

were positive for HCoV 229E and influenza A virus.

In this study, we simultaneously analyzed tissue specimens of paranasal sinuses and nasal polyps, as well as nasal swabs and blood samples, for a broad panel of viruses and atypical bacteria. To avoid seasonal bias, specimens were collected over a 1-year period and exclusively obtained from patients undergoing elective surgery in the absence of acute respiratory symptoms.

The finding that HBoV was present as a single virus in 18/22 virus-positive biopsy samples is intriguing. Moreover, the fact that no HBoV DNA was detected in nasal swabs or EDTA-blood samples indicates no active HBoV infection. In previous studies, HBoV DNA was frequently identified in the adenoids and tonsils of children (2,5,10). However, in contrast with our findings, detection of HBoV was mostly associated with other viruses, suggesting that co-virus-induced cellular damage might contribute to bocavirus reactivation and replication (5). Our findings indicate that persistence of viral nucleic acid in sinus mucosa might be a special advantage of HBoV, although the relevance of this observation remains unclear. Whether this presence as a single virus means a dead end for HBoV infection, true latency including the potential of reactivation, or a role in the pathogenesis of clinical conditions requiring surgery warrants future studies.

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Mixed Genotype Infections with Hepatitis C Virus, Pakistan

To the Editor: The prevalence of hepatitis C virus (HCV) infection is high (8% of the population) in Pakistan (1). HCV is an RNA virus that has a high mutation rate. This high rate results in extensive genetic heterogeneity, and HCV isolates are found as either quasispecies or genotypes (2). Humans can be co-infected with ≥ 1 genotype (mixed genotype infection) of this virus (3). The rate of HCV mixed genotype infections is extremely variable for different regions and for the same group of patients tested by using different assays (4). Thus, it is difficult to determine the prevalence of mixed genotype infections by currently available assays, including direct DNA sequencing, because they are designed to identify only the HCV genotype dominant in that particular population. Consequently, genotypes present at lower frequencies could be missed or mistyped (5).

To determine the prevalence of HCV mixed genotype infections, we retrospectively analyzed genotyping data for paired serum samples from 22,125 HCV-infected patients during the past 11 years (March 2000–May 2010) for all regions in Pakistan by using molecular-based genotype-specific methods (6,7). A total of 12,036 (54.4%) were male patients and 10,089 (45.6%) were female patients.

Table. Distribution of mixed genotype infections with hepatitis C virus in 1,007 patients, by age, Pakistan, March 2000–May 2010

Genotype	Mixed genotype infection	Patient age, y							Unknown	Total no. infections
		0–10	11–20	21–30	31–40	41–50	51–60	≥60		
1	1a + 1b	0	0	2	5	6	7	0	1	21
1 + 2	1a + 2a	0	0	2	1	2	0	0	0	5
	1b + 2a	0	0	0	1	2	1	0	1	5
	1b + 2b	0	0	1	0	0	0	0	0	1
1 + 3	1a + 3b	0	3	4	4	3	1	0	0	15
	1a + 3a	0	4	29	51	26	16	2	17	145
	1c + 3a	0	1	2	2	1	0	0	1	7
	1b + 3b	0	2	3	2	1	0	0	0	8
	1b + 3a	0	7	45	90	79	32	3	31	287
1 + 4	1a + 4	0	0	0	0	3	1	0	1	5
2 + 3	2a + 3a	0	2	5	9	4	3	0	1	24
	2a + 3b	0	1	5	8	5	3	0	1	23
	3a + 2b	0	0	1	2	2	0	0	1	6
3	3a + 3b	1	15	94	142	87	46	13	43	441
3 + 4	3b + 4	0	1	0	2	2	1	0	0	6
	3a + 4	0	0	0	2	4	0	0	0	6
3 + 6	3a + 6a	0	0	0	1	0	1	0	0	2
Total		1	36	193	322	227	112	18	98	1,007

The sensitivity and reliability of the assay we used has been assessed and found to be superior to restriction fragment length polymorphism analysis and serotyping methods for detection of mixed genotypes in a viral population. Our method can detect a small amount (8.3%) of HCV RNA in a mixed genotype population (7). Restriction fragment polymorphism analysis can detect 2 genotypes only if 1 of them represents $\geq 41.6\%$ of the genotypes in a mixed genotype population.

Of 22,125 HCV RNA-positive serum samples, type-specific PCR bands were observed in 18,181 (82.2%) samples and 3,944 (17.8%) were not typeable. A total of 1,007 (5.5%) patients had HCV mixed genotype infections.

The distribution of mixed genotype infections in 1,007 patients is shown in the online Appendix Figure (www.cdc.gov/EID/content/17/8/100950-appF.htm). Infection with mixed genotype 3a + 3b was most prevalent (43.79%). Age distribution of patients with mixed genotype infections is shown in the Table. Approximately 33% of patients with mixed genotype infections were 31–40 years of age and 22.5% were 41–50 years of age.

