the indications for surgical intervention, especially when many of these patients have ischaemic or rheumatic heart disease.

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Tropical eosinophilia presenting with neurological features

Hypereosinophilic syndrome is a condition of varied aetiology and comprises leukaemias, parasitic diseases, and collagenosis.¹ Tropical eosinophilia is a parasitic disorder in which pulmonary manifestations predominate. So far, neurological complications have not been reported. I report here two cases of tropical eosinophilia with associated neurological disorder but no detectable cerebral lesion.

Case reports

(1) A 37-year-old right-handed housewife developed impairment of memory and abnormal behaviour along with recurring numbness of the right half of her body. In the beginning she had cough, chest pain, and breathing difficulty. Within a week she became disorientated and started behaving abnormally, like undressing in front of her family. She had no fever, visual disturbances, weakness, or convulsions. She was well developed. System review was normal. Her memory was defective, especially for recent events, and she could not do even simple arithmetic. Speech showed anomic aphasia. Her writing was grossly abnormal with omission of letters and loss of spatial organisation. She also showed finger agnosia, right-left disorientation, and constructional difficulty. The cranial nerves were intact and optic fundi normal. There was no disturbance of motor function, but cortical sensory loss on the right side was conspicuous. The deep tendon reflexes were normal and plantar response flexor. Results of urine and blood analysis were normal. ESR was 68 mm in 1st h. She had an absolute eosinophil count of $11.5 \times 10^9/l$ ($11\,500/mm^3$) (table). Serum protein electrophoresis

Haemogram in case (case 1) of tropical eosinophilia

Date (1979)	Total white cell count ($\times 10^9/l$)	Absolute eosinophil count ($\times 10^9/l$)
January 25	19.5	11.5
January 31	17.0	9.1
February 12	15.0	6.0
March 21	10.0	1.5

Conversion: SI to traditional units—Blood cells: $1 \times 10^9/l \approx 1000/mm^3$.

was normal and LE cells were absent. No haemoparasites were seen but filarial complement-fixing antibody test was positive. The venereal disease research laboratory test was negative and coagulation profile normal. Bone marrow showed eosinophilic hyperplasia. Spinal fluid was normal. Stool showed a few ova of *Ancylostoma duodenale*. Skull and chest radiographs and ECG were normal. An electroencephalogram was mildly and diffusely abnormal with slow waves of 6-7 cps. Left carotid angiogram was normal. Treated with diethylcarbamazine 100 mg thrice daily she improved within two weeks. Her numbness disappeared first, then her finger agnosia and right-left disorientation. Her writing was the last to recover. Eight weeks later the patient had recovered completely.

(2) A 9-year-old boy was admitted with a two-month history of persistent, severe left temporo-occipital scalp tenderness. One week before he had developed double vision and loss of balance, controlled by closing his left eye. He had no fever, cough, chest discomfort, or convulsions. System review was normal. Mental functions, speech, visual fields, optic fundi, and pupils were normal. He kept his left eye constantly closed. On opening it he became ataxic. There was vertical and horizontal nystagmus and central facial paralysis of the right side. Other cranial nerves were intact. The neck

was supple. The scalp was hyperalgesic over the left temporo-occipital region. His motor and sensory functions were normal. He walked on a broad base. Plantar response was flexor and deep tendon reflexes normal. His urine and blood chemistry were normal. The absolute eosinophil count was $3.8 \times 10^9/l$ ($3800/mm^3$). ESR was 35 mm in 1st h and the coagulation profile normal. No haemoparasites were seen. Stool examination showed no parasites or ova. The bone marrow showed hypercellularity with 15% eosinophils. Antinuclear antibody and LE-cell tests were negative. Filarial complement-fixing antibody test was positive. Spinal fluid was normal. Skull radiographs were normal. Arch angiogram showed no abnormality except congenital absence of the right posterior inferior cerebellar artery. Amipaque (metrizamide) ventriculogram was also normal. On treatment with prednisolone and diethylcarbamazine the scalp hyperalgesia, ataxia, and nystagmus improved. Repeat haemogram on day 14 showed an absolute eosinophil count of $1.45 \times 10^9/l$ ($1450/mm^3$). By three weeks the ataxia had disappeared completely. Diethylcarbamazine was continued for 21 days and review after two months showed no neurological deficit.

