Genital herpes

Genital herpes is not only a distressing sexually transmitted infection but also a suspected cause of cervical carcinoma and a forerunner of serious disease in neonates. Furthermore, the incidence and pattern of infection is changing and yet there is neither a universally agreed system of management nor proved effective treatment.

Incidence

Reports of increasing prevalence of genital herpes were appearing in North American literature 10 years ago. Over the past 5 years there has been a 90% increase in patients with genital herpes who attend private physicians and an even greater rise in patients visiting clinics for sexually transmitted diseases.¹ There is a similar change in incidence in the United Kingdom—for example clinics reported 7547 cases of genital herpes in 1976 compared with 10 801 in 1980.²

Virology

Of the 2 herpes simplex viruses, type 2 (HSV-2) has always been the predominant cause of sexually transmitted infections. Herpes simplex virus type 1 (HSV-1) traditionally produces lesions above the waist, is the agent usually responsible for herpes labialis, and is frequently contracted in childhood largely as a consequence of kissing. The immunity acquired then cross reacts with the type 2 virus and may therefore modify a subsequent HSV-2 genital infection. In the 1950s most adults had antibodies to HSV-1 but since then this incidence has declined, particularly in upper socioeconomic groups. As a consequence the adult population has become more vulnerable to HSV-2 and at the same time the number of genital infections caused by HSV-1 rather than HSV-2 has increased. Recent reports from both sides of the Atlantic suggest this may now occur in a third of cases.

Clinical manifestations

A primary genital herpetic infection may be relatively mild or indeed completely asymptomatic. In these instances it is only recognised by screening for the presence of the virus, or by subsequent identification of HSV-2 specific antibodies, or by observing the characteristic cytological changes found in routine cervical smears. Alternatively it may be severe with large painful ulcers on the genitalia, inguinal lymphadenopathy, urinary re-

tention and systemic symptoms of headaches, generalised aching, and fever. Viral meningitis may occur in 4 to 8% of patients. Encephalitis when it occurs is often fatal but it is extremely rare outside the neonatal period.

Lesions appear within 3 to 7 days of sexual contact. In women these may affect the vulva, perianal skin, and the vaginal and ectocervical epithelium. The virus may have a predilection for metaplastic epithelial cells of the squamo-columnar junction and this is relevant to the hypothesis that genital herpes causes cervical carcinoma. In severe attacks multiple vesicles are seen, often surrounded by a red areola. These quickly macerate—within 24 to 48 hours in moist areas—and they may coalesce forming bullae and subsequently large shallow ulcerations. Vaginal and cervical infections sometimes present as raised fungating necrotic looking lesions. Healing may take up to 6 weeks and occurs without scarring.

A characteristic of herpes viruses is their ability to produce latent infections. This is true of HSV-2. In some patients the virus may persist, probably within sacral nerve ganglia, only to be reactivated by immunological, hormonal, biochemical, or physical changes. Even emotional disturbances may precipitate recurrent herpes genitalis. The symptomatology of these recurrent infections is rarely severe since lesions usually persist for less than 10 days. Nevertheless recurrent infections can be very distressing leading to apareunia, marital breakdown, severe depression, and suicide.

Management

Many antiviral preparations such as 5-iodo-2-deoxyuridine, adeninosine arabinoside, cytosine arabinoside, 2-deoxy-D-glucose, and photoreactive dyes have been used in the treatment of genital herpes but none has proved reliable in curing or preventing recurrent infection. Acyclovir is the subject of considerable current research. This drug acts as a selective substrate for viral thymidine kinase and it may shorten the healing time of vesicles and reduce viral shedding. It is unlikely, however, to influence latent infectiousness, it is potentially toxic, and most studies to date have been based on intravenous administration.

The disappointing results of chemotherapy have stimulated interest in vaccines and nonspecific stimulators of cell mediated immunity. For the

former to be successful they will need to sensitise cellular immunity since circulating antibodies are ineffective in preventing recurrent infections and in protecting the newborn. Trials with HSV-1 derived vaccines are in progress. It is hoped the cross immunity against HSV-2 will not only prevent genital herpes but also reduce the incidence of cervical carcinoma.

Clearly until improved treatments are available management has to be symptomatic and therefore analgesics and antibiotics to prevent secondary bacterial infection are used. Coitus is discouraged whenever lesions are present but it is difficult to guard against transmission from patients with asymptomatic infections. Prodromal sacral dermatomal irritation has been described with the recurrent disease. When recognised this should be taken as a warning to avoid sexual intercourse until the infection has cleared.

HSV in pregnancy

Genital herpes has been called the 'scourge of the sexual revolution' and if its incidence continues to rise it may prove an important factor in ending the current age of sexual freedom. To perinatologists, however, it is the potential transmission to neonates that represents the major concern.

Transplacental HSV infection of the fetus is uncommon even in women with severe primary disease. Abortion and congenital abnormalities presumed to be caused by HSV infections in the first trimester are rare³ and there are only isolated reports of HSV being cultured from amniotic fluid.⁴ The fetus is, however, susceptible to active virus present within the maternal birth canal during labour and delivery. Indeed modern techniques of labour management may increase this vulnerability because fetal skin is punctured either with scalp electrodes or at the time of blood sampling. A baby thus exposed has no effective resistance to the virus. Humoral antibodies acquired from the mother are inadequate and the cellular immune response is hopelessly immature. As a consequence 50% of these babies are likely to die and nearly half the remainder suffer serious neurological sequelae.³ Clearly modern chemotherapeutic regimes are far from satisfactory and spontaneous recovery seldom occurs.

Fortunately the experience of workers in the United States of America, where the incidence of neonatal HSV infection in some population groups is as high as 1 in 3500 live births,⁵ and where 1% of women probably shed HSV during pregnancy³ has shown that the neonatal disease can largely be prevented. It is recommended that all pregnant

women with either a primary genital herpes infection or with a history of recurrent herpes have cervical and vaginal swabs taken for viral culture weekly from 36 weeks until delivery. If any of these tests prove positive then the baby is best delivered by caesarean section. Indeed caesarean section is likely to prevent transmission of infection provided it is performed within 4 hours of the membranes rupturing. An operative delivery rate of nearly 33% may be anticipated but this is not considered a high price to pay for a healthy baby in such a high risk group. Breast feeding is not contraindicated.⁶

Hitherto this has been a rare neonatal problem in the United Kingdom, with only 66 cases reported between 1973 and 1980. With genital herpes fast becoming the most important sexually transmitted disease, however, and with the knowledge that 75% of cases of neonatal herpes can be traced to a maternal genital infection,⁷ the incidence may be expected to rise. Routine virological screening of all pregnant women is not feasible. Therefore obstetricians need to be alert to the possibility of asymptomatic recurrences and to the likely improved prognosis of babies delivered by caesarean section. Paediatricians are faced with the prospect of more seriously ill infected babies and an increasing prevalence of potentially lethal viruses that can be contracted not only from mothers but also from asymptomatic handlers within nurseries and special care units.

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