

Case Series

A New Perspective of Neuromyopathy to Explain Intractable Pancreatic Cancer Pains; Dry Needling as an Effective Adjunct to Neurolytic Blocks

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ABSTRACT

We present a new perspective of neuromyopathy in pancreatic cancer pain (PCP) referral to bodywall; proposal of new rationale to include ultrasound guided dry needling (USGDN) of body wall muscles as an effective adjunct to neurolytic coeliac plexus block (NCPB) or splanchnic nerve radiofrequency ablation (SRF) for comprehensive interventional management. Methods: PCP response to SRF in 2 patients and NCPB in 3 patients was documented on numerical rating scale (NRS) on post procedure days 3 and 15. If the residual pain was >5 NRS on day 15, USGDN of abdominal and back muscles was started on a thrice weekly basis. The response to USGDN documented on day 30 after approximately 6 sessions of DN, showed a significant pain reduction (0-2 NRS) with 50% reduction of pre-treatment opioid consumption. This was sustained at 6 months or till their demise. Convergence of visceral and somatic nerves at the dorsal horn (viscerosomatic neurons) causes referral of visceral pain to the back and abdominal muscles. This leads to formation of myofascial trigger points (MTrPs) in the muscles which sets up a parallel network of sensitized peripheral and central motor nociceptive processing (neuromyopathy). USGDN specifically addressed the MTrPs that develop as an epiphenomenon of self-perpetuating neuromyopathy while SRF/NCPB, analgesics and neuromodulators could address only visceral nociceptive afferents (pain mediated through coeliac plexus) which forms a meagre 10% of the total spinal cord afferent input. Thus, we conclude that combination of neuromyopathy and viscerosomatic convergence in PCP indicate a specific role for DN as an adjunct to SRF/NCPB in our patients

Key words: Myofascial trigger points, Neuromyopathy, Pancreatic cancer pain, Ultrasound guided dry needling, Viscerosomatic convergence

INTRODUCTION

The course of pancreatic cancer (PC) is characterized by severe intractable pain. As recommended by Christo and Mazloomdoost (2005),^[1] Tay and Ho (2009),^[2] Sharfman and Walsh (1990);^[3] the treatment of advanced PC is palliative care with medications by World Health Organization's Pain Relief Ladder and if necessary, interventions such as neurolytic coeliac

plexus block (NCPB) or radiofrequency (RF) ablation of splanchnic nerves (SRF), which is considered as step 4 in the analgesic ladder. Yan and Myers (2007)^[4] conducted a review of literature for RCTs conducted from 1996 to 2005 to compare the efficacy of NCPB with standard treatments in pancreatic cancer pain (PCP). They concluded that the data available on NCPB for

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How to cite this article: Vas L, Phanse S, Pai R. A new perspective of neuromyopathy to explain intractable pancreatic cancer pains; Dry needling as an effective adjunct to neurolytic blocks. *Indian J Palliat Care* 2016;22:85-93.

Access this article online

Quick Response Code:



Website:
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DOI:
10.4103/0973-1075.173957

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PCP were insufficient to judge for efficacy, long-term morbidity, or cost-effectiveness, since NCPB shows only a minimal clinical significance in improving pain control, reduced narcotic usage, and constipation when compared with medical management.

The neural pathways in PCP involve celiac plexus, as well as body wall innervations.^[5-8] A variety of pathophysiological processes have been identified to explain the referral of pain to parietal somatic structures of the body wall. NCPB/SRF targets only the pain mediated through celiac plexus but has no effect on the referred pain to the body wall, which persists even after NCPB/SRF.

Our therapy protocol for all abdominal pains in the last 10 years is a combination comprising SRF/NCPB to address visceral nociceptive nerve supply and ultrasound guided dry needling (USGDN) of the abdominal wall and back muscles to address the referred pain to the muscles of the body wall. We report its efficacy in achieving a comprehensive management of PCP in 5 patients specifically documented for the purpose of this report.

CASE REPORTS

Table 1 shows a brief description of the case presentation, medications, Numerical Rating Scale (NRS) of the pain, and quality of life scores (Short form-12 version 2 of Short form- 36 (SF-36) of health survey as formulated by Ware *et al.*^[9]) of our patients. Patients' consent was taken for SRF/NCPB as well as USGDN (an established treatment to relieve myofascial trigger points [MTrPs] known to cause myofascial pains) of the pain in abdominal and back muscles, if the pain 15 days after SRF/NCPB was more than 5 NRS despite continued opioid and neuromodulator medications. A thorough explanation of risks and expected outcomes of all the three procedures were given.

METHODS

The patients were positioned prone on the radiolucent table in the operation theater for SRF/NCPB. An intravenous line was secured for administration of a liter of Lactated Ringers solution, as well as 100 ml of paracetamol 10 mg/ml and tramadol 50 mg.

