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Original Research Article

Prospective Audit of a Pathway for In-Patient Pain Management of Chronic Abdominal Pain: A Novel and Cost-Effective Strategy

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Abstract

Background. Unexplained abdominal pain is a common cause of hospital admission and utilizes significant resource. Current in-patient pain management of acute exacerbation of chronic abdominal pain is primarily directed at pharmacological and psychological management strategies in this group of complex patients. We adopted a novel approach that proved to be both clinically effective and cost-effective.

Design. Adult patients admitted to a surgical ward with acute exacerbation of chronic abdominal pain referred to in-patient pain management were prospectively audited over a two-year period at a single tertiary centre.

Methods. Management strategy focused on a somatic source as the predominant pain generator. Patients were offered ultrasound-guided trigger point injection with steroids within 48 hours of referral and were discharged when pain control was achieved. Subsequent care by the pain physician included targeted treatment of somatic component (repeated trigger point injection with steroids or pulsed radiofrequency treatment of trigger points).

Results. We audited 43 patients referred to the inpatient pain management service over a two-year period. Four patients refused to undergo the diagnostic trigger point injection. Three patients with active visceral disease had a transient response to the injection. Thirty-six patients were diagnosed with abdominal myofascial pain syndrome, and two-thirds of these patients were discharged home within 36 hours of the intervention.

Conclusions. Abdominal myofascial pain syndrome is a poorly recognized cause of chronic abdominal pain, especially in patients with a past history of visceral inflammation. The novel strategy resulted in a significant reduction in opioid consumption, length of stay, and readmission rate.

Key Words. Myofascial Pain; Trigger Point Injection; Chronic Abdominal Pain; Viscerosomatic Convergance

Introduction

Unexplained abdominal pain is the sixth most common cause of hospital admission from any cause in women and the tenth most common cause in men [1]. In the United Kingdom, it has been estimated that nonspecific abdominal pain costs the economy in excess of £100 million per year [2]. Chronic abdominal pain as an independent predictor of suicidal behavior, after adjusting for comorbid psychiatric conditions, shows a risk that is three- to 11-fold greater than that in controls [3]. In our experience, a majority of these patients have a previous history of visceral disease with coexisting psychosocial issues, and most of them require high doses of opioid medication.

The mechanism of chronic abdominal pain is poorly understood. Current management is primarily focused on the treatment of underlying visceral inflammation if implicated in the etiology [2,4]. Chronic visceral abdominal pain is intermittent, diffuse, poorly localized, referred to

other locations, and accompanied by motor and autonomic reflexes [2]. In the absence of identifiable previous visceral disease, a diagnosis of functional abdominal pain syndrome is made [5]. These patients have undergone clinical, endoscopic, and imaging investigations with negative results, leading to frustration for both the patient and the clinician. Additional testing and investigations increase costs and patient morbidity and come with added risks [6,7]. Patients are discharged once the flare-up settles.

The in-patient pain management team at our hospital adopted a different strategy to manage these patients. This was based on our hypothesis that the pain is not visceral or functional but somatic in origin. Chronic somatic abdominal pain is a constant, dull localized pain with intermittent sharp flare-ups and a positive Carnett's sign (abdominal pain increases when the muscles of the abdominal wall are tensed) [8]. Common somatic pain generators include anterior cutaneous nerve entrapment (ACNES) and the myofascial structures [4,9]. Pain that arises from the myofascial elements of the abdominal wall is termed abdominal myofascial pain syndrome (AMPS) [4]. Other causes of chronic somatic abdominal pain include spigelian hernia, thoracic radiculopathy, rectus sheath haematoma and slipped rib syndrome [4]. Early distinction between visceral and somatic abdominal pain is necessary to tailor effective management and enhance patient experience.

This report discusses the pathophysiology of AMPS in visceral inflammation and suggests a novel way for the management of this difficult group of patients admitted with an acute exacerbation of chronic abdominal pain. We also present the results of a prospective audit on the management pathway in this subset of patients over a 24-month period.

