performed under fluoroscopic guidance. Pathologic examination of these specimens was consistent with discitis-osteomyelitis. Routine aerobic cultures of disc and bone both yielded *Streptococcus agalactiae* (also known as Group B *Streptococcus* or GBS). Anaerobic, fungal, and AFB cultures were negative, as was PCR for *Bartonella*. A tuberculin skin test and serum *Bartonella* antibodies were negative. Because GBS was unexpected from this site in a patient this age, a screen of the patient's immune status was undertaken. The HIV antibody was negative; serum immunoglobulins, total hemolytic complement, and neutrophil oxidative burst were normal; serum antibody values were consistent with an appropriate response to routine immunizations. The patient was placed in a thoracolumbosacral orthosis and he was treated with 8 weeks of intravenous antibiotics.

The rate of GBS infection in neonates has decreased since the institution of universal screening of pregnant women.[1] Invasive GBS disease remains a significant cause of morbidity and mortality in nonpregnant adults, almost exclusively in those with underlying immunocompromising conditions.[1,2] Although uncommon, discitis and vertebral osteomyelitis caused by GBS have been described, primary in elderly or immunocompromised patients.[3–5]

Our search of the literature did not reveal any cases of GBS discitis or vertebral osteomyelitis in children outside of the neonatal period. The isolation of GBS in our healthy adolescent patient from both the vertebral body and intervertebral disc makes his unusual presentation of discitis even more remarkable. Clinicians should be aware of the possibility of GBS as a pathogen in discitis and vertebral osteomyelitis, especially, although not exclusively, in immunocompromised patients.

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Kawasaki Disease Lacks Association With Human Coronavirus NL63 and Human Bocavirus

To the Editors:

Numerous reports have proposed an infectious etiology for Kawasaki disease (KD), but none of the discussed agents has been confirmed so far. Recently, an association of KD with the emerging respiratory pathogens, human coronavirus NL63 (HCoV-NL63), and human bocavirus (HBoV) was suggested.[1,2] However, ensuing studies could not find any correlation.[3] All trials employed PCR methods, though viral nucleic acids can usually be detected only during viremia or shortly thereafter. Hence, we used a serologic approach to examine a possible correlation between HCoV-NL63 or HBoV infections and KD.

Twelve children (10 male, 2 female) suffering from complete KD and 9 children (5 male, 4 female) with incomplete KD were identified according to diagnostic guidelines presented elsewhere.[4] Patients were aged 8 months to 14.9 years (mean: 4.5 years). Samples were obtained 3 to 10 days (mean: 5.5 days) after onset of fever, before medical treatment was commenced. As a control group, 33 children (18 male, 15 female; 6 months–14.5 years, mean: 5.2 years) were enrolled. All children originated from South-Eastern Germany and sera were collected between January 2006 and May 2008.

The collection of specimens was approved by the University of Regensburg Ethics Committee and written parental consent was obtained.

HCoV serology was conducted by use of a novel line-immunoassay, which allows the detection of antibodies to HCoV-strains 229E, NL63, OC43, HKU1, and SARS-CoV.[5] Screening of HBoV-specific humoral responses was performed by ELISA as described previously.[6] Additionally, all control sera and 19 of 21 samples obtained from KD children were screened for HBoV-DNA by quantitative real-time PCR as published elsewhere.[6]

Statistical analysis was performed by standard two-tailed χ^2 test. A $P \leq 0.05$ was considered statistically significant.

Seroprevalences of HCoVs and HBoV in KD children and in healthy controls are summarized in Table 1. IgG specific for HCoV-NL63 was found in 48% of children with KD but also in 67% of healthy children ($P = 0.27$). Comparison of antibody responses to any other HCoV in KD children and in healthy controls did not reveal any statistical relevance. Our data are in line with previously published findings, revealing that HCoV-NL63 and HCoV-229E seroconversion occurs on average before children reach the age of 3.5 years.[7] Recent coronaviral infections characterized by virus-specific IgM were neither overtly elevated in healthy controls (0%–3%) nor in KD children (5%–10%). However, due to the short time between onset of fever and sampling, some acute infections could have been missed. HCoV IgA-seroprevalence was not correlated with any of the 2 study groups. None of the tested individuals was detected positive for SARS-CoV.

HBoV-specific IgG and IgA were observed in 90% and 48% of samples obtained from KD patients, respectively. A comparable seroprevalence of bocavirus-specific IgG (82%, $P = 0.63$) and IgA (58%, $P = 0.66$) was found in the control group, confirming a previously reported high IgG seroprevalence in children between 4 and 5 years of age.[6] For the first time, IgA-antibodies against HBoV were detected in both healthy children and KD patients. Yet, IgA's role during viral infection and pathogen clearance remains to be elucidated. HBoV-specific IgM was not found in any of the samples, whereas in another trial IgM was detected in 67% of individuals suffering from productive HBoV infection.[6] Potential acute infections in the study groups were additionally excluded by PCR analysis. We detected productive infection in 1 healthy child (4.9×10^3 genome equivalents/mL serum), whereas no viral DNA was found in sera collected from children with KD (Table 1, HBoV PCR).

