Hepatitis B Outbreak Following a Mass-casualty Incident, Australia

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(See the editorial commentary by Ward and Averhoft, on pages 338–9.)

On 16 April 2009, a boat carrying 47 Afghan asylum seekers and 2 Indonesian crew exploded in Australian waters, resulting in mass casualties. Of these casualties, 23 persons who suffered significant burns were transferred to Royal Perth Hospital, Perth, Western Australia. One patient was subsequently shown to be a hepatitis B virus (HBV) carrier at the time of the explosion. Over the following months, 3 other patients received a diagnosis of acute hepatitis B, and an additional 4 patients showed serological evidence of recent HBV infection, including 1 patient who was transferred to another Australian city. Molecular typing determined that the strains from the HBV carrier and the acute and recent case patients formed a closely related cluster, and the investigation suggested that transmission occurred at or around the time of the boat explosion. This is the first report of confirmed transmission of HBV following a disaster, and it reinforces the importance of postexposure prophylaxis for HBV in mass casualty situations.

On the morning of 16 April 2009, a boat carrying 47 Afghan asylum seekers and 2 Indonesian crew was under escort by the Royal Australian Navy when it exploded near Ashmore Reef in northwestern Australian waters. The boat was believed to have departed Indonesia 5 to 6 days previously and to have exploded following the deliberate ignition of petrol on board the boat [1]. Five people died at the scene, while 44 survivors were recovered from the water via inflatable rescue boats and naval vessels within 15 minutes of the explosion [2]. Initial triage and treatment of the injured was performed at the scene on board the naval patrol boat. Images from the scene immediately after the explosion show multiple injured persons lying next to each other (with exposed skin touching) in the confines of the rescue vessels [3]. Because of the remote location where the incident occurred, 31 of the injured were transferred by boat to the nearest oil rig, where they arrived almost 7 hours after the rescue [4]. They were subsequently transferred by helicopter to a mainland airbase in Western Australia, where they were triaged and stabilized by a medical team. Eight individuals were then transferred to Royal Darwin Hospital and 23 were transferred to Royal Perth Hospital (RPH) by aircraft, a distance of >2000 km. Those patients transferred to RPH arrived between 22 and 36 hours after the incident. The remaining 13 individuals who were not landed in Western Australia were transported by sea to Darwin. Some patients who required further burn management were later transferred from Darwin to Brisbane. All injured persons admitted to RPH had burn injuries.

Between 3 and 5 months following the explosion, 6 new cases of hepatitis B virus (HBV) infection were identified within this group of patients, who were domiciled in Perth at the time of diagnosis. Because HBV infection is a notifiable disease in Australia, an investigation was undertaken to determine whether the
new HBV infections were part of a point source outbreak and, if so, the likely source and means of transmission.

METHODS

Case Ascertainment
The initial 3 cases were identified after serological testing for HBV was performed in response to the development of acute hepatitis illnesses. Those patients who were initially seronegative or had not had serological testing performed were screened for HBV. A newly acquired case was defined by either the detection of hepatitis B surface antigen (HBsAg) in a patient shown to be negative within the previous 24 months; detection of HBsAg and immunoglobulin (Ig) M to hepatitis core antigen (HbcAg), in the absence of prior evidence of HBV infection; or detection of HBV by nucleic acid testing and IgM to HbcAg in the absence of prior evidence of HBV infection.

Exposure Assessment
A review was undertaken of the management of the injured individuals before, during, and after hospital admission to determine possible means of HBV transmission. Medical records of all patients admitted to RPH were reviewed, and information was collected regarding serological HBV status, extent of burn injury, location and dates of in-hospital movement, and surgical treatment.

In addition, the patients with acute symptomatic cases were interviewed by staff from the public health unit, with the aid of an interpreter, at the immigration detention facility where they were housed upon discharge from hospital. As with all newly acquired cases of HBV, the 3 patients were seen as individual patient consultations and were questioned in regard to potential sources of exposure, including sexual exposure, sharing injecting equipment, tattooing, shared razors, biohazard injuries, and household members. Because of the unusual circumstances of the cluster, the interviews focused on potential exposures prior to and after departure from Indonesia to Australian waters and during the time since discharge from the hospital.

