's instructions as shown below:

Component	Volume
Respiratory adenovirus PCR reaction mix	15 µL
Respiratory enzyme mixture	5 µL
Template DNA	5 µL
Total volume	25 µL

Amplification was performed using the CFX96 Touch Real-Time PCR Detection System (Bio-Rad Laboratories, Hercules, CA, USA).

Thermal Cycling Conditions

The amplification protocol was carried out according to the manufacturer's recommended thermal cycling conditions as follows:

Step	Temperature	Time	Cycles
Initial denaturation	95°C	60 seconds	1
Denaturation	95°C	15 seconds	
Extension	60°C	30 seconds	45 cycles

Fluorescence detection was performed in the FAM channel during the extension phase.

Quality control and assay validation

Positive and negative controls supplied with the kit were included in each PCR run to ensure assay validity. A PCR run was considered valid only when the positive control amplified within the expected cycle threshold (Ct) range, and the negative control showed no amplification. All PCR runs satisfied the predefined validity criteria.

Interpretation of results

PCR results were interpreted based on the cycle threshold (Ct) values and amplification curve characteristics. Samples exhibiting a typical sigmoidal (S-shaped) amplification curve in the FAM detection channel with a Ct value < 35 were considered positive for adenovirus DNA. Samples showing no detectable amplification were considered negative.

Samples with Ct values between 35 and 38 were classified as indeterminate and were subjected to repeat testing. If repeat analysis produced a reproducible sigmoidal amplification curve with a Ct value < 35, the sample was classified as positive. Otherwise, it was classified as negative. In the present study, three samples yielded indeterminate results on initial testing and were subsequently retested; all three were confirmed negative following repeat analysis.

A PCR run was considered valid only when the positive control amplified within the expected Ct range and the negative control showed no amplification. All PCR runs met the predefined validity criteria.

Definition of respiratory tract infection

Respiratory tract infection was not classified into upper- or lower-respiratory-tract infection categories. It s defined as the presence of one or more of the following respiratory symptoms: cough, sore throat, rhinorrhea, nasal congestion, or rhinitis, with or without fever.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics software version 21.

Demographic characteristics were summarized using descriptive statistics. Categorical variables such as sex and age group were expressed as frequencies and percentages, while continuous variables were summarised using median and interquartile range (IQR). Prevalence was calculated as the proportion of PCR-positive children among all children tested.

Associations between adenovirus PCR positivity and categorical variables were assessed using Fisher's exact test due to the small number of positive cases. Odds ratios (OR) with corresponding 95% confidence intervals were estimated where appropriate to measure the Strength of associations. A p-value < 0.05 was considered statistically significant. Exact binomial 95% confidence intervals were calculated using the Clopper–Pearson method.

All enrolled participants had complete demographic data and valid PCR results.

Results

During the study period, 259 children presenting with respiratory tract infections were assessed for eligibility. Six children were excluded: three due to refusal of parental consent and three who were considered too ill to participate. The remaining 253 children were enrolled, provided adequate nasopharyngeal samples, were successfully tested by PCR for adenovirus, and were included in the final analysis. Of the 253 children enrolled in the study, 142 (56.1%) were female and 111 (43.9%) were male (a female-to-male ratio of 1.3:1). The largest proportion of participants was in the 1–5 years age group (100; 39.5%), followed by <1 year (57; 22.5%), 6–10 years (53; 21.0%), and 11–15 years (43; 17.0%). Children aged below 6 years, comprising the <1 year and 1–5 years groups, constituted the largest proportion of the study population. Table 1 shows baseline age and sex distributions.

Table I: Age and Sex distribution of the study population

Age (Years)	Sex Male	Female	Total	Percent (%)
<1	25	32	57	22.5
1-5	44	56	100	39.5
6-10	23	30	53	21.0
11-15	19	24	43	17.0
Total	111(44%)	142(56%)	253	100

Adenovirus was detected in 3 of 253 children, corresponding to a prevalence of 1.2% (95% CI: 0.24%–3.43%). Because the assay was limited to adenovirus detection, we did not assess the presence of other respiratory viral pathogens. (Figure 1).

