Spontaneous Recovery from Perinatal Infection Due to Hepatitis C Virus

Hepatitis C virus (HCV) infection is transmitted by the perinatal route, and chronic infection develops in >50% of exposed individuals. However, up to 50% of subjects with chronic HCV infection deny exposure to known risk factors [1]. Mother-to-infant transmission of HCV infection has been documented in several studies [2–4], but the natural history of vertical, perinatal infection is largely unknown, and it is uncertain to what extent this route of transmission may contribute to the rate of chronic infection in the general population. A recent study showed progression to chronic infection in seven children who acquired HCV from their mothers who were coinfected with HCV and HIV [5]. Herein, we report the persistent clearance of HCV RNA in a perinatally infected child.

The 32-year-old mother involved in viral transmission had a history of intravenous drug use and HCV infection was diagnosed during pregnancy. Results of tests for hepatitis B surface antigen and antibodies to HIV were negative. At delivery, the results of a third-generation recombinant immunoblot assay (RIBA 3.0, Ortho Diagnostic System, Raritan, NJ) were positive: anti-C100 (score, 2), anti-C33 (score, 2), and anti-C22 (score, 4). Serum HCV RNA was detected by PCR (Amplicor HCV, Roche Diagnostic Systems, Branchburg, NJ) and classified as genotype 3. The level of viremia, measured by competitive PCR [6], was \(10^{2.2}\) RNA copies/mL.

The female infant was born by vaginal delivery and breastfed. A PCR assay for HCV RNA was negative at birth; the RIBA 3.0 pattern was similar to that of the mother (figure 1). Abnormal alanine aminotransferase (ALT) levels, together with serum HCV RNA (genotype 3, \(10^7\) copies/mL) and anti-C22-indeterminate RIBA 3.0 (score, 2), were detected when the infant was 3 months old. The maternal origin of the infection was supported by the detection of an identical master sequence of the hypervariable region (E2 gene) in the mother-infant pair [4].

An increase in ALT levels and in anti-C22 score was detected in the infant at 4 months of age, and seroconversion to anti-C33 was observed at 7 months, together with diminishing ALT levels. The anti-C33 score increased at 16 months, with persistence of HCV RNA despite ALT levels within the normal range. The viral RNA was not detected in further samples collected at 24, 30, and 36 months of age. ALT levels were persistently normal and the anti-C33 score progressively decreased after the clearance of HCV RNA.

Follow-up observation of children with vertical, perinatal HCV infection has revealed wide variations in the ALT profile [4, 5].

Thus, the detection of normal ALT levels for prolonged periods cannot in itself be considered a reliable indicator of recovery from infection. However, since HCV RNA is detected consistently in children with chronic infection [4, 5], the absence of the viral RNA in three consecutive samples collected 6 months apart strongly suggests the persistent clearance of the virus. A recent study performed in our laboratory indicated a statistical correlation between reactivity to C33 in the RIBA test and persistence of serum HCV RNA in pregnant women with antibodies to HCV [4]. From this viewpoint, the hypothesis that the progressive decrease in anti-C33 score in the child is associated with recovery from infection is supported.

HCV transmission from mothers who are seronegative for antibodies to HIV is a rare event averaging 4.5%, whereas maternal coinfection with HIV-1 increases the risk of transmission to 18% [4, 7]. Maternal HIV-1 infection might play a role in HCV persistence and progression even in infants who acquire HCV infection alone. The only available data on the natural history of vertically, perinatally acquired HCV infection concern children of HIV-coinfected mothers, and these studies show chronic evolution in all cases [5]. By contrast, this report shows that HCV infection acquired from an HIV-1-seronegative mother...
Identification of *Shigella boydii* in Colonic Malacoplakia by Universal Bacterial 16S Ribosomal DNA–Based Amplification in a Human Immunodeficiency Virus–Infected Patient

Malacoplakia, a rare chronic infectious disease usually caused by *Escherichia coli*, most commonly affects the urinary tract. Its prevalence is increased among persons with immunosuppressive conditions. The principal underlying feature is probably a monocytic/macrophagic bactericidal defect. Malacoplakia is defined microscopically as a granulomatous accumulation of large granular macrophages containing the pathognomonic Michaelis-Gutmann (M-G) bodies [1]. We describe a case of colonic malacoplakia in an HIV-infected patient for which 16S rDNA–based amplification was used to search for a causative pathogen.

A 39-year-old man from Chad was determined to be HIV-positive in 1994 (CD4 cell count, 10/µL). Antiretroviral therapy was initiated with zidovudine and then didanosine. In June 1995 candidal esophagitis was diagnosed. In August 1996, he was readmitted to the hospital because of fever, weight loss, chronic diarrhea, and abdominal pain. His CD4 cell count at that time was 5/µL. Microbiological examinations of blood, stool, and urine specimens were negative. A colonoscopy demonstrated yellowish, ulcerative polypoid lesions involving the entire colon. Histopathologic examination of colonic biopsy specimens revealed a multifocal infiltration of the lamina propria, consisting mainly of large histiocytes with abundant granular cytoplasm that contained rare rounded eosinophilic inclusions, suggestive of M-G bodies. Electron microscopic evaluation showed numerous intracytoplasmic heterogeneous phagolysosomes and characteristic concentric multilaminated calcified spherules (M-G bodies, figure 1) leading to the diagnosis of malacoplakia. Intracellular or tissular bacteria were not seen. Cultures of the biopsy specimens were negative.

Universal bacterial 16S rDNA–based amplification was carried out by means of simple PCR with use of a primers set that recognized conserved bacterial 16S rDNA gene sequences and amplified divergent bacterial 16S rDNA regions: 91E and 13B, as previously described [2]. The final 475-bp amplification product was subjected to direct sequencing with fluorescent-labelled primers and dideoxynucleotide terminator approaches by using an automated DNA sequencer. The DNA sequence was aligned with use of the