Molecular epidemiological analysis of three hepatitis C virus outbreaks in Jammu and Kashmir State, India

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Outbreaks of hepatitis C virus (HCV) infection are associated with unsafe injection practices, intravenous drug abuse and other exposure to blood and body fluids. We report here three outbreaks of HCV infection from Jammu and Kashmir (J&K) State, India, which occurred over a period of 3 years and in which molecular epidemiological investigations identified a presumptive common source of infection, most likely a single healthcare venue. Representative blood samples collected from cases of hepatitis C were sent to the National Centre for Disease Control (NCDC) for molecular characterization. These samples were positive by HCV ELISA. Subsequently, specimens were also tested for the presence of HCV RNA by RT-PCR. Sequencing was carried out for all positive samples. A total of 812 cases were laboratory confirmed by HCV ELISA; a total of 115 samples were sent to the NCDC for RT-PCR, and 77 were positive. Subtype 3a of HCV was found in all samples from Anantnag (February 2013); and for subtype 3b, in all samples from Srinagar (May 2015). Subtypes 3a and 3g were identified from two samples from the Kulgam outbreak (July 2014). A detailed epidemiological investigation should be conducted whenever a cluster of HCV cases is revealed, as this potentially allows for the identification of larger outbreaks. Epidemiological investigations of outbreaks should be further supported by inclusion of molecular tests. Efforts to limit therapeutic injections to only those cases having strong medical/surgical indications and to restrict the use of non-sterile needles are essential to prevent transmission of HCV.

INTRODUCTION

The hepatitis C virus (species: hepatitis C virus; genus: Hepacivirus; family: Flaviviridae) (HCV) is a major global pathogen, and it is estimated that the worldwide public health burden of this infection is around 170 million cases which is expected to increase further (Davis et al., 2010; Negro, 2014; Razavi et al., 2014). India has not been spared by this scourge, and HCV infection is spreading rapidly there (NCDC Newsletter, 2014). India has not been spared by this scourge, and HCV infection is spreading rapidly there (NCDC Newsletter, 2014). According to a WHO report, as many as 12 million people may be infected with HCV in India, and most do not know that they are infected (WHO, 2002).

HCV may result in an acute or a chronic infection. Acute HCV infection is defined as the presence of clinical signs and symptoms of hepatitis within 6 months of exposure (Missiha et al., 2008). HCV usually remains clinically silent following acute infection, and in 15–45% of individuals with acute infection the virus clears on its own. The remaining 55–85% increase further (Davis of infected individuals carry the virus for the rest of their lives and are considered to have chronic infection (Hoofnagle, 2002; Leone & Rizzetto, 2005). Left untreated, chronic HCV infection causes liver cirrhosis, hepatocellular carcinoma and liver failure (Chen & Morgan, 2006).

The laboratory tests used to diagnose HCV infection consist of serologic assays that detect human antibodies against HCV (anti-HCV) and molecular assays that detect HCV nucleic acids (Chevaliez & Pawlotsky, 2005; Ghany et al., 2009). The serologic tests used to diagnose HCV include three different types of assay: enzyme immunoassay (EIA), chemiluminescent assay and recombinant immunoassay assay. The thir-
Molecular epidemiology of HCV outbreaks

Sequence analysis performed on isolates from different geographical areas has revealed that HCV can be classified into 11 major genotypes (designated 1–11) and many subtypes (designated a, b, c, etc.) based on the genomic sequence heterogeneity (WHO, 2002). The variation observed in the different genotypes is around 30–34% in the nucleotide positions over the entire viral genome, whereas the subtypes differ by about 23% in nucleotide sequence (Alexopoulo & Dourakis, 2005). In addition, HCV RNA circulates in the infected individuals as mixtures of closely related but distinct viral populations, referred to as quasi-species, which display up to 9% diversity from each other and are derived from an original strain by point mutations (Bréchot, 1996; Pawlotsky, 2003).

These genetic diversities have distinct consequences: different genotypes vary in their responsiveness to interferon/ribavirin combination therapy, and such heterogeneity also hinders the development of vaccines, since vaccine antigens from multiple serotypes are necessary for global protection (Ohno & Lau, 1996). This highlights the importance of determining the infecting genotype.