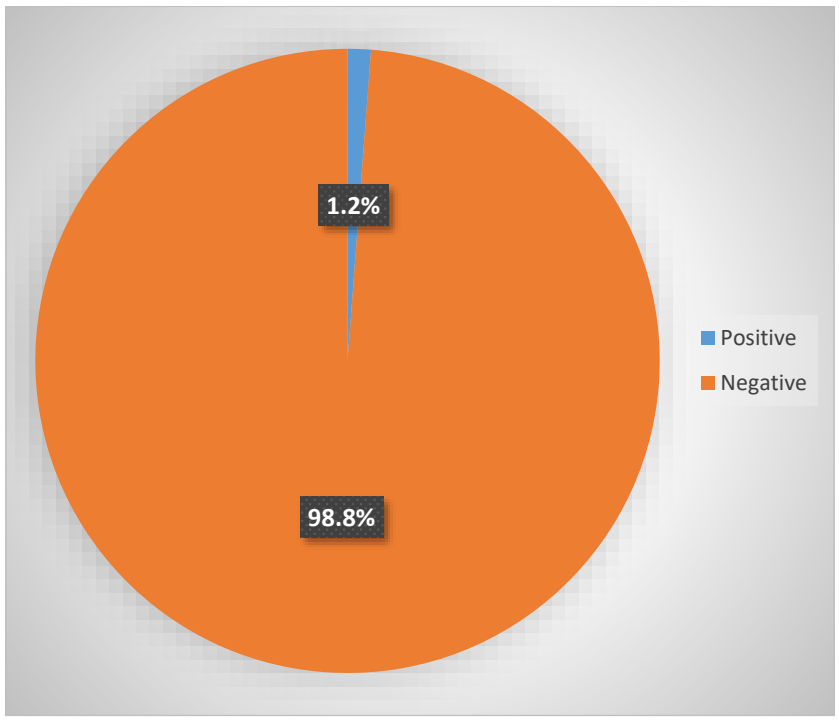

Figure 1: Showing prevalence of Adenovirus among the study population.

All three adenovirus-positive cases (3/3; 100%) occurred in children aged 1–5 years, with no cases detected in those younger than 1 year or in the 6–15 years age group. Of the three positive cases, two (66.7%) were female, and one (33.3%) was male. Given the very small number of positive cases, subgroup comparisons by age and sex were descriptive only and not powered for inferential statistical analysis. Fisher's Exact Test showed no statistically significant association between age group and sex distribution among adenovirus-positive children (p = 0.425). Therefore, any apparent differences across age or sex groups should be interpreted

with caution, and no meaningful statistical association can be inferred from these observations. These findings are summarised in Table 2.

Table II: Age and Sex distribution of children with *adenovirus* associated with Respiratory tract Infection in Jos

Age Year	Male	Female	Total	Percent	x^2	p-value
< 1 year	0	0	0	0	2.586	0.425
1-5 years	1	2	3	100		
6-10 years	0	0	0	0		
11-15 years	0	0	0	0		
Total	1(33.3)	2(66.7)	3	100		

Fisher's Exact Test: p = 0.425One of the three PCR-positive samples with sufficient DNA concentration was selected for sequencing of the hexon 1–7 region. Sequencing was performed using an ABI 3730xl DNA Analyser. The obtained sequence was edited and aligned, then compared with reference adenovirus sequences retrieved from GenBank. Reference sequences were selected based on BLAST similarity and their representation of relevant human adenovirus species and types. The study sequence was subsequently submitted to GenBank under accession number PX905945, and phylogenetic analysis was performed using MEGA version 11.

Phylogenetic analysis demonstrated that the study sequence clustered within the Human adenovirus C (HAdV-C) species. The study sequence clustered with reference strain MW558258 with bootstrap support of 88% and within the broader HAdV-C clade with bootstrap support of 100%. Other reference sequences clustered separately, as shown in Figure 2.