Methods

Patients with exacerbation of chronic abdominal pain referred to the in-patient pain service at Leicester General Hospital between January 2014 and Dec 2015 were included in this audit. Patients admitted to the surgical unit with a history of longstanding (more than three months) abdominal pain and negative investigations for active visceral disease and who had failed medical management were referred. This audit is a part of an ongoing three-year prospective audit of the management pathway of patients presenting with abdominal myofascial pain syndrome at the University Hospitals of Leicester NHS Trust. The audit is registered with Clinical Audit Safety and Effectiveness (CASE 7125), University Hospitals of Leicester NHS Trust, United Kingdom. The interventions and the questionnaires are standard care for all patients presenting with AMPS to the team. The objective of the ongoing audit is to identify an effective and durable interventional treatment for the individual patient and the patient satisfaction with the management pathway. Pharmacological management for patients in the audit included a trial of amitriptyline, pregabalin, and tramadol. The team was comprised of specialist nurses and a pain physician (NG).

The pain physician reviewed the patient in the surgical ward. ACNES was diagnosed if the patient reported localized discrete tender point(s) on the lateral border of the rectus abdominis muscle, cutaneous allodynia, and a positive Carnett's sign. Abdominal myofascial pain syndrome was diagnosed from the clinical history, presence of multiple tender trigger points not confined to the lateral border of the rectus abdominis muscle, absence of cutaneous allodynia, and a positive Carnett's sign in the presence of a past history of visceral inflammation. To confirm the clinical diagnosis, patients were offered a diagnostic ultrasound-guided trigger point injection with a mixture of local anesthetic (0.5% levobupivacaine) and steroids (60 mg depot methylprednisolone). The diagnostic intervention was performed within 48 hours of patient review. Each trigger point was treated with 2 mL of the mixture. The number and location of the trigger point(s) were documented. Following the procedure, the patients were reviewed by the referring team and discharged home if they reported significant pain relief (>50% relief).

Following discharge from the hospital, the patient's care was taken over by the pain physician. Management was directed at somatic pain and included two treatment modalities in succession: Trigger point injection with steroids and pulsed radiofrequency treatment. Pulsed radiofrequency treatment of trigger points has been shown to provide durable relief in this cohort [10,11]. The patients were followed up over telephone following each intervention as part of routine care by a specialist nurse. If the pain had returned to the baseline at three months, then the patient was booked to receive the next treatment in the pathway. If the patient reported greater than 50% relief at three months, then the same treatment was repeated at six months after completion of the two questionnaires. Once a durable treatment was identified, it was repeated at six to nine monthly intervals. Pulsed radiofrequency treatment was performed under real-time ultrasound guidance using an in-plane approach. A 20-gauge radiofrequency straight cannula with a 10 mm tip (Neuro Therm, Wilmington, MA, USA) was used. Pulsed radio frequency (PRF) treatment was initiated with an Radio Frequency (RF) generator (Neuro Therm, Wilmington, MA, USA) using the following parameters: voltage output 45 V; 5 Hz frequency; 20 ms pulses in a one-second cycle; impedance range between 150 and 450 ohm and 42°C plateau temperature. PRF was performed for six minutes. Following PRF, 2 mL of 0.5% levobupivacaine was injected into each trigger point.

Patients completed three questionnaires before they received the first diagnostic trigger point injection with steroids. They included Brief Pain Inventory–Short Form (BPI-SF), Euro Quality of Life Questionnaire (EQ-5D-3L), and Hospital Anxiety and Depression Scale (HADS). Following each treatment, the patient completed the questionnaires at three and six months. Patients who

had abnormal scores (>10) on HADS were referred for psychological workup. Clinical psychologists conducted an initial interview and, if appropriate, offered four to six sessions of therapy tailored for the patient.

In the audit, clinically significant pain relief was defined using the "Pain at its worst in the last 24 hours" construct from the BPI-SF questionnaire. This 11-point pain intensity scale has been found to have the strongest relationship with the pain interference scale [12,13]. Following Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT) recommendations, a two-point change (30–36%) at three months post-treatment was considered successful intervention [14].

Outcomes collected included data before and after the in-patient pain team saw the patient. This included the duration of chronic pain, number of hospital admissions in the preceding 12 months, length of hospital stay during each hospital admission, and the dose of opioid medication (oral morphine equivalent) during the current admission.