SRF procedure

Under C-arm guidance, three 15 cm, 22-gauge RF cannulae (Cosman [cannula] RFK TM) were positioned

so that the exposed curved active tip of 10 mm, lay at the anterior one-third of the vertebral bodies of 11th and 12th thoracic, and first lumbar vertebrae [Figure 1]. The tip position was reconfirmed with the spread of the nonionic dye Iohexol (Omnipaque® 300 mg l/ml GE Healthcare, Shanghai, China). A positive response to sensory stimulation at 0.6 V at 50 Hz (denoted by sensation of vibration in epigastrium) and a negative response to motor stimulation at 2.0 V at 2 Hz (denoted by the absence of visible twitches in abdomen or back or lower extremity) were documented. This was followed by injection of 2 ml of 2% lidocaine before activation of the RF generator (Cosman Medical Inc., Burlington, MA 01803, USA) for 2 min at 80°C. The procedure was repeated on the other side.

Transaortic neurolytic celiac plexus block procedure

Under C-arm guidance a 15 cm, 22-gauge Quincke needle was positioned at the antero-lateral instead of anterior lateral aspect of the vertebral body of L1 on the left side. The needle was advanced anteriorly till the back flow of arterial blood through a three way extension tubing was visualized. The needle was advanced till the back flow stopped, just as the needle exited the aorta with a gentle pop. The tip position was reconfirmed with the spread of iohexol around the pulsating aorta in lateral view [Figure 2]. Once the predominantly anterior periaortic spread was confirmed, 5 ml of lignocaine was injected. Five minutes later, after confirmation of pain reduction and well-maintained vital parameters, 15–20 ml of absolute alcohol was slowly injected. The alcohol was flushed out with another milliliter of lignocaine prior to needle withdrawal through the aorta and the paravertebral tissues. The vital parameters, bowel movements, and pain relief were monitored for another 2 h prior to discharge.

The response of pain to SRF/NCPB was assessed on post procedure days 3 and 15. The first assessment was done on day 3 when patients were able to perceive the effect of SRF/NCPB after the procedural pains had subsided. The second assessment was done at 15 days as presumably the neuritic phenomena from the thermal effects of RF would have subsided by then,^[10] enabling the patient to appreciate the extent of relief from SRF.

On day 15, if the residual pain was >5 by NRS, USGDN of abdominal and back muscles was started on a thrice weekly basis. The rationale the 15 days wait was to confirm the effect of SRF/NCPB prior to starting another intervention like DN.

The patient was put in a lateral position with the painful side up, and the back and abdomen were cleaned with betadine.

Table 1: Details of the patient presentation, medications, pain profile at various stages of treatment and patient perception of Health score summaries at various stages of treatment