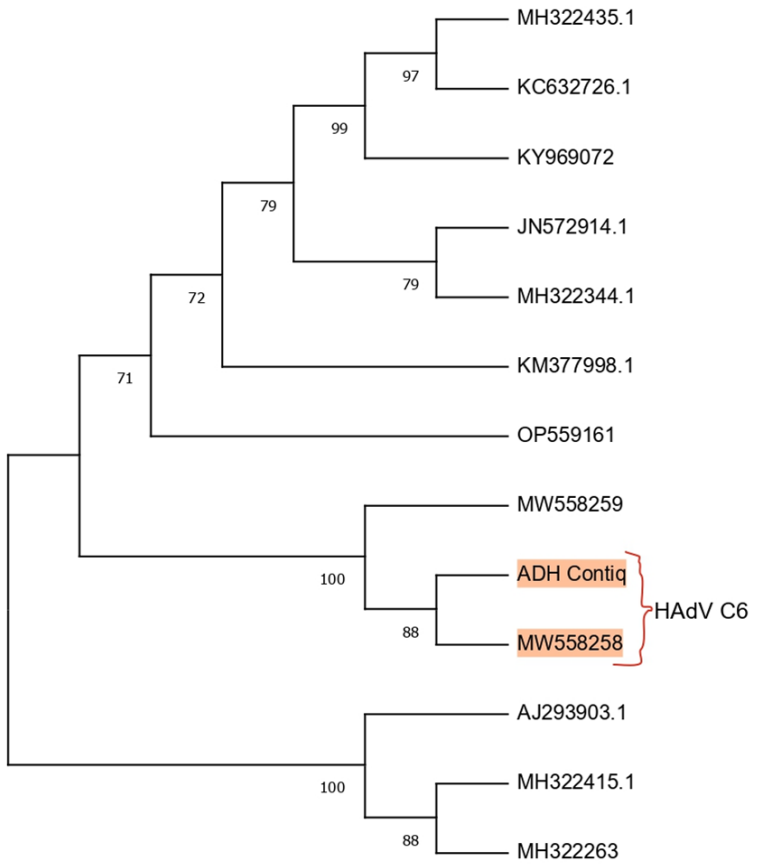

Figure 2: Phylogenetic tree showing the relationship between the adenovirus sequences obtained in this study (PX905945) and selected reference sequences from GenBank. The tree was constructed using the Neighbor-Joining method based on the Kimura 2-parameter model with 1,000 bootstrap replicates in MEGA 11.

Discussion

In this study, adenovirus was detected in 3/253 of paediatric patients presenting with respiratory tract infections (RTIs) at Jos University Teaching Hospital, Nigeria, using real-time polymerase chain reaction (PCR), corresponding to a prevalence of 1.2%.

This prevalence is lower than that reported in several studies conducted in other regions of the world, such as that conducted among hospitalised children in Hainan Province, China, from 2021 to 2024, with a higher prevalence of respiratory human adenovirus (HAdV) of 7.74%. The study found an 11.58% positivity rate in 2024, peaking at 19.42% in June 2024; [13] other studies conducted in parts of China also reported a higher prevalence rate of 3.71% in paediatric populations with respiratory illness.[14] Another study conducted in Brazil recorded a prevalence detection rate of respiratory adenoviruses of 2.8%, which is also higher than that of this study. However, this was in relation to other respiratory viruses. [15] The prevalence rate of 1.2% observed in our study among children is notably lower than a systematic review and meta-analysis conducted in China, which attributed 5–10% of respiratory adenovirus infections to the pediatric population and 1–7% to adult respiratory infections to adenovirus. [16] These comparisons are interpreted cautiously because the cited studies differed in population, sampling period, diagnostic approach, and clinical setting.

Similarly, a review of some African studies has documented a wide range in the prevalence of human adenovirus as a cause of pneumonia in children, identifying Human adenovirus respiratory infection as a causative agent in 1–31% of children with respiratory infections; this is in keeping with the prevalence of 1.2% obtained in this study.[17]

This discrepancy may stem from differences in diagnostic sensitivity, such as the use of antigen-based tests rather than more sensitive PCR methods, as well as seasonal climate variations and the specific age profile of our study population, which spanned larger age groups and may have further contributed to the comparatively low rate observed in our study.[16] These comparisons are again interpreted cautiously because the cited studies differed in population, sampling period, diagnostic approach, and clinical setting.

