Initial management, however, should vary with local experience and changing patterns. For example, New Zealanders of Caucasian and Maori origins have comparably high rates of testicular tumours,[5] and for them orchidectomy should be used. But in the USA, where different races have differing rates of testicular cancer,[1] management varies.

[1] Mostofi, F K, and Price, E B, *Tumors of the Male Genital System*. Washington, DC, Armed Forces Institute of Pathology, 1973.
[2] Jalota, R, Middleton, R G, and McDivitt, R W, *Cancer (Philadelphia)*, 1974, **34**, 464.
[3] Doll, R, *Proceedings of the Royal Society of Medicine*, 1972, **65**, 49.
[4] Burkitt, D P, *Proceedings of the Royal Society of Medicine*, 1973, **66**, 312.
[5] Petersen, G R, and Lee, J A H, *New Zealand Medical Journal*, 1973, **78**, 401.

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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.[1 2] After concentration by pressure dialyses (Amicon filter UM 10) the rubella antibody level was determined. Rubella antibodies were measured with the haemagglutination inhibition reaction,[3] using the Testerub test kit (RIT, Genval, 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 $MnCl_2$ 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 thrombocytopenia 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 at necropsy from different 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.[4 5] A maternal viraemia must have occurred, because there were antibodies in the IgM 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 viraemia.

[1] Gupta, J D, *et al, Journal of Clinical Pathology*, 1971, **24**, 547.
[2] Snijder, J A M, *Nederlandsch Tijdschrift voor Geneeskunde*, 1974, **26**, 995.
[3] Peetermans, J, and Huygelen, C, *Presse Médicale*, 1967, **75**, 2177.
[4] Haukenes, G, and Haram, K O, *New England Journal of Medicine*, 1972, **287**, 1204.
[5] Pead, P J, *Lancet*, 1973, **2**, 149.

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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 varicose 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 testicles 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 specimens from an arm nodule, left ear lobe, and left nostril showed typical lepromatous histopathology. Two of these biopsy specimens, however, also showed areas of vasculitis and infiltration by polymorphs. A lepromin test gave negative results at four weeks.

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

swelling of the legs. He was treated with rifampicin and dapsone from the outset, his reaction being controlled by prednisolone and thalidomide. Progress was uneventful, and nine weeks later leg oedema had virtually disappeared, superficial veins were normal, and he could breathe freely through his nose for the first time in several years.

Comment

This case is reported in detail because experience shows that in non-endemic areas, including Britain,[1] patients may present in extraordinary ways, often at special clinics, and with long delay before the diagnosis of leprosy is considered. The causes of brawny oedema of the legs in lepromatous leprosy almost certainly embrace factors concerned in non-specific panniculitis,[2] including cold, gravity, infection, stasis, and the unusual arrangement for the blood supply of subcutaneous fat. But additional factors may include (*a*) damage to autonomic nerves, causing increased capillary permeability[3]; (*b*) heavy parasitisation of endothelial lining cells by the bacillus; and (*c*) damage to blood vessel walls during immune-complex reactions.

Swelling of the hands and feet in leprosy may repay further study; the recent discovery[4] that after long periods of treatment for lepromatous leprosy the fingers may be the skin site with the highest bacterial load and the highest number of solid-staining bacilli originated in a clinical discussion on the significance of oedema in the fingers.

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[1] Powell, S, and McDougall, A C, *British Medical Journal*, 1974, **1**, 612.
[2] Barták, P, *British Journal of Dermatology*, 1970, **82**, Suppl 5, p 15.
[3] Jopling, W H, personal communication, 1976.
[4] Ridley, M, Jopling, W H, and Ridley, D S, *Leprosy Review*, 1976, **47**, 93.

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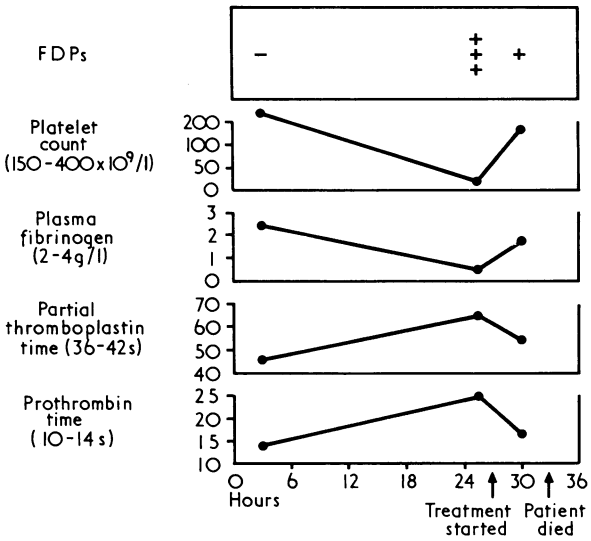
Dissecting aortic aneurysm and disseminated intravascular coagulation

