Diagnostic Microbiology and Infectious Disease 86 (2016) 153–159

Contents lists available at ScienceDirect

Diagnostic Microbiology and Infectious Disease

journal homepage: www.elsevier.com/locate/diagmicrobio

Virology

A molecular epidemiological study of human parainfluenza virus type 3 at a tertiary university hospital during 2013–2015 in Catalonia, Spain

Cristina Godoy [a], Paula Peremiquel-Trillas [b], Cristina Andrés [a], Laura Gimferrer [a], Sonia María Uriona [b], María Gema Codina [a], Lluis Armadans [b], María del Carmen Martín [a], Francisco Fuentes [a], Juliana Esperalba [a], Magda Campins [b], Tomàs Pumarola [a], Andrés Antón [a,*]

[a] Respiratory Viruses Unit, Microbiology Department, Hospital Universitari Vall d'Hebron, Vall d'Hebron Research Institute, Universitat Autònoma de Barcelona, Passeig Vall d'Hebron 119-129, 08035, Barcelona, Spain

[b] Preventive Medicine and Epidemiology Department, Hospital Universitari Vall d'Hebron, Vall d'Hebron Research Institute, Universitat Autònoma de Barcelona, Passeig Vall d'Hebron 119-129, 08035, Barcelona, Spain

ARTICLE INFO

Article history:
Received 4 April 2016
Received in revised form 20 July 2016
Accepted 21 July 2016
Available online 27 July 2016

Keywords:
Human parainfluenza virus type 3
HPIV-3
Immunocompromised
Children
Haemagglutinin-neuraminidase
Fusion protein
Molecular characterization
Phylogenetic analysis

ABSTRACT

Human parainfluenza virus type 3 (HPIV-3) is one of the most common respiratory viruses particularly among young children and immunocompromised patients. The seasonality, prevalence and genetic diversity of HPIV-3 at a Spanish tertiary-hospital from 2013 to 2015 are reported. HPIV-3 infection was laboratory-confirmed in 102 patients (76%, under 5 years of age). Among <5 years-old patients, 9 (11.5%) were under any degree of immunosuppression, whereas this percentage was significantly higher (19; 79.2%) among patients older than 5 years. HPIV-3 was detected at varying levels, but mainly during spring and summer. All characterized HN/F sequences fell within C1b, C5 and in other two closely C3a-related groups. Furthermore, a new genetic lineage (C1c) was described. Genetic similarity and epidemiological data confirmed some nosocomial infections, highlighting the importance of the HPIV-3 surveillance, particularly in high-risk patients. This study provides valuable information on HPIV-3 diversity due to the scarce information in Europe.

© 2016 Elsevier Inc. All rights reserved.

1. Introduction

Human parainfluenza viruses (HPIVs) are an important cause of acute respiratory tract infections (ARI). Human parainfluenza virus type 3 (HPIV-3) is an enveloped non-segmented, negative, single-stranded RNA virus, which is classified as member of the genus *Respirovirus* of the family *Paramyxoviridae* (Henrickson, 2003). HPIV-3 infection in immunocompetent adults usually courses as mild and self-limited. However, HPIV-3 is the second cause of viral lower respiratory tract infection (LRTI) in young children, only preceded by respiratory syncytial virus (RSV) (Counihan et al., 2001; Henrickson, 2003; Iwane et al., 2004). Children under 5 years of age, immunocompromised individuals and the elderly are at high risk for severe or even fatal infection (Falsey, 2012; Henrickson, 2003; Liu et al., 2013; Reed et al., 1997).

Unlike other HPIVs, HPIV-3 usually causes seasonal outbreaks during spring and summer months in community; as well, it has been sometimes reported to cause nosocomial outbreaks, in particular among hematopoietic stem cell transplantation (HSCT) recipients (Cortez et al., 2001; Harvala et al., 2012; Jalal et al., 2007; Piralla et al., 2009; Ustun et al., 2012).