Furthermore, the lower prevalence recorded in the present study may partly reflect the differences in seasonal circulation of respiratory viruses [14,17] with the study conducted around the rainy (April to July) season, which coincides with seasons in which this virus is in circulation, variations in study populations or the relatively limited sample size. [4,6,14,17]

Respiratory viruses exhibit seasonal fluctuations, and adenovirus transmission may vary across the year. [17] Therefore, the timing of sample collection could influence the detection rate observed in this study[17]. In addition, because this was a hospital-based study, the findings may not reflect adenovirus prevalence among children with milder RTIs managed in the community or primary care**.**[14,18]

The age distribution showed that all three adenovirus-positive cases occurred in children aged 1–5 years, with no positive cases detected in other age groups. However, given the small number of positives, this should be interpreted as a descriptive finding rather than evidence of increased age-specific risk.[14,16] These are general explanations from prior literature and are not findings demonstrated by this study [4,14,17]. Additionally, behavioural factors such as close contact in households, daycare centres, and preschool environments facilitate viral transmission among young children.[18]

This study observed that two of three adenovirus-positive cases were females and one was male; however, the small number of cases prevents meaningful inference about sex differences**.**[19,20] However, other literature conducted at the University of Ilorin, Nigeria, with reports on pediatric and HIV-co-infected populations suggests gender differences are often less pronounced or non-significant. 21 These observed differences in specific studies are often attributed to environmental exposure, behavioural factors, or small cohort sizes rather than inherent biological susceptibility. [16–18,21]

Therefore, the observed difference in this study may reflect random variation due to the limited number of cases detected. The relatively low detection rate observed in this study does highlight the possibility that other viral pathogens may play a more prominent role in paediatric respiratory tract infections in the study environment. [22–24] Respiratory viruses such as respiratory syncytial virus (RSV), influenza virus, parainfluenza virus, rhinovirus, and human metapneumovirus are known to account for a substantial proportion of respiratory infections in children worldwide.[24,25]

Molecular characterisation through sequencing is an important tool for identifying circulating adenovirus strains and understanding their epidemiological patterns. In this study, only one of the three PCR-positive samples had sufficient DNA concentration and quality for sequencing analysis. In comparison, the remaining two samples could not be sequenced due to insufficient nucleic acid content. This challenge is common in molecular virology studies, particularly when viral load in clinical samples is low or when nucleic acid degrades during sample handling, storage, or extraction procedures. 26 However, we successfully sequenced one of the adenovirus-positive samples, providing useful molecular information about the circulating strain in the study population. The fact that only one sample was successfully sequenced limits the ability to draw conclusions regarding the diversity or distribution of adenovirus genotypes circulating in the study area. More successfully sequenced samples would be needed to provide a more comprehensive understanding of the molecular epidemiology of adenovirus infections in this population.[1,6]

Molecular characterisation of the current isolate (PX905945) placed the successfully sequenced isolate within HAdV-C, clustering closely with a previously reported HAdV-C6 reference strain. confirms its placement within the Human adenovirus C (HAdV-C) species. Notably, phylogenetic analysis revealed a high degree of genetic similarity between PX905945 and the reference strain MW558258, supported by a robust bootstrap value. This reference strain was originally identified in a 2020–2021 study of pediatric patients in Tehran, Iran, where HAdV-C6 was documented in children both with and without acute respiratory symptoms. [27] This finding is consistent with previous reports identifying HAdV-C in paediatric respiratory samples, but the single sequence prevents broader inference**.** [27]

Further future studies incorporating multiplex molecular assays and Nanopore sequencing technology capable of detecting multiple respiratory viruses simultaneously will provide a more complete picture of the viral aetiology of paediatric respiratory infections in this setting .[1, 6]

Limitations of the Study

These findings should be interpreted in light of several limitations**,** such as the small number of adenovirus-positive cases, which restricted the ability to conduct robust statistical analyses and limited the exploration of associations with demographic or clinical variables. Moreover, only one sample was successfully sequenced; this further constrained the depth of molecular characterisation and prevented comprehensive assessment of circulating adenovirus genotypes in the study population. Furthermore, the study was conducted in a single tertiary healthcare facility, hence limiting the generalizability of the findings to the wider population. Clinical severity, diagnosis type, hospitalisation status, or outcomes were not analysed. Since sample collection did not cover all seasons (April to July, 2023), Adenovirus detection may vary over time, so restricted sampling can affect prevalence estimates. Again. Age and sex subgroup patterns are exploratory and descriptive because of the small number of positive cases. Lastly, because other respiratory viruses were not tested, this study cannot determine the relative contribution of adenovirus compared with RSV, influenza, rhinovirus, parainfluenza virus, or human metapneumovirus.

