Electrophysiological Features in the Management of Meralgia Paraesthetica

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Abstract

Meralgia paraesthetica is a condition which presents with pain and paresthesia over the anterolateral thigh, due to entrapment of the lateral femoral cutaneous nerve. Our local experience of 12 cases highlights the usefulness of antidromic sensory nerve conductions in the diagnostic and prognostic aspects of this condition. Follow-up studies suggest that patients with initial reduction rather than absent sensory amplitudes on the affected side more likely to experience symptomatic improvement over an 8 to 24 month period.


Key words: Antidromic sensory conductions, Diagnostic, Lateral femoral cutaneous nerve, Prognostic, Symptomatic improvement

Introduction

Meralgia paraesthetica is a condition resulting from entrapment of the lateral femoral cutaneous nerve at the region of the anterior superior iliac spine, when the nerve angulates sharply over the inguinal ligament.1 It presents classically with pain, paraesthesia and sensory loss over the anterolateral surface of the thigh.2 The term “meralgia paraesthetica” was first coined by Roth in 1895 when he described the characteristic syndrome.3 Pathological lesions range from focal demyelination changes to more severe injuries showing Wallerian degeneration.4 Various aetiologies have been implicated, with obesity and trauma being the most frequent.5

Anatomy

The lateral femoral cutaneous nerve is the first sensory branch of the lumbar plexus. It is derived from L2 ,L3 root levels and descends along the pelvic brim into a tunnel formed by the lateral attachment of the inguinal ligament and the anterior superior iliac spine. The nerve divides into anterior and posterior branches 12 cm below the exit from this tunnel. The branches supply the anterolateral and posterolateral aspect of the thigh respectively. Various anatomical differences have been implicated as predisposing factors for the development of meralgia paraesthetica.6

Materials and Methods

A total of 12 cases diagnosed as meralgia paraesthetica between January 1995 and December 1996 using clinical and electrodiagnostic methods were reviewed. The cases were studied and followed up over a period of 6 months to 2 years. Clinical and electrophysiological correlations were drawn with respect to diagnostic and prognosticative aspects.

Each patient had a single study of the lateral femoral cutaneous nerve performed bilaterally using the Dantec Counterpoint machine. A surface electrical stimulator delivered repetitive pulses 0.5 ms in duration at a rate of 1 Hz. Antidromic recording was employed using Dantec 13L20 surface electrodes, with an average of 20 trials in each study.

Nerve conductions were performed in accordance to the method of Stevens and Roselle.7 The active recording electrode was placed 20 cm distal to the anterior superior iliac spine on a line joining the lateral patella border and the anterior superior iliac spine. The stimulator cathode was placed about 1.5 cm medial to the anterior superior iliac spine. As this is a technically difficult study especially in obese subjects, repeated adjustment of the stimulator is required in order that a consistent sensory nerve action potential be obtained at the lowest stimulating current. The method just described is illustrated in Figure 1.

Results

Clinical Features

A total of 8 male and 4 female patients were studied. All patients had similar presentations of unilateral pain and numbness over the anterolateral aspect of the thigh of variable durations, ranging from 2 to 18 months. None

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of the patients studied had underlying diabetes mellitus or other medical conditions. There were no obvious aetiological factors apparent in all but 2 patients. One patient developed symptoms during pregnancy while the other experienced symptoms soon after laparoscopic inguinal hernia repair (Table I).

Electrophysiological Features

Sensory nerve action potentials were compared bilaterally in each patient studied. The nerve conductions on the unaffected side showed sensory latencies ranging from 2.7 msec to 4.6 msec and amplitudes ranging from 2.9 uV to 16 uV. A total of 9 cases out of 12 had absent sensory nerve action potential on the affected side (Group 1). Three out of the 12 cases studied had sensory amplitude reduction greater than 30% compared to the unaffected side (Group 2) (Table I).

Follow Up

All 12 patients were treated symptomatically upon initial diagnosis. They were followed up for a duration of 6 to 24 months for symptomatic improvement. Six of 9 cases (66.7%) in Group 1 did not experience symptomatic reduction over 6 to 18 months of follow up. However, all 3 cases (100%) in Group 2 had significant symptomatic improvement in pain or paraesthesia over 8 to 24 months of follow up. One patient in Group 2, despite having reduced numbness over 6 months of symptomatic treatment, opted for surgical intervention (Table I). The lateral femoral cutaneous nerve was found to be compressed in a narrow tunnel under the inguinal ligament. The nerve was freed, resulting in further reduction of pain postoperatively.