Case history	Medications	NRS - 1 st visit	Post-RF NRS	PHS/MHS	Final NRS 15 days post-DN
<p>Case No 1: A 55-year-old lady with terminal neuroendocrine tumor of the pancreas with severe pain in the epigastrium, radiating to flanks, and posteriorly to whole back up to the neck. She had extensive metastases in liver, lungs, sternum, spine, and pelvic bones. She had completed 5 cycles of chemotherapy and had undergone panhysterectomy in the past</p> <p>Abdominal examination showed severe guarding and rigidity of abdominal muscles. There was a severe spasm in her lumbar, thoracic, and cervical paravertebral muscles, which were palpable as ropey bands. Her liver and kidney functions were normal. Her consent for splanchnic RF and DN was secured</p> <p>Immediately after the RF, the patient who had remained fairly comfortable and cooperative during the RF procedure developed severe pain in epigastrium radiating to the upper and lower back. The pain was continuous with a score of 10/10 NRS. Refractory to additional tramadol 50 mg, fentanyl 50 µg bolus, and 100 µg infusion, and midazolam 2 mg. The patient became increasingly restless till USGDN of abdominal and back muscles was done. Following DN her pains appeared to reduce steadily along with her restlessness. At the end of 1 h, she was fairly comfortable, with almost no pain. She was sent to native place 15 days after twice weekly USGDN sessions with all NRS 0-1/10 with normal sleep and appetite. Medications continued except narcogin forte tramadol SR, fentanyl patch and oral transmucosal fentanyl SOS</p>	<p>Morcontin - 10 mg qds Fentanyl patch - 50 µg/h Lollipop SOS tramadol SR - 100 mg Narcogen forte - b.d Amitriptyline - 10 mg tds Pregabalin - 75 mg h.s</p>	<p>5-6/10-10/10 Every 15-20 min</p>	<p>10/10 constant 2 days post-RF and DN 50-60% pain relief</p>	<p>BT - 16/19 AT - 28/46</p>	<p>After 5 USGDN Session 90% pain relief, NRS 0-1/10 till date 3 m later at 40% of pre-DN opioid intake</p>
<p>Case No 2: A 52-year-old man with cancer of pancreas, extensive visceral, and bone metastases presented with severe pain in back radiating to the abdomen. Complete disruption of sleep and appetite. RF performed after consent. He reported 50% pain relief 3 and 15 days after RF with NRS of 3-4/10 with mild paroxysms of 5-6 once in 3-4 h. Sleep was still disturbed but was better than the pre-RF status, USGDN started 15 days after observing the RF effect. Totally, 5 sessions performed over 15 days relieved the pain to 0-2 NRS with normalization of sleep and return of appetite, which he maintained till death 5 months later</p>	<p>Morcontin - 20 mg qds Fentanyl patch - 25 µg/h Amitriptyline - 10 mg tds Pregabalin - 75 mg h.s</p>	<p>7-8/10-10/10 every hour</p>	<p>NRS 4/10 at 3 and 15 days but back to a steady 7/10</p>	<p>18/19 - BT 25/44 - AT</p>	<p>NRS - 0-2/10 till demise 5 m later at 40% of pre-DN opioid intake</p>
<p>Case No 3: A 48-year-old man with cancer of pancreas with extensive visceral and bone metastases presented with severe pain in abdomen radiating to the back. Complete disruption of sleep and appetite. Alcohol neurolysis of celiac plexus with single needle transaortic approach carried out after consent</p> <p>He reported 75% relief of pain over abdomen but continuing back pain of 6-7 NRS 3 and 15 days after neurolysis. USGDN started 15 days after observing the neurolysis effect. Totally, 5 sessions of USGDN performed over 15 days relieved the pain to 0 NRS with normalization of sleep and return of appetite, which he maintained till death 6 months later</p>	<p>Morcontin - 20 mg qds Fentanyl patch - 50 µg/h Amitriptyline - 25 mg tds Pregabalin - 75 mg b.d.</p>	<p>10/10 NRS</p>	<p>NRS 4/10 at 3 and 15 days but back to a steady 6-7/10</p>	<p>18/12 - BT 28/48 - AT</p>	<p>NRS - 0-2/10 till demise 6 m later at 50% of pre-DN opioid intake</p>
<p>Case No. 4: A 50-year-old man with cancer of pancreas, extensive visceral, and bone metastases presented with severe pain in abdomen radiating to the back. Complete disruption of sleep and appetite. Alcohol neurolysis of celiac plexus with single needle approach carried out after consent. He reported 80% relief of pain over abdomen but continuing back pain of 6-7 NRS 3 and 15 days after neurolysis. USGDN started 15 days after observing the neurolysis effect. Totally, 5 sessions of USGDN performed over 15 days relieved the back and abdominal pain to 0 NRS with normalization of sleep and return of appetite, which he maintained till death 8 months later</p>	<p>Morcontin - 20 mg qds fentanyl patch - 50 µg/h Ultracet qds Amitriptyline - 25 mg tds Pregabalin - 75 mg tds</p>	<p>8-9/10-10/10 every 10 min</p>	<p>NRS 3/10 at 3 and 15 days but back to a steady 6-7/10</p>	<p>8/10 - BT 26/54 - AT</p>	<p>NRS - 0-2/10 till demise 8 m later at 40% of pre-DN opioid intake</p>
<p>Case No. 5: A 60-year-old man with cancer of pancreas, extensive visceral, and bone metastases presented with severe pain in abdomen radiating to the back. Complete disruption of sleep and appetite. Alcohol neurolysis of celiac plexus with single needle approach carried out after consent</p> <p>He reported 90% relief of pain over abdomen but continuing back pain of 7-8 NRS 3 and 15 days after neurolysis. USGDN started 15 days after observing the neurolysis effect. Totally, 5 sessions of USGDN performed over 15 days relieved the back and abdominal pain to 0 NRS with good sleep and return of appetite, which he maintained till death 4 months later</p>	<p>Morcontin - 20 mg qds Ultracet qds Amitriptyline - 10 mg tds Pregabalin - 75 mg b.d.</p>	<p>7-8/10-10/10 every 2-3 h</p>	<p>NRS 1-3/10 at 3 and 15 day but back to a steady 7-8/10</p>	<p>18/20 - BT 25/50 - AT</p>	<p>NRS - 0-2/10 till demise 4 m later at 40% of pre-DN opioid intake</p>

qds: 4 times daily; tds: thrice daily; SOS: On demand for breakthrough pains; b.d.: Twice daily; TDP: Transdermal patch; µg: Micrograms/h; mg: Milligrams; h.s.: At night; Narcogen forte: Contains 30 mg codeine and 650 mg paracetamol; SF12 V2: Is short form 12 version 2; It shows a ratio of PHS over MHS; BT: Before treatment; AT: After treatment; PHS: Physical health summary; MHS: Mental health summary; RF: Radio frequency; DN: Dry needling; USGDN: Ultrasound guided dry needling; NRS: Numerical Rating Scale

