this research team (DR) collected the eggs from the hair shaft, they were found ≈3 3.5 cm from the hair follicle. Because hair grows ≈1.25 cm per month, the louse infestation occurred ≈3 months before egg collection (*6*).

Homeless persons that we have monitored for many years are often heavily infested by body lice but are also occasionally infested with head lice. Before genetic tools that differentiate the head and body louse lineages were available (*5*), it was speculated that body lice may have originated from head lice (*9*). From our study, it is clear that under conditions of massive infestation, body lice can migrate and colonize hair; the opposite may also be true. However, there is no evidence that body lice are capable of causing an outbreak of lice living on the head, as happens among schoolchildren that have been found to be infested only by head lice. This suggests that body lice cannot thrive in the environment of head lice, which infest millions of children worldwide (*10*), further suggesting that outbreaks of trench fever are most likely not linked to head lice in industrialized countries. In conclusion, by analyzing lice harvested from the heads and clothing of homeless persons, we have shown that the 2 ecotypes belong to the same body lice population.

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Rezak Drali, Abdoul Karim Sangaré, Amina Boutellis, Emmanouil Angelakis, Aurélie Veracx, Cristina Socolovschi, Philippe Brouqui, and Didier Raoult

Author affiliations: Aix Marseille Université, Marseille, France; and Institut Hospitalo-Universitaire Méditerranée Infection, 13005, Marseille

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Address for correspondence: Didier Raoult, Institut Hospitalo-Universitaire Méditerranée Infection, 27 boulevard Jean Moulin, 13385 Marseille CEDEX 5, France; email: didier.raoult@gmail.com

Myasthenia Gravis Associated with Acute Hepatitis E Infection in Immunocompetent Woman

To the Editor: Hepatitis E virus (HEV) is a common cause of acute hepatitis in developing countries. The course of acute hepatitis E is usually benign, except in pregnant women and in immunocompromised patients, who are prone to a lethal or chronic outcome of the disease. Since 2001, hepatitis E has been emerging in industrialized countries, and neurologic manifestations such as Guillain-Barré syndrome, brachial neuritis, transverse myelitis, and cranial nerve palsies have been reported in patients with acute or chronic forms of the disease (*1*–*6*). Most cases with neurologic manifestations have been characterized by infection with genotype 3 HEV. Data are not available to indicate whether this association between HEV infection and neurologic manifestations is related to a specific antigenic stimulus provided by HEV or is linked to the more comprehensive assessment for such neurologic conditions that is available in industrialized countries or to a reporting bias. We report a case of HEV infection in an immunocompetent woman who had muscle-specific kinase (MuSK) antibody–positive myasthenia gravis associated with HEV replication.

A 33-year-old woman was hospitalized in France for subacute asthenia and intermittent symptoms including dysarthria, dysphagia, muscle weakness, and diplopia. She had no family history of autoimmune disease and no notable personal medical history; she had not received any recent vaccinations and had not traveled outside France during the previous year. Physical examination showed no pyramidal, vestibular, or

cerebellar syndromes, and all tendon reflexes were typical.

On admission, the patient's liver function tests showed elevated alanine transaminase (190 UI/L). Test results were within reference ranges for aspartate aminotransferase, alkaline phosphatase, gamma-glutamyl transpeptidase, and creatine kinase levels. Antibody tests were negative for hepatitis B virus, hepatitis C virus, HIV, human T-lymphotropic viruses 1 and 2, and *Treponema pallidum*, but testing for cytomegalovirus and Epstein-Barr virus showed previous exposure. Serum samples were positive for HEV IgM (index 11) and negative for HEV IgG (index 0.71) by Adaltis microplate ELISA (EIAgen; Adaltis, Casalecchio Di Reno, Italy). HEV RNA was detected in serum and fecal samples by using HEV reverse transcription PCR (RT-PCR) (Ceeram, La Chapelle sur Erdre, France) on the SmartCycler II instrument (Cepheid, Sunnyvale, CA, USA). Sequencing studies showed that the HEV strain belonged to genotype 3f.