Conclusion

This study found a low prevalence of adenovirus among paediatric patients with RTIs at a tertiary hospital in Nigeria. All positive cases occurred among children aged 1–5 years, but the small number of cases limits inference about age-specific susceptibility—sequencing of one positive sample provided preliminary evidence of HAdV-C.

Larger, multi-centre studies using multiplex respiratory virus testing and broader sequencing are needed to define better the viral aetiology and molecular epidemiology of paediatric RTIs in Nigeria. The sequencing result should be interpreted as preliminary molecular evidence from a single isolate, not as evidence of the dominant adenovirus genotype in the study area. Although only one PCR-positive sample was successfully sequenced, the molecular analysis provided preliminary evidence of HAdV-C. These findings contribute valuable baseline data on adenovirus-associated respiratory infections among children in the study area. Strengthening viral surveillance systems and incorporating broader molecular diagnostic approaches for respiratory pathogen diagnosis would improve understanding of the epidemiology of paediatric respiratory infections in Nigeria and support the development of effective prevention and control strategies.

Conflict of Interest

The authors declare no conflict of interest.

Recommendation

Future studies should use a larger sample size, multiple sites, year-round sampling, multiplex viral testing, Ct value reporting, and sequencing of more positive samples.

Acknowledgements

The authors acknowledge the contributions of all the supervisors of the research work. The authors thank colleagues and technical staff, including those at PLASVIREC and Inqaba Biotech, Ibadan, for their assistance during laboratory analysis. Finally, the authors appreciate the support of family members whose encouragement contributed to the successful completion of this work.

