the patient 3 days before she died, a friend and a healthcare worker had contact with her on the day before her death, and the rest of the healthcare workers had contact with the patient on the day she died.

To our knowledge, this is the first imported case and the tenth case of HPS reported in British Columbia, Canada, since 1994 (2006 BC Annual Summary of Reportable Diseases, available from www.bccdc.org/content.php?item=33) (*4*). Six of these 10 cases were fatal. All cases except the 1 described here have been locally acquired Sin Nombre infections. Sin Nombre virus is endemic in the *Peromyscus maniculatus* (deer mice) population in most of British Columbia (*5*).

Worldwide, imported cases of HPS are unusual, although HPS has been reported in countries that are in close geographic proximity or in travelers to disease-endemic areas (*6–8*). Fortunately, none of the persons exposed to the patient reported symptoms consistent with HPS during the incubation period, and none who were tested seroconverted. Seroprevalence surveys in Chile among healthcare worker contacts of patients with HPS caused by the Andes virus showed a prevalence of 0% (*9*). A report from Argentina showed that cases due to secondary transmission occurred mostly in non-healthcare workers after prolonged close contact in the prodromal period (*10*). In conclusion, we describe an imported case of fatal HPS due to an Andes-like hantavirus with no evidence of secondary transmission.

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Disseminated Bocavirus Infection after Stem Cell Transplant

To the Editor: Human bocavirus (HBoV) (*1*) is increasingly recognized as a cause of respiratory infections worldwide. Children and infants appear to be most at risk (*2–7*), although HBoV's role in immunocompromised patients remains unclear. We report on a child with disseminated HBoV infection after hematopoietic stem cell transplantation (HSCT). HBoV DNA was detected at high levels in nasopharyngeal aspirates (NPAs) and in blood and stool samples.

A 4.5-year-old boy with dyskeratosis congenita was brought for treatment to our hospital due to severe persistent cytopenia. Allogenic HSCT was performed in August 2006 after conditioning with total body irradiation (200 cGy, day –8 before HSCT surgery), fludarabine (days –7 to –4), antithymocyte globulin (days –4 to –1), and cyclophosphamide (days –3

to –2). He received 7.16×10^8 nucleated bone marrow cells/kg body weight from a 9/10 human leukocyte antigen –matched unrelated donor. Graft-versus-host disease (GvHD) prophylaxis consisted of a short course of methotrexate and cyclosporin A. Neutrophil and platelet engraftment occurred on days 22 and 65 after surgery, respectively. Despite pre- and post-HSCT anti-infective prophylaxis with cotrimoxazole, colistin, acyclovir, and fluconazole, *Enterobacter cloacae* sepsis was diagnosed on day 2. After meropenem treatment, blood cultures remained negative. On day 12, fever reoccurred, elevated C-reactive protein values (229 mg/L) and reduced general health were noted, but no bacterial pathogen was isolated. During this period, the patient received antimicrobial drug therapy with meropenem, tobramycin, vancomycin, and amphotericin B. On day 16, his body temperature peaked to 40.6°C, and a cough and dyspnea without wheezing developed. Chest radiograph results suggested pneumonia with perihilar infiltrates. Reduced oxygen saturation (pO_2 86%) was recorded transcutaneously, and oxygen supplementation (maximum 4 L/min) was started by face mask (online Appendix Figure, available from www.cdc.gov/EID/content/13/9/1425-appG.htm). An NPA sample investigated by multiplex PCR (results provided by W. Puppe and J. Weigl; www.pid-ari.net) was negative for adenovirus, respiratory syncytial virus, human metapneumovirus, parainfluenza viruses 1–4, influenza viruses A and B, coronavirus, reovirus, enterovirus, *Clamydia pneumoniae*, *Mycoplasma pneumoniae*, *Bordetella pertussis*, *B. parapertussis*, and *Legionella pneumophila,* but positive for rhinovirus RNA. Retrospectively, the same NPA sample was reanalyzed for HBoV DNA by real-time PCR (*7*) and showed a viral load of 4.6×10^7 copies/mL (online Appendix Figure); specificity was confirmed by sequencing.

From day 19 on, the patient's general health improved and the chest radiograph results returned to normal. After neutrophil engraftment (day 22) and addition of erythromycin to the antimicrobial drug regimen, body temperature decreased and oxygen supplementation was discontinued. However, rhinitis, cough, and low-grade fever (<38.5°C) persisted until day 50 (online Appendix Figure), and HBoV DNA was detected in NPAs on days 37 and 44 at 2.4×10^{11} and 1.3×10^{14} copies/mL, respectively (online Appendix Figure). The NPA sample on day 37 was still rhinovirus positive.

Concurrent with the increased HBoV load in NPAs, cytomegalovirus (CMV) reactivation was first diagnosed by PCR on day 20 and peaked (58.250 copies/mL whole blood) on day 41 despite gancyclovir therapy. Switching to foscarnet led to temporary control of CMV replication (online Appendix Figure). Additionally, on day 22, acute GvHD grade I with skin manifestations developed. Treatment with steroids until day 60 led to complete resolution.