Brain magnetic resonance imaging results were normal, and results for analysis of cerebrospinal fluid were normal (leukocytes <5 cells/μL, glucose 3.2 mmol/L, protein 38 mg/dL, HEV RT-PCR negative). A nerve conduction study, performed even though the patient was not symptomatic, did not find any abnormality. Pharmacologic testing with prostigmine (0.5 mg intravenously) did not result in symptom improvement. Computed tomography scan of the mediastinum showed no thymoma. Test results for anti-acetylcholine receptor antibodies were negative; however, results were positive for MuSK antibodies (18.7 U/mL by radioimmunoassay; RSR Ltd., Cardiff, Wales, UK). These data, combined with physical examination, confirmed the diagnosis of myasthenia gravis.

Given this association of myasthenia and acute HEV infection, we suspected the potent role of HEV infection in the neurologic symptoms. Therapy was started with ribavirin (1,000 mg/day for 1 month) and intravenous immunoglobulin (IVIG) (1 g/kg/d for 2 days). Alanine transaminase levels rapidly decreased, and after 3 months, test results for HEV IgM and HEV RT-PCR in serum were negative. IVIG treatment relieved symptoms in the short term, but symptoms returned 15 days after treatment ended, requiring continuation of IVIG every 4 weeks for 6 months. Four months after the last infusion of IVIG, the patient reported mild 4-limb fatigability after exercise, but results of objective neurologic examination were normal. Results for MuSK antibodies were still positive, and treatment with azathioprine (150 mg/d) was started. Three weeks later, the patient required another infusion of IVIG for difficulty in swallowing, dyspnea, and 4-limb weakness, but she was free of symptoms for the remaining 5 months of follow-up.

In conclusion, we describe a case of anti-MuSK myasthenia gravis associated with acute HEV in a young, immunocompetent patient in France. Because myasthenia gravis with MuSK antibodies is rare (≈10% of myasthenia gravis cases) (*7*), the potential role of HEV infection as a trigger of autoimmune disorders should be investigated. Some cases of anti-MuSK myasthenia gravis associated with HIV (*8*) or Epstein-Barr virus (*9*) infections have been reported. Nevertheless, our findings do not enable us to draw conclusions regarding causality. Our observation might suggest a coincidental temporal association between HEV infection and myasthenia gravis or a triggering of autoimmunity by HEV. Moreover, anti-MuSK myasthenia gravis is usually characterized by a rapidly progressive course with moderate to severe symptoms (*7*). The initial unusually benign clinical course in this patient might be explained by the effect of early ribavirin treatment or, more likely, by a particular type of MuSK antibodies (*10*).

Aude Belbezier, Alban Deroux, Françoise Sarrot-Reynauld, Sylvie Larrat, and Laurence Bouillet

Author affiliations: Centre Hospitalier Universitaire de Grenoble, Grenoble, France (A. Belbezier, A. Deroux, F. Sarrot-Reynauld, S. Larrat, L. Bouillet); and Université Joseph Fourier Grenoble 1, Grenoble (F. Sarrot-Reynauld, S. Larrat, L. Bouillet)

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Address for correspondence: Françoise Sarrot-Reynauld, Clinique Universitaire de Médecine Interne, Pôle Pluridisciplinaire de Médecine, Centre Hospitalier Universitaire de Grenoble, CS10217, 38043 Grenoble Cedex, France; email: FSarrot-Reynauld@chu-grenoble.fr

Ciprofloxacin-Resistant *Salmonella enterica* Serotype Kentucky Sequence Type 198

To the Editor: Mulvey et al. (*1*) reported the emergence of ciprofloxacin resistance in *Salmonella enterica* serovar Kentucky of multilocus sequence type 198 (ST198) in Canada (*1*). Ciprofloxacin resistance in *S. enterica* ser. Kentucky was reported in 2011 in patients from Europe, most of whom had traveled to Africa and the Middle East (*2*). Since then, *S. enterica* ser. Kentucky ST198 with additional resistance to third-generation cephalosporins and carbapenems has been reported from France and Morocco, again associated with travel (*3*). Poultry has been implicated as the most likely vehicle for infection by this sequence type (*2*,*3*). Resistance to third-generation cephalosporins and carbapenems has not been seen in North America; however, the emergence of ciprofloxacin-resistant infections has been observed (*1*).