In India, all designated government-run primary healthcare centres, hospitals and private facilities serve as reporting units which collect and report data on HCV cases and outbreaks to district surveillance units, and finally, aggregate data are submitted to the Integrated Disease Surveillance Program (IDSP) via the Indian National Centre for Disease Control (NCDC), through a web portal (Kumar et al., 2015). Each suspected case of HCV infection is initially tested for anti-HCV antibodies by the ELISA test and, for those specimens scored as positive in this assay, confirmation is subsequently sought by testing for HCV-RNA using RT-PCR.

There are only a few reports of community-based HCV outbreaks from India. According to the data available in the division dealing with the IDSP of our institute, HCV outbreaks have been documented from the Bareilly district of Uttar Pradesh in June 2011; Kurukshetra district of Haryana in April 2012; districts Kaithal and Jind of Haryana in April 2013; and districts Barnala and Hoshiarpur of Punjab in 2013. We report here a series of three different outbreaks of HCV infection from the J&K state of India which occurred over a period of three consecutive years in the community/non-hospital setting, and in which the molecular epidemiological investigations identified a presumptive common source of infection, most likely a single healthcare venue. The community-based outbreak data reported in this study provide new insights into the magnitude of these outbreaks and the current prevailing genotypes in community settings. This will impact our knowledge on the contribution of HCV in the causation of liver disease and the associated morbidity and mortality, and strengthen the existing surveillance system and bring about changes in the legislation dealing with unqualified medical practitioners in such areas of India.

METHODS

Outbreaks of HCV infection were reported to the NCDC, Delhi, by investigators at the State Health Department of J&K State, seeking laboratory assistance for the molecular characterization of outbreak strains during a 3-year period from January 2013 to May 2015.

Serum specimens from a few representative HCV seropositive subjects were submitted to the Hepatitis Laboratory and the Biotechnology Department of NCDC for viral testing. All these samples were positive by third-generation HCV ELISA. Since these were all outbreak-related samples, no prior ethical approval was required for testing.

Sociodemographic data for each outbreak were collected in a standardized format and submitted to the NCDC along with each group of specimens. For this study, we also attempted to analyse the setting of the outbreak and the suspected source of the infectious agent, the size of the
outbreak and age of the individuals affected, by interviewing suspected cases. Data were not available for all of the above categories in all outbreaks.

Specimens were processed as follows: serum samples were tested using a commercially available third-generation ELISA for IgG to hepatitis C according to the manufacturer’s instructions at the public health laboratory of Srinagar. Representative specimens sent to the NCDC were further tested for the presence of HCV RNA by RT-PCR amplification of the 5'-UTR region of the HCV genome. RNA extraction was performed from each serum sample using the QiAmp Viral RNA Kit (Qiagen) according to the manufacturer’s protocol. 5'-UTR of HCV with amplicon size 249 bp was amplified using nested PCR. The nucleotide sequences of the primers (Ma et al., 2001) used for PCR amplification are shown in Table 1. The outer RT-PCR was performed using the GenAmp RNA PCR core kit (Applied Biosystems), while the inner PCR was carried out using the Green Master Mix PCR kit (Promega) according to the manufacturer’s instructions. The concentration of primers used in both rounds of PCR was 10 pmol. The programme for the outer PCR was as follows: cDNA synthesis at 45 °C for 45 min followed by initial denaturation at 95 °C for 10 min and 35 cycles of alternate 95 °C for 30 s, 48 °C for 30 s, 72 °C for 30 s and a final extension at 72 °C for 7 min. For inner PCR, initial denaturation was at 95 °C for 5 min followed by amplification comprising 35 cycles of alternate 94 °C for 30 s, 50 °C for 30 s, 72 °C for 30 s and a final extension at 72 °C for 7 min. Standard precautions recommended for PCR were strictly adhered to. Quality of the PCR products was checked on 1% (w/v) agarose gel electrophoresis in 1× Tris acetate EDTA buffer (40 mM Tris, 20 mM acetic acid and 1 mM EDTA), pH 8.3 (Merck). The agarose gels were stained with ethidium bromide. The PCR product bands in the gel were visualized under an ultraviolet transilluminator (Gel Documentation System; AlphaImager EC).

