Perinatal listeriosis

Perinatal listeriosis was first reported by Burn in 1933 and is now estimated to affect one in 20 000 births.[1] About a quarter of the 424 reports from England, Wales, and Scotland in 1983 to 1985 were in children less than 1 month old, and half of these were neonatal infections (Communicable Disease Surveillance Centre, unpublished data).

Listeria monocytogenes is a short, Gram positive, non-sporing, motile rod whose isolation is enhanced at 4°C. It is a facultative anaerobe, and there are four antigenic serotypes, of which types 1 and 4 are the main ones causing infection in man.[2] The epidemiology of listeria infections has remained obscure: although the organism infects many different animals and has been cultured from soil and faeces, infection in man does not result primarily from direct contact with infected animals.[3] Neonatal infection may be caused by cross infection,[4] and outbreaks often occur in clusters[5] or epidemics.[6-10] *L monocytogenes* may be a normal resident of the intestinal tract with potentially pathogenic properties, which would explain why antibodies to listeria are common in healthy people.[2] Rectal carriage by pregnant women was 10 times higher during an epidemic in New Zealand.[9] In Israel the symptomless carriage of *L monocytogenes* in the genital tract was associated with a history of recurrent abortion,[11] but this was not confirmed in Britain[12] or the United States.[13]

Listeric infection of the fetus during pregnancy is often associated with a non-specific, flu like, pyrexial illness in the mother.[14 15] Decidual microabscesses and characteristic foci of purulent villitis within the placenta have led some to believe that fetal infection is caused by the bloodborne transplacental passage of organisms from the mother[10 16]; severe chorioamnionitis is, however, in keeping with an ascending pathway of infection.[6 17] Fetal infection in early pregnancy results in abortion, and the extensive maceration seen at delivery, so soon after the maternal symptoms, suggests that the maternal bacteraemia may result from rather than be the cause of the fetal infection.[2] Fetal infection later in pregnancy causes stillbirth[15] or preterm labour associated with meconium stained liquor and an infected baby.[6-10]

Congenital listeria infection presents soon after birth as pneumonia and septicaemia, and *L monocytogenes* was isolated from the vagina of 37% of mothers of such infants.[1] The high mortality of 35-55% reflects associated prematurity.[1 6-10] Microabscesses and granulomas containing the organism are found throughout the body at necropsy[18]—particularly in the lungs, liver, and spleen—giving rise to the term granulomatosis infantiseptica.[16] A high rate of disability among survivors has been reported,[19] but this does not always occur.[6 9] Neonatal infection, acquired during delivery from the mother's genital tract[14] or later by cross infection,[1 4] presents as septicaemia and meningitis several days or even weeks after an uneventful birth.[7 8 19] A granulomatous macrocytic inflammation of the meninges may lead to microabscesses in the brain, but this form of neonatal listeriosis has a lower mortality,[7 9] and survivors are unlikely to have neurological sequelae.[9 19]

Treatment of suspected neonatal infection usually begins before firm bacteriological confirmation. The combination of ampicillin or penicillin with gentamicin or kanamycin is more effective against listeria than other agents alone or in combination.[8 20] Listeric septicaemia in the mother during pregnancy has been successfully treated with ampicillin, and such treatment prevented perinatal listeriosis.[10 21] Success in managing this condition depends not so much on considering the diagnosis[10] but more on the degree of fetal or neonatal infection at the time of presentation.[15]

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Make way for the new genetics

Although recombinant DNA technology may not be easy to understand, the new genetics is important for clinical practice.[1] Much better information can be given to potential parents worried about genetic disease, while genetic diseases are increasingly preventable and may eventually be treatable.

The potential is vast and there are so many diseases that are mainly genetic in origin that one in 50 children suffers death or chronic disability from these. Premature coronary artery disease, cancer, and other common diseases also have an important genetic component and are candidates for renewed genetic exploration. For example, recent DNA studies with rare familial Alzheimer's presenile dementia[2] and manic depressive psychosis[3] have introduced a new genetic and biochemical approach to common psychiatric problems. At the present rate all genetic disorders will probably be mapped to their chromosomal location by the year 2000,[4] and most important genes will eventually be sequenced and their products identified.