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Influenza C Virus in Children With Acute Bronchiolitis and Febrile Seizures

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Background: Influenza C virus (ICV), a lesser-known member of the *Orthomyxoviridae* family, usually causes mild respiratory illness in children. Due to its low prevalence and clinical similarity to other respiratory infections, it is not routinely screened. This study investigates the detection of ICV in children with acute bronchiolitis (AB) and febrile seizures (FS), comparing the findings with a healthy control group.

Methods: Between October 2009 and September 2011, 499 nasopharyngeal swabs were collected from children up to 6 years old at the University Medical Center Ljubljana. The study group included 307 children diagnosed with AB and 192 with FS, and 150 healthy control children. Respiratory viruses, including ICV, were detected using real-time polymerase chain reaction. ICV-positive samples were further analyzed by Sanger sequencing and phylogenetic analysis.

Results: ICV was detected in 1.3% (4/307) of children with AB and 2.6% (5/192) of those with FS, but in none of the healthy controls. Coinfections occurred in 56% (5/9 cases) of ICV-positive children. ICV was the only virus identified in 1 AB and 3 FS cases. Among AB children, ICV positivity was associated with a more severe disease, including 1 child requiring 5 days of oxygen therapy. Phylogenetic analysis demonstrated that all Slovenian ICV strains belonged to clade I, closely related to the Yamagata lineage.

Conclusions: ICV was detected at low frequencies but exclusively in hospitalized children with AB and FS, not in healthy controls. These findings suggest a possible role of ICV in pediatric respiratory illnesses and seizure-associated infections.

Key Words: influenza C virus, acute bronchiolitis, febrile seizures, pediatric patients, clinical study

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Influenza C virus (ICV) is an enveloped, negative-sense RNA virus belonging to the *Orthomyxoviridae* family, along with influenza A and B viruses. Structurally, it differs from influenza A and B viruses in several ways. Its single-stranded RNA genome is segmented into 7 parts, encoding 9 viral proteins.[1–3] Among these is the hemagglutinin-esterase-fusion glycoprotein, which is unique to ICV. This protein is responsible for receptor binding, sialic acid cleavage, and membrane fusion—functions performed by hemagglutinin and neuraminidase in influenza A and B viruses.[4–7]

ICV is less common and less contagious than influenza A and B viruses.[8] It is primarily transmitted through respiratory droplets, aerosols and contact transmission.[9] In children under 6, especially those under 2, ICV commonly causes mild to moderate lower respiratory tract infections, such as pneumonia, bronchiolitis and bronchitis.[10,11] Additional presentations include vomiting, diarrhea, acute otitis media and even acute encephalopathy.[12–14] In contrast, ICV typically results in asymptomatic or mild upper respiratory tract infections in adults.[8]

Codetection with other respiratory viruses is frequent in hospitalized children, complicating the interpretation of ICV's role in disease severity.[10,11,15] ICV circulates year-round, but its activity peaks during late fall and winter, similar to other influenza viruses, even though it often remains overshadowed by influenza A and B.[13,15–18]

Due to its relatively rare occurrence and milder symptoms, there are no vaccines specifically developed for ICV. Infections are generally managed with supportive care.[19] Several real-time polymerase chain reaction (RT-PCR) assays offer improved sensitivity over traditional culture methods because virus isolation remains challenging due to the weak cytopathic effect of ICV.[16,20–25] As a result, ICV is typically excluded from routine diagnostic screenings. Nonetheless, understanding its distinct features is essential for a comprehensive picture of influenza viruses and their public health implications.

MATERIALS AND METHODS

Ethics

The study protocol was approved by the National Medical Ethics Committee of the Republic of Slovenia (no. 87/08/09) and registered with ClinicalTrials.gov (NCT00987519). Written informed consent was obtained from all the participants' parents. The study followed the principles outlined in the Declaration of Helsinki, the Oviedo Convention on Human Rights and Biomedicine and the Slovenian Code of Medical Deontology.

Patients, Controls, Samples

This study aimed to detect ICV in nasopharyngeal (NP) swabs from children under 6 with acute bronchiolitis (AB)—defined as nasal discharge, cough, wheezing and/or crackles on auscultation, and febrile seizures (FS)—defined as fever-associated cerebral paroxysms without signs of central nervous system infection. Seizures were classified as simple if they were generalized, lasted <15 minutes, and occurred only once within 24 hours, and as complex if they were focal, persisted for 15 minutes, or recurred within 24 hours.[26] Samples were collected from children admitted to the Department of Infectious Diseases at the University Medical Center Ljubljana, between October 2009 and September 2011. The healthy control group included children admitted to the Department

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of Pediatric Surgery and Intensive Care for elective procedures (eg, inguinal hernia, testicular retention or hydrocele testis) during the same period.

