Twenty-four hours after admission the patient showed moderate signs of meningeal irritation. He remained comatose for four days, then regained consciousness, but showed psychomotor agitation, mental confusion, and visual hallucinations. A gradual improvement was observed in the following days, and 20 days after admission he was discharged clinically recovered. Among virological tests, carried out by the virological service of the infectious and tropical diseases department, the haemagglutination-inhibition test for rubella, carried out on blood samples taken during the 20 days' stay in hospital, showed a clearcut increase of the antibody titres from 1/512 to 1/2048. The complement fixation test results for measles, mumps, adenovirus, and herpes were negative. Lumbar puncture was carried out 24 hours after the onset of neurological symptoms. The CSF was inoculated into SIRC cells and monkey kidney cells at the patient's bedside. No cytopathic effects could be shown on monkey kidney cells, whereas the culture on SIRC cells showed cytopathic effects. A subculture was carried out on RK 13 and four days later a clear positive cytopathic effect was evident.

The virus was identified by the neutralisation test on RK 13 using a specific antirubella immunoserum diluted at 10^{-1} and 10^{-2} titre.

Comment

The failure of previous attempts at isolating the virus from the CSF in acquired rubella encephalitis contrasts with the 33%-positive isolation in congenital rubella. This may be due to the longer lifetime of the virus in the nervous system in cases of congenital rubella,[3] but we should like to emphasise the relevance of technical problems. Most previous attempts at isolating the virus have been carried out under unfavourable technical conditions, such as shipment of the specimen, lack of appropriate cells for culture, collection of specimen in advanced stages of the disease, and when the presence of high specific antibody titre may interfere with recovery of the virus.[1]

A successful isolation of the virus from the CSF in acquired rubella requires not only specific cell lines (SIRC, RK 13, and Vero) but also the immediate inoculation of CSF into the cells. The location of a virology laboratory within the hospital (as occurs here) makes it easy to comply with this requirement.

The isolation of the virus from CSF, apart from its virological interest, may be relevant for understanding the pathogenesis of acquired rubella virus encephalitis. Direct viral invasion of the central nervous system has been considered as an alternative to activation of a latent encephalotropic virus or to immunologic processes as the mechanism underlying encephalitis.[4] The recovery of rubella virus in CSF supports the view that direct virus invasion of the central nervous system does in fact occur.

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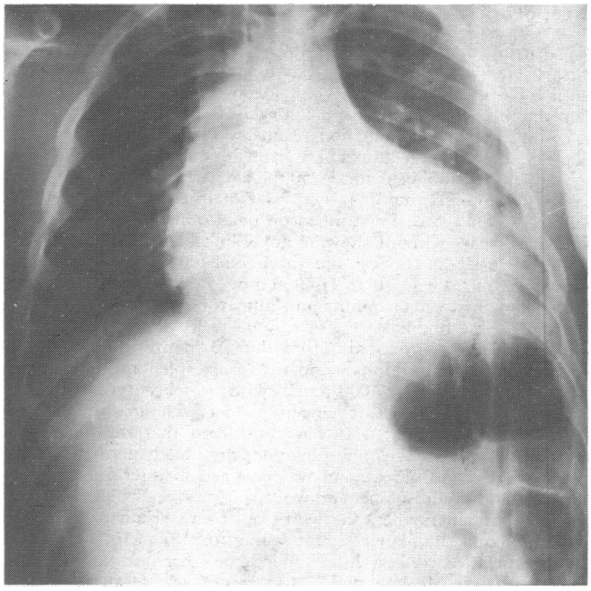
Meningococcal pericarditis without meningitis

The number of reported cases of meningococcal pericarditis without clinical evidence of meningitis is very small. We report a further case additionally complicated by peritonitis, arthritis, and urethritis.

Case report

The patient was a 14-year-old Negro schoolboy. Five weeks before admission he spontaneously developed painful swelling of his right knee, lasting five days before resolving completely. The night before admission he suffered a rigor and woke with severe retrosternal chest pain exacerbated by respiration and movement. He also admitted to generalised abdominal pain and to a painful right ankle. He denied any headache or neck stiffness. He and his family were living in council accommodation in East London. Neither he nor his family had travelled abroad in the previous year.