Comment

Tropical eosinophilia is a hypersensitivity response to an exciting allergen invariably parasitic, the commonest being filaria.² Neurological manifestations are very rare, in contrast to hypereosinophilic syndromes associated with severe vasculitis like polyarteritis nodosa and Wegener's granulomatosis. The patients presented here had definite eosinophilia, fulfilling all the criteria of the hypereosinophilic syndrome.³ With ascariasis excluded by stool examination, it is postulated that both patients had the eosinophilic syndrome due to filariasis. The positive filarial complement fixation test was confirmatory. Neither patient had a detectable neurological abnormality in spite of signs of focal disease. This, in combination with a rapid recovery, suggested a reversible disorder like a benign vasculitis or eosinophilic infiltration.

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Cervical myelopathy after metrizamide myelography

Metrizamide (Amipaque), a water-soluble contrast medium, is increasingly being used for cervical and thoracic myelography as well as lumbar myelography. It differs from other water-soluble contrast media in being non-ionic, and side effects of metrizamide occur less often and are less severe than with other agents.¹ We report a case of cervical myelitis occurring after lumbar myelography with metrizamide.

Case report

A 29-year-old man presented to the orthopaedic department of a peripheral hospital with a one-year history of low backache exacerbated by lifting heavy weights. Examination showed limited movement of the lumbar spine, with bilateral reduction of straight-leg raising. Neurological findings were normal. Lumbar myelography was performed with 10 ml metrizamide introduced atraumatically at the L3-4 level. The spinal canal was narrowed but no disc protrusion was seen. Within minutes he developed severe headache associated with paraesthesiae in the hands and back of the legs. These symptoms resolved after about 30 minutes, but he noticed flaccid weakness of the left forearm and hand. He was transferred to Nottingham General Hospital.

Examination confirmed weakness of all muscle groups below the elbow in the left arm, predominantly of a C8-D1 distribution, with associated hyperreflexia. The right arm was normal. Abdominal reflexes were symmetrical, and there were no motor signs in the legs. Sensory examination disclosed subjective impairment of pain sensation over the outer aspect of the left arm and forearm within the C6 dermatome. Cranial nerves were normal. Radiography of the skull showed no abnormality at the craniocervical junction. Cervical spine radiography showed a narrow but adequate canal.

Oblique views showed osteophyte encroachment on the left exit foramina of C3-4 and C4-5. No appreciable disc-space narrowing was noted and there was no evidence of cervical ribs.

He was given a course of intensive physiotherapy and fitted with a wrist splint. Six months after myelography, however, there was still pronounced weakness in the left forearm with associated reflex emphasis.

Comment

Side effects of metrizamide when used for both lumbar and cervical myelography have been extensively reviewed.¹⁻³ Minor side effects such as headache, nausea, and vomiting occur fairly often. Transient hyperreflexia after lumbar myelography with metrizamide has also been reported.⁴ One case of areflexia after thoracolumbar myelography was presumed to reflect a direct neurotoxic effect on the cauda equina.⁵ Our patient developed acute symptoms shortly after metrizamide myelography. Neurological signs suggested cervical myelopathy, which may have resulted from a direct toxic effect of metrizamide. Spinal canal stenosis, however, may have been contributory.

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Erythema nodosum and infectious mononucleosis

Erythema nodosum is associated with many conditions, the commonest being sarcoidosis.¹ Other examples include drug reactions; inflammatory bowel disease; and some conditions with an established infective cause such as tuberculosis, leprosy, ornithosis, and streptococcal infections. *Yersinia enterocolitica* infection has also been implicated.² I report a case in which erythema nodosum occurred in association with infectious mononucleosis.