The linear 6–13 MHz probe of Sonosite™ MSK (USA) was used to guide solid 32-gauge needles of 25–50 mm length into all the abdominal and paravertebral muscles that form the

ellipse of abdomen [Figures 3a and b]. The needle trajectory into the relevant muscle was visualized on the US as shown in Figure 3c-e. These needles can be clearly visualized in-plane

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with experience. The tip of the needle seen as bright spots in out-of-plane view was constantly kept in view to confirm entry into specific muscles, as well as avoid accidental needle injury to underlying peritoneum and viscera.

The paravertebral muscles including psoas were also visualized from a T3-4 level down to the origin of erector spinae on the sacrum to place needles in the muscle mass [Figure 3b].

In the abdominal wall, about 7 pairs of needles were placed in rectus abdominis equidistantly from the xiphisternum to symphysis pubis [Figure 3a]. Care was taken to visualize these needles in plane superficial to the easily visible peritoneum and gut. The needles were kept *in situ* for 20–30 min and then removed.

RESULTS

In Table 1, patient 1 [Table 1] complained of a severe exacerbation of pain immediately post-SRF. She was given intravenous tramadol 100 mg followed by fentanyl 50 µg as bolus along with midazolam 2 mg. A further 150 µg of

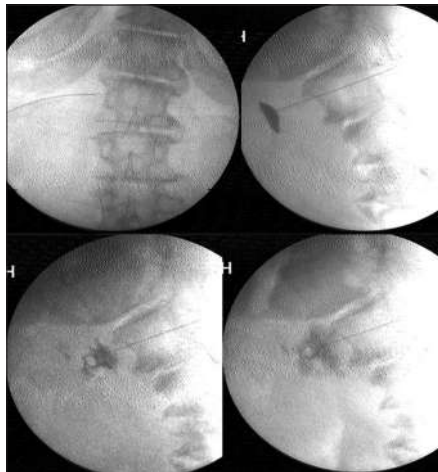


Figure 1: Transaortic approach to celiac plexus

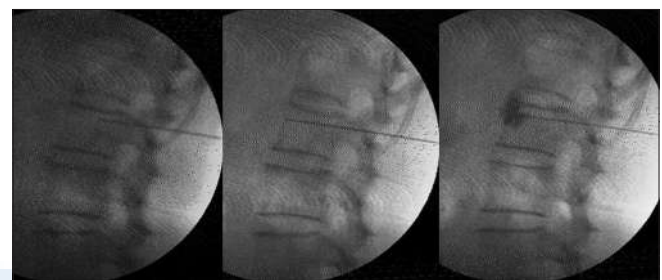


Figure 2: Splanchnic radiofrequency at T12

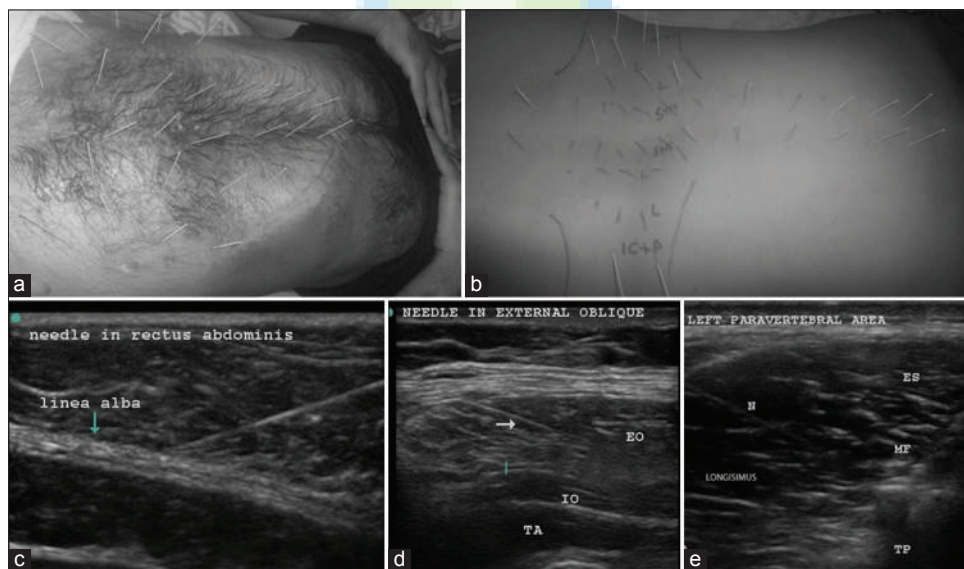


Figure 3: Upper row; abdominal muscle DN (a); in the abdominal wall about 7 pairs of needles were placed in rectus abdominis of each side equidistantly between the xiphisternum to the pubis. Three pairs were supraumbilical, 3 pairs were infraumbilical, and 1 pair was on either side of the umbilicus. About 3–4 needles were placed at a distance of 3–4 cm lateral to the needles in rectus abdominis, and another 3–4 needles were placed a further 3–4 cm lateral to the latter to target external and internal oblique muscles, as well as transversus abdominis between the costal margin and the inguinal ligament. The dark stains are betadine used as a medium for USG probe. The costal margin is marked with a blue line. Paravertebral muscle DN (b); the point of insertion was about 1 cm lateral to the spinous process on either side and medial to the facet for the spinalis and multifidi. An out of plane USG was used to visualize the needle tips clearly in the spinalis. The Figures b and e shows the length of the needle “in plane” in the longissimus. Longissimus and iliocostalis part of erector spinae were needled 4–6 cm lateral to the spine below the costal margin. The psoas at L3-5 on either side was visualized to place 3–4 needles in the muscle mass by starting far laterally so that the needle could slip beneath the transverse process (6–8 cm from the spinous process). The muscles targeted by the needles are marked on the figure as follows: S + M – Spinalis + Multifidus, L – longissimus, IC + P – Iliocostalis + Psoas. The costal margin and the iliac crests are also marked, the second row; USGDN: Needles (indicated by N or arrow) are visualized in rectus abdominis (c) EO: External oblique; (d) IO: Internal oblique, TA: Transversus abdominis, ES: Spinalis part of erector spinae, MF: Multifidus, TP: Transverse process; N: Needle in the longissimus (e)

