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Non-Hodgkin's lymphoma with left suprascapular neuropathy on magnetic resonance imaging

非霍奇金淋巴瘤患者由磁共振成像發現左肩胛神經病

Magnetic resonance imaging is gaining importance in the diagnosis of nerve and muscular disorders. The ability of magnetic resonance imaging to delineate the different muscles and the nerve in any plane has made the differentiation between the changes of neuropathy, denervation, and nerve entrapment possible. Although findings on magnetic resonance imaging are non-specific, their use, coupled with clinical symptoms and electromyographic findings, allow an accurate diagnosis to be made without resorting to invasive biopsies.

磁共振成像在神經和肌肉紊亂的診斷中越來越重要。磁共振成像能描繪任何剖面上的不同肌肉及神經，這有助分辨神經病變化、去神經支配及壓迫性神經損害。雖然磁共振成像不能顯示特定狀況，若以此技術配合臨床徵狀及肌電描記結果，仍可達至準確的診斷而無需進行活組織切片檢查。

Introduction

Neuropathy is a known sequela of intensive chemotherapy regimens including vincristine administered to patients with non-Hodgkin's lymphoma. The condition commonly presents as a peripheral neuropathy.[1,2] Traditionally, electromyography (EMG) has been used to diagnose neuropathy. Magnetic resonance imaging (MRI) is becoming popular as an adjunct to EMG, however, and may ultimately eliminate this invasive technique. We report a patient with non-Hodgkin's lymphoma who developed left suprascapular nerve denervation during the course of chemotherapy. Appearances and differential diagnosis on MRI are discussed.

Case report

A 57-year-old Chinese man initially presented with a right-sided cervical lymphadenopathy. Biopsy of the mass revealed stage IIA high-grade non-Hodgkin's lymphoma. The patient was treated with six courses of chemotherapy, consisting of cyclophosphamide 1200 mg/m^2, epirubicin 130 mg/m^2, vincristine 2 mg/m^2, and prednisolone 100 mg/m^2, given at 3-weekly intervals. No radiotherapy treatment was planned.

Four months following commencement of chemotherapy, the patient complained of stiffness and weakness in the left shoulder, and an inability to abduct and flex the shoulder. He had no history of trauma and previous surgery had been limited to the right side of the neck. Examination of the left shoulder revealed a power grade of 3/5 for abduction and flexion. There was no sensory deficit evident. Muscle bulk appeared normal. An EMG was performed for both the right and left upper limb, which showed fibrillation activity, with loss of motor unit action potentials in the left supraspinatus and infraspinatus muscles. Denervation-reinnervation changes were also noted in these affected muscles, consistent with neuropathy.

Magnetic resonance imaging of the cervical spine revealed no significant abnormality and, subsequently, MRI of the left shoulder was performed. All

Key words:
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of the left supraspinatus and infraspinatus muscles appeared oedematous, and were homogenously hypointense on T1-weighted images and hyperintense on both T2-weighted images (Fig 1) and short tau inversion recovery (STIR) images (Fig 2). Moderate enhancement of these muscles on post-gadolinium T1-weighted MRI was seen (Fig 3). The fat planes were intact and the rest of the shoulder was normal.

The patient was seen for follow-up at the hospital haematology clinic and was scheduled for a biopsy. However, he subsequently failed to attend for further treatment.

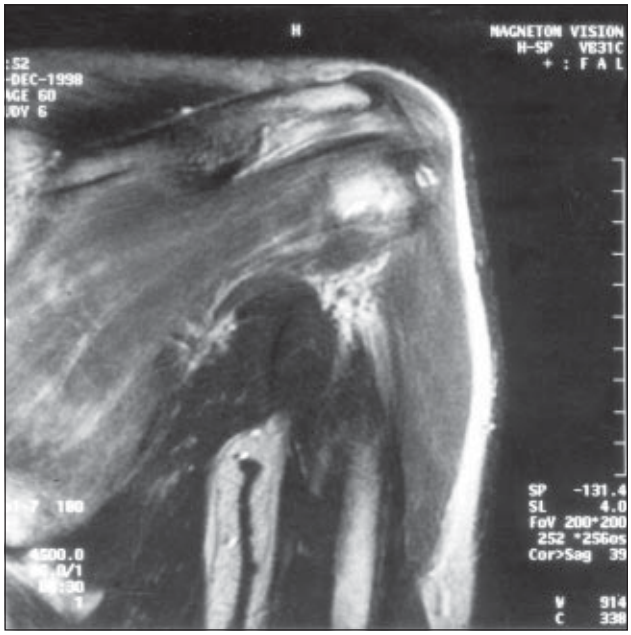

Fig 1. Coronal turbo spin echo T2-weighted magnetic resonance image of the left shoulder shows the left supraspinatus and infraspinatus muscles as hyperintense compared to other muscles

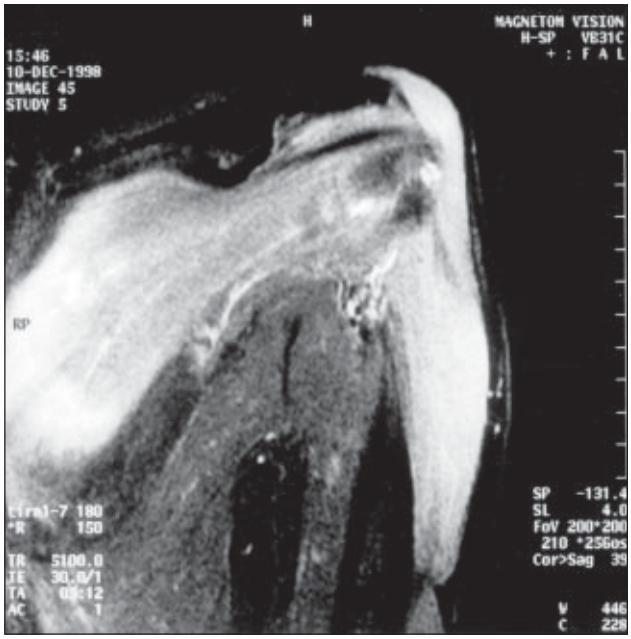

Fig 2. Coronal short tau inversion recovery image showing a homogenous increase in intensity in both the left supraspinatus and infraspinatus muscles