Serological Testing
Serum samples were obtained either at the time of admission to the hospital immediately following the incident, at the time of readmission 4 months later for those with acute HBV infection, or, in the case of asymptomatic asylum seekers, as part of a lookback survey following identification of the symptomatic acute HBV infection cases. Serological testing for HBsAg, hepatitis B e antigen (HBeAg), hepatitis B e antibody (HBeAb), hepatitis B core antibody (HbcAb), and HbcAb-IgM was performed using the Architect 2000 SR immunoassay analyzer (Abbott Laboratories) in a nationally accredited laboratory.

Genome Amplification and Sequencing
To generate polymerase chain reaction (PCR) products for subsequent DNA sequencing analysis from the 8 HBsAg-positive asylum seekers, viral nucleic acid material extracted from 200 μL serum samples (QIAmp viral RNA Mini Kit; Qiagen) was added to PCR assays using previously described primers directed against portions of the S [5, 6] or X and pre-core [7] regions of the HBV genome and in-house–designed primers designed to detect the pol region (nucleotides 1818–2467 of HBV genome; GenBank accession number AY236162) (Table 1). The nucleotide sequences on both strands of the PCR products were determined using the AB Big Dye Terminator, version 3.1, sequencing kit on the AB 3130xl Genetic Analyzer (Applied Biosystems).

Table 1. Primer Sequences Used for Phylogenetic Analysis

<table>
<thead>
<tr>
<th>Target region, primer name</th>
<th>Primer sequence (5’ → 3’)</th>
<th>PCR product (base pairs)*</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>S gene</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FHBS1</td>
<td>GAGTCTAGACTCTGTTGGAACCTTC</td>
<td>448</td>
<td>2</td>
</tr>
<tr>
<td>RHBS1</td>
<td>AATKGCACHTAATCTGACCTCCCA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FHBS2</td>
<td>CGTGTTGACTTCTCTCATTTTC</td>
<td>417</td>
<td>2</td>
</tr>
<tr>
<td>RHBS2</td>
<td>GCCGCAAGAAACGCTGAGGCC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>X and pre-C region</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CF1</td>
<td>CATAGAGGTGGTTTGGGA</td>
<td>720</td>
<td>3</td>
</tr>
<tr>
<td>CR1</td>
<td>AGCCGGAGGGTTTCTCT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CF2</td>
<td>TAAGAGGACTTTGGACT</td>
<td>714</td>
<td>3</td>
</tr>
<tr>
<td>CR2</td>
<td>GCGAGGGAGTTCTTC</td>
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<td></td>
</tr>
<tr>
<td>pol region</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HBV-18</td>
<td>AAGGTATGTTGGCCGTTTGT</td>
<td>655</td>
<td>In-house</td>
</tr>
<tr>
<td>HBV-649</td>
<td>TTGGCGAGAAAGTAAGG</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE. PCR, polymerase chain reaction.

* The approximate size of the PCR amplicon as some variation may occur amongst genotypes.

† The primer positions, compared with the hepatitis B virus (HBV) genome (GenBank accession number AY236162) are HBV-18 (positions 1818–1837) and HBV-649 (positions 2449–2467).
Phylogenetic Analysis

The HBV genotypes were determined by nucleotide sequence comparison to existing sequences from the GenBank public database, and all were found to be of genotype D. On the basis of the nucleotide sequences from the S, X, and pre-core and pol regions, genetic analysis of the 8 HBV strains was undertaken. The sequences for the 3 gene regions were compared with those from a database of 32 genotype D HBV strains. Phylogenetic analysis was performed using MEGA software, version 3.0, by the neighbor-joining method with the Kimura 2- parameter model and 1000 bootstrap replicates.