In summary, our serologic data strongly suggest that there is no association between infections with HCoVs and/or HBoV and KD in children. It yet remains to be determined whether KD is caused by a single pathogen or whether it is the result of interaction of more than 1 etiologic agent. Furthermore, genetic predisposition and environmental factors might promote development of full blown KD.

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TABLE 1. HCoV and HBoV Serology and PCR Detection of HBoV in KD Children and Healthy Controls

Study Subjects	N	Age	HCoV Serology												HBoV Serology			HBoV PCR pos. (%)
			229E pos. (%)			NL63 pos. (%)			OC43 pos. (%)			HKU1 pos. (%)						
			IgG	IgM	IgA	IgG	IgM	IgA	IgG	IgM	IgA	IgG	IgM	IgA	IgG	IgM	IgA	
HC	33	5.2	7 (21)	1 (3)	2 (6)	22 (67)	1 (3)	7 (21)	10 (30)	1 (3)	4 (12)	4 (12)	0	2 (6)	27 (82)	0	19 (58)	1 (3)
KD	21	4.5	4 (19)	2 (10)	2 (10)	10 (48)	2 (10)	6 (29)	11 (52)	1 (5)	4 (19)	6 (29)	1 (5)	5 (24)	19 (90)	0	10 (48)	0*
$P^{\dagger}$			0.85	0.68	0.66	0.27	0.68	0.77	0.18	0.74	0.76	0.25	0.81	0.14	0.63	—	0.66	0.44

*HBoV PCR was accomplished with 19 of 21 serum samples.

†Two-tailed χ^2 test.

HCoV indicates human coronavirus; HBoV, human bocavirus; PCR, polymerase chain reaction; N, number of individuals tested; Age, mean age in years; pos., positives; Ig, immunoglobuline; HC, healthy control; KD, Kawasaki disease.

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Acute Encephalopathy Associated With Influenza C Virus Infection

To the Editors:

Influenza C virus infection is considered to be milder than the infections caused by influenza viruses A and B; there are no reports of severe complications associated with influenza C virus infections.[1] Influenza C virus is distributed worldwide, and the seropositive rate in people aged above 10 years is approximately 100%. However, the clinical diagnosis of type C influenza is complicated by the rarity of specific symptoms and the dearth of facilities equipped with the resources for performing efficient viral isolation. Here we report the first case of acute encephalopathy associated with influenza C virus infection.

The patient (age, 2 years and 4 months) presented with hyperpyrexia. Several hours after the onset of hyperpyrexia, the patient had a generalized convulsion that lasted for approximately 10 minutes, after which the patient exhibited severely disturbed consciousness and symptoms of compensatory shock. His body temperature was 42.0°C.

The pharyngeal and nasal swabs collected on admission tested positive for the influenza C virus but negative for the other viruses. The serum hemagglutination-inhibition titer of antibodies against the isolated virus increased from less than 8-fold at the onset of hyperpyrexia to 128-fold on day 24.

CSF analysis revealed a normal cell count. The cytokine profile on admission revealed markedly elevated serum and CSF concentrations of interleukin (IL)-6 (1527.2 pg/mL and 951.3 pg/mL, respectively) and IL-10 (582.3 pg/mL and 49.3 pg/mL, respectively).

Diffusion-weighted imaging of the brain, performed on day 7, revealed diffuse high-intensity signals over the subcortical white matter. Diffusion-weighted imaging performed on day 24 revealed that the high-intensity signals indicating dendritic forms had disappeared; however, mild diffuse brain atrophy persisted.

Acute encephalopathy with prolonged febrile seizure and late reduced diffusion (AESD) has been suggested to be associated with infection by some viruses (eg, influenza A, influenza B, and human herpes virus type 6).[2] AESD is considered the primary form of excitotoxicity-induced acute encephalopathies. The MRI findings obtained in the present case are compatible with those noted in patients with AESD. AESD usually exhibits a biphasic clinical course, with status epilepticus at the onset. However, our patient underwent a monophasic clinical course, and status epilepticus was not noted. This could be attributable to the immediate intensive care that the patient received during the early phase. We considered that the diagnostic criteria for AESD were satisfied in the present case.

The invasion, uncoating, and proliferation mechanisms of the influenza C virus are fundamentally identical to those of the influenza A virus,[3] and we can assume that the influenza C encephalopathy is not associated with any unique pathophysiology. A unique feature of our case is the concomitant elevation in the CSF levels of IL-6 and IL-10. The marked elevation in the patient's IL-10, which is not observed in common AESD,[4] indicated CNS inflammation.

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