To date, neither antiviral drugs nor vaccines are approved for clinical use against HPIV-3 infection (Falsey, 2012; Guillon et al., 2014). In most severe cases the administration of ribavirin has been reported, but its efficacy and benefits have not still been demonstrated (Falsey, 2012). Regarding prophylaxis, two chimeric bovine/human PIV-3 vaccines based on haemagglutinin-neuraminidase (HN) and fusion (F) proteins were found to be well tolerated and immunogenic (Karron et al., 2012; Schmidt et al., 2011), but remained under study in clinical trials. HPIV-3, like other RNA viruses, has the capability to acquire random point mutations throughout its genome, mainly in the envelope glycoproteins (HN and F) that are under the selective pressure from human immune response. The rate of molecular evolution of HPIV-3 based on HN protein was estimated at 1.10×10^{-3} substitutions per site per year, similar to that of other virus genes such as the *G gene* of respiratory syncytial virus (Mizuta et al., 2014). The pattern of HPIV-3 evolution, based on the analysis of *F* gene sequences, it is quite similar to influenza virus B (Prinoski et al., 1992). Since the rest of the genome remains highly conserved, the non-coding region of *F* gene and the coding region of *HN* gene are often used to perform molecular epidemiological studies

* Corresponding author. Tel.: +34-665941722.
E-mail address: aanton@vhebron.net (A. Antón).

http://dx.doi.org/10.1016/j.diagmicrobio.2016.07.023
0732-8893/© 2016 Elsevier Inc. All rights reserved.

Table 1
Primers and PCR amplification and sequencing protocols of complete HN protein-coding region and partial *F* gene sequences. The M13 primer binding sites used for sequencing are underlined.

Gene (Fragment position*)	Primer name	Primer sequence (5′ - 3′)
PCR amplification protocol		
HN**	HN1-M13F	TGTAAAACGACGGCCAGTAACTTAGGAGTAAAGTTACRCA
(6708–7658)	HN1-M13R	CAGGAAACAGCTATGACCATCAAGTACAATTTCTTCTATGCC
HN**	HN2-M13F	TGTAAAACGACGGCCAGTCTGTAAAYTCAGAYTTGGTACCTG
(7474–8320)	HN2-M13R	CAGGAAACAGCTATGACCGCTGTTGAGTAAGTTATGACTGG
HN**	HN3-M13F	TGTAAAACGACGGCCAGTCATAATGTGCTATCAAGACCAGG
(8139–8572)	HN3-M13R	CAGGAAACAGCTATGACCCTGATTGCTGATTACTTATCATATACTTG
F	F-M13F	TGTAAAACGACGGCCAGTACTTAGGACAAAAGARGTCA
(4855–5243)	F-M13R	CAGGAAACAGCTATGACCACCACAAGAGTTAGARTCTTC
Thermal profile:	50 °C × 30 min – 95 °C × 15 min – 45 cycles (95 °C × 15 sec – 52 °C × 20 sec – 72 °C × 1 min) – 72 °C × 10 sec	
Sequencing protocol		
M13	M13F	TGTAAAACGACGGCCAGT
	M13R	CAGGAAACAGCTATGACC
PCR Thermal Profile	96 °C × 1 min – 30 cycles (96 °C × 10 sec – 50 °C × 5 sec – 60 °C × 4 min)	

* Nucleotide position relative to KF530250 sequence from GenBank.
** Primers as previously described [27], with minor modifications.

(Almajhdi, 2015; Côté et al., 1987; Mao et al., 2012; Prinoski et al., 1992; Storey et al., 1987).

The aim of this study was to describe the prevalence, the seasonality and the genetic diversity of HPIV-3 detected in respiratory specimens from patients attended at the Hospital Universitari Vall d'Hebron (HUVH) in Barcelona (Spain) from the 2013–2014 season to the 2014–2015 season.

