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 $11{\cdot}5 \times 10^9$/l (11 500/mm^3) (table). Serum protein electrophoresis 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 $3{\cdot}8 \times 10^9$/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 $1{\cdot}45 \times 10^9$/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.

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: 1×10^9/l $\approx$ 1000/mm^3.

(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 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.

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 craniovertebral junction. Cervical spine radiography showed a narrow but adequate canal.