HBoV infection in this patient was not restricted to the respiratory tract. Diarrheic stool samples obtained on day 21 and, after resolution of respiratory symptoms, on day 75 showed substantial HBoV DNA (2.5×10^6 and 6.0×10^5 copies/mg, respectively; online Appendix Figure). Tests for rotavirus and adenovirus antigens were negative, and no bacterial pathogen was isolated. Moreover, HBoV DNA was detected at lower levels (3.7×10^3 to 7.8×10^4 copies/mL) in 4 EDTA plasma samples taken days 21–47. Subsequent plasma (days 61, 68, 75, 88, 219), NPA (day 219), and stool (day 219) samples were negative for HBoV DNA. However, the ability of HBoV to cause persistent infection, as do other members of the *Parvovirinae* subfamily, cannot be excluded. Future investigations are needed to address this hypothesis.

Here, we report on disseminated HBoV infection in an immunocompromised patient. Whether the clinical course in this case was more severe or prolonged than it would have been for HBoV infections in non-HSCT children remains unknown due to the lack of long-term observations in immunocompetent children. The dramatic increase of HBoV load in NPAs and viral dissemination most likely resulted from progressive impairment of cellular immunity as indicated by simultaneous CMV reactivation. Moreover, the increased viral load might have also been a consequence of steroid addition to immunosuppressive therapy to control GvHD. The contribution of HBoV to respiratory disease remains ambiguous because 2 NPA samples were also rhinovirus positive. Additional studies are required to investigate the pathogenic role of HBoV in double or multiple infections. Association of HBoV with the patient's continued diarrhea is in accordance with previous studies (*8–10*). Prolonged fecal shedding has important implications for isolation measures in transplantation units. More studies in immunocompromised patients are required to evaluate the spectrum of pathology caused by this emerging virus.

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Pure Red Blood Cell Aplasia and Isoniazid Use

To the Editor: Isoniazid is a first-line drug in the treatment of tuberculosis; its uses include prevention as well as cure. Isoniazid is usually well tolerated, although its common side effects include gastrointestinal discomfort, rash, allergy, hepatitis, and peripheral neuropathy. Hematologic disorders such as eosinophilia, thrombocytopenia, and autoimmune hemolytic anemia are rarely reported (*1*). Pure red cell aplasia (PRCA) is an uncommon disorder in adults. PRCA occurs secondary to drug exposure in 5% of patients; ≈30 drugs have been implicated (*2*), and few reports involving isoniazid have been published. We report a case of isoniazid-induced PRCA.

In 2006, a 79-year-old woman sought care at a public assistance hospital in Paris; she reported having had asthenia, breathlessness, and decreased appetite for 2 weeks. She had had node-negative, localized gastric adenocarcinoma 2 years earlier, which had been treated by partial gastrectomy, and pulmonary tuberculosis in 1948, which had been treated by partial pneumonectomy. As a result of a pleuropulmonary tuberculosis relapse diagnosed 6 weeks earlier, she was receiving antituberculous treatment with rifampin (400 mg/day), isoniazid (150 mg/day), ethambutol (800 mg/day), and pyrazinamide (1,000 mg/day). She received no other concomitant medications.

Her initial physical examination showed nothing abnormal. Laboratory analysis showed hemoglobin 6.3 g/dL, leukocyte and thrombocyte counts within normal limits, 1% reticulocytes, and zero schizocytes. Other test results (liver and renal function, serum folate and vitamin B12 levels, lactic dehydrogenase levels, C-reactive protein, serum protein electrophoresis, direct and indirect Coombs tests, and antinuclear antibody tests) were within normal limits, as were viral serologic test results (HIV, hepatitis B virus, hepatitis C virus, parvovirus B19). Upper and lower digestive tract endoscopic examination and carcinoembryonic antigen showed no abnormalities, which lessened the likelihood of a tumor relapse. Bone marrow aspiration from the sternum showed a hypocellular marrow with complete absence of the erythroid series, a normal myeloid series, and megakaryocytes. The marrow findings were consistent with PRCA.

In view of previous reports of isoniazid-induced PRCA (*3*–*5*), we suspected this drug to be responsible in this case. Isoniazid was withdrawn, and other antituberculous drugs were continued. The patient's hemoglobin level rose to 10.6 g/dL and reticulocyte count to 3% over 10 days; no blood transfusion was required.

Drug-induced PRCA is a rare blood disorder in adults and has already been reported in isoniazid-treated patients (*3*–*5*). For this patient, other causes for anemia (e.g., drug-induced hemolytic anemia, digestive malignancies, viral causes known to date, hematologic malignancies, and autoimmune disorders) were excluded (*2*).

The favorable outcome after isoniazid withdrawal increased the likelihood that isoniazid was the cause. Although rechallenge with isoniazid could have confirmed isoniazid as the cause, it is not an ethical option because of the hazardous adverse effects.

The exact mechanism for isoniazid-induced PRCA remains unclear, but the demonstration of antibodies reacting with nucleated red blood cells in ≈50% of cases suggests an induction of autoimmunity (*4*,*5*). This hypothesis is supported by previously reported cases in which PRCA relapses occurred when treatment with isoniazid was resumed (*3*,*5*). The intrinsic imputability of isoniazid also relies on the lack of a dose-effect relationship and the delay between the introduction