Importance of IgM determination in cord blood in cases of suspected rubella infection

We report here two cases showing the importance of measuring IgM in the cord blood of immature neonates.

Methods

IgA, IgM, and IgG levels were determined with the radial immunodiffusion method. Sephadex G 200 was used to separate the IgM and IgG fractions in sera. After concentration by pressure dialyses (Amicon filter UM 10) the rubella antibody level was determined. Rubella antibodies were measured with the haemagglutination inhibition reaction, using the Testebur test kit (RIT, Gentwal, Belgium). Briefly 0.2 ml serum was mixed with 0.2 ml buffer solution (pH 6.4) and inactivated. After cooling 0.1 ml pigeon erythrocytes 50+, were added and after incubation the mixture was centrifuged. The liquid was poured off and mixed with 1.1 ml kaolin suspension (25%). After one hour the liquid was separated from the suspension and used as the first serum dilution (1:8) in the inhibition test. From this dilution the succeeding dilutions: 1:16, 1:32, etc. were made. Antigen solution (containing four haemagglutination units) was added to every dilution, and after incubation erythrocyte suspension was added. The test was read after two hours. For the IgM and IgG fractions we used a heparin MNL Cl mixture instead of kaolin.

Case 1

The child was born at 38 weeks gestation, weighed 1780 g, and was 45 cm long. The spleen and liver were enlarged, and a severe thymocytopenia had developed. Immunoglobulin levels in cord blood were: IgA trace, IgM 3.0 g/l and IgG 8.1 g/l. This represented a steep increase in the IgM level (normal 0-2 g/l). Titres of rubella antibodies in IgM and IgG fractions were 1:64 and 1:128 respectively. After four days the child died, and rubella virus was isolated from necropsy organs.

Case 2

This child was also born at 38 weeks gestation, weighed 1760 g, and was 45 cm long, but seemed perfectly healthy. The immunoglobulin values in the cord serum were: IgA trace, IgM 4.1 g/l and IgG 9.0 g/l. Again the IgM level was increased greatly. The titre of rubella antibodies in IgM and IgG fractions were 1:128 and 1:64 respectively. A rubella infection in the child seemed likely. Cultures from nasopharynx and urine, however, remained negative. Rubella titres in the mother’s serum rose from 1:16 in the eighth week of pregnancy (only IgG antibodies) to above 1:512 in the 38th week of pregnancy (also only IgG antibodies).

Comment

The diagnosis of rubella infection in case 1 seemed clear. From the anamnesis we concluded that the infection had occurred in early pregnancy. Case 2 was probably a case of reinfection. A maternal viremia must have occurred, because there were antibodies in the IgG fraction of the cord blood. Or a primary infection might have occurred in the eighth week of pregnancy. The absence of congenital disorders, the absence of exanthema in pregnancy, and the fact that antibodies were found only in the IgG fraction are arguments against this.

Three conclusions can be drawn from these two cases. Firstly, determining the IgM level in the cord blood can be useful in the case of suspected congenital rubella infections. Secondly, if the IgM level is increased in the child’s serum rubella antibodies should be looked for in the IgM fraction. Thirdly, it is doubtful whether a titre of haemagglutinating antibodies of 1:16 offers sufficient protection against reinfection with viremia.

Lepromatous leprosy presenting with swelling of the legs

In the various forms of leprosy, and including the adverse reactions associated with either cell-mediated or humoral (immune-complex) responses, oedema is well recognised, though its cause is not fully understood. We report a case in which seven years after arriving in Britain a Pakistani man was found to have lepromatous leprosy after presenting with swelling of the legs and superficial phlebitis.

Case report

A 50-year-old Pakistani man presented at a varicos vein clinic with swelling of the legs and giving a vague history of varicose eczema and recurrent phlebitis. Examination confirmed pitting oedema of the feet and ankles and tender areas of inflammation along the superficial veins. Although there was neither eczema nor varicose ulceration he was referred to a dermatological clinic by which time he was feverish, looked ill, and had soft-tissue swelling of the nose, cheeks, forehead, and ears. There were tender nodules, about 1 cm in diameter, over the thighs, lower legs, and extensor surface of the forearms, and larger, less well-defined lesions—raised, red, not tender on palpation—on the calves, knee regions, and thighs. Oedema of the legs was unusually firm and reached almost to the knees; the overlying skin had a shiny “mahogany woodgrain” pattern. Nothing abnormal was found on palpation of the peripheral nerves but cotton-wool testing showed anaesthesia of an incomplete “glove-and-stocking” type in the hands and feet. The tincticles were atrophic and painless on pressure. Fresh blood was present in the right nasal vestibule.

From 1970 to 1975 he attended an ear, nose, and throat clinic on several occasions with deafness and ear inflammation, and in the year before diagnosis he developed nasal blockage and epistaxis, the latter needing cauterisation and packing on several occasions.

Ziehl-Neelsen staining of slit-skin smears from ears, cheeks, and arm and leg lesions disclosed numerous Mycobacterium leprae. Nasal smears and nose-blow material were strongly positive for acid-fast bacilli, and biopsy specimen confirmed the diagnosis of lepromatous leprosy.

The type of leprosy had been judged to be lepromatous with features of immune-complex reaction, as evidenced by erythema nodosum lepromin in the skin, increased erythrocyte sedimentation rate, fever, malaise, pain, and