Case report

A 15-year-old Caucasian girl presented with a six-day history of pharyngitis, arthralgia, lethargy, and a rash on both legs. After the rash appeared she was given ampicillin 250 mg four times daily for four days. There was no history of illness or of drug treatment. The only abnormalities found on examination were an inflamed fauces and erythema nodosum affecting both legs. A throat-swab culture was sterile and the antistreptolysin O titre under 200 IU/ml. Haemoglobin concentration was 12.0 g/dl, erythrocyte sedimentation rate 34 mm in first hour, and white cell count $6.8 \times 10^9/l$ (6800/mm³) with 7% atypical mononuclear cells. A Paul-Bunnell test was positive with titres of 224 in saline, 224 with guinea-pig kidney absorption, and under 7 with ox red-cell absorption. Cytomegalovirus titre was under 10, and agglutination tests for *Y enterocolitica* and *Y pseudotuberculosis* were negative. Chest radiography showed no abnormalities, and a Mantoux test gave a grade I-II reaction. There was no response to a Kveim test. Ten weeks later the rash had disappeared and she felt well without needing any specific treatment.

Comment

Infectious mononucleosis has been associated with erythema multiforme³ but not with erythema nodosum. The present patient showed no evidence of sarcoidosis or any of the other conditions associated with erythema nodosum. The rash occurred during an attack of infectious mononucleosis, a condition thought to be causally related to the Epstein-Barr virus, which is one of the herpesvirus group. A link between herpes-like infection and sarcoidosis was suggested by Hirshaut *et al.*⁴ Patients with sarcoidosis were later shown to have the same prevalence of raised antibody titres to

Epstein-Barr virus as control subjects but the titres were much higher than those in the controls⁵; patients with non-sarcoid erythema nodosum had about the same antibody titres to all herpesviruses as controls.

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Lack of transmission of viral hepatitis type B after oral exposure to HBsAg-positive saliva

Hepatitis B has been transmitted to a human¹ and to gibbons² and chimpanzees³ by subcutaneous or intravenous injections of saliva containing hepatitis B surface antigen (HBsAg). Attempts to transmit hepatitis B by nasal and oral exposure of gibbons to saliva containing HBsAg failed.² The possibility that cardiopulmonary resuscitation (CPR) training manikins act as fomites in the transmission of viral hepatitis type B by contact with HBsAg-positive saliva has not been investigated. We report a study in which viral hepatitis type B was not transmitted to people exposed to HBsAg-positive saliva during CPR training.

Subjects, methods, and results

Twenty-two hospital trainees were studied. They had participated in a CPR training programme eight days before one of them developed clinical hepatitis B. The training programme included two all-day classes with intensive practice sessions on five different manikins. All trainees had used a manikin after it had been used by the infected trainee. Ten trainees had also participated in two-rescuer CPR with him during practice or at the time of the final test. Manikin heads had been washed with water and rinsed with 70% isopropyl alcohol after every practice session but they were not dismantled. Their faces and inside mouth area were wiped with a clean absorbent material wetted with 70% isopropyl alcohol after every use. But during two-rescuer CPR there had been no cleaning of the manikin between trainees.

The infected trainee's serum and saliva were positive for HBsAg at the time of his illness. The radioimmunoassay ratios (counts per minute/negative control mean) for the serum and saliva were 40.92 and 7.34 respectively. His saliva was also negative for occult blood. His serum was positive for HBeAg. The trainees were tested for HBsAg and antibody to hepatitis B surface antigen (anti-HBs) two weeks, six weeks, and six months after training. All were negative for HBsAg at two weeks and one was positive for anti-HBs. At six weeks and six months only 17 trainees were tested. All were negative for both HBsAg and anti-HBs and none reported illness. The other four trainees ceased employment with the hospital between two and six weeks after training (including the one with existing anti-HBs). They were contacted at six months and none reported illness during that time. None of the four who ceased employment were among those who participated in two-rescuer CPR with the infected trainee.

Comment

Manikins used in training programmes are a potential vehicle for transmitting hepatitis B. Mouth-to-mouth or mouth-to-nose artificial respiration requires physical contact. Trainees are instructed to disregard sanitary and hygienic precautions to save the victim's life. They therefore commonly practise on manikins contaminated by the oral secretions of others. Manikins may also be contaminated from hands during practice removal of upper airway obstructions. The lack of transmission of hepatitis under these conditions in our study may have