Prolotherapy for Recalcitrant Lumbago

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Abstract

Recalcitrant lumbago with a mean duration of 5.5 years in 41 consecutive patients presenting over a one-year period was treated effectively with a series of subcutaneous prolotherapy treatments. 90% improved by more than 50% from an initial mean Visual Analogue Score (VAS) of 7.6, with 29%, reaching VAS 0 at a mean treatment length of 8.3 weeks. Subcutaneous prolotherapy treatment has been shown to be highly effective in the treatment of a variety of peripheral neuropathic painful conditions. This clinical practice is founded on a proposed working hypothesis that subcutaneous prolotherapy is a suitable, effective and cheap treatment for prolonged pathological peripheral neurogenic inflammation.

Introduction

The subcutaneous prolotherapy protocol was in the first instance developed by the author for the effective treatment of Achilles tendinosis.[1, 2] Widening the scope of subcutaneous prolotherapy to treat chronic exertional compartment syndrome (CECS) was published in 2005.[3] Subsequently the protocol was tested on shoulder, knee and lateral elbow pain with promising results. This was documented and published in 2007.[4]

The challenge of treating lumbago with the same protocol seemed more difficult at first. A series of different approaches evolved into a working hypothesis satisfactorily addressing the key clinical demands of "how to diagnose and treat", "safety" and "rationale".

Quintessential to the working hypothesis that subcutaneous prolotherapy treats prolonged pathological peripheral neurogenic inflammation is the work by Douglas W Zochodne from the Neuroscientific Research Unit at Calgary University.[5, 6]

In their Chronic Constriction Injury (CCI) animal model the resulting peripheral neuropathic pain was treated with "percutaneous near nerve injections". Their solution targeted μ opioid receptors (MOR) on neuropathic peripheral nerves, effectively modulating neuropathic pain.

Closer scrutiny of the most effective way to administer subcutaneous prolotherapy suggests that "percutaneous near nerve injections" of neuropathic peripheral cutaneous nerves gives the best outcome.

In the clinical setting of a lumbago consultation focussing on peripheral neuropathic pain, pain history and examination strongly suggested the intermediate and superior cluneal nerves to be the most frequently affected peripheral nerves. The ramifications of these findings are still evolving.

The safety of subcutaneous prolotherapy has been well documented in the past,[1, 2, 3, 4] and is no different in this clinical study. Adverse effects of localized tenderness and bruising are no different from other injection treatments and there were no reports of worsening low back pain as a result of the treatment.

Methods and results

Every patient was prospectively monitored with a recovergram.[7] This is a quick and useful evidence-based monitoring tool and has been used in this practice for over five years on all treated patients.

All individual recovergrams were collated in a "study" recovergram. Mean values were calculated for age, length of symptoms, initial VAS, VAS at last treatment, length of treatment in weeks and number of treatments.

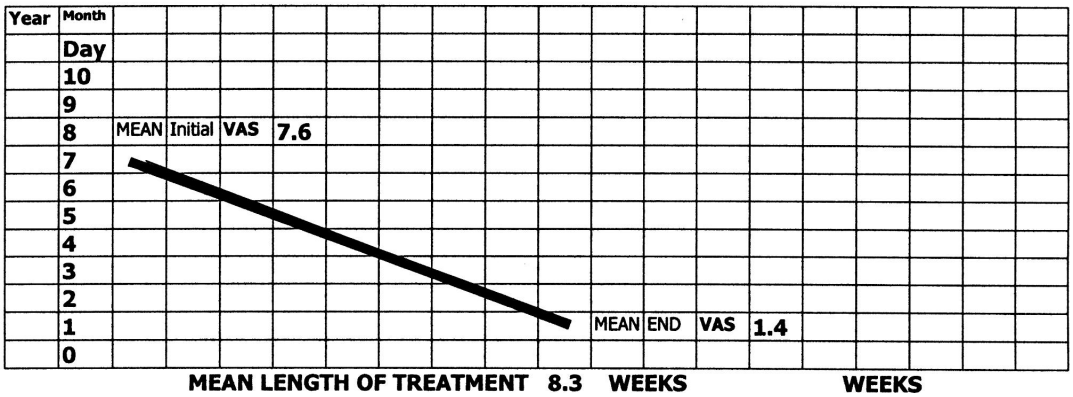
The solution used in the earlier part of the audit was hypertonic dextrose 20-40%, mixed with 0.1% lignocaine and/or ropivacaine 0.1% in normal saline.

Towards the end of the treatment the solution consisted of dextrose 20%, lignocaine 0.1% and cholecalciferol 1000 IU/ml in normal saline.

In the earlier phase of the audit period "tender points" were targeted mainly along the latissimus dorsi tendons, the gluteus maximus origin and the supraspinous ligament.