Following the diagnostic trigger point injection, the outcomes included date of discharge, hospital readmission(s) and length of stay for the same condition, reduction in opioid medication at six-month telephone review, pain severity, and pain interference scores. An independent clinician collected the data on length of stay and hospital readmission.

Cost-Effectiveness

NHS reference cost guidelines published by the Department of Health for the year 2014 to 2015 were used to calculate the average cost of each hospital admission. The Finished Consultant Episode (FCE)–based average cost for nonelective inpatient stay is £1,565 and for each excess bed day is £303 [15]. An FCE is the time a patient spends in the care of one consultant. These figures were used to calculate the costs for the hospital stay. This costing included the in-patient diagnostic trigger point intervention. Table 2 shows a savings of £347,358 by reducing readmission and length of hospital stay over a projected 12-month period.

Following hospital discharge, the patient was seen in the outpatient pain clinic (£168) [15]. Subsequent interventions were performed in the day case procedure (TPI: £497; PRF: £643) [15]. Each patient was projected to have not more than two day case interventions over a 12-month period (£41,040). Each patient received two nurse-led telephone reviews (£80) [15]. Cost of the clinical psychology care tariff was estimated as £340 per patient. Nineteen patients were referred for therapy (£6,460). The projected annual cost in the outpatient setting for our cohort was calculated at £56,428.

Statistical analysis of the results was performed using Stata version 13.1 (StataCorp LLC, College Station, Texas, USA) statistical package for Windows (Microsoft Corp.). The paired *t* test was used to compare baseline pain numeric rating scale (NRS) to NRS at each follow-up period (three months, six months). The same statistical approach was used for HADS and EQ-5D-3L scores at baseline and at six-month follow-up. Differences were considered significant for $P < 0.05$. Missing data was imputed using the "last observation carried forward" method.

Results

Over a 24-month period, a total of 43 patients with acute exacerbation of chronic abdominal pain were referred to the in-patient pain service at the Leicester General Hospital. Three patients had markers of ongoing visceral inflammation.

Clinical examination by the pain physician revealed myofascial trigger points in the abdominal muscles and an absence of cutaneous allodynia, and Carnett's sign was positive in all 43 patients. Diagnostic ultrasound-guided trigger point injection was offered to all patients. Four patients refused the diagnostic procedure. Reasons for refusal included needle phobia, improvement in pain with conservative management, and patient belief that the source of pain was from an underlying visceral disease (endometriosis).

Thirty-nine patients underwent diagnostic ultrasound-guided trigger point injection with a mixture of local anesthetic and steroids. Trigger points were observed in the rectus abdominis (most common), internal oblique, and transversus abdominis muscles.

Three patients had an underlying active visceral inflammation. Two patients had features of active pancreatic inflammation while one patient presented with unilateral flank pain secondary to recurrent renal calculi–induced urinary tract inflammation. Trigger point injection (TPI) provided transient relief (eight to 12 hours). Subsequent management was directed at visceral pain management.

Active visceral disease markers were absent in 36 patients. Follow-up data were collected in these 36 patients. The diagnostic trigger point injection with steroids provided greater than 50% relief within one hour in 97% (35/36) of the patients. Two-thirds of the patients diagnosed with AMPS were discharged home within 36 hours of the trigger point injection. The procedure failed to provide any improvement in one patient while in the other patient the pain relief lasted three weeks.

Sixteen patients (45%) received pulsed radiofrequency treatment. Clinically significant pain relief (≥30%) at three months was reported by 70% (25/36) following trigger point injection (TPI) with steroids and by 69% (11/16) following pulsed radiofrequency (PRF) treatment. Substantial clinical relief (≥50%) at three months was reported by 25% (9/36) with TPI steroids and 31% (5/16) following PRF treatment. Six-month post-treatment outcomes were