**RESULTS**

The 23 patients who were admitted to RPH were all male with no known family relationships between the patients. They were 16–56 years of age (median age, 30 years), with the age of those who contracted HBV infection ranging from 29 to 46 years (median age, 30 years). The patients had burns affecting 5%–60% of total body surface area (TBSA) (median TBSA, 22%). Case patient 1 (the HBV carrier) had burns affecting 12% TBSA, whereas the 6 case patients at RPH who developed HBV infection had burns affecting 15%–45% TBSA (median TBSA, 35%). The duration of hospitalization for all patients was 11–60 days (median hospitalization, 18 days), and for those who contracted HBV infection, the duration of hospitalization was 17–32 days (median hospitalization, 23 days).

All patients underwent at least 1 operation. Twenty-one (91%) of 23 patients required intravenous antibiotics for secondary burn infection or pneumonia for 1–32 days (median duration of antibiotic therapy, 9 days). There were no deaths.

No breakdown in infection control practices could be identified in a review of the management at RPH. All patients were nursed with contact precautions in place from the time of admission to the hospital. In addition, all 4 patients treated in the intensive care unit were in single isolation rooms. Of the 6 patients with incident HBV infection identified following hospital discharge, 3 had been nursed on the same ward as case patient 1 at some point during their hospitalization, whereas 3 had not. The index case patient underwent 1 operation, with the attending surgeons operating on 2 of the 6 subsequent patients with HBV infection.

Twenty-two of the patients who were determined to be asylum seekers were accommodated in Perth after discharge from the hospital. The detention center where they were housed consisted of 3 houses located on the same premises, with 2 of the houses connected with a shared bathroom and dining area. All detainees had separate bedrooms. On arrival at the center, detainees were provided with separate razors and toothbrushes and were advised not to share these items. There was no evidence of exposure risk during their time in detention, and this was deemed as an unlikely source of exposure. Similarly, no potential transmission events were identified in the histories of the asylum seekers during their time in Indonesia prior to departure or during the voyage to Australia before the explosion that could explain the cluster of cases. No history of previous HBV vaccination could be established.

After diagnosis of the 3 symptomatic acute HBV infection cases, to prevent ongoing transmission within the detention center, staff reeducated detainees on the mode of transmission of HBV and on appropriate infection control practices. All detainees still present in Perth were tested for HBV status, and those who were nonimmune were offered HBV vaccination.

Serological Results

Hepatitis B serological testing was performed for 18 (78%) of the 23 persons admitted to RPH within several days after the refugee boat explosion. Seven (39%) of these showed evidence of past infection with or vaccination against HBV (HBsAb ≥10 mIU/mL) and were considered to be immune, 10 (56%) were nonimmune, and case patient 1 had serological test results consistent with a high infectivity (HBeAg positive) HBV carrier state (HBsAg positive, HBeAg-IgM negative). This asylum seeker was therefore the potential source of infection for the nonimmune patients.

Hepatitis B Ig and vaccination were not administered to any of the admitted patients. When case patients 2, 3, and 4 were readmitted to the hospital 110–127 days after the explosion with jaundice, their serological test results were consistent with acute HBV infection (HBsAg, HBeAg, and HBcAb-IgM positive). These cases were notified to the local public health unit by the treating doctors as a notifiable disease. Following this, the remaining 12 asylum seekers, who were domiciled in Perth and were known to be nonimmune (9) or had not had baseline serological testing performed (3), were screened for HBV. The patient who did not remain in Perth after hospital discharge was known to have immunity. Three asymptomatic asylum seekers (case patients 5–7) were found to have serological evidence of recent HBV infection. Case patients 5 and 6 were HBsAg, HBeAg, and HBcAb-IgM positive, and case patient 7 was HBsAg and HBeAg positive but HBcAb negative, which suggested a pre-seroconversion phase prior to the appearance of HBcAb. Additional blood samples from case patient 7 were not available to demonstrate subsequent HBV seroconversion. Serological data for all case patients are detailed in Table 2.

