titre of 1/8, which did not rise. Chlamydia were not isolated from cerebrospinal fluid or urethral swabs after cefotaxime was started.

Dexamethasone was given to reduce cerebral oedema, which improved over three weeks, his mental state returning to normal. He was unable to pass urine spontaneously, however, and remained slightly feverish. Cefotaxime was stopped after one week when there was no bacterial growth in blood, cerebrospinal fluid, or urine. After a two-week course of oxytetracycline by mouth his fever settled and he passed urine normally on removal of the catheter.

Comment

Microimmunofluorescence antibody tests for *Chlamydia* are replacing complement fixation tests in diagnosis as they are type specific. The tests are simplified by pooling the serotypes for trachoma, urethritis and inclusion conjunctivitis, lymphogranuloma venereum, and psittacosis.[1] Our patient's rising IgG titre was strong evidence of recent infection. IgM is short lived, and its absence does not exclude current infection.[2] IgG titres in genitourinary infection rarely exceed 1/64.[3] Our patient's titre was 1/1024, which suggests systemic infection. Meningoencephalitis was shown by clinical findings and results of lumbar puncture and computed tomography. As other possible causes were excluded we would suggest that this was due to *C trachomatis*. Three similar cases have been reported from Sweden.[4 5] We believe that *C trachomatis* merits serious consideration as an aetiological agent in encephalitis in sexually active adults.

We thank Dr J D Treharne, Institute of Ophthalmology, London, for performing the microimmunofluorescence tests and for helpful advice.

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(*Accepted 2 November 1982*)

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Capillary flow velocity in leukaemia

With the growth of interest in the rheological properties of blood attention has centred on the deformability characteristics of the red cell. In-vitro measurements, however, suggest that white cells are considerably less deformable than red cells,[1] a concept supported by the leucocyte plugging temporarily blocking capillary flow that has been described in several preparations, including human pedicle skin graft.[2] In disease states in which the number of white cells is increased or white cells are stiffer than usual, both conditions that may prevail in leukaemia, this phenomenon of leucocyte plugging may become clinically relevant.[3] We therefore studied capillary flow characteristics in unselected patients with leukaemia with raised peripheral white counts using a non-invasive technique.

Patients, methods, and results

We studied six patients. Four had chronic granulocytic leukaemia, one had follicle centre cell lymphoma in leukaemic phase, and one had acute lymphoblastic leukaemia. All patients were studied on two occasions, the second study visit being after a spontaneous rise in white cell count (cases 3 and 5) or a fall in white cell count after chemotherapy (cases 1, 2, 4, and 6). Vascular studies were performed under identical conditions at both study visits after acclimatisation in a room at constant temperature. Capillary flow was assessed in the finger nailfold microcirculation by television videomicroscopy.[4] Velocity of capillary blood flow was determined by frame to frame analysis of the video record by measuring the distance moved by plasma gaps or red cells in recorded time. A minimum of 20 such estimations from five different capillaries were used to derive the mean velocity. The proportion of stationary vessels was estimated by recording the number of capillaries visible in a stationary frame and assessing the proportion then observed to be flowing when the record was played. Total finger arterial inflow was determined by mercury strain gauge plethysmography (Periflow, Janssen Scientific Instruments).

The table gives the results. In all patients the velocity of capillary blood flow was lower in both arterial and venous limbs of the capillary loop at the time of highest count ($p < 0.05$, Wilcoxon's signed rank sum test). The absolute (mean ± SEM) capillary blood flow velocity at the time of highest count (0·20 ± 0·06 mm/s in the arterial limb, 0·11 ± 0·04 mm/s in the venous limb) was considerably lower than normal values recorded under these conditions in our laboratory (0·92 ± 0·42 mm/s in the arterial limb, 0·56 ± 0·28 mm/s in the venous limb). Five patients showed a greater proportion of stationary vessels at the time of highest count. In contrast, plethysmographic studies showed no consistent relation between total arterial inflow and white cell count. In five patients arterial inflow was normal or high at the time of highest count despite the coexistence of stationary capillaries.