Table 1 Demographic data of 36 patients with abdominal myofascial pain syndrome

Patient ID	Gender, Age, y	Duration of Pain, y	Oral Morphine Equivalent Before, mg	Reduction in Oral Morphine Equivalent at 6 mo, mg (%)	Primary Visceral Inflammation
1	M,48	2	230	0 (100)	Pancreatitis
2	F, 37	6	300	12 (96)	Appendicitis, postsurgery
3	M,23	6	170	90 (47)	Gastritis, IBD
4	F, 45	1	100	0 (100)	Biliary, postsurgery
5	F, 22	1	60	0 (100)	Gastritis
6	F, 63	4	120	60 (50)	Postsurgery
7	F, 24	6	60	0 (100)	Pancreatitis
8	F, 27	1	500	0 (100)	Biliary
9	M,41	1	140	0 (100)	Biliary, postsurgery
10	M,34	17	320	45 (86)	Gastritis, postsurgery
11	F, 24	3	120	20 (84)	Pancreatitis
12	M,72	2	250	150 (40)	Pancreatitis, postsurgery
13	M,43	17	25	25 (0)	Pancreatitis
14	M,45	5	200	120 (40)	Pancreatitis
15	F, 22	3	230	0 (100)	Pancreatitis
16	F, 39	3	300	12 (96)	Appendicitis
17	F, 45	3	370	300 (19)	Pancreatitis
18	F, 22	1	120	40 (66)	Biliary
19	F, 36	4	120	120 (0)	Gastritis
20	F, 53	1	100	100 (0)	Gastritis, biliary
21	M,60	2	40	12 (70)	Gastritis
22	F, 42	2	100	12 (88)	Pancreatitis, gastritis
23	F, 22	6	40	40 (0)	Pancreatitis
24	M,44	3	320	170 (47)	Pancreatitis
25	F, 58	1	110	60 (46)	Biliary
26	F, 40	1	50	0 (100)	Biliary
27	M,22	1	100	40 (60)	Gastritis
28	F, 22	4	60	60 (0)	Pancreatitis
29	F, 77	3	120	20 (84)	Pancreatitis
30	F, 29	1	360	100 (72)	Pancreatitis, postsurgery
31	F, 44	1	0	0	Biliary, postsurgery
32	M,79	4	100	100 (0)	Biliary
33	M,52	1	140	80 (43)	Pancreatitis, postsurgery
34	F, 66	10	200	40 (80)	Pancreatitis
35	F, 23	1	60	40 (33)	Recurrent urinary infection
36	F, 67	1	500	500 (0)	Appendicitis, postsurgery

IBD = inflammatory bowel disease.

complete in 78% of patients (28/36). The number of trigger points treated ranged from six to 14 triggers.

Nineteen patients (52%) reported abnormal baseline scores on the HADS and were referred to the Clinical Psychology Department. Twelve patients attended the initial interview. Five patients reported significant ongoing benefit from trigger point treatment and were discharged. Seven patients underwent therapy.

Table 1 includes demographic data, duration of pain, oral morphine equivalent dose of opioids at the baseline, and percentage reduction in opioid consumption at the six-month follow-up period. There was a significant reduction in opioid use.

The novel strategy of identifying a somatic pain generator and directing treatment targeting the myofascial trigger point was successful in reducing the length of stay as well as repeat admissions (Table 2). It also proved to be a cost-effective process. Cost savings as a result of a reduction in repeat hospital admission(s) over a projected 12-month period after deducting costs for the outpatient appointment, nurse telephone reviews, day case interventions, and clinical psychology management were over £290,000.

Table 2 Hospital admission, length of hospital stay, and cost saving data of 36 AMPS patients before and after seeing the in-patient pain physician