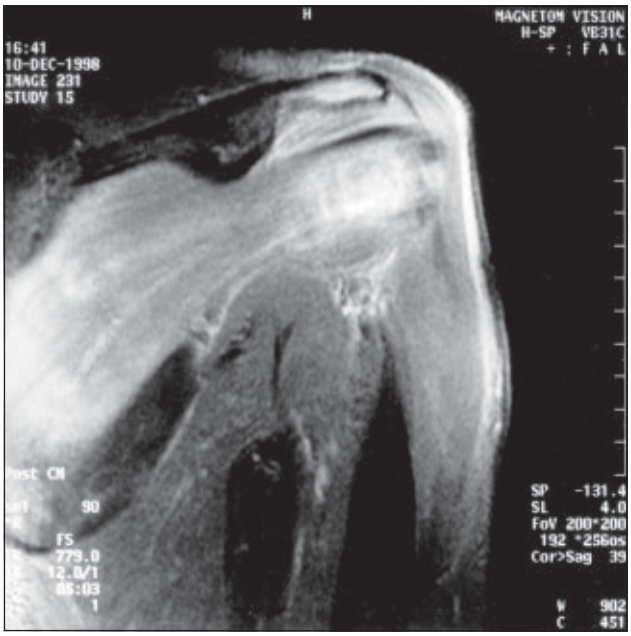

Fig 3. Post-gadolinium T1-weighted magnetic resonance image shows moderate enhancement of the left supraspinatus and infraspinatus muscles

Discussion

A motor unit consists of the motor neuron cell body located in the anterior horn of the spinal cord; the axons of the motor neuron in the peripheral nerve; the neuromuscular junction; and the muscle fibres innervated by the motor neuron.[3] Diseases of the motor unit are characterised by muscle weakness, fatigue, cramps, pain, or stiffness.[3] Denervation of the skeletal muscle may result from lesions in the spinal cord, nerve root, plexus, or the peripheral nerve. Causes include trauma, various forms of peripheral neuropathy, and spinal radiculopathy.[4]

Vincristine administration as part of a chemotherapy regimen is known to cause primary toxic myopathy,[3] myelopathy,[5] and peripheral neuropathy.[5] DeAngelis et al[2] reported that intensive administration of intravenous vincristine (total dose 2 mg/m^2 every other week), and corticosteroids for 12 weeks to patients with non-Hodgkin's lymphoma resulted in neuropathy and myelopathy in all 27 evaluable patients studied. Symptoms were most apparent in the distal extremities, involving mainly the extensor muscles. Correale et al[6] reported toxic polyneuropathy in 39 (3.9%) of 989 patients with lymphoma undergoing cytotoxic treatment.

Peripheral neuropathy can be due to axonal loss, demyelinating injury, or both. Vincristine-induced neuropathy usually presents with axonal loss on EMG, leading to loss of amplitude of nerve action potentials and evidence of denervation on needle examination of affected muscles.[7] Hamilton et al[8] demonstrated vincristine-induced peripheral neuropathy in a dog, with an EMG result consistent with muscle denervation. In most reported studies, neuropathy associated with vincristine therapy affected more than

one nerve (that is, resulted in polyneuropathy), and involved the distal extremities. This patient had neuropathy involving the left suprascapular nerve only, however.

Although EMG has remained the gold standard for assessment of the neurological integrity of individual muscles, it is a painful and operator-dependent examination.[9] Magnetic resonance imaging has the advantage of providing a better indication of the extent of muscle involvement. In a study by Fleckenstein et al,[9] muscle denervation was defined as acute (if less than 1 month in duration), subacute (between 1 and 12 months' duration) and chronic (present for more than 1 year). These authors found that MRI demonstrated poor sensitivity in acute denervation. In subacute denervation, however, the affected muscles were shown to be hypointense on T1-weighted images, and hyperintense on T2-weighted images and STIR images. In chronic denervation, the muscles were noted to be markedly atrophic and replaced by fat, with relatively little hyperintensity on STIR images. Uetani et al[4] reported that characteristic MRI signal intensity patterns in denervated muscles were observed at 15 days or more after onset of symptoms. Changes noted were normal signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Appearance on MRI after intravenous gadolinium administration has not been well documented. In recent years, the importance of muscular oedema as a significant sign of neuropathy on MRI has been highlighted.[10] It has been shown to be more specific (100%) and more sensitive (94.5%) than muscle atrophy and fatty changes.[10]

Although MRI is a useful aid in the diagnosis of muscular neuropathy, MRI findings are non-specific. Similar T1-weighted and T2-weighted appearances are seen in other conditions, including exertional muscle injury, muscle necrosis, intramuscular haemorrhage, oedema associated with tumour, polymyositis, effects of radiation, and even as a short-term reaction to exercise.[9] Careful correlation with patient history and clinical findings is imperative. Magnetic resonance imaging can depict changes that suggest denervation, and plays a valuable role in evaluating and mapping the extent of nerve and muscular involvement. Follow-up MRI scans can also be used to assess the efficacy of treatment or the progression of the condition.

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