fentanyl was added in the infusion, but her pain continued to escalate over the next 1 h. Hence, an emergency USGDN session was performed with the assumption that this pain crescendo was a myofascial pain syndrome (MPS) from an exaggerated referral to abdominal muscles, since the visceral component had just been addressed by SRF. This immediately reduced her pain to 3–4 NRS. Subsequently, she continued to receive thrice weekly USGDN for next 2 weeks when she reported 0–1 NRS pain.

Patients 2–4 [Table 1] reported a persistent guarding, rigidity, and pain of at/above 5 NRS 15 day post-SRF/NCPB, and hence, USGDN was started. Our patients reported significant pain relief (to 0–2 NRS), within a week (after 3 DN sessions). Opioid consumption was reduced to 50% of the pre-treatment intake. We did DN for another week to ensure that most/all MTrPs were resolved.

Thus, we started DN 15 days after SRF/NCPB and did 6 DN sessions over 15 days, thereby completing the whole treatment within 30 days. All the patients reported continued pain relief at 6 months or until their demise.

DISCUSSION

PCP is a complex syndrome caused by many mechanisms. About 70–85% patients report severe neuropathic pain resulting from PC.^[11-12] Metastases and treatments such as chemotherapy and radiation can also result in pain.^[13,14] Neuropathy in PC starts with tumor spread along pancreatic nerves causing nerve damage, loss of neural sheath, and stretching of neural tissue at local ganglia up to the celiac axis. The concomitant release of neurolytic pancreatic enzymes causes pancreatitis. The increase in interstitial and ductal pancreatic pressure induces pancreatic ischemia or “pancreatic compartment syndrome.” All these chemical and mechanical stimuli lead to peripheral and subsequent central sensitization.^[15] Hitherto “silent” mechanically insensitive afferents acquire mechanosensitivity to further increase the nociceptive barrage.^[16-19]

Nociceptive afferent A delta and C sensory fibers accompany both sympathetic and parasympathetic ramifications on the viscera into the celiac plexus, which acts as a relay station for the sympathetic, parasympathetic, and intrinsic visceral afferents. The greater (T5-T9), lesser (T10-T11), and the least splanchnic (T12) nerves carry the nociceptive fibers from celiac plexus to their cell bodies in laminae 1 and 5 of the dorsal horn through the white rami.^[20,21]

“Phenotype switch” described by Sengupta,^[18] and Regan and Peng^[22] is a second mechanism, whereby there is

axonal sprouting of A beta fibers into nociceptive C-fiber locations with an expression of nociceptive mediators such as substance P from A beta fibers. Consequently, the low-threshold information from large A beta afferents that is normally perceived as touch may now be misinterpreted by the nervous system as pain.^[22-23] This leads to a state of hyperexcitability in the “wide dynamic range” neurons of dorsal horn with multisegmental spread of noxious input, which eventually overwhelms the “gate control” allowing the sensitization to proceed to cortical centers.^[21,23,24] The altered supra-spinal facilitatory and inhibitory impulses also modulate the dorsal horn, further aggravating the symptoms.^[25-27] Thus, in “centrally sensitized” visceral pain states, there is a generalized amplification of pain even in the reciprocally innervated body wall structures.^[5,19,22,23,25,26,28]