Patient ID	Data Period Prior to Seeing Pain Physician, mo	Number of Admissions over Data Period	Total Length of Stay, d	Total Cost, £	Data Period After Seeing Pain Physician, mo	Number of Admissions	Total Length of Stay, d	Total Cost, £	Cost Savings over a 12-mo Period, £
1	12	4	12	8,684	12	2	11	5,857	2,827
2	12	2	20	8,584	12	0	0	0	8,584
3	12	4	8	7,472	12	5	6	8,128	–656
4	12	3	35	14,391	12	2	7	4,645	9,746
5	12	10	66	32,618	12	3	12	7,422	25,196
6	12	3	15	8,331	12	0	0	0	8,331
7	12	3	18	9,240	12	2	10	5,554	3,686
8	12	2	29	11,311	12	0	0	0	11,311
9	12	4	21	11,411	12	2	9	5,251	6,160
10	12	7	15	13,379	12	2	3	3,433	9,946
11	12	19	110	57,308	12	3	20	9,846	47,462
12	12	3	41	20,398	12	3	13	7,725	12,673
13	12	14	43	30,697	12	7	17	13,985	16,712
14	12	1	6	3,080	12	0	0	0	3,080
15	12	4	21	17,671	12	2	18	7,978	9,993
16	12	2	10	5,554	12	0	0	0	5,554
17	12	4	50	14,391	12	4	21	11,411	2,980
18	12	6	27	15,753	12	6	14	11,814	3,939
19	12	3	27	11,361	12	0	0	0	11,361
20	12	2	12	6,160	12	0	0	0	6,160
21	12	7	19	14,591	12	1	2	1,868	12,723
22	12	5	20	12,370	12	0	0	0	12,370
23	12	14	61	36,151	12	5	18	11,764	24,387
24	12	4	18	10,502	12	0	0	0	10,502
25	12	3	16	8,634	12	0	0	0	8,634
26	12	2	4	3,736	12	0	0	0	3,736
27	12	2	19	11,411	12	0	0	0	11,411
28	12	3	13	7,725	12	1	1	1,565	6,160
29	12	4	16	9,896	12	1	5	2,777	7,119
30	9	8	37	21,307	9	2	13	6,463	14,844
31	6	1	21	7,625	6	2	3	3,433	4,192
32	6	3	11	7,119	6	0	0	0	7,119
33	6	1	2	1,868	6	1	25	8,837	–6,969

(continued)

Table 2 Continued

Patient ID	Data Period Prior to Seeing Pain Physician, mo	Number of Admissions over Data Period	Total Length of Stay, d	Total Cost, £	Data Period After Seeing Pain Physician, mo	Number of Admissions	Total Length of Stay, d	Total Cost, £	Cost Savings over a 12-mo Period, £
34	6	1	8	3,686	6	0	0	0	3,686
35	6	5	38	17,824	6	0	0	0	17,824
36	6	1	11	4,595	6	0	0	0	4,595

The Finished Consultant Episode–based average cost for nonelective inpatient stay is £1,565 and for each excess bed day is £303.[11]
AMPS = abdominal myofascial pain syndrome.

The mean baseline NRS score (pain at its worst in the last 24 hours) was 8.6 (SD = 1.1), and the mean score at three months post-treatment was 5.6 (SD = 2.6, $P < 0.001$). Changes in the three outcomes (pain, HADS, and EQ-5D-3H) at six months from the baseline are summarized in Table 3. Figure 1 illustrates the change in NRS pain scores over time.

Rates of patient satisfaction with the management pathway were 56% excellent, 27% good, 11% fair, and 6% poor. Complications included transient nightmares (steroid = 1) and postprocedural pain flare-up lasting more than one week in four patients (steroid = 1, PRF = 3).

Discussion

Current management of acute exacerbation of chronic abdominal pain is primarily directed at pharmacological and psychological management strategies in this group of complex patients. Undue significance is given to a previous history of visceral inflammation [2].

Abdominal myofascial pain syndrome develops as a result of trigger points in the abdominal musculature. The abdominal trigger point can develop as a result of either trauma (physical or surgical) or due to the phenomenon of viscero-somatic convergance (VSC) [16–19]. Quinter et al. hypothesize that the development of trigger point(s) in the muscle is a secondary phenomenon and represents sensitization as a result of primary neurogenic inflammation [20].

In patients with a history of visceral inflammation, AMPS could occur as a result of VSC. VSC is a physiological phenomenon and describes the convergence of somatic and visceral inputs onto the central nervous system neurones. Acute visceral inflammation results in a massive barrage of afferent visceral signals to the convergent viscero-somatic neurons in the spinal cord. This results in the process of central sensitization that presents as referred muscle pain and hyperalgesia [19]. Thus, there can be tenderness in the abdominal muscles during this phase of active visceral inflammation. Nevertheless, the predominant source of pain is the viscera and treatment should be directed at the visceral pain. Treatment of myofascial pain at this stage may be ineffective, as seen in the three patients who had active visceral markers.

