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VERTICAL TRANSMISSION OF HUMAN IMMUNODEFICIENCY VIRUS M Bosch, I Puig, M Bonastre, O Altirriba, N Margall, G Verges, J Cubells. Hospital 'St Creu i St Pau, Barcelona, Spain.

Between November 1985 and January 1988, a total of 18 children born to HIV-infected mothers were identified. Seventeen of these pregnant women were IV drug abusers, one was a sexual partner of a high risk male and four of them were also prostitutes. All 18 women were symptom-free during pregnancy and only two developed HIV infection. Nine children were delivered by caesarean section and nine vaginally. All newborns were HIV-seropositive by ELISA and Western-blot at birth. Seven children had lost antibody at a median time of nine months, were negative for HIV antigen and remained asymptomatic at a mean age of 20 months. Six children were HIV-antibody positive at a median age of 7.3 months but were HIV-antigen negative and were clinically well. Four children developed HIV infection; two of them had serum HIV-antigen. Another child remained seropositive at 24 months and a fourth patient had persistent HIV-antibody together with a cellular and humoral immunodeficiency and clinical stage of P-2-A. Of these four HIV-infected children, only two were delivered by caesarean section and none of them were breastfed. One child did not come to follow-up control. Prevalence of vertical transmission of HIV from infected mothers to children in our seroepidemiological study was 23.5%, however this percentage may become higher if closer contact of all high risk pregnant women is achieved. In our small series, neither the mode of delivery, nor breast-feeding, were related to vertically transmitted HIV infection.

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KAWASAKI SYNDROME IN BERNE, SWITZERLAND, 1977-1986; REVIEW OF 26 PATIENTS. Urs B Schaad, Karin Odermatt, JW Weber and FP Stöcker. Department of Pediatrics (Inselhospital), University of Berne, CH-3010 Berne, Switzerland.

The purposes of this retrospective study of the 26 patients (17 male (67%), 9 female (35%); mean age SD = 2.8 2 years) treated for Kawasaki syndrome at our centre since 1977 were to describe their clinical and laboratory presentation, and to evaluate high-risk factors for the development of coronary arterial complications and effects of different therapies in preventing the coronary involvement. The diagnostic criteria were met by 100% for fever and rash, by 96% for digital desquamation, by 92% for bilateral conjunctival injection and mucous membrane changes, by 81% for cervical lymphadenopathy, and by 54% for peripheral edema/erythema. Treatment included aspirin, in the beginning also antibiotics, and since 1986 high-dose intravenous immune globulins (IVIG). Regular routine electrocardiogram and echocardiogram controls recorded 9 cardiovascular complications: 6x carditis (23%) and 3x coronary artery aneurysm (12%). The 3 patients with coronary aneurysm were all males, and compared to the others both younger (22 vs. 33 mths) and febrile over a longer period of time (22 vs 12 dys); elevated acute-phase reactants such as ESR, leukocyte and platelet counts were similar in both groups and did not affect prognosis. Since the therapeutic use of IVIG no more cardiac complications were detected. All patients are clinically cured. The findings of carditis in 6 and of coronary aneurysm in 2 patients completely resolved; one patient with regressive coronary aneurysm shows myopathic cardiomegaly after 2 yrs.

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KAWASAKI SYNDROME, TREATMENT WITH INTRAVENOUS GAMMAGLOBULINS J.C. Borderon, Y. Lebranchu Centre de Pédiatrie Gatien de Clocheville 49, Bd Beranger 37000 TOURS (France)

Twenty eight infants and children (3-63 months) with Kawasaki disease received intravenous immunoglobulins (IVIG) in a multicentric study: Veinoglobulin (Merieux, France) in 24 cases or Sandoglobulin (Sandoz, Switzerland) in 4 cases. The total amount administered was in the range 1-2.5g/Kg and varied from 1 to 5 daily infusions. In 16 cases the patients received 0.4 mg/Kg/day during 5 consecutive days. All of them were given orally acetylsalicylic acid 30 mg/Kg/day during a 2 month period. A dramatic improvement in the patients' general condition was observed, with resolution of fever within 48 hours. Hyperfibrinemia and hyperleucocytosis returned to normal in one week and increased platelets within 2-3 weeks. An important feature was that follow-up echocardiograms over more than one year have demonstrated no coronary artery abnormalities. Five out of 6 children studied immunologically during the acute phase presented with a deficiency of circulating CD8 T cells with an increased CD4/CD8 ratio. High doses of IVIG were followed with an increase of serum IgM, CD8 T cells and a normalisation of CD4/CD8 ratio. High dosage IVIG represents the treatment of choice of Kawasaki syndrome; this activity focuses the deficiency of the immunoregulatory function in Kawasaki syndrome.

