Bilateral foot drop, weight loss and rectal bleeding as an acute presentation of Crohn's disease

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Summary: We report a 71 year old lady who presented with weight loss, rectal bleeding and bilateral foot drop having been previously fit and well. Clinical examination, laboratory investigation and postmortem examination confirmed the diagnosis of active Crohn's disease and acute peripheral neuropathy. The clinical course of this patient suggests that the peripheral neuropathy might have resulted from the common pathogenesis for Crohn's disease.

Introduction

Crohn's disease has numerous extra-intestinal manifestations with notable sparing of the nervous system, especially the peripheral nerves. In this report we describe a patient who presented acutely with active Crohn's disease and peripheral neuropathy, a presentation that raises a question about the possible association of the two disorders.

Case history

A 71 year old woman, previously fit and independent, presented with a 2 week history of unsteadiness and lower limb weakness, and a one week history of rectal bleeding and loose motions 5–6 times/day. She also gave a history of weight loss of around one stone in the previous month. Prior to this she enjoyed good health apart from hypertension for a number of years for which she took atenolol, chlorthalidone, amlodipine 5 mg/day and aspirin 75 mg daily.

On general examination the only abnormal physical sign was bilateral foot drop with marked weakness of the dorsiflexors bilaterally; tendon reflexes in the lower limbs were present but sluggish. Proctosigmoidoscopy revealed normal colour stools but red inflamed mucosa. A rectal biopsy was taken that concern the diagnosis of acute Crohn's disease.

Other laboratory tests included ESR 82 mm/hour, C-reactive protein 168 mg/l (normal < 6 mg/l, albumin 24 g/l, potassium 2.6 mmol/l, haemo-

globulin 11 g/dl. The remainder of the biochemistry was normal as were immunoglobulins, proteins, electrophoresis, B₁₂ folate rheumatoid factor, antinuclear and anti-neutrophil cytoplasmic antibodies. Stool microscopy and culture were negative on three occasions. Barium enema and lumbar puncture were unsuccessful. Sural sensory studies showed small units with normal velocity suggestive of an axonal type of neuropathy. Both lateral popliteal (common peroneal) nerve motor studies showed evoked response in extensor digitorum brevis stimulating at the ankles. More proximal studies were within normal limits except for an increased latency of 6.4 ms from the neck of fibula to the upper tibialis anterior on the left. Electromyography of the left extensor digitorum brevis showed no activity at rest and no units under voluntary control, whilst the left tibialis anterior showed no activity at rest and the pattern on maximum voluntary effort was reduced. Left posterior tibial nerve motor studies to the abductor hallucis were normal. In conclusion there were some changes of peripheral nerve dysfunction in both motor and sensory studies, with most changes in the distal lateral popliteal nerves.

As soon as the diagnosis of acute Crohn's disease had been made she was started on treatment with oral prednisolone 30 mg/day, and prednisolone enema, and mesalazine 400 mg three times/day and potassium supplement. She showed considerable improvement in her general well-being and physical ability, notably her foot drop.

At the end of her fourth week in hospital she suddenly collapsed and died. Postmortem examination revealed acute Crohn's disease involving the entire colon and the last 10 cm of the terminal ileum and a large pulmonary embolus secondary to

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right leg deep vein thrombosis. Postmortem examination did not reveal any other lesions that could have caused or contributed to this patient's peripheral neuropathy.

Discussion

Crohn's disease and ulcerative colitis are chronic inflammatory bowel diseases that manifest many complications outside the gastrointestinal tract. Among these, nervous system involvement has rarely been noted, despite the presence of predisposing factors such as malabsorption, vasculitis, thromboembolism and amyloidosis.

Peripheral nerve involvement associated with Crohn's disease has been previously described. Duffy *et al.*¹ reported a group of children with Crohn's disease who developed a reversible sensory peripheral neuropathy due to metronidazole. Nemni *et al.*² described two patients who suffered from distal sensory neuropathy with a fluctuating course parallel to the exacerbations of the Crohn's enteritis. Neither of these two cases had vitamin B₁₂ deficiency or received metronidazole. A similar fluctuating course of sensori-motor polyneuro-

pathy was described by Humbert *et al.*³ in a patient with Crohn's disease in whom an immunological mechanism was suggested because of concurrence with cutaneous vasculitis.

Lossos *et al.*⁴ reported a case of severe sensory peripheral neuropathy associated with low serum folate in whom the diagnosis of Crohn's disease was made 2–3 years later. Cases of peripheral nerve involvement in the form of polyneuropathy and mononeuritis multiplex have also been reported in relation to Crohn's disease.^{5–8}

In the present case, we found no evidence of antecedent infection or other disease that might have caused or contributed to the clinical condition. A drug effect is also unlikely as she had not taken any drug that is known to cause peripheral neuropathy. We conclude that the clinical course of our patient supports a possible causal relationship between Crohn's disease and peripheral neuropathy.

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References

1. Duffy, L.F., Daum, F., Fisher, S.E. *et al.* Peripheral neuropathy in Crohn's disease patients treated with metronidazole. *Gastroenterology* 1985, **88**: 681–684.
2. Nemni, R., Fazio, R., Corbo, M., Sessa, M., Comi, G. & Canal, N. Peripheral neuropathy associated with Crohn's disease. *Neurology* 1987, **37**: 1414–1417.
3. Humbert, P., Monnier, G., Billerey, C., Birgen & Dupond, J.L. Polyneuropathy: an unusual extraintestinal manifestation of Crohn's disease. *Acta Neurol Scand* 1989, **80**: 301–306.
4. Lossos, A., Argov, Z., Ackerman, Z. & Abramsky, O. Peripheral neuropathy and folate deficiency as the first sign of Crohn's disease. *J Clin Gastroenterol* 1991, **13**: 445–447.
5. Contamin, F., Ollat, H., Levy, V.G. & Thierman-Duffaud, D. Atteinte des systemes nerveux peripherique et pyramidal au cours d'une maladie de Crohn: role determinant d'une carence folique. *Sem Hop Paris* 1983, **59**: 1381–1385.
6. Rankin, G.B., Watts, H.D., Melnyk, C.S. & Kelley, M.L. National cooperative Crohn's disease study: extraintestinal manifestation and perianal complications. *Gastroenterology* 1979, **77**: 914–920.
7. Danzi, J.T. Extraintestinal manifestation of idiopathic inflammatory bowel disease. *Arch Intern Med* 1988, **148**: 297–302.
8. Cohen, M.G. & Webb, J. Brachial neuritis with colitic arthritis. *Ann Intern Med* 1987, **106**: 780–781.