

SHORT COMMUNICATION

PHENOL NEUROLYSIS FOR RELIEVING INTERMITTENT INVOLUNTARY PAINFUL SPASM IN UPPER MOTOR NEURONE SYNDROMES: A PILOT STUDY

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Objective: The aim of this study was to assess the efficacy of phenol neurolysis in relieving intermittent attacks of involuntary painful muscle spasm in patients with upper motor neurone syndromes.

Design: Case series.

Patients: Nineteen patients with intermittent involuntary painful muscle spasm of one of the following muscles: extensor hallucis longus ($n=13$), psoas major ($n=3$), tensor fascia lata ($n=1$) or vastus lateralis ($n=2$).

Methods: Phenol neurolysis was performed for the target muscles using a Teflon-coated stainless-steel injection needle (connected to a block stimulator). The change in frequency and severity of intermittent involuntary painful muscle spasm was assessed 1, 2, 8 and 24 weeks after neurolysis.

Results: The frequency and severity of intermittent involuntary painful muscle spasm decreased in all patients for 24 weeks following neurolysis. Analgesic drugs were not required for the intermittent involuntary painful muscle spasm and no serious side-effects were observed following neurolysis.

Conclusion: Phenol neurolysis gave promising results in treating intermittent involuntary painful muscle spasm in patients with upper motor neurone syndromes.

Key words: muscle spasticity, neurolysis, rehabilitation, pain management.

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INTRODUCTION

Spasticity has some disadvantages, including pain (1). Pain associated with spasticity may take the form of intermittent involuntary painful muscle spasm (IIPMS) involving a muscle or a group of muscles. We observed IIPMS at the extensor hallucis longus (EHL), psoas major (PM), tensor fascia lata (TFL) and vastus lateralis (VL). The IIPMS disturbed the patients' life and interfered with their activity of daily living and/or sleep.

Transcutaneous electrical nerve stimulation and oral baclofen may be of little benefit in treating pain related to spasticity (2). Intrathecal baclofen administration might be promising in controlling pain associated with generalized spasticity (3), but this invasive technique may be associated with a relatively high rate of complications (4). Botulinum toxin injection has been used in managing painful spasticity (5). Little is known, however, about the efficacy of phenol neurolysis in treating pain related to spasticity. The aim of this study was to assess the efficacy of phenol neurolysis in relieving IIPMS in patients with upper motor neurone lesions.

METHODS

Patients

Nineteen patients (age range 27–57 years) with daily attacks of IIPMS that involved mainly one muscle, for 3–12 months duration. There were 4 groups: (i) 3 women and 10 men with partial spinal cord injury (PSCI), and IIPMS of the EHL; (ii) 3 women with PSCI and IIPMS of the PM; (iii) one woman with PSCI and IIPMS of the TFL; and (iv) 2 patients with IIPMS of the VL (one man with cervical myelopathy and one woman with multiple sclerosis). All participating patients were ambulatory, had grade 2–3 spasticity according to the modified Ashworth scale at the involved extremity, and experienced some intolerable attacks of IIPMS (lasting for minutes) for which they had to take rapidly acting analgesic drugs. The selected patients had no prior history of botulinum toxin injection, phenol neurolysis or surgery for spasticity. Once enrolled in the study, patients did not take any anti-spasticity medication.

Neurolysis of the muscular branches

The stimulator reference electrode was fixed over the medial malleolus during neurolysis of the EHL or over the anterior superior iliac spine (ASIS) for neurolysis of PM, VL or TFL. The negative pole of the stimulator was connected to the needle. The needle was inserted at the appropriate neurolysis point (NP) for the target muscle according to Awad NP charts (6). Using low intensity electrical stimulation (1–3 mA), the needle was manipulated until maximal response at the target muscle occurred with the least current intensity (approximately 1 mA). When proper localization was achieved, 1 ml of 5% phenol solution in water was injected at each NP. Aspiration was carried out prior to phenol injection in order to avoid intravascular injection.

The NP of the EHL was located at the anterolateral surface of the middle third of the leg. The 2 NP for the PM are located at the paralumbar sulcus at the level of the 2nd and 3rd lumbar vertebrae. The needle was directed medially and forward at a 45° angle with the sagittal plane and advanced until it reaches the bone, then retracted, reintroduced at

decreasing angles and manipulated until PM contraction occurred. The NP for the TFL is located approximately 50 mm posterior and distal to the ASIS. The NP for the VL is lateral to the femoral artery and is located 75 mm distal and medial to the ASIS (6).

Assessment

The IIPMS frequency (the number of IIPMS during the last 2 days prior to the assessment day) was assessed just prior to neurolysis and 1, 2, 8 and 24 weeks after neurolysis. Pain relief at the time of assessment (compared with pre-neurolysis pain severity) was assessed (in a single blind way) 1, 2, 8 and 24 weeks following neurolysis as complete, marked, moderate, minimal, or no relief. The paired *t*-test was used in data analysis for the change in the frequency of the IIPMS of the EHL (patient numbers =13). The need for analgesic drugs during any IIPMS, and the appearance of any side-effect (e.g. pain, paraesthesia, swelling and/or weakness) were recorded. Failure of neurolysis was considered when the patient did not experience complete-moderate pain relief within 2 weeks following neurolysis. This study was carried out in accordance with ethical standards for studies involving human subjects.

RESULTS

The severity and frequency of the IIPMS decreased in all patients 1–24 weeks after neurolysis. Failure of neurolysis was not encountered in any patient. In patients with partial improvement after neurolysis, the IIPMS became tolerable without the need for taking any analgesic drug as IIPMS became not only less severe, but also of short duration (few seconds).

Following neurolysis of the VL, PM and TFL, there was at least a marked decrease in the severity of the IIPMS for 24 weeks, except in 2 patients with PM IIPMS (who showed moderate improvement). The frequency of the EHL IIPMS decreased significantly ($p < 0.01$) 1–24 weeks after neurolysis. Also, there was complete-marked pain relief in 11 (85%) patients 1–2 weeks following neurolysis of the EHL. At 8–24 weeks following neurolysis, complete-marked pain relief of the EHL IIPMS was encountered in 12 (92%) patients.

All patients reported mild-moderate local pain following neurolysis, which disappeared within 1–3 days. Mild ankle oedema was observed in 4 (30%) patients after neurolysis of the EHL. This disappeared within 1 week. Paraesthesia at the front of the thigh occurred in 1 patient after neurolysis of the PM. It had disappeared completely by the third post-neurolysis assessment. No change in muscle power was observed following neurolysis.

DISCUSSION

Phenol neurolysis was successful in decreasing the frequency and severity of IIPMS. However, it may be less promising in treating PM IIPMS; in this condition two-thirds of patients experienced only moderate improvement. In addition, phenol neurolysis had a relatively long duration of effect (24 weeks). This is consistent with the opinion of other authors (7). The absence of motor weakness following neurolysis could be due to the induction of partial and not complete neurolysis (6). The absence of any persistent swelling may have been due to the avoidance of intravascular injection. The mild paraesthesia at the thigh that occurred in only one patient following neurolysis of PM may be due to the fact that the injected muscular branches for the PM were close to the lumbar nerve roots.

In conclusion, phenol neurolysis appeared to be promising and safe in relieving the IIPMS when the neurolysis was performed in the appropriate way.

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