any anatomic defect that could predispose this patient to invasive disease due to this unlikely pathogen, a history of suspected immunodeficiency, or evidence of bloodstream infection, it is unclear what mechanism underlies this patient’s infection. Although nontypeable Ha. influenzae is rarely found in the gastrointestinal tract [1], given this patient’s abdominal complaints and pseudocyst formation, the most likely source of her infection is her gastrointestinal tract. Studies suggest that certain nontypeable Ha. influenzae strains possess one or more virulence factors capable of mediating bloodstream invasion or resistance to host immune defenses [6]. Whether the nontypeable Ha. influenzae isolated in our case with recurrent ventriculoperitoneal shunt infection possesses such virulence determinants remains to be determined.

Mary E. Lim, Jill A. Hoffman, and Kwang Sik Kim
Division of Infectious Disease, Childrens Hospital Los Angeles; and University of Southern California School of Medicine, Los Angeles, California

Herpes Simplex Virus Encephalitis: Chronic Progressive Cerebral Magnetic Resonance Imaging Abnormalities in Patients Despite Good Clinical Recovery

Herpes simplex virus encephalitis (HSVE) is a life-threatening encephalitis. Cranial MRI is sensitive in the detection of morphological abnormalities in acute HSVE. We aimed to prospectively study MRI changes in patients with HSVE, who were treated early in the course of the disease, and continued the studies for up to 6 months following onset of symptoms.

All patients (five male, one female) had received acyclovir (three doses daily, 10 mg/kg of body weight) for at least 14 days. HSV DNA in the CSF was confirmed by PCR assay, done as described recently [1]. Patients were clinically assessed daily during hospitalization and 6 months later. MRI and neuropsychological assessment [2–4] were done on admission, at discharge, and at 6 months.

Clinical symptoms during acute disease were recorded as mild (no impairment of consciousness, minor cognitive dysfunction), moderate (focal neurological signs, disoriented, or stuporous), and severe (coma, severe dysfunction after short-term recovery) [1]. Magnetic resonance images were obtained (1.0 T; Picker-Vista; Cleveland) with the following sequence parameters: proton density, T2-weighted spin echo (SE) (repetition time [TR], 2400 ms; echo time [TE], 20 ms, 80 ms, respectively); T1-weighted SE (TR, 560 ms; TE, 20 ms). MRI was done before and after administration of gadopentetate dimeglumine, 0.2 mmol/kg of body weight. Mild MRI changes were defined as abnormal areas without space-occupying effect in ≤30% of characteristic locations (temporal lobe, cingulate gyrus, frontobasal lobe). Moderate changes involved ≤50% of the described areas, but >30% with an occasional space-occupying effect. Severe findings involved >50% and showed a space-occupying effect.

Neurological and neuropsychological assessment. On admission, two patients were graded as mildly impaired (Glasgow coma scale [GCS] 14), two patients had moderate clinical and neuropsychological findings (GCS 9, 10), and two were graded severe (GCS 7, 8). At discharge, MRI of five of six patients revealed improvement. All patients were significantly improved after 6 months.

Cranial MRI findings. Abnormalities on admission and at discharge from hospital were graded as mild (in 2/6), moderate (in 3 and normal (in 1). After 6 months, MRI findings were moderate (in 5) and normal (in 1). Two patients had progressive abnormalities (figure 1). One of these was clinically mildly impaired and the other moderately impaired at 6 months. Because both showed significant clinical improvement, repeated CSF analysis was not done.

Kapur et al. [5] described a positive correlation between long-term cognitive impairment and involvement of limbic structures on MRI. Consistent with that, in five patients we found abnormalities in limbic structures and neuropsychological impairment. In two patients we found chronic progressive MRI changes despite clinical improvement. The histological background of these findings is not known. One may hypothesize that these changes represent relapsing or chronic HSVE [6]. However, relapsing, chronic HSVE is characterized by clinical deterioration and is therefore unlikely. Progressive changes after early antiviral treatment may be secondary to tissue damage not directly mediated by the virus. Evidence for such secondary immune-mediated tissue damage comes from experimentally induced HSVE [7]. Clinicians should be aware of the possibility of chronic progressive MRI changes in HSVE despite clinical improvement and antiviral therapy.

Uta K. Meyding-Lamadé, Wolfram R. Lamadé, Brigitte T. Wildemann, Klaus Sartor, and Werner Hacke
Departments of Neurology, Neuroradiology, and Surgery, University of Heidelberg, Heidelberg, Germany

References

Informed consent was obtained from all patients or their guardians included in this prospective follow-up study. The guidelines for human experimentation were followed in the conduct of the clinical research.

Financial support: German Society of Virology (Glaxo Wellcome) and Forschungsförderung, University of Heidelberg, Germany.

Reprints or correspondence: Dr. Uta K. Meyding-Lamadé, Department of Neurology, INF 400, 69120 Heidelberg, Germany (J79@ix.urz.uni-heidelberg.de).

Clinical Infectious Diseases 1999;28:148–9
© 1999 by the Infectious Diseases Society of America. All rights reserved.
1058-4838/99/2801–0029$03.00
Figure 1. Progressive MRI findings: A, C, spin echo (SE) 560/20, before (A) and after (C) administration of contrast agent; section thickness, 3 mm; B, D, SE 2400/20; section thickness, 5 mm; coronal plane. At discharge from hospital, findings were graded mild (B, arrowheads). At 6 months (C, D), abnormalities were graded as moderate. Additional pathological contrast enhancement (small arrows) was present in the mediobasal temporal lobe (C). Lateral to the inferior horn of the ventricle, a sharply defined area of tissue loss secondary to herpes simplex virus encephalitis had the same signal intensity as CSF (C [curved arrow], D). High signal abnormality in the medial temporal lobe had extended at 6 months (D). Note additional involvement of the gyrus cinguli, gyrus frontalis inferior, and insula (small arrows).

References