Of the 6 patients with acute hepatitis B identified in Western Australia, 3 (case patients 3, 5, and 7) had baseline serological test results available from the time of admission to RPH, which allowed seroconversion to be clearly demonstrated (Table 2). The initial HBV status of the remaining 3 patients (case patients 2, 4, and 6) was unknown, but their serological test results 4 months later were indicative of recent HBV infection, and they were also likely to have been nonimmune at the time of the boat explosion.

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Based on initial HBV screening results, 7 of the asylum seekers residing in Western Australia were immune to HBV, and 10 were nonimmune at the time of the incident, including case patients 3 and 7 and case patient 5, who had an HBsAb level of 4.9 IU/L with a negative HBcAb test result. His vaccination status was not known. The initial HBV status of the remaining 3 asylum seekers (case patients 2, 4, and 6) was unknown but, because of their serological evidence for recent HBV infection 4 months later, they were almost certainly nonimmune at the time of the boat explosion. Of the patients with unknown baseline serological test results, 3 developed HBV infection, with the serological test results for the remaining 2 patients not available to determine whether they were immune or nonimmune (Figure 1). Therefore, among the 13 definite or probable nonimmune patients treated in Western Australia who had follow-up serological test results, 6 became infected, resulting in a transmission rate of 46% in this cohort. Had the 2 patients for whom neither baseline nor follow-up serological test results were available been nonimmune at baseline and remained so at follow-up, the transmission rate would have been 40%.

The HBV serological status of the patients transferred to Darwin was not checked during their hospitalization. Initial HBV screening in Brisbane of the 7 admitted patients did not identify any acute or chronic cases of HBV infection. However, after the notification of new cases of HBV infection, testing of the remaining asylum seekers domiciled outside of Western Australia identified a patient who had been transferred to Brisbane who had serological evidence of recent HBV infection (case patient 8), giving a minimum total of 7 incident HBV infections following the boat explosion.

### Molecular Epidemiology

Hepatitis B DNA was detected from the serum of the index carrier case patient and the 7 incident case patients, and all 8 patients were found to be of genotype D. We obtained sequences for the S (312–1003 base pairs), pol (630 base pairs), X and pre-core (533–717 base pairs) gene regions for these cases. These gene regions were targeted to represent the most-variable regions of the HBV genome to give the best discrimination for delineating the chains and sources of infection [7–9], but full length sequences were not available for all isolates, and sufficient sequence for the X and pre-core region for case 1 could not be

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**Table 2. Hepatitis B Serological Test Results for the Index Carrier and 7 Incident Antigen-Positive Asylum Seekers**

<table>
<thead>
<tr>
<th>Case patient</th>
<th>Days after incident</th>
<th>HBsAg</th>
<th>HBeAg</th>
<th>HBcAb</th>
<th>HBcIgM</th>
<th>HBsAb</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>DET</td>
<td>DET</td>
<td>DET</td>
<td>NOT</td>
<td>DET</td>
</tr>
<tr>
<td>2</td>
<td>110</td>
<td>DET</td>
<td>DET</td>
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<td>DET</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>NOT DET</td>
<td>DET</td>
<td>NOT DET</td>
<td>ND</td>
<td>&lt;2.0</td>
</tr>
<tr>
<td>4</td>
<td>121</td>
<td>DET</td>
<td>DET</td>
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<tr>
<td>5</td>
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<td>DET</td>
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<td>ND</td>
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<tr>
<td>6</td>
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</tr>
<tr>
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<td>&lt;2.0</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>136</td>
<td>DET</td>
<td>DET</td>
<td>NOT DET</td>
<td>NOT DET</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>

**NOTE.** Case patient 1 is the index case patient. Case patients 2, 3, and 4 had symptomatic acute HBV infection. Case patients 5–8 had evidence of seroconversion or recent infection. DET, detected; HBcAb, hepatitis B core antibody; HBcIgM, hepatitis B core immunoglobulin M; HBeAg, hepatitis B e antigen; HBsAb, hepatitis B surface antibody; HBsAg, hepatitis B surface antigen; ND, not done; NOT DET, not detected.