Specimens from Anantnag (n=42, designated ANT 1–42), Kulgam (n=2, KLG 6, KLG 7) and Srinagar (n=22, SNG 1–22) were sequenced. Automated nucleotide sequencing was carried out for all positive samples. Sequencing PCR was carried out for purified double-stranded PCR fragments using the Big Dye Terminator cycle sequencing Kit 3.1 (Applied Biosystems). Each sample was sequenced using inner PCR, forward and reverse primers. Purification of the extension products was carried out manually using a sodium acetate and ethanol precipitation method. The purified DNA was lyophilized and re-suspended in 12 μl Hi-Di formamide (Applied Biosystems) followed by heat (95 °C for 2–3 min) and immediate chilling on ice (+4 °C) for 5–10 min. The DNA was finally loaded onto a 3130xl Genetic Analyzer (Applied Biosystems), and data were collected.

Sequences were analysed for their corresponding peaks in the electropherogram of the sequence analyser. All the sequences collected from Genetic Analyzer were resolved with the help of bio-informatics software MEGA version 6.06 (Tamura et al., 2013) and BioEdit version 7.0.9.0 (Hall, 1999), and multiple sequence alignment of study sequences was performed (Fig. 1). Basic Local Alignment Search Tool (BLAST) (Source: https://blast.ncbi.nlm.nih.gov/Blast.cgi?PAGE_TYPE=BlastSearch) analysis of HCV sequences was performed to compare the data to the globally available sequences in the National Center for Biotechnology Information database. The resolved sequences of all study cases were submitted to GenBank, and accession numbers were obtained (GenBank accession numbers of the sequences of this study are KU166923–KU166986). For nucleotide sequence analysis, all the HCV outbreak sample sequences were compared to the HCV prototype H77 sequence (GenBank accession number AF009606).

RESULTS

Outbreaks

In total, three outbreaks of HCV infection were reported from January 2013 to May 2015 with requests for molecular characterization of the outbreak strains. These outbreaks were reported from Takya Magam village and the adjoining villages of Block Larnoo, Anantnag district, J&K, in January and February 2013; from Checkpora village of Kulgam district in June 2014, and from Srinagar city in May 2015.

Epidemiological characteristics

The most common setting reported was a community setting (Table 2). In January 2013, during routine investigations, some cases were found to be positive for hepatitis C IgG antibodies by ELISA in the Anantnag district. The Chief Medical Officer of Anantnag district informed the state surveillance officer (SSO) about these cases in Block Larnoo. A team consisting of an epidemiologist, SSO, a physician and state microbiologist visited the area, and a mass screening of the community in this district was performed by conducting a house-to-house survey. Temporary medical camps were organized in this district to collect blood samples from suspected cases. Samples were tested at the public health laboratory of Srinagar by ELISA, and 79 representative samples positive for HCV IgG were sent to the NCDC for genotyping.

Following reports of jaundice and chronic liver disease among adults in the village of Checkpora, district Kulgam, in June 2014, an investigation was conducted on the suspected population to examine the exact burden of hepatitis in this area.

In May 2015, six documented cases of HCV infection were reported from the leper colony of Srinagar; therefore, a mass screening was initiated by the J&K state health authorities in this colony.

Molecular and phylogenetic characteristics

Of the 79, 6 and 30 samples received from the Anantnag, Kulgam and Srinagar districts in the biotechnology division for molecular characterization of HCV, the presence of HCV-specific 5'-UTR was confirmed by RT-PCR in 53 (67.1%), 2 (33.3%) and 22 (73.3%) samples, respectively.

The length of the sequences in the study varied from 248 to 251 nt (concordant with nt positions 63–313 of the
H77 prototype sequence of HCV). All 42 sequences from the HCV outbreak in Anantnag belonged to subtype 3a, whilst all 22 sequences from Srinagar belonged to subtype 3b. Of the two sequences from Kulgam, one belonged to subtype 3a whereas the other belonged to a comparatively rare subtype, 3g. Phylogenetic and molecular evolutionary analysis was conducted using MEGA version 6. Phylogenetic analysis also revealed that all 64 study sequences from the HCV outbreaks in Anantnag, Kulgam and Srinagar districts of Kashmir belonged to HCV genotype 3. The dendrogram shows that there was separate clustering of HCV outbreak study sequences (Fig. 2). All the study sequences were grouped together, but in different clades.