The third and the most important mechanism in PCP is “viscerosomatic convergence,” which is the rule in visceral pain. All dorsal horn cells that receive input from the viscera also receive input from receptors in the body wall (viscerosomatic neurons).^[5,16-18,25] Further, the relative contribution of visceral afferent fibers to the total spinal cord afferent input is < 10% while somatic afferent fiber contribution from the body wall muscle is abundant, paving the way for visceral nociception to be referred to the body wall. Thus, true visceral pain from pancreas forms only a small component of the severe pain experienced by the patient. The larger component of the pain perceived by the patient results from the referred pain to the muscles of abdomen and back, or extremities, and creates a regional MPS. As described by Sikandar and Dickenson,^[5] and Gerwin,^[25] the genesis of MPS starts with the formation of MTrPs in corresponding somatic segments of referred visceral pain. Mechanisms such as dichotomizing or split sensory fibers, afferent-afferent interactions with orthodromic and antidromic impulses, and sympathetic reflexes to the skin causing fluid extravasation and edema have been proposed to explain the trophic changes and formation of MTrPs. Initial sustained muscle contraction/tightness (which may be visible as a taut band), causes latent MTrPs, which are not necessarily painful. However, the latent MTrPs once formed progress to active MTrPs,^[29] which are painful and respond with severe exacerbation of pain with each triggering wave of visceral nociception. Active MTrPs are the areas of increased spontaneous electrical activity (SEA) or end-plate noise (EPN).^[30] The increase in EPN results in a reduced pain threshold and increased pain intensity as described by Kuan *et al.* (2007).^[31] MTrPs become an independent source of persistent nociceptive input to the spinal cord, which is reported to be especially effective in inducing

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neuroplasty in the dorsal horn.^[23,29] This muscle nociception is independent of the viscerosomatic convergence and maintains a parallel central sensitization.^[24,26,29,32] In PCP, the shared innervation between pancreas and muscles leads to MPS in these muscles. The fascia covering abdominal muscles is in anatomical continuity with thoraco-dorsal fascia enveloping the back muscles.^[20] Tightening of the abdominal ellipse formed by muscles, fascia, and skin is perceived as abdominal guarding and rigidity [Figure 4]. Sensitized muscle nociceptors in the abdominal and back muscles with a lowered stimulation threshold manifest with symptoms such as allodynia and mechanical hyperalgesia with muscle movement (on breathing, coughing, movements, etc.). Thus, a vicious cycle of combined visceral and parietal nociception sets in, leading to abdominal and back muscle spasm, which in turn leads to more pain, and more sensitization (diagrammatically represented in Figure 5). Descriptions by Sikandar and Dickenson,^[5] Chen and Wai,^[15] Regan^[22], and Gerwin^[25] confirm our observation that the referred pain and muscle hyperalgesia in PCP occurs early, is accentuated by repeated

episodes of colic, and remains long after the original visceral nociceptive stimulus has resolved.

We have proposed the concept of “neuromyopathy” as a novel perspective of neuropathy in nerves responsible for both muscle pain, sensation, and motor action. We have proposed that neuromyopathy mediates the secondary MPS, which causes the pain in various diverse pain syndromes. Some examples are knee pain, presently referred to as pain of knee osteoarthritis^[33] and postsurgical neuropathic pain after knee replacement surgery,^[34] and in a case of refractory camptocormia occurring as a complication of RF ablation of medial branch to facet.^[35] We believe that it plays a major role in the causation of complex regional pain syndrome as well.^[36-39] We have also proposed neuromyopathy in the pain of interstitial cystitis^[40] and in “writer’s cramp.”^[41]

We have described results in all these conditions with dry needling. Pain in abdominal cancer is yet another condition that appears to involve not just visceral neuronal pathways but also a global activation of body wall muscle innervation