Disseminated intravascular coagulation has numerous diverse causes.[1] Large clots, as in abruptio placentae and giant cavernous haemangioma, occasionally result in this syndrome. Severe disseminated intravascular coagulation is very rarely reported in acute aortic dissection.[2]

Case report

A 62-year-old woman with a history of hypertension was admitted to hospital as an emergency with acute pulmonary oedema. The previous day she had experienced mild retrosternal pain radiating to both arms. Examination showed acute heart failure with an early diastolic murmur maximal at the left sternal edge. All pulses were present and equal. The chest *x*-ray film showed widening of the aortic shadow in addition to pulmonary oedema. There was left ventricular hypertrophy and general T-wave flattening on the electrocardiograph. An arch aortogram showed a small false lumen in the media of the ascending aorta, without involvement of the major aortic branches. Surface scanning after intravenous ^{131}iodine-labelled fibrinogen showed a large clot in the false lumen. Conservative hypotensive therapy was instituted with methyldopa and guanethidine. Twenty-four hours later petechiae and bleeding from venesection and intravenous infusion sites were noted. The haemoglobin was 8·9 g/dl. Schistocytes and microspherocytes were seen on the blood film. The figure shows other coagulation parameters. The aspartate transaminase was 2400 IU/l (normal 10-30). Bleeding persisted



Results of coagulation studies. Normal ranges are given in parentheses.

and low-dose heparin (5000 units subcutaneously twice daily) treatment together with clotting factor and platelet supplements was begun. The bleeding diathesis improved over the next six hours (figure), but she then developed cardiac tamponade, collapsed, and died three hours later. Necropsy showed a 0·5-cm tear in the posterior wall of the ascending aorta and a 440-g clot containing ^{131}iodine-labelled fibrinogen in the false lumen. Aneurysmal rupture into the pericardium had occurred. The liver was intensely congested and microscopic examination showed considerable centrizonal necrosis. Fibrin clots were found in the microvasculature of the liver and kidneys.

Discussion

Aortic aneurysm is an unusual cause of disseminated intravascular coagulation,[2] only two cases having been reported.[3 4] Abruptio placentae and giant cavernous haemangioma may also cause disseminated intravascular coagulation. The common denominator in all these conditions is the formation of a large extravascular blood clot, as seen in our patient.

The aetiology of disseminated intravascular coagulation may be classified into three processes[1]: (1) vascular endothelial cell damage, which activates Hageman factor and the intrinsic clotting system; (2) tissue injury, which activates extrinsic clotting; and (3) red-cell or platelet injury with the release of coagulant phospholipids. Probably all three processes were active in this patient. Large clots alone, as in abruptio placentae,[5] do not cause consumption coagulopathy: probably thromboplastic substances are released, initiating generalised intravascular coagulation as shown in this patient by the presence of fibrin clots in the microvasculature of both liver and kidneys. A further factor was probably liver injury, which, though rarely the sole cause of disseminated intravascular coagulation, is often a contributory aetiological factor.[1]

The definitive treatment for disseminated intravascular coagulation is removal of the cause: nevertheless, the administration of clotting factors, platelets, or heparin may be of temporary benefit.[1] Heparin therapy, although hazardous, was used with good results in the patient of Bieger *et al* as a prelude to surgery. In our patient low-dose heparin used together with clotting factors and platelets was beneficial and did not apparently contribute to her death.

[1] Colman, W R, Robboy, S J, and Minna, J D, *American Journal of Medicine*, 1972, **52**, 679.
[2] Merskey, C, Johnson, A J, and Kleiner, G J, *British Journal of Haematology*, 1967, **13**, 528.
[3] Bieger, R, *et al*, *New England Journal of Medicine*, 1971, **285**, 152.
[4] Fine, N L, *et al*, *Archives of Internal Medicine*, 1976, **119**, 522.
[5] Prichard, J A, in *Treatment of Haemorrhagic disease*, ed by O D Ratnoff. p 175. New York, Harper and Rowe, 1968.

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