and pneumonia. The patient was treated initially with cefotaxime (50 mg/kg po q8h) and oxacillin (37 mg/kg iv q6h) and then with vancomycin (10 mg/kg iv q6h), clarithromycin (7.5 mg/kg po q12h), and ampicillin-sulbactam (50 mg/kg po q6h), but his clinical condition worsened and sepsis developed, at which time he was referred to our institution (West General Hospital, Caracas).

At our institution, several cultures were performed, including cultures of sputum, pleural fluid, blood, urine, and stool specimens. The findings of coproparasitological studies were all negative. A. hydrophila was isolated from blood cultures, and no other pathogen was isolated from any culture. The isolated strain was initially tested for antimicrobial susceptibility with the disk-diffusion technique and then by determination of MICs, in accordance with NCCLS standards; it was found to be susceptible to imipenem and cefoperazone-sulbactam but resistant to amikacin, cefotaxime, levofloxacin, gentamicin, oxacillin, piperacillin, ampicillin-sulbactam, and extended-spectrum cephalosporins. The isolate was an ESBL-producing strain (as determined by the double-disc technique, although this method is nonstandardized for this pathogen). Antimicrobial drug therapy was changed to imipenem (15 mg/kg iv q6h), with a successful clinical evolution. Additional culture results were all negative.

Despite the abundant knowledge about inducible, chromosomally mediated β-lactamases among Aeromonas species, ESBL-producing A. hydrophila strains isolated in clinical practice have been rarely reported [4]. There are recent reports of A. hydrophila strains resistant to tetracyclines, cotrimoxazole, aminoglycosides, and extended-spectrum cephalosporins [5–7]. In the case we describe here, the clinically isolated strain was susceptible to imipenem and cefoperazone-sulbactam but was resistant to the other β-lactams tested. This report supports the importance of bacteriological diagnosis of Aeromonas respiratory tract infection, as well as the epidemiological relevance of stool investigation for certain cases—namely, those in which ingestion of contaminated water is suspected and in which diarrhea was present before respiratory tract infection developed, especially if an antibioticogram reveals a drug-resistant pathogen that commonly produces sepsis and fatal outcomes [8], especially in children.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

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ected in CSF specimens but not in PBMC specimens.

In immunocompromised patients—particularly of HIV-1-infected patients and organ transplant recipients—HHV-6 re-infection and reactivation of HHV-6 infection have been widely reported; thus, HHV-6 may be considered an important opportunistic pathogen [2]. In addition, concurrent infections with HHV-6 and other pathogens, including measles virus, herpes simplex virus, cytomegalovirus, Epstein-Barr virus, parvovirus B19, coxsackievirus B, and varicella-zoster virus, have been reported [4, 11]. It is interesting to note that these viral infections concurrent with HHV-6 infection have not been specifically associated with an immunosuppressive effect.

On the other hand, reactivation or reactivation of HHV-6 infection is trickier to demonstrate in immunocompetent adults with meningitis/encephalitis than in others, even though HHV-6 seems to be a resident virus of the human brain and is able to cause a restricted or minimally productive infection of brain cells, including microglial cells, astrocytes, and oligodendrocytes [12].

In our opinion, however, the pathogenic mechanism involved in HHV-6 meningitis/encephalitis in immunocompetent adults may be related to the ability of HHV-6 to evade host immune responses through (1) induction of CD4 lymphocyte depletion via apoptosis [13], (2) down-regulation of CD3 expression on in vitro–infected T cell clones [14], (3) decrease of peripheral blood lymphocyte proliferation by HHV-6 via transcriptional down-regulation of IL-2 [15], and (4) decreased generation of reactive oxygen intermediates from monocytes that were infected with HHV-6 in vitro [16]. In addition, a transient immunocompromise of T cell immune response—particularly of natural killer cell activity and of IFN-α production—cannot be ruled out in adult immunocompetent patients with meningitis/encephalitis [4].

In conclusion, in adult immunocompetent patients, HHV-6 may behave as an immunosuppressive pathogen, to evade host immune responses, and in immunocompromised patients, it also behaves as an opportunistic pathogen. Thus, the ability of HHV-6 infection to remain latent or to be reactivated in certain conditions, along with the ability of HHV-6 to evade host immune responses through various mechanisms, render it an opportunistic and serious pathogen in immunocompromised patients as well as in adult immunocompetent patients with meningitis/encephalitis.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

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Clinical Infectious Diseases 2005; 41:422–3

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