He was distressed and dyspnoeic with an oral temperature of 38°C. No rash, lymph node enlargement, or abnormality of the mucous membranes was detected. Chest examination showed left basal dullness and bronchial breathing (see figure). The pulse was regular at 120/min with a lying blood pressure of 110/70 mm Hg; the venous pressure was raised 3 cm without paradoxical respiratory change; and the apex beat displaced 2 cm. A loud pericardial rub was audible. The abdomen was tense with guarding and reduced bowel sounds. The right ankle was swollen, hot, and tender. Neurological examination showed no abnormalities and, in particular, there was no neck stiffness.



Chest radiograph on admission showing a large pericardial effusion.

Initial investigations showed haemoglobin: 14·9 g/dl; white cell count: $15{\cdot}7 \times 10^9$/l (15 700/mm^3) (93% neutrophils); repeated blood cultures: negative results on days 1 and 2 of admission; chest radiography: enlargement of the heart with collapse and consolidation of the left lower lobe; and electrocardiogram: widespread ST elevation consistent with acute pericarditis.

In the next 12 hours he deteriorated with increasing dyspnoea, development of pronounced pulsus paradoxus, and further raising of venous pressure. Percutaneous needle aspiration was performed to relieve the tamponade and 150 ml of serosanguinous fluid obtained. Gram stain showed profuse polymorphs with intracellular Gram-negative diplococci. Culture grew *Neisseria meningitidis*, identified by fermentation reactions and by positive agglutination with meningococcal type C antisera. Disc diffusion methods showed sensitivity to penicillin, cephaloridine, and sulphafurazole. A urethral smear on the second day showed scanty pus cells and a few intracellular diplococci, which failed to grow on culture. Further questioning disclosed no history of dysuria, urethral discharge, or sexual activity. Needle aspiration of peritoneal fluid also disclosed a pronounced neutrophil leucocytosis but without identifiable organisms. Culture of postnasal swabs from the patient and his family failed to show meningococci.

After pericardial aspiration he received parenteral cephaloridine, 1 g four times a day (because of penicillin sensitivity), and sulphadimidine, 1 g four times a day, but within 24 hours tamponade recurred. An indwelling percutaneous pericardial drain was therefore inserted and 1500 ml of pus drained during the next five days. After this he made an uneventful recovery and remains clinically normal six months later.

Comment

Neisseria meningitidis infection has widespread manifestations, including urethritis,[1] arthritis, and pericarditis. Pericarditis accompanying meningococcal meningitis is uncommon but recognised. Pericarditis occurring as the major feature of a meningococcal infection without clinical meningitis is much rarer, and we have found only nine such cases reported.[2–5]

Pericarditis has been considered to be a later manifestation of meningococcal infection resulting as part of a "polyserositis" from a supposed hypersensitivity to the organism or to endotoxin.[4] Others,

conversely, have reported it as the presenting feature of the disease after direct pericardial infection.[3]

In our case the history of arthritis might suggest a preceding meningococcal illness with pericarditis, arthritis, and peritonitis as later complications. The undoubted presence of organisms in the pericardial fluid, however, implies a direct bacterial mechanism.

Corticosteroids have been advocated to aid resolution of the serous effusions of meningococcal infection. Our patient, however, responded satisfactorily to antibiotics and drainage alone. Effective eradication of the organism is obviously the object of treatment and whether steroids offer further benefit seems still open to question.

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The unsupported arm: a cause of falsely raised blood pressure readings

Many technical errors can occur in measuring blood pressure with a sphygmomanometer.[1 2] We have noticed that many doctors, while measuring blood pressure in a sitting or standing patient, fail to support the arm being measured. Indeed, a survey of 40 doctors in our hospital showed that only five supported the arm. To obtain an accurate blood pressure measurement the cuff must be at heart level.[2] In the sitting or standing position this means that the upper arm must be extended forward to an angle of 45°. A person holding his arm in this position without support is undergoing isometric exercise, which causes an appreciable increase in blood pressure and heart rate.[3-5] We studied the effects of the arm being unsupported on the blood pressure in normal volunteers.