2. Materials and Methods

2.1. Sample collection

A descriptive observational study was conducted from upper and lower respiratory tract specimens received to our laboratory for viral diagnosis, which were collected from patients, children and adults, attended at the emergency care unit or hospitalized in HUVH, a tertiary 1,200-bed university hospital, from week 40/2013 (2013–2014 season) to week 20/2015 (2014–2015 season), including the 2014 inter-seasonal period. In addition, demographic and clinical data from these patients were collected from the hospital clinical records. Institutional review board approval (PR_AG_156/2015) was previously obtained from the HUVH Clinical Research Ethics Committee.

2.2. Detection of HPIV-3 and RNA extraction

The detection of HPIV-3 and other respiratory viruses was routinely performed either by direct immunofluorescence antigen detection (D^3*Ultra* 8™ DFA Respiratory Virus Screening & Identification Kit Diagnostic HYBRIDS, USA) or by a multiplex real-time PCR assay (Anyplex II RV16 Detection Kit, Seegene, Korea). Total nucleic acids (NA) were purified by using NucliSENS® EasyMag® (BioMérieux, Marcy l'Etoile, France) from 400 µl of respiratory specimens and eluted to 100 µl according to the manufacturer's instructions. Eluted NAs were kept at −80 °C until use.

2.3. HN and F genes sequencing

From laboratory-confirmed HPIV-3 specimens, overlapping fragments that span the entire coding sequence of HN protein and partial *F* gene sequence were amplified by using a one-step RT-PCR-based

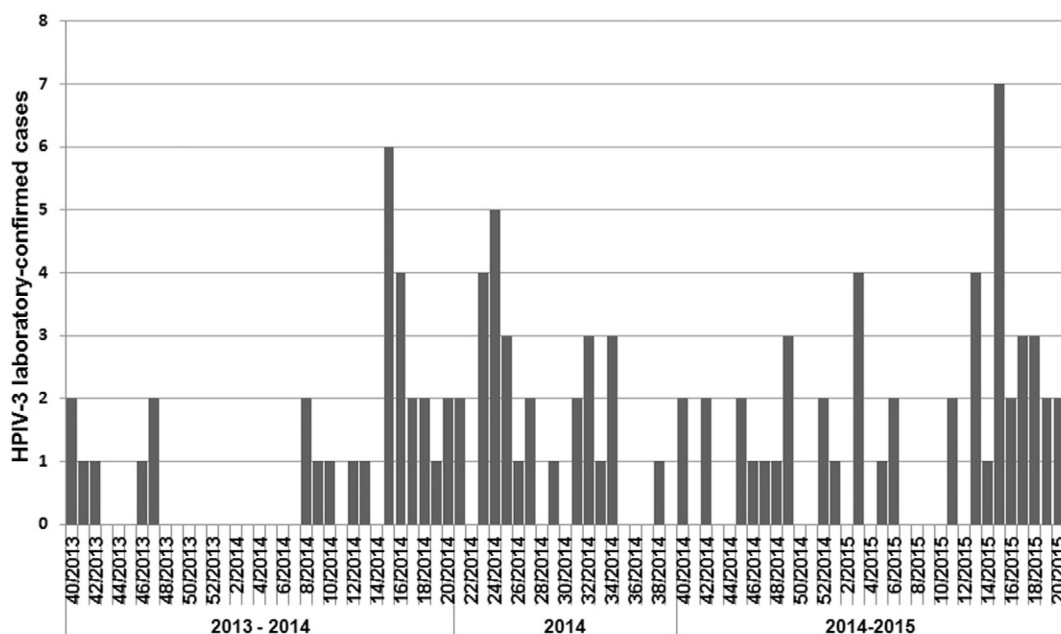

Fig. 1. Weekly distribution of laboratory-confirmed HPIV-3 cases detected from week 40/2013 (2013–2014 season) to week 20/2015 (2014–2015 season).

Download English Version:

https://daneshyari.com/en/article/3346759

Download Persian Version:

https://daneshyari.com/article/3346759

Daneshyari.com