NP swabs were taken using flocked-tip swabs and transported in Copan Universal Transport Medium (UTM-RT; Copan Italia, Brescia, Italy). Sample preparation and nucleic acid extraction followed procedures described by Jevsnik et al.[27] In a previous study, real-time RT-PCR was used to detect several respiratory viruses, including human respiratory syncytial virus, influenza A and B virus (FluA-B), parainfluenza viruses 1–3, human metapneumovirus, human bocavirus, human adenovirus and human rhinovirus.[27] For ICV testing, total nucleic acids were isolated from 300 µL of archived NP swab material using the Maelstrom 9610 system (Taiwan Advanced Nanotech Inc., Taoyuan City, Taiwan).[16]

Molecular Detection and Sequencing of ICV

To amplify a 64-bp fragment of the ICV matrix gene (M1), a real-time RT-PCR assay was performed using the QuantStudio 5 Dx Real-Time PCR System (Thermo Fisher Scientific, Grand Island, NY). A total of 5 µL of extracted nucleic acid was combined with 15 µL of a reaction mixture containing TaqMan Fast Virus 1-Step Master Mix, 0.8 µM of each primer and 0.2 µM of the probe as described by Pabbaraju et al.[16] The thermal cycling conditions were as follows: 5 minutes at 50 °C, 20 seconds at 95 °C, followed by 41 cycles of 3 seconds at 95 °C and 30 seconds at 60 °C.

Samples with a Ct value below 30 were considered suitable for sequencing. RT-PCR was performed using forward primer 5′–AAAAATGTTGCTCCTGAGACCAG-3′ and reverse primer 5′-CATCTCAACCAAGCTGTGATTGT-3′, 0.4 µM each and 2.5 µL of nucleic acid. Primer design was performed using PrimalScheme v3.0.2. The amplification was conducted with the PrimeScript One Step RT-PCR Kit (Takara Bio Inc., Shiga, Japan) on a Veriti 96-Well Fast Thermal Cycler (Thermo Fisher Scientific). Cycling conditions were 30 minutes at 50 °C, 20 seconds at 94 °C, followed by 40 cycles of 30 seconds at 94 °C, 30 seconds at 57.8 °C and 60 seconds at 72 °C.

The resulting 856 bp amplicons were verified by 1.5% agarose gel electrophoresis and sequenced using the Sanger method. PCR products were enzymatically purified with FastAP and Exonuclease I (Thermo Fisher Scientific), and sequencing reactions were performed using the BigDye Terminator v3.1 Cycle Sequencing Kit (Thermo Fisher Scientific), according to the manufacturer's protocol. Sequencing was carried out on an ABI-3500 Genetic Analyzer (Thermo Fisher Scientific).

Nucleotide sequences were compared with known ICV matrix gene sequences in the GenBank database using the NCBI BLAST tool (https://blast.ncbi.nlm.nih.gov). A 279 bp region was aligned using the ClustalW algorithm in MEGA-X software (v10.1.7). A maximum-likelihood phylogenetic tree was constructed with MEGA 6.06.

Statistical Analysis

Continuous variables were summarized using median values and ranges or interquartile ranges (IQRs), and discrete variables using frequencies and percentages (with 95% confidence intervals). Fisher exact test was used to compare differences in categorical variables.

RESULTS

In prior testing that did not include ICV, viral etiology was established in 385/499 (77.1%) patients: in 263 of 307 (85.7%) children with AB and in 122 of 192 (63.5%) children with FS.[27]

In this study, which was performed on the same patient group, ICV was identified in 9 of 499 (1.8%) children: in 4 of 307 (1.3%) children with AB and in 5 of 192 (2.6%) children with FS. ICV was codetected with other respiratory viruses in 5 cases (3 AB, 2 FS) and was the sole agent detected in 4 cases (1 AB, 3 FS). More detailed information is presented in Figure 1. None of the 150 healthy control children tested positive for ICV. However, the difference between patients and controls was not statistically significant (9/499 vs. 0/150; $P = 0.127$).

The virus appeared to circulate more frequently during the fall and spring months (Fig. 2). However, due to the low prevalence of ICV, no clear seasonality could be established.

