SHORT REPORTS

Brachial plexus neuropathy associated with human parvovirus infection

Human parvovirus is a recognised cause of arthritis.[1] A recent report suggests that it may also cause brachial plexus neuropathy.[2] We describe a patient who presented with both articular and neurological manifestations of infection.

Case report

A previously fit 23 year old nurse presented in January 1986 with pain in both arms. Two weeks earlier she had noticed an erythematous rash which spread from the legs to the abdomen and arms but spared the upper trunk and face. Within hours she developed pain in the ankles and knees, and by 48 hours she had pain in the neck and shoulders, weakness of the arms, and numbness and dysaesthesia of the right forearm. There was no history of recent immunisations. She had in the past been immunised against poliomyelitis.

On examination she was found to be alert with no fever or meningeal irritation. The rash was no longer apparent and the joints were normal. There was pronounced tenderness of both trapezius muscles, deltoids, and biceps and severe weakness of shoulder abduction. Both biceps reflexes and the left supinator reflex were reduced. An area of sensory loss was detected over the radial border of the right forearm.

Full blood count, erythrocyte sedimentation rate, biochemical profile, blood glucose concentration, and serum creatine kinase activity were normal. An autoimmune profile was negative and no circulating immune complexes were detected. Cerebrospinal fluid contained normal amounts of cells and protein. Electromyography showed severe denervation in deltoid, supraspinatus, and infraspinatus muscles bilaterally. The table gives the serological results.

Serological results

Agent	Type of test	Acute serum (2 weeks from onset)	Convalescent serum (6 months from onset)
Mycoplasma pneumoniae	Complement fixation	1/10	1/10
Borrelia burgdorferi	ELISA*	Negative	—
Rubella	ELISA (IgG)	460 μg/l (normal)	—
Cytomegalovirus	Complement fixation	Negative	Negative
Adenovirus	Complement fixation	1/10	1/80
Varicella zoster	Complement fixation	1/10	1/10
Herpes simplex	Complement fixation	1/20	1/20
Epstein-Barr virus	Monospot	Negative	—
Parvovirus B19 (IgM)	Radioimmunoassay	31 Units	2·5 Units
Parvovirus B19 (IgG)	Radioimmunoassay	40 Units	17 Units

*ELISA = Enzyme linked immunosorbent assay.

The pain resolved within four weeks but severe wasting of both deltoids and the left supraspinatus and infraspinatus and right biceps muscles occurred and the biceps and supinator reflexes were completely lost. Six months later wasting and weakness had improved and sensory loss had recovered.

Comment

Acute neuropathy of the brachial plexus (neuralgic amyotrophy) is characterised by the sudden onset of pain around the shoulder girdle followed by weakness and wasting of periscapular and arm muscles. Sensory impairment is common. Complete recovery taking up to three years is the rule.[3] An association with various inoculations, infections, and surgical procedures has been described.[3]

Viraemia due to human parvovirus was first described in 1975, and the virus has subsequently been incriminated as a cause of aplastic crises in people with various chronic haemolytic anaemias, of erythema infectiosum, and of arthralgia or arthritis.[4,5] There have been no reports of neurological illness other than brachial plexus neuropathy due to parvovirus in man.

Our patient showed serological evidence of recent parvovirus infection coincident with the development of brachial plexus neuropathy. There was also evidence of adenovirus infection occurring between the two tests; adenovirus, however, have rarely been implicated as a cause of arthralgia and never as a cause of brachial plexus neuropathy, nor did the patient recall typical adenovirus symptoms during the period. From the clinical and laboratory findings we believe that parvovirus was the cause of her illness and may therefore be implicated as a cause of brachial plexus neuropathy. We suggest that testing for parvovirus should be undertaken in cases of brachial plexus neuropathy.

We thank Dr M Anderson, of University College Hospital, London, for undertaking parvovirus serology; Dr M Ogilvie, of Southampton General Hospital, for other serological tests; and Mrs B Hatch for typing the manuscript.

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(Accepted 8 December 1987)

Southampton University Rehabilitation Unit, Southampton General Hospital, Southampton SO9 4XY
KEVIN J WALSH, MA, MRCP, lecturer in rehabilitation

Southampton General Hospital, Southampton SO9 4XY
R D ARMSTRONG, MA, MRCP, consultant rheumatologist

Wessex Neurological Centre, Southampton General Hospital, Southampton SO9 4XY
A M TURNER, MB, MRCP, consultant neurologist

Correspondence to: Dr Kevin Walsh, MRC Epidemiology Unit, Southampton General Hospital, Southampton SO9 4XY.

Intraperitoneal calcium for resistant symptomatic hypocalcaemia after parathyroidectomy in chronic renal failure

Patients with chronic renal failure may require parathyroidectomy to correct the complications of hyperparathyroidism.[1] Postoperative hypocalcaemia occurs in most cases,[2-4] and calcium and vitamin D supplements are necessary to maintain serum calcium concentrations. A recent review of 17 patients receiving long term dialysis who underwent subtotal parathyroidectomy reported improvement in symptomatic, biochemical, and radiological abnormalities in 14; postoperative hypocalcaemia occurred in 15 and was symptomatic in seven.[2] We report on one of the patients in that series who was receiving continuous ambulatory peritoneal dialysis and developed severe and resistant symptomatic hypocalcaemia requiring prolonged calcium treatment, including intraperitoneal administration of calcium chloride.

Case report

A 25 year old white man with familial nephritis began haemodialysis in 1978 and changed to continuous ambulatory peritoneal dialysis in February 1982. Itching, bone pain, irritation of the eyes, hypercalcaemia, and pronounced radiological bone changes led to subtotal parathyroidectomy. Preoperative serum concentrations (reference ranges in parentheses) were: total calcium 2·63 mmol/l (2·29-2·44); phosphate 2·97 mmol/l (0·8-1·66); magnesium 1·87 mmol/l (0·76-0·99); alkaline phosphatase 674 U/l (<110); and parathyroid hormone 64 μg/l (0·10-0·35) by C terminal direct immunoradiometry.

The operation resulted in rapid relief of symptoms and complete resolution of skeletal abnormalities by 10 months. Postoperatively the patient was given oral calcium gluconate 1 g thrice daily but developed severe paraesthesia, muscle twitching, and Chvostek's and Trousseau's signs. Serum total calcium concentration was 1·64 mmol/l (fig 1). Symptoms were rapidly controlled with oral 1,25-dihydroxyvitamin D_3 0·25 μg/day and intravenous infusion of calcium gluconate.

Stopping the calcium infusion while increasing oral treatment resulted in symptomatic hypocalcaemia and florid evidence of depersonalisation. The calcium in the dialysis fluid (about 1·6 mmol/l) was therefore supplemented with a 10% solution of calcium chloride (20 mmol per two litre dialysis bag; added under sterile laminar flow, and the serum calcium concentration entered the normal range for the first time after operation. Thereafter, intraperitoneal calcium 60 mmol/day, oral calcium gluconate 6 g/day, and 1,25-dihydroxyvitamin D_3 1·5 μg/day maintained serum calcium concentration in the low normal range.

Absorption of calcium from the peritoneal cavity was calculated at intervals