Letters to the Editor

Screening for herpes simplex virus in infertile women

Herpes simplex virus (HSV) infection is among the most common causes of sexually transmitted diseases worldwide. Symptomatic infection with HSV causes much physical discomfort, psychological disorders and interferes with sexual relations. Subclinical infections, which are not infrequent, contribute to the sexual transmission of the disease. Besides, transmission of the virus from mother to child when passing through the birth canal may cause severe disease in the neonate, resulting with death or serious sequelae.

Two hundred and seventy-nine infertile women attending an in vitro fertilisation and embryo transfer (IVF-ET) programme in the Ege University Hospital (Turkey) were included in the study. The patient group consisted of asymptomatic women from the middle socioeconomic class, aged between 25-40 years, having no history of genital herpes. Endocervical specimens were obtained from these women on their first visit to the hospital. Specimens were tested by a commercially available direct immunofluorescence test kit (Pathfinder, Kallestad Laboratories, USA), according to the manufacturer’s recommendations.

Forty-one out of 279 specimens (14.69%) were considered inadequate, owing to insufficient number of cells. Of the remaining 238, three were positive for HSV-1 (1.26%), 11 for HSV-2 (4.62%), and one was both HSV-1 and -2 positive (0.42%). The overall positivity rate for HSV was 6.30%.

Genital HSV infections have shown a rising trend all over the world in recent years. However, it is difficult to assess the real percentage of genital herpes due to the frequency of subclinical infections. Koutrakis et al. indicated that, in a patient group having serologic or virologic evidence of HSV-2, 22% had symptomatic clinical genital infection, 16% reported past episodes of genital herpes, but were asymptomatic at the time material was collected, while 62% had unrecognised infection, of whom 4% had asymptomatic viral shedding at the time of examination. In another study, Kulhanjian et al. stated that, detailed questioning of the participants revealed that only 35% of the women, and 46% of the men with serologic evidence of HSV-2 infection had had symptoms of genital herpes.

In the present study, none of the patients had past or present evidence suggestive of genital herpes. However, 6-3% were asymptotically shedding the virus. Barlas et al. from Turkey, detected the HSV positivity rate in asymptomatic young women from a low socioeconomic class as 7.7%. In another study from Turkey, HSV-2 incidence in sex workers was found to be 6%.

As stated by several authors, asymptomatic infection plays a major role in the transmission of the virus to seronegative sexual partners or more importantly, to the neonates.

The development of antiviral therapy has made it possible to improve the outcome of the neonatal disease and the knowledge of the epidemiology of herpes infections in a certain population is essential to take preventive measures.

The outcome of the pregnancy and the survival of the newborn in an IVF-ET programme bears both physical and moral importance. Hence, the significance of genital herpes in such a patient group increases.

The data in the present study indicate that genital HSV infection is a challenge in our population as in many others, and that HSV-2 and less frequently HSV-1 can produce asymptomatic infections. Also, dual infections with both viruses can be encountered, although very rare.

Since there is no control programme on a national basis at present in our country, further studies on different patient groups are needed to disclose the epidemiological distribution of genital HSV infections. Meanwhile, screening of patients attending IVF-ET programmes, before any intervention is planned, may be beneficial for applying preventive measures beforehand.

Address correspondence to: Dr D Serter, Ege University, Faculty of Medicine, Department of Clinical Bacteriology and Infectious Diseases, 35100 Bornova, Izmir, Turkey

We are grateful to Ahmet Hüdaverdi and Ali Köse for technical assistance.

More severe course of delta hepatitis in HIV-infected patients [L]

HIV infection alters the course of both hepatitis B and C virus infections, increasing carriage states and viral replication. Interestingly, the effect is the opposite in terms of liver disease. Milder liver injury is seen in patients with chronic hepatitis B who become severely immunosuppressed, despite extremely high levels of HBV viremia. In contrast, rapidly progressive liver disease seems to occur in HIV-infected patients with chronic hepatitis C; while HCV viremia rises as the CD4+ T cell count falls.

In regions such as Spain, where drug addicts make up a large part of the HIV-positive population, delta hepatitis is the main cause of severe liver disease in HIV-infected patients. However, little is known on the effect of the interaction of HIV and hepatitis delta virus (HDV). Since HIV infection may be an important predisposing factor for premature liver cirrhosis in patients with chronic hepatitis D, we analysed the features of 27 HIV-positive patients suffering chronic delta hepatitis and compared them with those from 10 patients with chronic hepatitis D without HIV infection.

We reviewed the clinical charts of 37 patients attending our institution from 1989 to 1993, fulfilling the criteria for chronic hepatitis D (persistent hypertransaminemia, positive HBSAg in sera, and presence of delta antibody). As shown in the table, gender and age were similar in both HIV-positive and -negative individuals. However, previous or current drug addiction practices were admitted by 96% of the former and only by 30% of the latter. Probably for this reason HCV coinfection was also more prevalent in the HIV-positive than in the HIV-negative population (85% vs 20%). Mean alanine aminotransferase levels were higher (twice) in subjects with HIV infection than in those HIV-negative, and previous episodes of hepatic decompensation (ascites, encephalopathy, and/or jaundice) were recognised more often in the former. Indirect signs of portal hypertension (as manifested at physical examination, ultrasonography and gastroduodenal endoscopy) also tended to be more frequent in HIV-infected patients with chronic hepatitis D than in HIV-negative. Liver biopsy had been performed in 13 patients (8 HIV-positive and 5 HIV-negative), and histological findings were not significantly different comparing the groups, as chronic active hepatitis with cirrhosis was the most common diagnosis.

The reasons for this apparent more severe course of chronic hepatitis D in HIV-infected patients were investigated. We found a higher level of HDV replication in them, as reflected by the recognition of circulating delta antigen in a quarter of HIV-positives but only in one out of 10 HIV-negative patients. Interestingly, HBV replication which usually is suppressed in HDV superinfection, remained elevated in these HIV-immunodeficient patients (mean CD4+ T cell count was 172, SD 86 per mm3): serum HBeAg was detected in nearly half of the HIV-infected patients and in none of those HIV-negative, and HBV-DNA was positive in two thirds of the former and in none of the latter. In seven cases, delta antignaemia coexisted with detectable HBeAg in sera, and all these patients showed very high aminotransferase levels.

In immunocompetent individuals, the presence of multiple hepatitis virus infections seems to favour the predominant replication of one virus instead of the others. We recently demonstrated that in HIV-positive patients with severe immunodeficiency, this reciprocal inhibitory effect seems to be lost. Thus, high replicative kinetics are recognised for all coincident hepatitis viruses. Since impaired cellular immune function does not affect chronic HDV or HCV liver injury, as characteristically is seen for chronic hepatitis B, the direct cytopathic effect of these viruses could be enhanced with higher levels of HDV and HCV replication, leading to rapidly progressive liver disease.

In conclusion, we found a more severe