Patterns of HCV mixed genotype infections in Pakistan are similar to those reported from India and Iran (8). However, the prevalence of HCV mixed genotype infections was lower (2%) (8) for Iran than for Pakistan. This lower rate may have been caused by use of a genotyping kit that can detect only genotypes 1a, 1b, 2, and 3a. Thus, mixed infections with other genotypes would not have been detected. A recent study in Brazil reported that mixed genotype infections were detected in 3.9% of intravenous drug users and 7.1% of former injecting drug users (9). These rates were similar to those in our study. In contrast, data from Sweden and Russia showed no mixed genotype infections in serum samples of chronically infected intravenous drug users, hemodialysis patients, and patients with hemophilia (10).

Women (288/7,390, 3.89%) in Pakistan had significantly fewer HCV mixed genotype infections than men (719/10,791, 6.66%) ($p < 0.01$). This finding might be the result of women having fewer risk factors for contracting mixed genotype infections. Possible risk factors for infection with mixed genotype infections analyzed were blood transfusions and use of blood products (51.3%); multiple use

of needles or syringes (18.4%); sharing razors during shaving or circumcision, piercing instruments, nail clippers, and toothbrushes (13.7%); and major or minor dental surgery (9.5%). Mode of transmission was not clear for 7.1% of the patients.

In conclusion, the prevalence of HCV mixed genotype infections in Pakistan is higher than previously reported and higher among men ($p < 0.01$). Comprehensive and detailed investigations are warranted to evaluate the clinical role of chronic HCV mixed genotype infections, provide essential information that can be used to determine type and duration of therapy needed, and predict disease outcome.

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West Nile Virus Aseptic Meningitis and Stuttering in Woman

To the Editor: West Nile virus (WNV), a mosquito-borne flavivirus, is closely related to St. Louis encephalitis virus and Japanese encephalitis virus (JEV). Most cases of WNV have been mild, but neuroinvasive disease has been observed, especially among older persons and immunocompromised persons (1,2). The most common neurologic manifestations of WNV are aseptic meningitis, meningoencephalitis, and encephalitis with or without acute flaccid paralysis (3). Other less common neurologic manifestations include Guillain-Barré syndrome, chorioretinitis, stroke-like symptoms, and unilateral brachial plexopathy (4,5).

We report a case of WNV aseptic meningitis in a 39-year-old immunocompetent woman who had severe headache with new-onset stuttering. Her medical history included lumbar disc herniation and migraines, for which she was taking sumatriptan. Her symptoms started ≈2 weeks before hospitalization and included a severe generalized headache initially thought to be a migraine, but sumatriptan resulted in no improvement. A few days later, she had fever and was intermittently stuttering. She denied recent travel or animal exposure but admitted to

having received multiple mosquito bites during the preceding weeks.

At admission, she had a temperature of 101.3°F, pulse rate of 92 beats/min, blood pressure of 130/80 mm Hg, and respiratory rate of 16 breaths/min. She appeared mildly ill but was alert and oriented with no nuchal rigidity, photophobia, rash, or limb weakness. Results of a physical examination were unremarkable, and results of a neurologic examination were notable only for stuttering. Laboratory test results included a leukocyte count of 12,300 cells/mm³ (63% neutrophils, 29% lymphocytes, 7% monocytes, 1% basophils) and a platelet count of 204,000 cells/mm³. Other laboratory values were unremarkable, and levels of serum transaminases and creatinine phosphokinase were within reference ranges. Cerebrospinal fluid (CSF) was clear and contained 37 leukocytes/mm³ (2% neutrophils, 78% lymphocytes, 20% monocytes), 2 erythrocytes/mm³, a glucose level of 68 mg/dL, a protein level of 36 mg/dL, and a lactic acid level of 2.1 meq/L. No abnormalities were found on a cranial computed tomography scan.

The patient began treatment with acyclovir, 10 mg/kg intravenously, every 8 hours for 3 days. On hospital day 2, she underwent magnetic resonance imaging of the brain; results were within reference limits. On hospital day 3, her headache began to improve and she became afebrile, but she still stuttered occasionally. Results of CSF tests for enterovirus, herpes simplex viruses 1 and 2, and varicella zoster virus and PCR for human herpesvirus 6 were negative, and acyclovir was discontinued. On hospital day 5, she was discharged. Three days later, serum and CSF ELISA results for WNV were positive. A WNV ELISA was performed at ViroMed Laboratories (Minnetonka, MN, USA) by using a Focus Test Kit (Focus Diagnostics, Cypress, CA, USA), and the result was positive. The