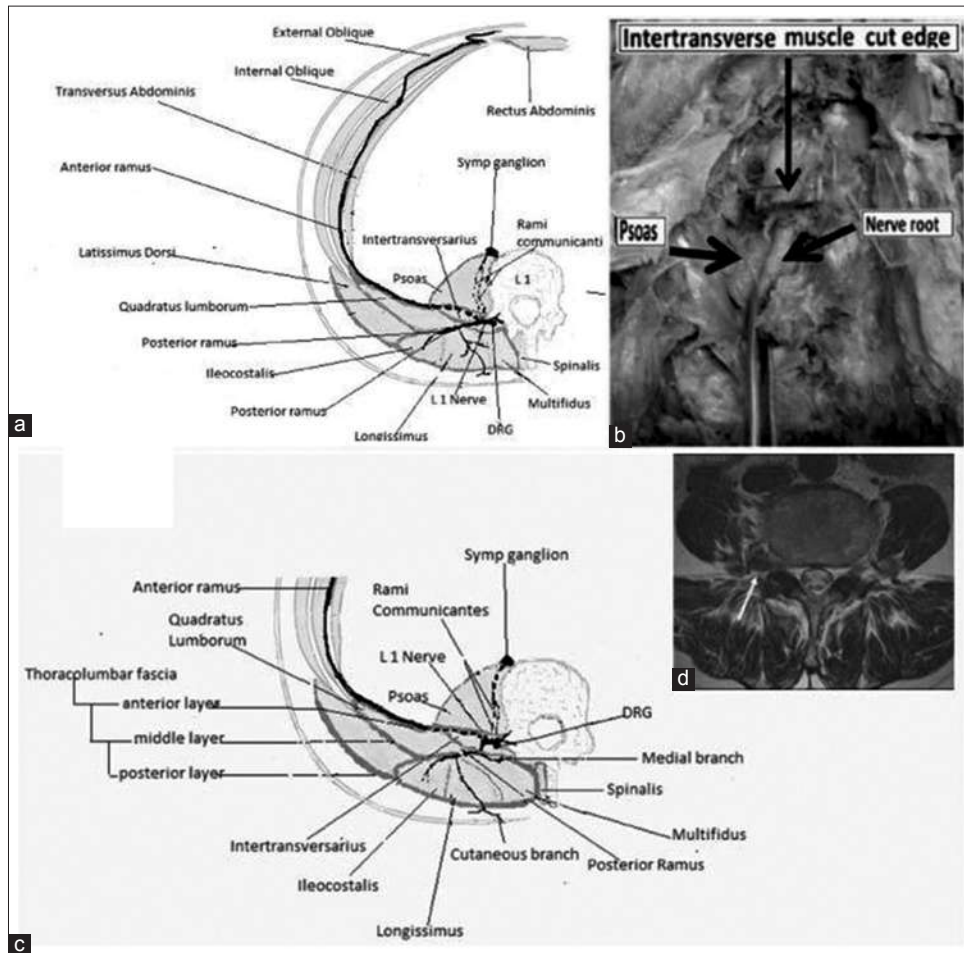


Figure 4: (a) Anterior nerve root; (b) Cadaveric dissection, lateral lamina removed; (c) Main nerve and anterior ramus sandwiched between psoas and intertransversarius muscles; (d) MRI - Muscles sandwiching the nerve root

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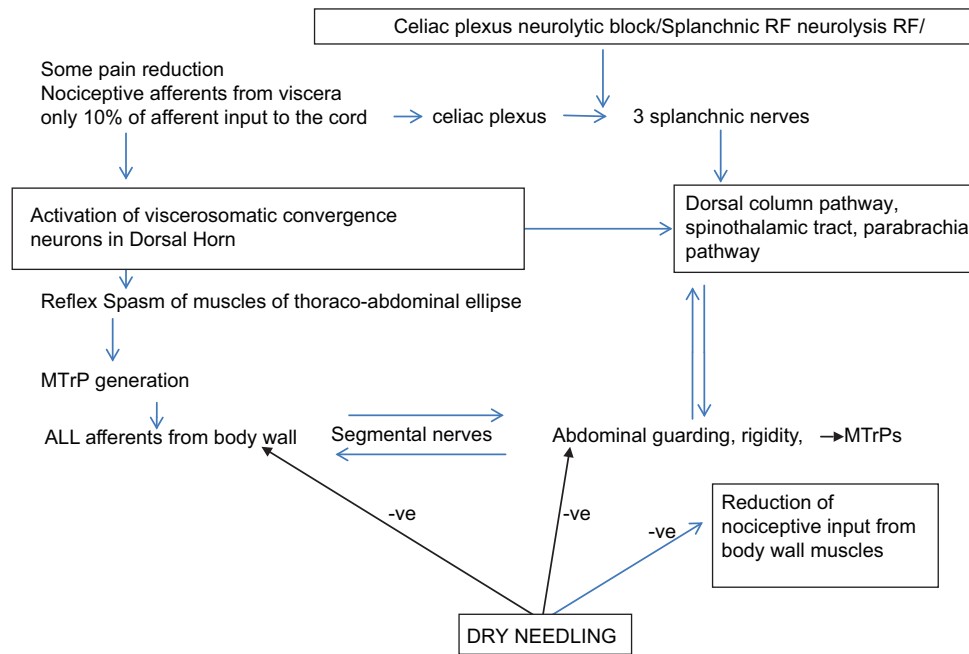


Figure 5: Diagrammatic representation of the way body wall muscles become the expressor organs of visceral pain

causing a secondary MPS with severe spasm, guarding, and rigidity through the viscerosomatic convergence making it a neuromyopathic condition.