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**Figure 1.** Demonstration of patient movement and state of hepatitis B virus immunity after the boat explosion.
produced. There was a high degree of similarity between the isolates from the 8 asylum seekers. The sequences for cases 2 to 8 were identical to those from case 1 except for a 1 base pair difference in the S region for case patients 5–7 and a 1 base pair difference in the pol region for case 7.

The sequences were then compared with a database composed of genotype D HBV isolates identified through local viral hepatitis clinics and collected from Middle Eastern immigrants, including immigrants from Afghanistan and the Indian subcontinent. Of the 32 genotype D HBV strains in this database, partial or full-length sequences were available for all 32 strains from the X and pre-core region, for 21 isolates from the S region, and for 9 isolates from the pol region. Phylogenetic trees for the S gene, the X and pre-core genes, and the pol gene for ~391, 669, and 573 base pair segments, respectively (Figures 2–4), demonstrate that the 8 isolates from asylum seekers clustered separately from the other genotype D isolates. The results demonstrate that the asylum seeker strains form a cluster of very closely related sequences distinct from the other hepatitis B genotype D strains. These molecular sequence data supports the epidemiological enquiry and suggest that the 8 asylum seekers were involved in a common HBV transmission chain.

**DISCUSSION**

HBV infection is a notifiable disease in Australia, and individuals with newly diagnosed cases undergo contact tracing by state- or territory-based public health units. The notification of 3 individuals with symptomatic acute cases of HBV infection admitted to RPH within a 3-week period, all of whom were residents at the same immigration detention facility in Perth, alerted the local public health unit to the possibility of a point source exposure among asylum seekers from a refugee boat that had exploded in Australian waters [1]. The ensuing investigation identified a likely carrier source case patient and an additional 4 asymptomatic asylum seekers with recent HBV infection, all of whom had been injured in the explosion.

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**Figure 2.** Phylogenetic tree of hepatitis B S gene region of asylum seekers, compared with the D genotype database. The partial gene sequence is approximately 391 base pairs in length. The scale bar represents genetic distance (substitutions per nucleotide).

**Figure 3.** Phylogenetic tree of hepatitis B X and pre-core gene region of asylum seekers, compared with the D genotype database. The partial gene sequence is ~669 base pairs in length. The scale bar represents genetic distance (substitutions per nucleotide).
The molecular epidemiology suggested a point source cluster of HBV transmission, because all 8 case strains were of the same genotype and were closely related over 3 HBV gene regions. Hepatitis B genotypes are geographically distributed around the world. Genotype D, which was found in these cases, is more prevalent in the Middle East than in Australia, where genotype B and C are more commonly found [10]. This makes HBV transmission to the asylum seekers from Australian HBV carriers (for example, during hospital treatment) very unlikely. Transmission from other asylum seekers from the Middle East, who were colocated at the detention center, was excluded by the fact that an asylum seeker in another Australian city, who had been separated from the other case patients soon after the explosion, was also found to have a closely related HBV strain. The isolation of this closely related strain of HBV from the patient in Brisbane also makes transmission during hospitalization at Royal Perth Hospital very unlikely.

Substantial genetic variation has previously been found in epidemiologically unrelated HBV genotype D isolates, allowing a link to be established between outbreak cases [9]. Our investigation included the S, X, and pre-core and pol regions to improve the resolution of the phylogenetic analysis, because the pre-core and pol regions have greater nucleotide diversity than the S region [7, 9]. When we compared these 3 gene regions in the isolates from the 8 asylum seekers to our database of 32 genotype D isolates, including case patients from the Middle East, we found the isolates from the asylum seekers to all be closely related and distinct from other isolates. This supports a common HBV transmission chain. The most likely transmission event was at the time of the boat explosion or in its immediate aftermath, as indicated by the likelihood of blood cross-contamination following an explosion in which many persons in close confinement experienced acute traumatic injury with significant burns and bleeding; consistency of the time from the explosion to onset of symptoms in the 3 symptomatic case patients with the known HBV incubation period of 45–180 days (average, 60–90 days) [11]; the epidemiological look-back investigation finding no other likely routes or sources of HBV transmission; and the fact that a case patient who had been transferred to a distant part of Australia demonstrated a closely related strain.