In the United States, *S. enterica* ser. Kentucky is the most common serotype isolated from chickens and the second most common found among retail chicken, but ciprofloxacin resistance has not been documented among these sources (*4*). We sought to determine if ciprofloxacin- or ceftriaxone-resistant *S. enterica* ser. Kentucky has emerged in humans in the United States. We examined isolates and data from the National Antimicrobial Resistance Monitoring System to document antimicrobial resistance and sequence type and to assess possible risk factors for acquiring infection.

Participating state and local public health laboratories submit every 20th nontyphoidal *Salmonella* (NTS) isolate to the Centers for Disease Control and Prevention for susceptibility testing. MICs of ≥15 antimicrobial agents were determined by using broth microdilution (Sensititer, Cleveland, OH, USA) according to the manufacturer's instructions. Where available, Clinical and Laboratory Standards Institute performance standards were used for interpretation of MICs; otherwise, interpretations established by the National Antimicrobial Resistance Monitoring System were used (*5*,*6*).

During 2009–2012, a total of 21 (0.2%) of the 9,225 NTS isolates tested were *S. enterica* ser. Kentucky. Six (29%) were resistant to ciprofloxacin; all were susceptible to ceftriaxone (Table) (*5*). As was observed in Canada, the 6 resistant isolates were >80% similar by pulsed-field gel electrophoresis analysis (*Xba*I; data not shown), and all 6 were ST198. Although a rare cause of human infection, *S. enterica* ser. Kentucky represented 23% (6/26) of all ciprofloxacin-resistant NTS detected during 2009–2012.

The median age of the 6 patients with ciprofloxacin-resistant *S. enterica* ser. Kentucky infections was 32 years (range 9 months–56 years); 5 (83%) were female. Of the 4 patients for whom information was available, 2 were hospitalized, and 1 died. Specimen sources were stool (n = 3) and urine (n = 3). Travel histories were obtained for 5 patients, and all had traveled internationally in the 7 days before specimen submission: 2 were residents of other countries (Saudi Arabia and Ethiopia), and 3 were US residents who had returned from travel to India. By comparison, only 3 of 10 patients with ciprofloxacin-susceptible infections had traveled ($p = 0.02$).

Resistance to ciprofloxacin in *Salmonella* is a growing concern because it limits treatment options for invasive disease. We describe ciprofloxacin-resistant *S. enterica* ser. Kentucky isolated from 6 patients in the United States. The emerging global story of *S. enterica* ser. Kentucky ST198 demonstrates the need for international integration of surveillance for antimicrobial drug resistance.

Table. Patient and isolate information for 6 cases of infection with ciprofloxacin-resistant *Salmonella enterica* serotype Kentucky sequence type 198 detected by the National Antimicrobial Resistance Monitoring System, United States, 2009–2012*

Isolate ID	Patient age, y/sex	Patient race	Patient travel history	Year specimen collected	Specimen type	Antimicrobial resistance†
AM41047	<1/F	Black	Ethiopia	2009	Stool	AMP, FIS, GEN, NAL, STR, TET
AM45820	54/F	Unknown	Unknown	2010	Urine	AMP, COT, FIS, GEN, NAL, STR, TET
AM47052	56/F	Asian	India	2011	Urine	AMP, FIS, GEN, NAL, STR, TET
2012AM-1081	2/F	Asian	India	2012	Stool	AMP, NAL
AM50773	37/M	Asian	India	2012	Stool	AMP, AUG, FIS, GEN, NAL, STR, TET
2012AM-0353	42/F	White	Saudi Arabia	2012	Urine	AMP, FIS, FOX, KAN, NAL, STR, TET

*ID, identification; AMP, ampicillin; AUG, amoxicillin-clavulanic acid; COT, trimethoprim-sulfamethoxazole; FIS, sulfisoxazole; FOX, cefoxitin; GEN, gentamicin; KAN, kanamycin; NAL, nalidixic acid; STR, streptomycin; TET, tetracycline.
†Resistance of isolate from infected patient to antimicrobial agents other than ciprofloxacin.