Discussion

The diagnosis of unilateral cases of meralgia paraesthetica is most often made clinically. However, side to side comparison of sensory nerve action potentials provide a useful confirmatory step in making the final diagnosis. It is important for the clinician to bear in mind several conditions which can mimic a case of meralgia paraesthetica. An underlying chronic neuropathic process, like diabetic polyneuropathy, must be excluded. Likewise, lumbar radiculopathy affecting the L2 and L3 sensory roots must also be ruled out. In our experience, the likelihood of an underlying condition present is

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**Table I: Clinical and Electrophysiological Features of 12 Patients with Meralgia Paraesthetica**

<table>
<thead>
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<th>No.</th>
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<td>A</td>
<td>A</td>
<td>3.3</td>
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<td>A</td>
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<td>Amplitude (uv)</td>
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<td>6.4</td>
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<td>A</td>
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<td>A</td>
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<td>7 mo</td>
<td>7 mo</td>
<td>9 mo</td>
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L: left; R: right; A: absent; HR: hernia repair; P: pregnancy; S: surgery; I: improved
mostly seen in cases with marginal reduction in sensory nerve action potential compared to the uninvolved side. Therefore, additional studies of the peripheral nerves, needle electromyogram and neuroimaging of the lumbar sacral spine may be necessary in these cases.

Four of the cases presented in this study (Cases 3, 7, 8 and 10) showed diminished sensory amplitudes of less than 5 uV on the unaffected side. The co-existence of subclinical involvement on the asymptomatic side is speculated. Subclinical entrapment is a well-known and autopsy-proven entity. Bilateral cases have been well documented and rarely exhibit equal involvement of both sides. It is interesting to note that of these 4 cases, only Case 3 had an aetiology of pregnancy. Nonetheless, we still find comparisons of sensory amplitudes useful as a diagnostic adjunct, even in cases showing low sensory amplitudes on the uninvolved side.

It would be incomplete in any study of meralgia paraesthetica not to mention the technical difficulties in performing conduction studies of the lateral femoral cutaneous nerve. Its anatomical position, gross obesity and variations in its course contribute to this. Various methods have been devised in order to overcome these difficulties, each with its own advantages and disadvantages. Butler et al. employed specially constructed lead strip recording electrodes and a needle electrode as a stimulator. Lagueny et al. compared both antidromic and orthodromic stimulation and found that the stimulation artifact was greater with orthodromic stimulation. In this study, we employed antidromic stimulation in accordance to the technique of Stevens and Roselle, and avoided the need for painful, invasive needle electrodes which prove impractical for routine usage. This method provided adequate sensitivity with least discomfort to the patient and is fairly easy to master.

The aetiology of meralgia paraesthetica is probably the point of greatest interest in this condition. Associations ranging from obesity to lipomas have been reported. None of our cases were in any way obese, and implied associations of trauma and pregnancy were apparent in 2 of the 12 cases (Table I). However, it is not uncommon to see that no aetiology seem apparent in a great majority of cases. One large series of 67 cases reported an idiopathic cause as the second commonest finding after obesity. It leads one to speculate the extent that repeated, unrecognised microtrauma plays as a cause of symptoms, resulting from damage to small unmyelinated pain fibres.

The value of the sensory nerve amplitude as a predictor of recovery had only been addressed in one retrospective study. Lagueny et al. concluded that the sensory nerve amplitude was not predictive of long-term symptomatic outcome in this condition. However of the 17 patients in their series, only 6 patients were treated conservatively as compared to all 12 patients in our series. Of the 10 patients with absent sensory amplitudes, 4 (40%) did not experience symptomatic reduction on follow up. This contrast with our experience where 6 of 9 cases (66.7%) did not report symptomatic improvement over 6 to 18 months follow up. All 3 cases (100%) with sensory amplitude reduction showed symptomatic improvement over 8 to 24 months of follow up. This contrasts with Lagueny et al.’s finding where only 5 of 7 cases (71%) had symptomatic improvement. We feel that the likely reason for this disparity lies in the different methodologies employed and the pathogenesis of the disease itself. As it is well documented that the condition preferentially affects sensory fibres of the largest diameter which transmit touch and pressure sensations, their sensory amplitudes would have been adequately reflected with surface recordings in our study. However, the use of invasive needle recordings by Lagueny et al results in sensitive detections of small late components of unmyelinated or regenerated fibres, and its over-representation in the sensory nerve amplitude recordings is likely to give rise to different correlations. Further research into the relation between the aetiology, pathogenesis and appropriate sensory nerve recording technique is necessary in order that a better understanding of this interesting condition be possible.

REFERENCES