References

1. Cao Y, Kong F, Zhou F, Xiao M, Wang Q, Duan Y, et al. Genotyping of human adenoviruses using a PCR-based reverse line blot hybridisation assay. Pathology. 2011; 43(5):488-94.
2. Zaki MES, Eid AR, Faried OA. Clinico-pathological study of adenovirus associated with respiratory infections in children. Open Microbiol J. 2020; 14:48-52.
3. Stasiak AC, Stehle T. Human adenovirus binding to host cell receptors: a structural view. Med Microbiol Immunol. 2020; 209(3):325-33.
4. Chen HL, Chiou SS, Hsiao HP, Ke GM, Lin YC, Lin KH, et al. Respiratory adenoviral infections in children: a study of hospitalized cases in Southern Taiwan in 2001-2002. J Trop Pediatr. 2004; 50(5):279-84.
5. Zou L, Yi L, Yu J, Song Y, Liang L, Guo Q, et al. Adenovirus infection in children hospitalized with pneumonia in Guangzhou, China. Influenza Other Respir Viruses. 2021;
15(1):27-33.
6. Gray GC, McCarthy T, Lebeck MG, Schnurr DP, Russell KL, Kajon AE, et al. Genotype prevalence and risk factors for severe clinical adenovirus infection, United States 2004-2006. Clin Infect Dis. 2007; 45(9):1120-31.
7. Robinson CM, Singh G, Lee JY, Dehghan S, Rajaiya J, Liu EB, et al. Molecular evolution of human adenoviruses. Sci Rep. 2013; 3:1812.
8. Sarantis H, Johnson G, Brown M, Petric M, Tellier R. Comprehensive detection and serotyping of human adenoviruses by PCR and sequencing. J Clin Microbiol. 2004; 42(9):3963-9.
9. Zhao S, Wan C, Ke C, Seto J, Dehghan S, Zou L, et al. Re-emergent human adenovirus genome type 7d caused an acute respiratory disease outbreak in Southern China after a twenty-one year absence. Sci Rep. 2014; 4:7365.
10. Alharbi S, Van Caeseele P, Consunji-Araneta R, Zoubeidi T, Fanella S, Souid AK, et al. Epidemiology of severe pediatric adenovirus lower respiratory tract infections in Manitoba, Canada, 1991-2005. BMC Infect Dis. 2012; 12:55.
11. Möller L, Holland G, Laue M. Diagnostic electron microscopy of viruses with low-voltage electron microscopes. J Histochem Cytochem. 2020; 68(6):389-402.
12. Karaivanova GM. Viral respiratory infections and their role as public health problem in tropical countries (review). Afr J Med Med Sci. 1995; 24(1):1-7.
13. Zeng X, Shi S, Chen M, Weng X, Wen J, Fu X, et al. Epidemiology of respiratory adenovirus infections among hospitalized children in Hainan Province, China, 2021-2024. Pathogens. 2025; 14(12):1246.
14. Zhao MC, Guo YH, Qiu FZ, Wang L, Yang S, Feng ZS, et al. Molecular and clinical characterization of human adenovirus associated with acute respiratory tract infection in hospitalized children. J Clin Virol. 2020; 123:104254.
15. Pscheidt VM, Gregianini TS, Martins LG, Veiga ABG. Epidemiology of human adenovirus associated with respiratory infection in southern Brazil. Rev Med Virol. 2021; 31(4):e2189.
16. Liu MC, Xu Q, Li TT, Wang T, Jiang BG, Lv CL, et al. Prevalence of human infection with respiratory adenovirus in China: a systematic review and meta-analysis. PLoS Negl Trop Dis. 2023; 17(2):e0011151.17. Puenpa J, Tantipraphat L, Aeemjinda R, Vichaiwattana P, Korkong S, Poovorawan Y. Seasonal dynamics and genetic diversity of human adenoviruses in patients with acute respiratory infection in Thailand, 2024. PLoS One. 2025; 20(12):e0338450.
18. Collins JP, Shane AL. Infections associated with group childcare. In: Long SS, Prober CG, Fischer M, editors. Principles and practice of pediatric infectious diseases. 5th ed. Philadelphia (PA): Elsevier; 2018. p. 25-32.e3.
19. Peer V, Mandelboim M, Jurkowicz M, Green MS. Sex differences in acute respiratory tract infections—multi-year analysis based on data from a large tertiary care medical center in Israel. Front Public Health. 2025; 13:1502036.
20. Huang Y, Wang C, Ma F, Guo Q, Yao L, Chen A, et al. Human adenoviruses in paediatric patients with respiratory tract infections in Beijing, China. Virol J. 2021; 18(1):191.
21. Kolawole OM, Oladosu TO, Abdulkarim AA, Okoh AI. Prevalence of adenovirus respiratory tract and HIV co-infections in patients attending the University of Ilorin, teaching hospital, Ilorin, Nigeria. BMC Res Notes. 2014; 7:870.
22. Yunusa T, Egah DZ, Banwat EB, Shwe DD, Kolade-Yunusa OH. Prevalence of H. influenzae among under-five children presenting at the Emergency Paediatric Unit (EPU) of two teaching hospitals in Jos, Plateau State, Nigeria. IOSR J Dent Med Sci. 2014; 13(4):73-9.
23. Albishi NS, Alenazi AO, Alshammari WO, Al Ruwaili JA, Al Samti AMS, Alharbi KHD, et al. Respiratory infections in children: an updated review of pathophysiology, diagnosis, treatment, biochemical aspects, and nursing interventions. J Med Chem Sci. 2024; 7(12):1847-60.
24. Sari IÖ, Bolaç Özyılmaz LG, Günyüz Türe E, Kazaz TG, Türkmen Karaağaç A, Güven Ş. Viral infections among hospitalised children during the COVID-19 outbreak in a tertiary hospital in Turkey: relationship with non-pharmaceutical interventions. Niger Postgrad Med J. 2025; 32(2):88-91.
25. Khales P, Razizadeh MH, Ghorbani S, Moattari A, Saadati H, Tavakoli A. Prevalence of respiratory viruses in children with respiratory tract infections during the COVID-19 pandemic era: a systematic review and meta-analysis. BMC Pulm Med. 2025; 25(1):135.
26. Kathinzi CM, Njenga PK, Mutai JK. Barriers to effective sample management in viral load testing in Machakos County, Kenya: a convergent parallel mixed-method study.
Pan Afr Med J. 2025; 51:26.
27. Zadheidar S, Yavarian J, Heydarifard Z, Nejati A, Sadeghi K, Ghavami N, et al. Molecular epidemiology of human adenoviruses in children with and without respiratory symptoms: preliminary findings from a case-control study. BMC Pediatr. 2022; 22(1):583.