Various specific patterns of transitions and transversions were observed in the HCV outbreak sample sequences, as shown in Table 3. The most notable findings were: at position 175, there was transition of T to C in all sequences of ANT and KLG and some SNG sequences. Nucleotide 178 showed C to T transition in all ANT sequences, but none in sequences KLG and SNG. A di-nucleotide mutation at nt 182 and 183 showed consistent transition of A to G and C to T, respectively, in all sequences of ANT and KLG. Another di-nucleotide mutation at nt 203 and 204 showed consistent transversion of T to A and A to C in all sequences of the study. Nucleotide 217 showed G to A transition in all sequences of ANT and KLG. All ANT samples had a point mutation, transition of G to A at nt 221, which was not seen in any of the KLG and SNG sequences. Nucleotide 224 showed consistent transversion of G to A in all outbreak sequences. A very prominent tri-nucleotide transition

![Fig. 1. Multiple nucleotide sequence alignment of 5'-UTR of HCV in comparison to prototype H77.](http://jmm.microbiologyresearch.org)

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mutation of C, T, G to T, C, A, respectively, was observed in all the outbreak samples of ANT, KLG and SNG at nt positions 247–249.

**DISCUSSION**

Our epidemiological and molecular investigations revealed a series of three outbreaks of HCV infection identified in the community through the public health disease reporting and notification system of J&K State in India. Based on molecular evolutionary analysis by the construction of a phylogenetic tree and interviews of the suspected patients, we can only speculate that the use of inadequately sterilized needles and syringes by an unqualified medical practitioner was the most likely risk practice associated with these sets of outbreaks. The common modalities of HCV infection are blood transfusions, injection drug use, unsafe therapeutic injections and healthcare-related procedures. In developed countries, the predominant cause of HCV infection is intravenous drug use, whereas in India, blood transfusions and unsafe therapeutic injections are the predominant means of transmission (Verma et al., 2014).

Although outbreaks of HCV infection in both hospital and community-based/non-hospital settings have been reported from various parts of the world (Luby et al., 1997; Comstock et al., 2004; Savey et al., 2005; Thompson et al., 2009), this is, to the best of our knowledge, the first report of a series of community-based/non-hospital HCV outbreaks in India. By
non-hospital/community setting, we mean small outpatient clinics, day care centres, polyclinics and specialty clinics. As shown in Tables 2 and 4, the most common age group was 25–34 years in all three outbreaks. This correlates well with the observations of other researchers who investigated blood-borne viral hepatitis outbreaks (Patel et al., 2012; Verma et al., 2014). From this, we conclude that this age group, being the most productive, seeks injections more often than others for any illness, based on the belief that injections are a more effective treatment than oral medications and will achieve a faster cure and hence decrease morbidity and disability days.

In this study, we identified subtype 3a of HCV in all samples from the outbreak in Anantnag (February 2013) and subtype 3b in all samples from the outbreak in Srinagar (May 2015). Subtypes 3a and 3g were discerned, respectively, from the two samples from the Kulgam outbreak (July 2014). This geographical distribution of HCV genotypes 3a and 3b is similar to previous reports from North India (Singh et al., 2004; Chakravarti et al., 2011, 2013). The atypical subtype 3g found in one of the Kulgam outbreak samples has also been reported by other investigators from both India and other countries (Panigrahi et al., 1996; Lu et al., 2013, Mohanraj et al., 2015).

