

A subacute onset inflammatory axonal-demyelinating polyradiculoneuropathy after hemicolectomy: a case report.

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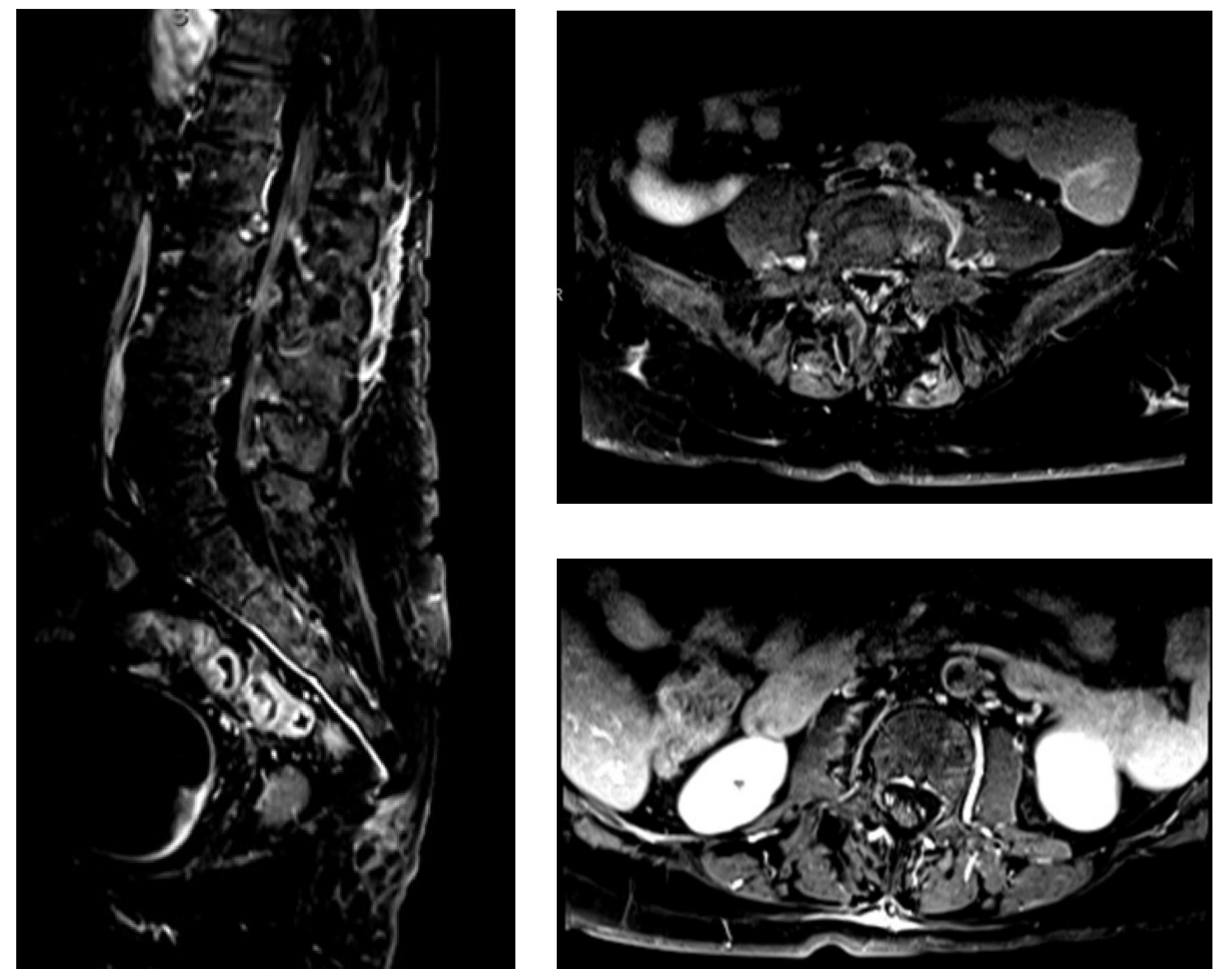
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BACKGROUND and OBJECTIVE: Post-surgical inflammatory neuropathies usually started within thirty days after surgery and present with acute pain, weakness and axonal damage. We here report a patient with an inflammatory axonal-demyelinating polyradiculoneuropathy appeared five months after hemicolectomy for colon adenocarcinoma.

PATIENT and METHODS: A 70-year-old woman subacutely developed pain, paresthesias and sensory impairment on distal upper limbs (UP) and, subsequently, on distal lower limbs (LL) five months after hemicolectomy. One month later, she started to complain of worsening proximal-distal muscle weakness and abnormal gait. Neurological evaluation showed areflexic tetraparesis, distal UL and LL superficial and deep sensory loss.

RESULTS: Anti gangliosides and onconeural antibodies as well as tumor markers were normal. EMG-ENG revealed LL>UP sensory-motor neuropathy with reduction in CMAP amplitude and mild increase in distal latency. CSF analysis showed a mild increase in protein content. A 5-day IV immunoglobulin cycle was administered with no significant improvement. Two months later, EMG-ENG revealed marked axonal and demyelinating changes in peripheral nerves. Spinal cord MRI showed a marked enhancement of cervical and dorsal roots and cauda equina, suggesting an inflammatory polyradiculoneuropathy. High dose steroid therapy (prednisone 1 mg/kg) and plasma exchange (PE) were started and a marked improvement in strength and neuropathic pain was obtained. Three months later, patient was ambulant, pain was gone and strength markedly improved.

DISCUSSION AND CONCLUSION: Post-surgical inflammatory neuropathies have been described in a small number of patients with onset usually within thirty days after surgery and typically presenting with pain and weakness. In these cases, electrophysiology showed axonal damage and nerve biopsies detected epineurial perivascular lymphocytic inflammation or changes suggestive of microvasculitis. The pathogenic mechanisms remain poorly understood and an immune-mediated response to a physiological stress (i.e. surgery, anesthesia) has been evoked. Prolonged high-dose steroids usually improve clinical picture. Our patient presented with an inflammatory neuropathy which should be clinically comparable to a post-surgery neuropathy (subacute onset with pain and weakness, axonal injury at early stages). However, onset was more than 30 days and electrophysiological findings evolved towards myelin involvement, resembling an inflammatory CIDP-like polyradiculoneuropathy. Although we can not document with certainty the relationship between the surgery and neuropathy in this patient, the temporal relationship between the two events should raise suspicion that a surgical stress may cause autoimmune nerve injury even longer distance from surgery and may trigger pathogenic mechanisms involving myelin. This observation deserves further larger studies.



T1-weighted images following an intravenous gadolinium administration showed an enhancement in different cervico-dorsal roots and cauda equina

Laughlin RS et al., *Neurology* 2009
Nobile-Orazio et al., *Lancet Neurol* 2012