Average cycle threshold (Ct) values for solely ICV-positive and codetected samples were comparable (25.7 vs. 28.9 in AB and 29.2 vs. 31.8 in FS). ICV Ct values detected in AB and FS cases were also comparable (28.1 vs. 30.2, respectively). However, due to the small sample size, statistical comparison between groups was not performed.

Of the 9 samples with Ct values below 30, 5 were deemed suitable for sequencing, and 4 yielded interpretable sequence data. Phylogenetic analysis of the matrix gene revealed that all Slovenian ICV strains clustered within clade I, which includes evolutionarily more recent isolates and is closely related to the Yamagata lineage. Three strains showed the highest similarity to the C/Yamagata/10/89 strain and 1 to C/Iwate/2016, both from Japan (Fig. 3).

Children with ICV as the sole detected virus had a median age of 26.2 months (IQR: 9.4–29.3). All presented with fever >38 °C, sore throat, and oxygen saturation above 90%. None had underlying conditions, such as prematurity (<37 weeks), atopy or chronic pulmonary or cardiovascular diseases. The only child with AB and ICV mono-infection was a 3.7-month-old seriously ill girl, with a bronchiolitis severity score of 7 and Wang respiratory score 2, who required 5 days of oxygen therapy. Diarrhea was observed in 1 child with AB and 1 with FS, both of whom were

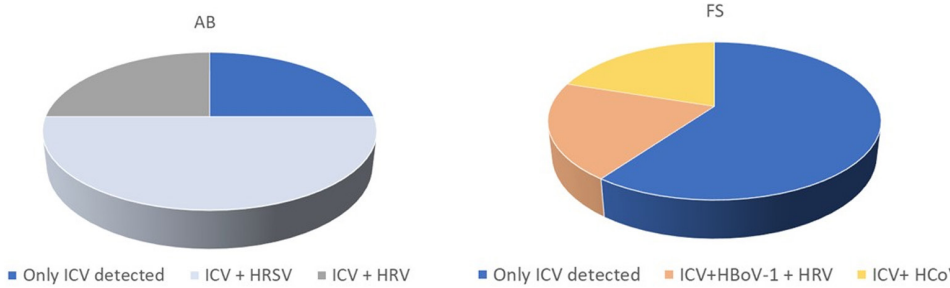

FIGURE 1. Distribution of ICV infections (only and codetected with other respiratory viruses) in children with AB and FS. AB indicates acute bronchiolitis; FS, febrile seizures; HBoV-1, human bocavirus 1; HCoV, human coronavirus; HRSV, human respiratory syncytial virus; HRV, human rhinovirus; ICV, influenza C virus.

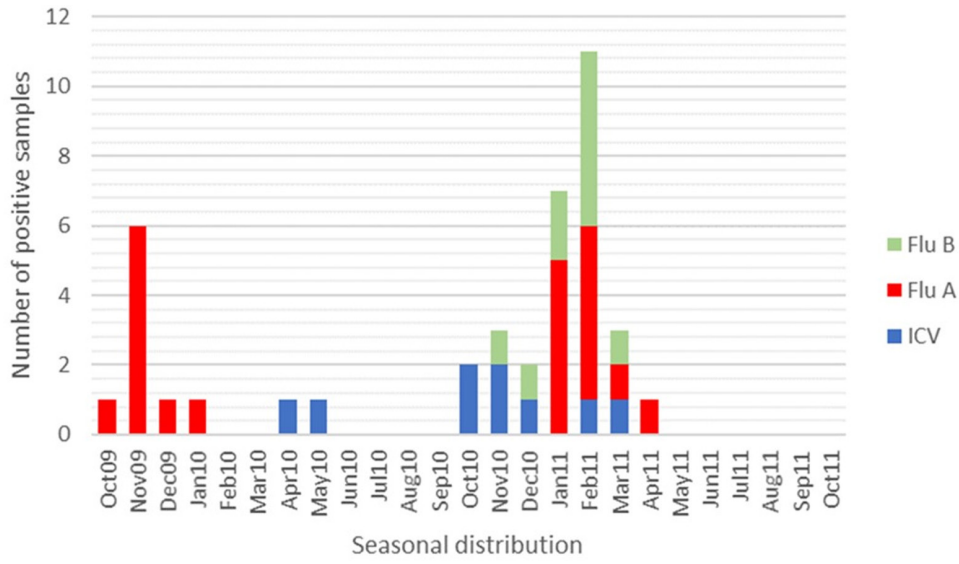

FIGURE 2. Seasonal distribution of ICV-positive samples compared with influenza A (Flu A) and influenza B (Flu B) viruses in children with AB and FS. AB indicates acute bronchiolitis; Flu A, influenza A virus; Flu B, influenza B virus; FS, febrile seizures; ICV, influenza C virus.