The concept of neuromyopathy explains why efficacy of SRF/NCPB has been reported as minimal in relieving PCP by Sharfman and Walsh (1990),^[3] Wyse *et al.* (2014),^[7] and Wiechowska-Kozłowska *et al.* (2014).^[12] It also explains immediate post-RF pain crisis in patient 1 where we surmised that the SRF had eliminated the sympathetic action on the gut leading to un-opposed vagal propulsive action on bowel movement. Second, the existing pain was probably magnified after the fresh thermal injury to the nerves from SRF which cut off only the small visceral component (10%) of the viscerosomatic convergence, whereas the dominant somatic input continued unabated. These two mechanisms presumably led to the sudden escalated barrage of afferent nociceptive impulse traffic to and from the active MTrPs in the already spasmodic abdominal and paravertebral muscles immediately after SRF. According to Dommerholt,^[29] Kuan *et al.*,^[31] and Dommerholt *et al.*,^[42] an increase in SEA and EPN results in a reduced pain threshold and increased pain intensity, which explains the excruciating pain experienced by patient 1. The USGDN led to the deactivation of the MTrPs presumably by reducing the EPN at the region of the MTrPs with consequent relief of the vicious circle of pain-spasm-more pain, which had remained unresponsive to intravenous opioids. Our findings confirm the observations made by Dommerholt *et al.*,^[42] Chou *et al.*,^[43] Chen *et al.*,^[44] Srbely *et al.*,^[45] and Lewit *et al.*,^[46] that dry needling restores muscle

activation patterns and exerts an anti-nociceptive effect resulting in a reduction of local, referred, and widespread pain.

In patients 2–5, NCPB/SRF had provided only partial pain relief probably because of elimination of only the visceral contribution to pain. However, the numerous active MTrPs and their well-established network of sensitized peripheral and central motor nociceptive processing remained unabated, resulting in persistent pain > 5 NRS 15 days after SRF/NCPB.

We also suggest another mechanism to explain the severe pain crisis in patient 1, as well as the persistent pain in others. The main nerve roots are sandwiched between the psoas and the intertransversarius muscles, which firmly encircle the nerve root foramen [Figure 4c]. In fact, we found in cadaveric dissections that it is difficult/impossible to visualize the nerve root unless the intertransversarius muscle has been removed [Figure 4b]. Spasm of these muscles could exert a pincer-like effect causing nerve entrapments at the main nerve roots causing yet another vicious cycle of pain-spasm-more pain. The iliocostalis, longissimus, and spinalis components of erector spinae are supplied by the lateral branch of the posterior rami of spinal nerves and the multifidi and the small muscles of the back are supplied by the medial branch of the posterior rami of the corresponding thoracic spinal nerves [Figure 4a and c]. The anterior abdominal wall muscles are supplied by the anterior rami of inferior six thoracic nerves.^[20] All these nerves could suffer further

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entrapment by the muscles of abdomen tautened by the neuromyopathy [Figure 4]. Relaxation of back and anterior abdominal muscles probably reduced these nerve entrapments at neural foramina and along the course of the nerves, as well as diminishing the compressive effect of the muscle ellipse on the abdominal viscera.

Thus, it is clear that by the time a patient is referred for palliative care, there is a dysfunctional but self-sustaining feedback system of complex, interlinked somatic, sensorimotor, visceral, and sympathetic pathways responsible for viscerosomatic convergence and neuromyopathy. Woolf and Mannion,^[47] and Planjar-Prvan *et al.*^[48] recommend that the treatment of neuropathic pain in cancer should target not the etiological factors or the symptoms, but the mechanisms that operate to produce those symptoms. These mechanisms need to be understood, defined, and addressed separately by a multimodality approach. We suggest that USGDN is one such approach that can be used along with neurolytic blocks and medications for a comprehensive pain relief in PCP.

CONCLUSION

The referral of visceral pain to the body wall leads to the formation of MTrPs in the muscles as an epiphenomenon of neuromyopathy. This referred pain and muscle hyperalgesia in PCP occurs early and is accentuated by repeated episodes of colic, and remains long after the original visceral nociceptive stimulus has resolved or has been eliminated by neurolysis. USGDN appeared to be the only modality that specifically addressed the myopathy in our 5 patients to achieve complete pain relief. A study of DN in a large number of patients with residual pain after neuroablative procedures for visceral cancer would provide confirmation of this surmise.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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