### Table 2. Epidemiological characteristics of the three outbreaks

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>No. of suspected cases</td>
<td>2051</td>
<td>187</td>
<td>187</td>
</tr>
<tr>
<td>No. of confirmed cases by third-generation ELISA</td>
<td>767</td>
<td>6</td>
<td>39</td>
</tr>
<tr>
<td>No. of samples sent to the NCDC for RT-PCR</td>
<td>79</td>
<td>6</td>
<td>30</td>
</tr>
<tr>
<td>No. of samples positive by RT-PCR</td>
<td>53</td>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>No. of samples for which sequencing was carried out</td>
<td>42</td>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>Median age of affected persons (years)</td>
<td>33</td>
<td>23</td>
<td>33</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>29</td>
<td>2</td>
<td>17</td>
</tr>
<tr>
<td>Female</td>
<td>50</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Seasonality</td>
<td>January–February</td>
<td>July</td>
<td>May</td>
</tr>
</tbody>
</table>

### Table 3. Tabular representation of nucleotide mutations in 5’-UTR of HCV outbreak sample sequences

<table>
<thead>
<tr>
<th>Nucleotide position</th>
<th>From</th>
<th>To</th>
<th>Sample nos.</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>G</td>
<td>A</td>
<td>ANT (93–98, 102–108)</td>
</tr>
<tr>
<td>119</td>
<td>A</td>
<td>C</td>
<td>ANT (74–76, 84–86, 89, 91–94, 106–107), KLG (ALL)</td>
</tr>
<tr>
<td>175</td>
<td>T</td>
<td>C</td>
<td>ANT (ALL), KLG (ALL), SNG (2, 4, 9, 11, 19, 21, 22, 23, 25)</td>
</tr>
<tr>
<td>178</td>
<td>C</td>
<td>T</td>
<td>ANT (ALL)</td>
</tr>
<tr>
<td>179</td>
<td>A</td>
<td>G</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL except 3, 10, 14, 15, 24)</td>
</tr>
<tr>
<td>182</td>
<td>A</td>
<td>G</td>
<td>ANT (ALL), KLG (ALL)</td>
</tr>
<tr>
<td>183</td>
<td>C</td>
<td>T</td>
<td>ANT (ALL), KLG (ALL), SNG (15)</td>
</tr>
<tr>
<td>203</td>
<td>T</td>
<td>A</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL)</td>
</tr>
<tr>
<td>204</td>
<td>A</td>
<td>C</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL)</td>
</tr>
<tr>
<td>217</td>
<td>G</td>
<td>A</td>
<td>ANT (ALL), KLG (ALL)</td>
</tr>
<tr>
<td>220</td>
<td>T</td>
<td>C</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL except 3, 10)</td>
</tr>
<tr>
<td>221</td>
<td>G</td>
<td>A</td>
<td>ANT (ALL)</td>
</tr>
<tr>
<td>222</td>
<td>A</td>
<td>C</td>
<td>SNG (ALL except 7, 16, 17)</td>
</tr>
<tr>
<td>224</td>
<td>G</td>
<td>A</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL)</td>
</tr>
<tr>
<td>243</td>
<td>A</td>
<td>G</td>
<td>ANT (ALL except 61, 63, 66–71, 73, 75–80, 84–89, 92, 94, 95, 98, 103–105, 107), KLG (ALL), SNG (ALL)</td>
</tr>
<tr>
<td>247</td>
<td>C</td>
<td>T</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL)</td>
</tr>
<tr>
<td>248</td>
<td>T</td>
<td>C</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL)</td>
</tr>
<tr>
<td>249</td>
<td>G</td>
<td>A</td>
<td>ANT (ALL), KLG (ALL), SNG (ALL)</td>
</tr>
</tbody>
</table>
Fig. 2. Phylogenetic tree, depicting relationship of HCV outbreak sample sequences with that of global and Indian reference sequences of different genotypes, generated by neighbour-joining method using MEGA software (version 6.06) based on the
A detailed analysis of the phylogenetic relationships of the outbreak sequences by construction of an evolutionary tree and comparison to known sequences suggested a point source cluster of HCV as transmission in two of the outbreaks, because all case strains from a single outbreak were of the same genotype and were closely related over the 5′-UTR of HCV. Although the precise determination of the mechanism of transmission is not possible, the collected data allow for some hypotheses. As per the information available after interviewing suspected cases, these infections most probably occurred due to administration of injections using unsterilized needles and syringes by medical personnel at small-scale healthcare settings in the community. J&K state, being a remote and far-flung area, is a fertile ground for unscrupulous elements that in the name of medical practitioners are spreading the disease. Strict interventions by government organizations are needed to limit administration of injections to only those where clinically indicated.