Subjects, methods, and results

Twenty normal adults (10 men and 10 women) aged 25 to 60 were studied. Their blood pressures were measured by an automated blood pressure device (Arteriosonde 1217-Roche), which uses the ultrasound principle. All blood pressures were measured in the left arm, the upper part of which was extended forward 45° so that the cuff was at heart level. The forearm was parallel to the ground. Three studies, each of which were divided into three stages, were performed. In each study the subject was seated. In the first stage the blood pressure cuff was placed on the left arm, which was then placed on a support with the cuff at heart level, and the patient was allowed to sit quietly for three minutes. During the next four minutes (stage 2) the patient continued to sit quietly but the blood pressure and heart rate were measured every minute. The mean of these four measurements was taken as the control blood pressure. The test period (stage 3) then followed for two minutes: the blood pressure was measured at 30 and 90 seconds and the heart rate every minute. In the first study the left arm was supported at heart level for these two minutes; in study 2 it was unsupported for the two minutes; and in study 3 it was supported but the right arm was raised and was unsupported. The purpose of the third study was to assess the effect on the blood pressure of isometric contraction in the arm that was not being measured.

The results are shown in the table. The blood pressure and heart rate rose moderately when the arm was unsupported (study 2). In particular, the diastolic pressure measured 90 seconds after the arm was left unsupported increased by 10·6% over the control value.

Comment

Both heart rate and blood pressure showed the greatest increase when the left arm was unsupported. The diastolic pressure rose by up to 10·6%, which may be enough to cause a patient to be considered as hypertensive. Such an increase may also influence the amount of treatment being given.

The change in blood pressure that occurs during isometric exercise is due to a combination of cardioacceleration (due to both reduced vagal and increased alpha-adrenergic stimulation) and increased peripheral resistance (due to increased alpha-adrenergic stimulation).[3] The increase in blood pressure after isometric exercise is greater in hypertensive patients than in normotensive ones[4 5] and is even more exaggerated after treatment with beta-blocking drugs.[5] Clearly, therefore, great care must be taken in measuring the blood pressure to avoid the isometric exercise that occurs when the arm is left unsupported.

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Acute infectious lymphocytosis as a T-cell lymphoproliferative syndrome

Acute infectious lymphocytosis, known in France as maladie de Carl Smith[1] because of xenophilia, is an illness (most probably viral) affecting children from 1 to 14 years of age, predominantly young infants. Symptoms are variable but include upper respiratory tract infections, diarrhoea, abdominal pain, and rashes. Lymphadenopathy and splenomegaly are always absent. The ill-defined symptoms contrast with an appreciable peripheral blood lymphocytosis varying from 40 000 to 100 000 $\times 10^9$/l with a normal haemoglobin and platelet count. We have recently determined the nature of this lymphoproliferation in term of T, B, or null cells.

Case report

A 16-month-old infant, of consanguinous parents, who had been seen at the department of paediatrics for repeated infections, was admitted to hospital with fever and diarrhoea. The result of clinical examination was normal. Initial blood count showed white cells 16·9 $\times 10^9$/l, with 30%

Mean blood pressure and heart rate (±SE of mean) during 4-minute control period and 2-minute test period in each study

	Blood pressure in control period (mm Hg)		Change in systolic pressure in test period (mm Hg)		Change in diastolic pressure in test period (mm Hg)		Heart rate in control period (beats/min)	Change in heart rate in test period (beats/min)	
	Systolic	Diastolic	30 s	90 s	30 s	90 s		30 s	90 s
Study 1	109·6 ± 2·40	72·6 ± 1·24	0 ± 1·22	+ 0·35 ± 1·2	+ 0·05 ± 0·56	+ 1·30 ± 0·92	73·65 ± 1·63	− 0·35 ± 0·60	+ 0·55 ± 0·68
Study 2	111·8 ± 1·74	73·6 ± 1·78	− 1·25 ± 1·74	+ 2·25* ± 1·74	+ 4·95 ± 1·28	+ 7·80* ± 1·13	73·3 ± 2·19	+ 4·45* ± 0·80	+ 5·25* ± 0·85
Study 3	109·3 ± 3·02	73·10 ± 1·45	+ 3·70* ± 1·09	+ 5·95* ± 1·28	+ 3·95* ± 0·83	+ 5·15* ± 1·39	73·6 ± 1·72	+ 0·9 ± 0·87	+ 2·85* ± 0·73

*P < 0·05.