Table 1 Pituitary evaluation

	Stimulation by insulin induced hypoglycaemia							
	Glycaemia (mg/dl)		*GH (0.5–5 ng/ml)**		*Plasma cortisol (80–230 μg/dl)**		*ACTH (9–52 pg/ml)**	
Time (min)	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*
0	90	99	0.9	1.3	359	195	ND	61.8
20	40	35	4	0.7	286	150	ND	51.8
60	120	89	12.1	0.8	585	207	ND	141.3

	Stimulation by TRH and LHRH									
	*LH (2–14 IU/l)**		*FSH (2–12 IU/l)**		*Prolactine (2–14 ng/ml)**		*TSH (0.12–5 μU/ml)**		*T4 (4–11 μg/dl)**	
Time (min)	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*	*B*
0	5.2	0.3	2.1	0.2	11.6	30.1	2.29	1.2	11.1	0.89†
20	13.2	1.3	5.8	2.8	143.1	34.5	22.4	4.64	—	—
60	11	2	4.9	3.7	44	31.8	14.5	4.68	—	—

A = Patient number 1; B = patient number 2. ND = not detected. *Normal values. †Free T4 (ng/dl).

hypothalamus or hypophysis stalk. Some authors have suggested a relation between adrenal insufficiency resulting from glandular infarctions and ACA in patients with or without SLE.[7–9] It could be that the patients presented here had vascular impairment, not because of inflammatory causes, but because of thrombotic events. Finally we cannot rule out a direct autoimmune lesion. Schgeerbaum and Botazzo[10] published the existence of autoantibodies against vasopressin producing neurons in some patients suffering from NDI. However, the antibodies against hypophysis structures were negative by IIF in our two cases.

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Neuralgic amyotrophy and polyarthritis caused by parvovirus B19 infection

Infection with human parvovirus B19 causes a wide range of clinical manifestations, which may vary according to the immune status of the patient. They include erythema infectiosum (fifth disease), symmetrical polyarthritis, vasculitis, transient aplastic crisis in patients with low red cell production, and chronic infection in immunocompromised patients. We report a patient treated with immunosuppressive drugs in whom bilateral brachial plexitis and polyarthritis were associated with parvovirus B19 acute infection.

A 33 year old woman presented with severe pain in the arms. She had a two year history of severe Crohn's disease and was treated with azathioprine (125 mg/day) and prednisone (10 mg/day). The patient developed fever (38°C), then a maculopapular rash, which resolved within 24 hours and a lancinating pain in both arms that persisted. Physical examination disclosed bilateral polyarthritis involving the fingers and a symmetrical flexor tenosynovitis. No motor weakness was detected but there was an area of sensory loss covering the radial border of both forearms. Both the right biceps reflex and the right supinator reflex were absent. The pain resolved progressively within three months but severe wasting of both deltoids developed. Electromyography showed patchy denervation of the upper limbs. Erythrocyte sedimentation rate was normal. Rheumatoid factor and antinuclear antibody were negative. Cerebrospinal fluid contained normal amounts of cells and protein. Anti-parvovirus B19 antibody in serum samples was measured by ELISA and is reported as an optical density (OD) index. Three days after the onset of the febrile illness, the IgM (OD) was positive at 0.521 (IgM (OD) threshold 0.071), and the IgG (OD) was 0.394 (IgG(OD) threshold 0.315). Six weeks later, the IgM (OD) had decreased to 0.187 whereas the IgG (OD) had increased to 0.981, consistent with serological conversion caused by acute infection. Parvovirus DNA was not detected by polymerase chain reaction in the serum collected at either of these two dates.

Neuralgic amyotrophy is an axonal disorder of unknown aetiology. It is characterised by the sudden onset of shoulder girdle pain followed by weakness and wasting of scapular and arm muscles. It may follow surgery,[1] vigorous exercice, heroin misuse, radiation therapy,[2] chemotherapy,[3] immunisation or viral infection.[4] Four previous cases of neuralgic amyotrophy have been reported during the acute phase of parvovirus B19 infection.[5–8] As in this case report, all cases were young women (mean age, 27.5 years) who presented with a transient rash and arthralgia rapidly followed by the onset of neuralgic amyotrophy. They were all previously healthy and none developed polyarthritis, in contrast with our patient who was severely disabled by bilateral symmetrical polyarthritis associated with the plexitis.

We conclude that a diagnosis of parvovirus B19 infection should be considered in patients presenting with brachial plexus neuropathy, particularly those who are young women with a history of arthralgia and rash. In addition, the association of a neuralgic amyotrophy with a symmetrical polyarthritis, which appears not to have been reported previously, might be linked to the specific characteristics of the host.

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