At the time of admission to the hospital, 3 days after the boat explosion, case patient 1 was shown to be HBeAg positive and, although no HBV load was determined, he is the likely point source of the transmission event. The HBeAg-positive status indicates high infectivity in regard to the risk of transmission of HBV, measured at 22%–40% following a large-bore needle-stick injury [12]. TBSA burns in these patients ranged from 12% through 45%, representing a significant loss of protective barriers and likely blood loss. The high HBV transmission rate to nonimmune asylum seekers of 40%–46% seen in the cohort transferred to Western Australia is similar to that reported for needlestick injuries with large-bore needles, indicating a high-risk situation for HBV transmission.

Regarding case patient 5, who had an HBsAb level of 4.9 mIU/mL but later developed acute HBV infection, the likely explanation is an inadequate response to previous HBV vaccination or, possibly, false HBsAb reactivity rather than past infection. This is supported by the lack of detectable HBcAb and, if case patient 5 had previously been infected with HBV, the immunological memory, despite waning antibody titers, should have been protective and produced an anamnestic HBsAb response [13, 14].

Although outbreaks of HBV infection in both hospital and nonhospital health care settings are well reported [15, 16], this is, to our knowledge, the only report of an outbreak of HBV infection following a mass casualty disaster to be confirmed by molecular methods. Although the potential for transmission of HBV via bone fragments in blast injuries and, in particular, in suicide bombings has previously been reported, in those cases, prophylaxis was administered to those exposed, and there was no transmission of virus [17, 18]. Additionally, in those situations, there was physical transfer of body tissue from one person to another. In contrast, many of the patients from the Ashmore Reef incident were thrown into the ocean as a result of
the blast [1] and predominantly suffered burns without fractures or amputation of body parts. We postulate that transmission occurred via either direct blood contact from the source patient or via an intermediary as medical assistance was being given, because the injured, who experienced burns and blood loss, were bought together during rescue. Although we have not been able to determine the exact time or mode of transmission of HBV, it seems likely that it occurred either at the time of the blast, in the immediate aftermath of the rescue from the water and transfer of patients by small boat to the naval ship, or during the initial triaging and first aid of the patients on the naval ship or oil rig. This process took many hours because of the remote location of the incident, and because such wounds and injuries were not definitively treated during this time, there was the potential for blood-borne transmission of infection because of the close proximity of the injured to each other. Unfortunately, it was not possible to determine which persons were located next to each other during the rescue process; this information would have been important in confirming this period as the likely time of transmission and, in particular, which persons were in closest proximity to the index case.

Hepatitis B vaccination is recommended for postexposure prophylaxis following bombings and mass casualty situations [19]. This is particularly important considering that there is a high prevalence of HBV in some regions of the world, yet the status of individual persons at the time of a mass disaster will not be known. The rate of chronic HBV infection varies between 8.3% and 60.8% in Afghans in refugee camps, suggesting high prevalence of the disease in selected Afghan populations [20, 21]. Because of the remote location where the boat explosion occurred, most of the patients reached definitive medical care after the recommended 24-hour period for administration of HBV immunoglobulin [11]; however, there may be benefit associated with administration of HBV immunoglobulin up to 72 hours after exposure [22]. In this instance, a window of opportunity was therefore present in which infection of nonimmune asylum seekers may have been prevented by HBV immunoglobulin administration and vaccination.

By confirming the transmission of HBV in this scenario, the current recommendations to give postexposure prophylaxis should continue to be encouraged in mass casualty situations and in emergency departments that receive multiple casualties. In addition, staff working in the area of disaster management should be adequately protected against infection due to blood-borne viruses, such as HBV, by vaccination or postexposure prophylaxis (where relevant) and appropriate infection control practices, including the use of personal protective equipment.

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References