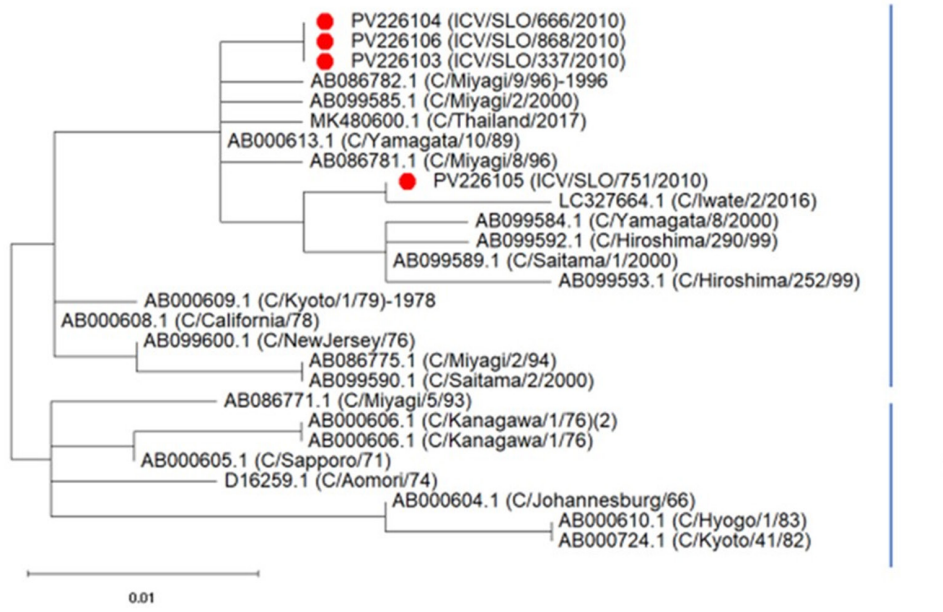

FIGURE 3. Phylogenetic tree based on 279 bp sequences of the matrix M1 gene, including 4 Slovenian ICV strains (●) and reference strains from GenBank (with accession number, country and year). The tree was constructed using the neighbor-joining method with 1000 bootstrap replicates.

positive only for ICV. No differences in clinical presentation were noted between FS cases with solely ICV and those with coinfections. Detailed information on ICV-positive children is presented in Table 1.

DISCUSSION

This study investigated the presence of ICV in children diagnosed with AB and FS, comparing findings with those from a healthy control group. While the rationale for studying children with AB is rather straightforward, the rationale for studying children with FS may appear less obvious. However, the majority of children with FS have viral infections, and the influenza virus is among those most strongly associated with FS.[28] The overall prevalence of ICV was low: it was detected in 1.3% of children with AB and 2.6% of those with FS. Notably, ICV was not identified in any of the healthy controls, suggesting a potential association between ICV and these clinical conditions. However, the difference between the presence of ICV in patients and controls was not statistically significant ($P = 0.127$).

TABLE 1. Clinical, Laboratory and Demographic Characteristics of Influenza C Virus-positive Children With Acute Bronchiolitis or Febrile Seizures

Characteristic Demographic Data	AB (n = 4)	FS (n = 5)
Age (months), median (IQR)	11 (5.1–22.0)	25.4 (15.6–28.6)
Sex (F:M ratio)	1:3	1:1.5
Clinical findings		
Fever > 38 °C	3 (75%)	5 (100%)
Conjunctivitis	2 (50%)	0
Otitis	0	0
Sore throat	4 (100%)	5 (100%)
Diarrhea	1 (25%)	1 (20%)
Hearth rate, median (IQR) (bpm)	168.5 (147–179)	138 (140.5–188.5)
Respiratory rate, median (IQR) (/min)	52.5 (37.5–60.0)	All normal
Saturation, median (IQR) (%)	90.5 (88–94)	98 (96–98.5)
Oxygen therapy	3 (75%)	0
Oxygen therapy duration (days)	5 (5–7)	0
Length of hospitalization (days), median (range)	6 (2–8)	2 (1–4)
Laboratory findings		
CRP, median (range) (mg/L)	24 (19.5–32.5)	20 (3.5–60.5)
Leukocyte count, median (IQR) ($\times 10^9$/L)	10 (8.5–12.7)	16.5 (10.2–18.6)

AB indicates acute bronchiolitis; CRP, C-reactive protein; FS, febrile seizures; IQR, interquartile range.