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INTRAVENOUS IMMUNOGLOBULIN IN KAWASAKI DISEASE Eeva Salo. Children's Hospital, University of Helsinki, SF-00290 Helsinki, Finland.

In November 1984 Furusho et al published their results suggesting that IV IGG prevents coronary artery aneurysm formation in Kawasaki Disease. These results were confirmed in an open controlled multicenter study by Newburger et al in 1986. In Finland new cases of KD have been registered since 1981. To determine the value of IV IGG in Finnish KD patients the records of patients registered in 1985-87 were reviewed. Seventy-one patients were registered. Data on both treatment and findings of coronary echocardiography were available on 63 patients. Coronary aneurysms were detected in 3/44 patients treated with aspirin + IV IGG and 3/19 patients treated with aspirin only. Two of the untreated children with aneurysms fulfilled only 4 of the 6 criteria of KD. These data are in accord with the suggested benefit of IV IGG in preventing aneurysm formation in KD. When treating Kawasaki patients with IV IGG we are facing the following problems:

- patients with diseases other than KD will be treated;
- KD patients not fulfilling the criteria will not be treated or their treatment will be delayed;
- an occasional patient with IgA deficiency will be treated;
- the final diagnosis may be obscured as the treatment modifies the clinical picture and invalidates the results of bacterial and viral serology.

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C-REACTIVE PROTEIN IN KAWASAKI DISEASE J. Bouilloche,\* E. Mallet,\* F. Pollet\*\*et al.\*\*\* Collaborative study from EVREUX (\*\*), LISEUX (\*\*\*) ROUEN (\*) Children's Hospitals - France.

C- reactive protein (CRP) levels were investigated in 8 cases of Kawasaki disease (KD) all treated with high doses of intravenous gammaglobulin (400 mg/kg) for 3 days). Medium initial value of CRP was 190 mg/100 ml (range 129-284). The delay between the onset of the disease and treatment was 12.5 days (5-24). In all cases CRP normalized dramatically with a mean delay of 3.5 days (range 0-7dys) after the end of treatment. This biologic parameter normalized before other inflammatory tests, such as erythrocyte sedimentation rate, platelet count, fibrinogen and IgE. Both apyrexia occurred and skin lesions evolution stopped. One patient with coronary artery involvement was treated after a delay of 24 days when cardiac lesions were already present. CRP value was normal 24 hours after the onset of treatment and coronary lesions stabilized. Monitoring of CRP appears to be an accurate index in clinical practice. Furthermore dramatic normalization of CRP should be considered of interest in immunoglobulin efficacy and in indirect approach of KD pathophysiology.

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AN OUTBREAK OF BELLS'S PALSYP CAUSED BY BORRELIA BURGdorFERI IN HUNGARY. Andras Lakos, Agnes Herczegfalvi, Eva Veress Central Hospital for Infectious Disease and Heim Pal Children's Hospital, Hungary

The first LGB patients in Hungary were diagnosed in 1984, and a few more in 1985. But only following the introduction of serological tests (indirect immunofluorescence) in 1986 that interest in the illness was aroused. During the last two years more than 2000 sera and CSF samples from 1400 patients have been tested for Borrelia burgdorferi antibody. 92 of 1400 patients suffered from peripheral facial palsy. 35/92 were found to be serologically positive. 19 of them were seen during the summer and fall of 1987. In this year twice as many Bell's palsy patients were seen in our hospitals than in previous years. Of the 35 clinically and serologically proven cases: 14 showed ECM; 10 had other neurological manifestations (Bannwarth's syndrome); 1 had frank arthritis; 1 had carditis. 18/33 seropositive Bell's palsy patients were children. The clinical picture, epidemiology, serology and CSF findings of these cases will be discussed. Almost all of the seropositive patients became free of symptoms. The longest period of recovery was 100 days. Residual symptom was found only in those patients that remained seronegative during a minimum of 45 days serological follow-up period or did not receive adequate parenteral antibiotic therapy. Half of the seropositive patients had lymphocytic meningitis. Only one seronegative case had pleocytosis in the CSF.