4.28 × 10^6 cells/L, a hemoglobin level of 86 g/L, and a leukocyte count of 8.28 × 10^6 cells/L with 17% neutrophils and 78% lymphocytes.

The patient was given symptomatic supportive treatment, methylprednisolone sodium succinate, and 5 g of γ-globulin. He was not given any antimicrobial drugs. On August 4, he was discharged from hospital after symptoms had resolved.

In conclusion, our results confirm that mimivirus is an unlikely cause of human respiratory infections in China, as reported in other countries (5–9). Sporadic detection of mimivirus in 1 child who was born with a compromised respiratory system and had numerous hospitalizations was most likely caused by colonization of the child with this virus during numerous hospitalizations and critical care stays. In addition, parainfluenza virus 3 and bocavirus were detected in the mimivirus-positive child. Because parainfluenza virus 3 causes pneumonia and bocavirus causes infections with respiratory symptoms, particularly in children of his age, these 2 pathogens probably caused the illness in the child.

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Severe Pneumonia Associated with Adenovirus Type 55 Infection, France, 2014

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To the Editor: Human adenoviruses (HAdVs) comprise 70 recognized genotypes (as of February 15, 2016; http://hadvwg.gmu.edu/) and are frequently associated with mild and acute upper respiratory tract infections, depending on virus type and host immune status (1). HAdV type 55 (HAdV-55) has recently reemerged as a highly virulent pathogen, causing severe and sometimes fatal pneumonia among immunocompetent adults, particularly in Asia (2–4). Formerly known as HAdV-11a, HAdV-55 is a genotype resulting from recombination between HAdV-11 and HAdV-14 (5). We report 2 cases of severe pneumonia associated with HAdV-55 infection in France.

In November 2014, two immunocompetent women, 71 (patient A) and 36 (patient B) years of age, sought care 4 days apart at the emergency unit of the University
Hospital of Clermont-Ferrand, France, for an influenza-like syndrome characterized by fever, cough, and dyspnea. Laboratory investigations at admission revealed thrombocytopenia (98 and 88 × 10^9 thrombocytes/L for patients A and B, respectively; reference range 150–450 × 10^9 thrombocytes/L) and elevated C-reactive protein concentrations (71.6 and 45.8 mg/L, respectively; reference range <3 mg/L). Chest radiographs and thoracic tomodensitometry images showed acute left lobar pneumonia in each patient. Therapy with intravenous antimicrobial drugs (cefepime and levofloxacin) and oxygen was initiated. Patient A was transferred to the intensive care unit 4 days after admission because of unimproved respiratory function; patient B was transferred 5 days after admission because of acute respiratory distress syndrome.

Results for all bacteriologic analyses were negative (blood cultures, bronchoalveolar lavage fluid cultures, PCR for Mycobacterium tuberculosis [Xpert MTB/RIF; Cepheid, Sunnyvale, CA, USA] of bronchoalveolar lavage fluid, and urinary antigen testing [BinaxNOW Legionella and Streptococcus pneumoniae; Alere, Scarborough, ME, USA]). No specific antibodies were detected against Chlamydia pneumoniae (Anti-C. pneumoniae; Euroimmun, Lübeck, Germany) and Mycoplasma pneumoniae (Platelia M. pneumoniae IgM; Bio-Rad, Hercules, CA, USA). For each patient, HAdV was the only pathogen detected in nasopharyngeal secretions collected at admission and in bronchoalveolar lavage fluids collected while in the intensive care unit (molecular multiplex assay [FilmArray Respiratory Panel; bioMérieux, Durham, NC, USA]). HAdV DNA was also detected in whole blood (Adenovirus R-gene; bioMérieux); viral load was 280,524 copies/mL for patient A 9 days after hospital admission and 951,146 copies/mL for patient B 4 days after admission. During hospitalization, transient hepatitis developed in each patient; serum aspartate aminotransferase levels were elevated up to 6–10 times reference range, and leucocyte counts indicated leukopenia (2.17 and 1.28 × 10^9/L for patients A and B, respectively; reference range 4–10 × 10^9/L). Patient B had acute pancreatitis and hyperlipaemia (lipase 1,697 U/L; reference range 73–393 U/L). Healthy respiratory function was restored for both patients, who were discharged 26 (patient A) and 19 (patient B) days after admission.

A partial region of the hexon gene was amplified and sequenced from DNA extracts of respiratory and blood

**Figure.** Phylogeny of 13 complete-genome sequences of human adenovirus type 55 (A) and of 21 sequences of hexon genes (B). The complete genome tree (A) is rooted to a human adenovirus type 14 isolate (GenBank accession no. JX892927). The strains from patients A and B (immunocompetent women with human adenovirus infection) reported in this study are indicated. The phylogenetic tree was calculated by using the maximum-likelihood method in MEGA6 (http://www.megasoftware.net). The best algorithm was chosen by the criterion score of the Bayesian information criteria. The statistical robustness of branches was estimated by 1,000 bootstraps. Only bootstrap values >70% are indicated. The tree is drawn to scale; branch lengths are measured in number of substitutions per site (scale bar). All positions containing gaps and missing data were eliminated. The sequence of the hexon gene from patient B was partially complete (2,821/2,841, 99.3%).
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Spotted Fever Group

Rickettsia in the Pampa Biome, Brazil, 2015–2016

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Technical Appendix

HAdV Full-Genome Sequencing and Analysis

Because HAdV-55 isolation on A549 cells was unsuccessful for both patients, the full genome sequence was assessed directly from clinical samples by a metagenomic approach with the MiSeq Reagent kit v2 on a Miseq 2000 Illumina system (Illumina, San Diego, CA). Three different strategies were used and combined to obtain maximum genome coverage. Except for nucleic acid purification steps, libraries were generated as previously described (Naccache S. et al., Genome Res 2014) with Nextera XT DNA Sample Preparation Kit (Illumina). Libraries were sequenced on an Illumina MiSeq 2000 according to standard Illumina protocols, creating 250-nt paired-end reads. 1) Total nucleic acids from plasma samples were extracted with EasyMag® (bioMérieux). 2) To remove most of the host DNA and thus to enrich the viral sequences, plasma samples were first incubated with Turbo DNAsE (Ambion). We then followed the protocol mentioned above. 3) After extraction of total nucleic acids from plasma samples as described in 1), host DNA was removed by MBD2-Fc-bound magnetic Bead and the remaining nucleic acids were purified with the Zymo DNA Clean and Concentrator kit.

For all MiSeq runs, after a quality filtering step with FastQ Quality Trimmer tool implemented on Galaxy (Blankenberg D. et al., Bioinformatics 2010), reads were aligned on HADV-55 JX491639 genome by Bowtie 2 (Langmead B. et al., Nat methods 2012). As no approach managed to obtain sufficient coverage of the reference genome, aligned reads of the three strategies were combined by Geneious R8 (Biomatters) (Kearse M. et al., Bioinformatics 2012) to process a reference-based assembly.