In the latter phase the focus became the "inflamed" superior and intermediate cluneal nerves and thoracic spinal nerves where clinically indicated. The swollen and tender nerves were clinically identified and treated with "percutaneous near nerve injections" approximally 1 ml

Lumbago 2008 audit recovergram. VAS: visual analogue scale 0 = no pain 10 = worst imaginable pain



every 2 cm. The objective of the treatment was to achieve a complete local anesthetic response for all low back pain at the time of the treatment.

All 46 patients presenting with lumbago with or without leg pain from 1 February 2007 to 1 February 2008 were included in the clinical audit. Two patients were subsequently diagnosed with an underlying hip pathology and referred for surgical treatment. Two patients did not return after the initial consultation and one patient preferred chiropractic treatment after two prolotherapy sessions.

Of the remaining 41 patients 24 (58%) were male and 17 (42%) female. Mean age was 48.3 (range 23-73) years. Mean duration of symptoms was 5.5 years (range 1- 264 months). Mean initial VAS was 7.6 (range 5-10). Mean VAS at last treatment was 1.4 (range 0-6), mean duration of treatment was 8.3 weeks (range 1-17) and the mean number of treatments was 6.2 (range 2-16).

Ninety percent of patients improved more than 50%, and 10% less than 50%. Twenty-nine percent of patients reported no pain at the last consultation.

Discussion

Peripheral neuropathic pain is the subject of a recent avalanche of clinical and basic scientific research papers.[5, 6, 8-13]

"Peripheral nerve damage arising from trauma and disease is common and disabling",[5] according to Douglas Zochodne in his seminal review on peripheral nerve injury.

He goes on to say that "peripheral nerves respond to injury in a unique fashion". Injured peripheral nerves develop hyperemia with increased endoneurial blood flow (EBF) rather than ischemia as in other tissues following trauma. The nervi nervorum innervating the connective tissue of peripheral nerves are thought to be responsible for the ensuing "neurogenic inflammation", with the clinical signs of rubor, calor, dolor and tumor caused by "dumping" of neuropeptides calcitonin gene related peptide (CGRP), substance P (SP) and nitric oxide from axonal endbulbs, originally described by Cajal.

CGRP, SP and nitric oxide are potent vasodilators and markedly increase permeability of nearby blood vessels, resulting in painful swollen peripheral nerves.

In contrast to other tissue trauma where cell necrosis and arachidonic acid release induce "tissue inflammation", "neurogenic inflammation" is caused by nervi nervorum alone.

The "efferent secretory" or "peptidergic noceffector"[11] phenotype of nervi nervorum causing neurogenic inflammation through the dumping of CGRP, SP and nitric oxide is followed by chemo-attraction of mast cells and macrophages initiating a "trophic" phase,[5] with an ever-increasing number of newly discovered trophic factors. If successful, this phase is followed by regeneration and resolution of pain, swelling and disability.

"The mechanisms involved in the transition from acute to chronic pain are complex, with involvement of interacting receptor systems and intracellular ion flux, second messenger systems, new synaptic connections and apoptosis", according to Vadivelu and Sinatra from Yale University of Medicine.[13]

One of the clinical implications of unsuccessful regeneration and development of chronic peripheral neuropathic pain is inflammation of the nerve trunk. This is best diagnosed by pressure palpation as described by Dr Geoffrey Bove, a neurophysiology researcher at Harvard Medical School. "If a nerve is compressed, one gets numbness. If an inflamed nerve is compressed, one often feels pain." Bove is also the co-author of an article identifying nervi nervorum as nociceptors.[14]

Pressure palpation then becomes a useful clinical tool for identifying neuropathic pain in the low back arising from peripheral subcutaneous nerves.

The author's clinical experience suggests that the intermediate and superior cluneal nerves are most often involved but not exclusively so.

Treating the pathological peripheral neuropathic nerves with "percutaneous near nerve" injections with local anesthetic, resulting in elimination of all clinical symptoms of lumbago, confirms the diagnosis.

The above forms the basis for a successful subcutaneous prolotherapy treatment.

During the course of the one year audit, a number of recent articles identified vitamin D hormone (VDH) deficiency as a probable etiological factor in low back pain and other musculoskeletal pain.[15-17] Subsequent testing of vitamin D levels showed more than 90% of patients with lumbago to be VDH deficient. Because cholecalciferol has been shown to be neuroprotective through its modulating action on voltage-gated calcium channels (VGCC), cholecalciferol was added to the prolotherapy solution at a very low dose of 1000 IU per ml towards the end of the audit period. In addition patients were prescribed a standard oral cholecalciferol treatment.

Initial clinical impressions of the addition of cholecalciferol are favourable but not enough data have been collected to make any firm conclusions.

Conclusion

Peripheral neuropathic pain caused by neurogenic inflammation is a well-documented clinical entity. It is clinically identified by palpation of inflamed peripheral cutaneous nerves. Subcutaneous prolotherapy with a series of percutaneous near nerve injections has been shown to be an effective treatment for a variety of recalcitrant painful conditions caused by prolonged neurogenic inflammation.

Good results are achieved in this clinical audit on recalcitrant lumbago.

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