Comment

Preston *et al* described the occurrence of neurological symptoms in patients with leukaemia attributable to cellular hyperviscosity.[5] Our study is the first description of subclinical capillary hypoperfusion associated with leukaemia. Interestingly, conventional assessment of blood flow using plethysmography gave no indication of the limitation of the nutritional capillary circulation. The limitation of capillary flow probably represents partial plugging of capillaries with large numbers of poorly deformable white cells. Television videomicroscopy promises to be a valuable technique for the study of blood rheological behaviour in vivo as opposed to in a viscometer. Our results serve to emphasise that the white cell may be of greater rheological importance than has previously been appreciated.

We thank the British Heart Foundation for research grant number 80/58; and Dr J A Child and Dr B E Roberts for permission to study patients under their care.

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Capillary blood flow velocity (CBV) and results of plethysmography related to white cell count in six patients with leukaemia

Case No	Age (years) and sex	Diagnosis	Highest white cell count: White cell count (× 10⁹/l)	CBV in arterial limb (mm/s)	CBV in venous limb (mm/s)	% of stationary vessels	Finger arterial inflow (ml/100 ml/min)	Lowest white cell count: White cell count (× 10⁹/l)	CBV in arterial limb (mm/s)	CBV in venous limb (mm/s)	% of stationary vessels	Finger arterial inflow (ml/100 ml/min)
1	73 F	CGL	300	0·23	0·11	5	13·0	10	0·56	0·16	0	23·6
2	66 F	CGL	290	0·08	0·02	50	2·6	5	0·19	0·10	0	1·4
3	29 F	CGL	109	0·46	0·28	0	26·4	83	1·13	0·48	0	11·2
4	58 F	CGL	164	0·18	0·11	15	10·8	54	0·54	0·47	0	13·9
5	57 M	FCCL	900	0·03	0·03	25	17·8	766	0·19	0·12	12	5·4
6	19 F	ALL	325	0·20	0·12	50	11·7	90	0·44	0·30	0	6·8

CGL = Chronic granulocytic leukaemia. FCCL = Follicle centre cell lymphoma. ALL = Acute lymphoblastic leukaemia.

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(*Accepted 1 November 1982*)

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Severe myocardial ischaemia induced by intravenous adrenaline

The dose of adrenaline usually recommended for the treatment of anaphylactic shock is 0·3 mg administered subcutaneously: an additional 0·1 mg may be given intravenously, but intravenous doses in excess of 0·1 mg are normally reserved for cardiac emergencies.[1] We describe a case of severe myocardial ischaemia caused by 0·3 mg adrenaline given intravenously.

Case report

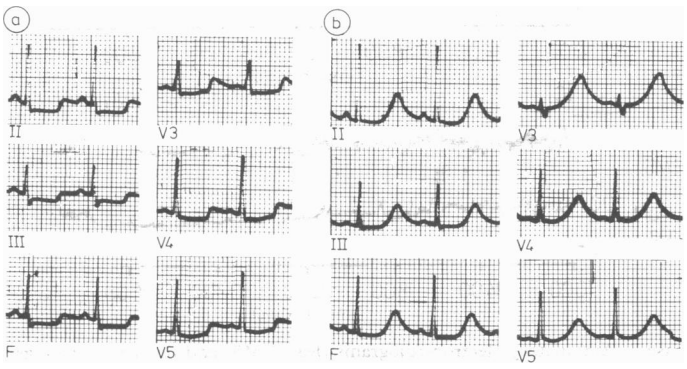
A 23 year old Asiatic woman presented to her doctor with acute pharyngitis. After an intramuscular injection of 600 000 units procaine penicillin she became pale and felt dizzy. Her pulse rate was 120 beats/min and blood pressure 110/70 mm Hg. Suspecting an anaphylactic reaction, the doctor administered betamethasone 4 mg intravenously, promethazine 25 mg intramuscularly, and adrenaline 0·3 mg intravenously. Immediately thereafter she developed severe retrosternal chest pain associated with sweating and dizziness. Sublingual nitroglycerine afforded no relief.