However, when the visceral inflammation subsides, the predominant pain generator appears to move from the viscera to the abdominal wall muscle overlying the viscera. Central sensitization has been primarily implicated in this phenomenon. Some authors suggest the possibility of peripheral sensitisation as an additional factor [19,21–23]. Examples of VSC are flank pain in patients with ureteric colic, upper abdominal pain in chronic pancreatitis, right upper abdominal quadrant pain in biliary colic, and lower abdominal pain in chronic pelvic pain [24,25].

Table 3 Outcomes at six months following the treatment of abdominal myofascial trigger points

Outcomes	No.	Baseline	6 mo	Change Mean (95% CI)	*P*
Worst Pain in 24 h	28	8.6 (1.1)	5.7 (2.4)	–2.9 (–3.7 to –1.9)	<0.001
HADS Anxiety	27	10.6 (4.6)	8.7 (4.2)	–1.9 (–2.8 to –1.0)	<0.001
HADS Depression	27	10.3 (3.4)	7.7 (3.1)	–2.6 (–3.5 to –1.6)	<0.001
EQ-5D-3L	28	9.8 (1.2)	8.1 (1.6)	–1.7 (–2.5 to –1.0)	<0.001

Values are mean (SD) or number.
CI = confidence interval; EQ-5D-3L = Euro Quality of Life Questionnaire; HADS = Hospital Anxiety and Depression Scale.

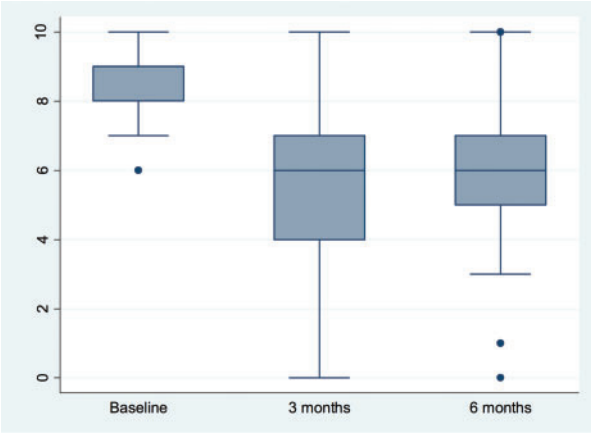

Figure 1 Change in numeric rating scale score (pain at its worst in the last 24 hours) at baseline, three months, and six months following treatment of abdominal myofascial trigger points.

A third of our patients had a previous history of pancreatitis. There is evidence to suggest that pain in chronic pancreatitis could be nonvisceral [26–28]. Using differential neuraxial block, one study of 23 patients found that a majority of patients had nonvisceral pain (78%) while only 22% had visceral pain [26].

A common cause of somatic abdominal pain is ACNES [4,9,29]. Clinical features mirror myofascial pain, and Carnett's sign is positive. However, a history of previous visceral inflammation, trigger points not confined to the lateral border of the rectus muscle, absence of cutaneous allodynia, and elicitation of a myofascial twitch on trigger point injection may help differentiate AMPS from ACNES. Although none of our cohort presented with features suggestive of ACNES, this condition should be excluded.

There were two novel processes in our practice. First, we actively looked for a somatic focus as a source of pain in patients admitted to a surgical ward with a past history of visceral inflammation. Thereafter, we were able to perform the diagnostic block within 48 hours of seeing the patient. Response to the trigger point injection was crucial in confirming whether the pain generator was somatic or visceral and charting the appropriate management course. The current practice in the United Kingdom is to perform TPI treatment in an outpatient setting with a recognized waiting time. We believe that a delay in performing TPI would have resulted in another flare-up and readmission to the hospital. Subsequent treatment targeting the myofascial trigger point enabled a significant reduction in the number of readmissions as well as the length of stay for patients diagnosed with AMPS. We recommend that trigger point interventions should be performed under ultrasound guidance as this could increase the safety and efficacy of the procedure [30–32].

Our data is observational in a small cohort of patients presenting with acute exacerbation of chronic abdominal pain. We did not study other reported treatment approaches in managing myofascial pain including acupuncture, botulism toxin A injection, and injection with neurolytic agents. We are aware that there is inadequate basic science evidence to explain the development of myofascial trigger points as a result of primary visceral inflammation. Based on our limited experience, we suggest that pain physicians managing this subset of patients consider the possibility of AMPS and actively look for myofascial trigger points.

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