There were a number of genotype 3-specific nucleotide changes (Masante et al., 2008) observed in the 5′-UTR of the outbreak sample sequences in our study, at nt positions 175, 203 and 224, in almost all the sample sequences from all three outbreaks. Some subtype-3a-specific nucleotide changes (Li et al., 2011) were also seen in our study at nt positions 178, 179, 182, 183, 204, 217, 220, 221, 243, 247, 248 and 249 in the sample sequences from both the Anantnag and the Kulgam outbreaks. However, the mutations at nt positions 179, 204, 220, 243, 247, 248 and 249 were also seen in 3b sample sequences isolated from the Srinagar outbreak. Apart from the above-mentioned mutations, at nt position 100 a G to A transition was seen in a few of the sample sequences from the Anantnag outbreak; at nt position 119, A to C transversion was seen in a few of the Anantnag sample sequences, and both sequences from the Kulgam outbreak and at nt 223, A to C transversion was observed in most sample sequences from the Srinagar outbreak. These mutations were not genotype 3-specific or subtype 3a-specific as has been reported previously by researchers (Masante et al., 2008; Li et al., 2011). The first two mutations fall with in the stem of the domain II of 5′-UTR, and the third mutation falls with in the stem of domain III, which have been shown to have an enhanced effect on translation and are associated with the internal ribosome entry site (IRES) function of 5′-UTR (Fukushi et al., 1994; Honda et al., 1996). IRES is an RNA element in the 5′-UTR of HCV which serves as the direct binding site of the 40S ribosomal subunit and is involved in the translation of the HCV genome (Pestova et al., 1998). Bioinformatic tools and structural probing have revealed four different domains in the 5′-UTR of HCV. Domain I (nt 5–20) is not required for IRES activity, domain II spanning nt 43–119 assists the IRES activity of domain III (nt 120–320) and represents the most important region of IRES activity, whilst domain IV (nt 321–341) harbours the AUG codon (Friebe et al., 2001; Masante et al., 2008). Thus, sequence modifications arising in domains II and III may impair the efficiency of viral translation and may also have an effect on replication capacity of the virus. Thus, it is important to understand the possible clinical role of these transition and transversion mutations observed in the 5′-UTR of the HCV, as they may have functional roles in both ribosome binding/translation initiation and viral replication and propagation.

**Conclusions**

A detailed epidemiological investigation should be conducted whenever there is clustering of hepatitis C cases, as detected cases allow for identification of larger outbreaks. Epidemiological investigations of outbreaks should be further supported by inclusion of molecular tests.

Our findings strongly suggest a single origin/source of HCV infection for all of the investigated outbreaks. Our findings also confirm that the genotype in all three outbreaks is the same as that known to circulate predominantly in North India, which is genotype 3. Notably, HCV genotype 1 is known to be more prevalent in southern parts of the country.

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**Table 4. Age distribution of cases in the three outbreaks**

<table>
<thead>
<tr>
<th></th>
<th>Anantnag, n=79</th>
<th>Kulgam, n=6</th>
<th>Srinagar, n=30</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>F</td>
<td>M</td>
</tr>
<tr>
<td>0–14</td>
<td>4</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>15–24</td>
<td>4</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>25–34</td>
<td>8</td>
<td>16</td>
<td>1</td>
</tr>
<tr>
<td>35–44</td>
<td>6</td>
<td>13</td>
<td>1</td>
</tr>
<tr>
<td>45–54</td>
<td>4</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>≥55</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

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These outbreaks indicate a wider and growing problem of breach in infection control practices and aseptic techniques, as healthcare is increasingly being provided in the outpatient settings in our country in which infection control training and oversight may be inadequate. The present study highlights the importance of a comprehensive approach involving better viral hepatitis surveillance and case investigation, healthcare provider education and training, professional oversight, licensing and public awareness to ensure that patients are always afforded basic levels of protection against viral hepatitis transmission.

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