She presented to the emergency unit some five hours later, still suffering from chest pain; pulse rate was 105 beats/min and blood pressure 140/80 mm Hg. Jugular venous pressure was not raised, and her chest was clear. The apical impulse was normal in position and contour, and normal heart sounds with a grade 3/6 late systolic murmur maximal at the fourth left intercostal space were heard at auscultation. Electrocardiography (figure a) showed sinus tachycardia, a normal PR interval and axis, and extensive 1·5 mm planar ST-segment depression while the QT interval corrected for rate was prolonged (0·62 s): this reaction has been reported after administration of adrenaline.[2] Total plasma calcium and protein concentrations measured at the same time were within the normal range.

She was admitted to the coronary care unit, and the chest pain continued to be severe despite treatment with sublingual nitroglycerine (1·5 mg) and oral nifedipine (60 mg). The pain was relieved by an intravenous infusion of nitroglycerine (50 μg/min), with a simultaneous decrease in the degree of ST-segment depression. Subsequent electrocardiograms showed progressive normalisation of the ST segments and the development of tall, peaked T waves in the chest leads (figure b).

Her condition remained stable after her pain had been relieved, and the nitroglycerine infusion was subsequently stopped. Serial estimations of the creatinine phosphokinase MB fraction were all within the normal range.

Results of a submaximal effort stress test incorporating a multigated equilibrium blood pool scan were normal. M mode and two dimensional echocardiography disclosed normal left ventricular wall motion and size with evidence of mild prolapse of the mitral valve. No risk factors for ischaemic heart disease could be found. She remained asymptomatic with a normal electrocardiogram six months later.



Electrocardiogram at time of admission (a) and after administration of intravenous nitroglycerine (b).

Comment

Typical ischaemic chest pain with appropriate electrocardiographic changes in a young woman with no risk factors for ischaemic heart disease after intravenous injection of adrenaline indicates that the adrenaline was almost certainly the cause of the subsequent myocardial ischaemia. This supposition was further substantiated by the results of subsequent investigations, which largely excluded the presence of preexisting ischaemic heart disease. The mechanism of myocardial ischaemia lasting for more than five hours after a single bolus of adrenaline is unclear.

The ability of catecholamines to cause myocardial damage is well established, and these hormones have also been implicated in the myocardial necrosis occurring in patients with phaeochromocytoma.[3] The mechanism is thought to be either a direct effect on the myocardial cell or myocardial damage resulting from ischaemia caused by, among other things, constriction of the coronary artery. An additional mechanism whereby adrenaline may cause myocardial ischaemia is localised coronary artery spasm, as suggested by evidence showing that adrenaline added to strips of large coronary arteries increases the tension through a mechanism susceptible to alpha-antagonistic agents,[4] which reverse coronary artery spasm in some patients.[5]

This report emphasises the potential hazards of injudicious use of adrenaline and suggests that intravenous administration of adrenaline should be confined to cardiac emergencies.

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(*Accepted 2 November 1982*)

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Late development of incisional hernia: an unrecognised problem

A long term follow up study was carried out to determine the incidence of incisional hernia after major abdominal operations; we report the results.

Patients, methods, and results

A total of 831 patients undergoing major abdominal operations at four centres in south Wales in 1972-3 were entered into a long term follow up study. Patients were examined by a single observer at one, three, and five years for the development of incisional hernia. The details and composition of the original cohort have been described elsewhere.[1] Altogether 564 surviving patients were willing to enter the study at one year. Loss of patients to follow up after one year was negligible and due mainly to death and to a few patients moving out of Wales. Information was obtained from the case notes to compare the prevalence of possible aetiological factors in patients in whom hernias developed early and those in whom they developed late. The table shows the total number of new hernias.

Full details of nine possible aetiological factors were available for 38 of the patients (21 with a hernia at one year, 14 with a hernia at three years, and three with a hernia at five years). Postoperative wound infection was the only