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Human bocavirus DNA detected in a boy with plastic bronchitis

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Case report

A 14-month-old boy, previously healthy, was taken to our hospital with a five-day history of cough, a two-day history of high fever and increasing dyspnea with no medication. A chest X-ray showed atelectasis of the left lung, and chest computed tomography showed occlusion of the left main bronchi. The patient underwent flexible bronchoscopy with general anesthesia. The procedure demonstrated total obstruction of the main left bronchus by extremely thick secretions. The secretions were removed by vacuuming and the bronchus showed no further evidence of disease. After the endoscopic interventions, the patient progressed satisfactorily and was discharged from the hospital after 9 days, without sequelae.

Histopathology of the bronchial cast revealed a large quantity of eosinophils, some Charcot-Leyden crystals, and Curschmann spirals. Nothing was cultured from the cast. The intratracheal aspirate was subjected to real-time PCR screening for six bacterial pathogens, and real-time RT-PCR screening for 11 viral pathogens as previously described (1,2). Human bocavirus (HBoV) was the only pathogen detected (9.5×10^5 copies/reaction tube). Serum samples were studied for HBoV DNA using a PCR assay in the acute phase (first day of hospitalization) and the convalescent phase (46 days after) and the levels of IgG antibody to the unique part of virus protein 1 (VP1; a capsid protein of HBoV) were measured using an immunofluorescence assay as previously described (3). HBoV DNA was detected in the acute phase (2.10×10^4 copies/ml) but not in the convalescent phase ($<4.0 \times 10^2$ copies/ml). The titers of the IgG antibody against HBoV VP1 increased from $<1:40$ (acute phase) to $1:2560$ (convalescent phase).

Discussion

After the first description in 2005, HBoV is detected worldwide in children with acute respiratory tract infection with PCR-based methods (4). However, the causative role of HBoV remains unclear. HBoV is also detected in children without respiratory illness, and co-infection with other viruses has been examined in several studies.

In our case, we observed HBoV DNA in intratracheal aspirates, and no other causative pathogen was found. HBoV DNA was also detected in blood only in the acute phase. The titers of IgG antibody against HBoV VP1 increased. Thus, we diagnosed the case as acute HBoV infection.

In the pediatric age group, plastic bronchitis is often associated with inflammatory lung disease, sickle-cell anemia, congenital heart disease and so on (5). In our case, the histopathological findings suggest that the boy had allergy state such as asthma, but he have no episode of wheezing before and after hospitalization. HBoV DNAs were detected from the children of wheezing, but bronchial histopathology of HBoV or relation with bronchial asthma remains unknown. In this case, he has no underlying condition, nor other causative pathogen is detected, so we speculate that acute HBoV infection caused inflammatory lung disease followed by mucous plugging.

So far, there is no case report of plastic bronchitis related to HBoV infection. As methods such as real-time PCR or serological studies that can easily detect HBoV infection become widely available, there will be increasing numbers of reports with varied symptoms.

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