Interestingly, ICV was more frequently detected among children with FS than those with AB. Among ICV-positive cases, some were coinfected with other respiratory viruses, whereas others had ICV as the only pathogen detected. Mono-infection was more common in FS cases (3/5, 60%) than in AB (1/4, 25%), however, the difference was not statistically significant ($P = 0.523$). Nevertheless, this finding supports previous observations that ICV often cocirculates with other respiratory viruses, making it difficult to determine its independent pathogenic role.[10,11,15]

Children with ICV mono-infection had a median age of 26.2 months and they uniformly presented with fever (>38 °C) and sore throat. None had underlying health conditions, such as prematurity, atopy or chronic pulmonary/cardiovascular diseases. Among AB cases, the only child with ICV mono-infection was a 3.7-month-old infant that experienced a more severe clinical course, requiring 5 days of oxygen therapy. This supports earlier studies suggesting that ICV may contribute to moderate-to-severe respiratory illness, particularly in infants and young children under 2.[10,11]

ICV is not routinely included in standard respiratory virus diagnostic panels. As demonstrated by the AB case with no other identified pathogen, this omission may lead to a missed etiological diagnosis. Especially during periods of influenza A and B circulation, additional testing for ICV may be warranted (Fig. 2). Of note, diarrhea was present in 2 mono-infected cases—1 with AB and 1 with FS—indicating a possible gastrointestinal manifestation, although rare. Similar findings have been reported in cases involving influenza A and B viruses and may be associated with more severe illness.[11,13,15,29–34]

Although the low number of ICV-positive cases precludes definitive conclusions regarding seasonality, our data suggest higher circulation during the fall and spring months—consistent with prior reports.[13,15–18]

Phylogenetic analysis of the ICV matrix gene revealed that all Slovenian strains belong to clade I, which includes evolutionarily recent isolates closely related to the Yamagata lineage. This finding is consistent with global trends, in which clade I viruses continue to circulate with relative genetic stability. To date, 6 ICV lineages have been identified based on HE gene sequencing: C/Yamagata, C/Mississippi, C/Kanagawa, C/Taylor, C/Aichi and C/São Paulo.[16,35–40] Three Slovenian isolates showed the closest genetic similarity to the C/Yamagata/10/89 strain, indicating a strong link to older Japanese strains. The 4th was more like the more recent C/Iwate/2016 strain, suggesting either viral evolution within Slovenia or multiple introductions of ICV from different regions. These findings emphasize the global persistence and movement of ICV strains, with the potential for regional variation driven by transmission dynamics and viral adaptation. The phylogenetic tree (Fig. 3) supports this, showing that Slovenian isolates cluster with clade I strains from other regions. The use of the neighbor-joining method with 1000 bootstrap replicates provides confidence in the tree's topology. Even though the analysis was based on a relatively short sequence (279 nucleotides), it yielded useful insights into the genetic relationships among global and local ICV strains.

This study has several limitations. The small number of ICV-positive cases restricts the ability to draw strong conclusions about its clinical relevance. In addition, the frequent codetection with other viruses complicates efforts to isolate ICV's specific pathogenic role. Larger studies and longitudinal surveillance are needed to better understand the epidemiology, seasonality and clinical spectrum of ICV. Moreover, full genome sequencing and broader geographic sampling could provide deeper insights into the virus's evolutionary dynamics and global circulation patterns. Ongoing surveillance is also essential to monitor potential genetic shifts that may affect ICV's transmission or virulence.

This study documents the presence of ICV in Slovenian children with AB and FS—constituting the first such report from this region. Even though the overall prevalence was low, the absence of ICV in healthy controls and its detection as a mono-infection in some cases support a possible role in pediatric respiratory and febrile illness. Importantly, ICV may contribute to more severe clinical presentations in younger children, particularly in cases of bronchiolitis. Given that ICV is not included in routine diagnostic panels, its role as an underrecognized respiratory pathogen deserves further investigation. Additional testing should be considered in children hospitalized for respiratory illness with negative results for common respiratory viruses. Longitudinal studies and expanded molecular surveillance are needed to better define the clinical and epidemiological impact of ICV.

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