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.

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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.

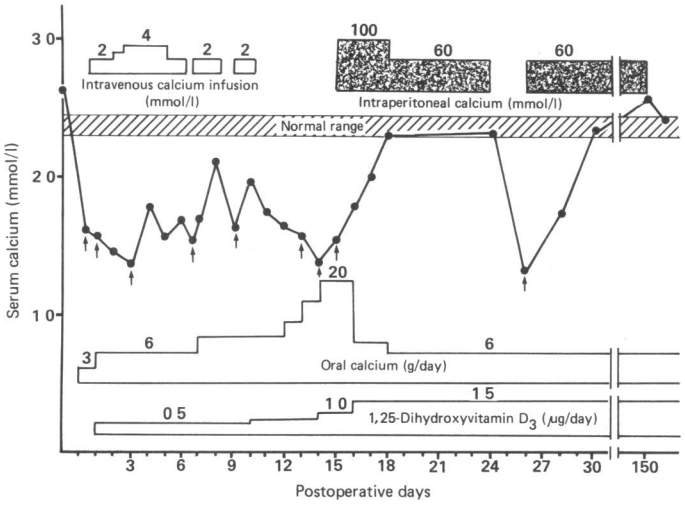
The operation resulted in rapid relief of symptoms and complete resolution of skeletal abnormalities by 10 months. Postoperatively the patient was given oral calcium gluconate 1 g thrice daily but developed severe paraesthesia, muscle twitching, and Chvostek's and Trousseau's signs. Serum total calcium 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

during the first 14 days of this treatment. To eliminate the risk of contaminating the fluid the calcium concentration was not measured before instillation. The estimated mean amount of calcium instilled during each cycle was 23·2 mmol and that in each bag after drainage was 7·4 mmol (range 4·1-9·8). The estimated mean amount of calcium absorbed per bag was therefore 15·8 mmol (range 13·4-19·1), roughly 69% of the total in the bag. Fasting serum calcium concentration was measured daily before instillation of the first bag of dialysis fluid. Two attempts to stop the intraperitoneal calcium supplements resulted in symptomatic hypocalcaemia. The supplements were finally stopped on the 150th postoperative day, the oral calcium and 1,25-dihydroxyvitamin D_3 supplements being continued.

Serum magnesium concentration in the months preceding operation averaged 1·54 mmol/l (range 1·21-1·87). Symptoms were not due to hypomagnesaemia as the serum magnesium concentration did not fall below the normal range at any time postoperatively.



Clinical course after subtotal parathyroidectomy. Arrows indicate symptomatic hypocalcaemia.

Comment

Although hypocalcaemia often occurs after parathyroidectomy for secondary hyperparathyroidism, the associated symptoms are seldom as severe or protracted as in our patient. Previous studies have shown that calcium supplementation may be necessary for more than a year after operation,[4,5] but parenteral treatment is seldom required beyond the first week.[3,5]

Intraperitoneal administration of calcium has a distinct advantage over intravenous administration in patients receiving continuous ambulatory peritoneal dialysis who have protracted symptomatic hypocalcaemia not controlled by oral supplements as it can be managed by the patient on an outpatient basis with minimal medical supervision. In the immediate postoperative period, however, intravenous infusion is better suited to the unstable situation. The peritoneal route is effective, convenient, and safe in patients receiving continuous ambulatory peritoneal dialysis who require parenteral calcium for more than a few days after operation.

We thank the department of endocrinology, Auckland Hospital, for measuring parathyroid hormone concentration.

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Department of Medicine, Wellington School of Medicine, Wellington, New Zealand
T J THOMPSON, MB, CHB, registrar
T J NEALE, MD, FRACP, associate professor of medicine

Correspondence to: Dr Neale.

Rash mediated by immune complexes associated with ranitidine treatment

Ranitidine has established an important place in the treatment of peptic ulceration: rates of healing at six weeks are between 80 and 90%.[1] In its standard dose of 300 mg daily it is well tolerated and has few side effects. We report a vasculitic rash occurring in association with ranitidine.

Case reports

Case 1—A 65 year old man was treated with ranitidine 150 mg twice daily as the sole treatment for duodenal ulcer that had been proved endoscopically. One month after starting treatment he developed a maculopapular rash on his arms, legs, and trunk. The rash was itchy and scaly and made up of discrete papules, some of which had coalesced to form lesions up to 3 cm in diameter. Full blood count, erythrocyte sedimentation rate, biochemical profile, and serum immunoglobulin and complement concentrations were all normal, while tests for circulating immune complexes and autoantibodies yielded negative results. Routine histological examination of a skin biopsy specimen showed nothing remarkable, but immunofluorescence showed deposition of C3 and IgA in the dermal capillaries, which suggested that the condition was mediated by immune complexes. The rash faded two weeks after ranitidine was stopped. After giving his informed consent he was re-exposed to the drug several weeks later. Within one week of challenge a similar rash developed and the findings on immunofluorescence were identical with those seen previously.

Case 2—A 79 year old man with a benign gastric ulcer that had been proved endoscopically was treated with ranitidine 150 mg twice daily. Four weeks after starting treatment he noticed erythematous, itchy, scaly lesions on his arms and legs. No haematological, biochemical, or immunological abnormalities were found. Routine histological examination of a skin biopsy specimen showed perivascular lymphocytic infiltration, and immunofluorescence showed C3 and IgA in the smaller vessels, findings consistent with vasculitis. The rash cleared completely within two weeks of the drug being stopped.

Case 3—A 60 year old man with rheumatoid arthritis and ulceration of the pyloric channel was started on ranitidine 150 mg twice daily. Within two weeks he developed an itchy, scaly, erythematous rash on his trunk, arms, and legs. Histological examination of a biopsy specimen of the skin lesion showed nothing remarkable; immunofluorescence, however, showed fine granular deposition of IgA at the dermal-epidermal junction, a finding consistent with a diagnosis of dermatitis herpetiformis. Duodenal biopsy showed no evidence of enteropathy, and his rash resolved completely on withdrawal of ranitidine.

Comment

All three patients seem to have had a rash mediated by immune complexes and associated with treatment with ranitidine. The rash cleared on withdrawal of the drug, and an identical rash developed in the one patient rechallenged. Immunofluorescence showed deposition of C3 and IgA in the small vessels, which is typical of a vasculitic rash; drugs are a well recognised trigger for such a reaction.[2] Rashes such as urticaria that are due to a type I allergic reaction have been described in association with treatment with ranitidine[3] and have been reported to the Committee on Safety of Medicines, but in extensive studies such lesions were associated with ranitidine only slightly more commonly than with placebo.[4] Nevertheless, of the 87 adverse reactions probably due to ranitidine that had been reported to the Committee on Safety of Medicines by September 1987 (personal communication), 23 affected the skin, but none seem to have been a vasculitic reaction. With increasing use of ranitidine, particularly as maintenance treatment, awareness of this type of rash is important. We have reported these cases to the Committee on Safety of Medicines and to the manufacturer.

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Department of Geriatric Medicine, Cardiff Royal Infirmary, Cardiff CF2 1SZ
N HABOUBI, MB, MRCP, senior registrar

Alastair Fraser and John Squire Clinical Investigation Unit, East Birmingham Hospital, Birmingham B9 5ST
P ASQUITH, MD, FRCP, consultant physician

Correspondence to: Dr Asquith.