Retrospective Review

The Impact of Local Steroid Administration on the Incidence of Neuritis following Lumbar Facet Radiofrequency Neurotomy

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Background: Pain arising from the lumbar facet joints is a common cause of axial back pain in adults. Radiofrequency neurotomy (RFN) of the medial branches of the spinal dorsal rami has been used as a treatment option. The most common side effect is transient, localized, burning, neuritic-type pain, termed post-neurotomy neuritis (PNN). Corticosteroids have been administered through the radiofrequency cannula after neurotomy to prevent PNN, but no study has examined the effects of this on PNN.

Objectives: We investigated the incidence of PNN in patients who received corticosteroids after RFN and in those patients who did not receive corticosteroids.

Study Design: Retrospective evaluation.

Setting: Single-site interventional pain management practice in an urban tertiary academic medical center.

Methods: One hundred and sixty-four patients were included in this study and were categorized into non-steroid (n = 87) and steroid (n = 77) groups. Patient's age, gender, body mass index (BMI), laterality of procedure, use of neuropathic pain medications, baseline pain, and duration of pain were all recorded. PNN was determined if the patient self-reported transient burning or neuropathic pain at the site prior to or at the 6-week routine follow-up encounter.

Results: There was no significant difference in demographic characteristics between the 2 groups in age, gender, baseline pain, and duration of pain. The proportion of patients in the steroid treated group with PNN was 5 out of 77 (6.4%) and the non-steroid group was 6 out of 87 (6.9%). There was no statistically significant difference between the groups. There was no statistically significant difference in the incidence in neuritis between individuals taking neuropathic agents and individuals not taking neuropathic agents.

Limitations: This study has several limitations including small sample size, patients' self-reported neuropathic symptoms, and inability to draw strong conclusions due to the retrospective study design. A single interventionalist performed all the procedures in this retrospective study and variations in technique amongst others are inevitable.

Conclusion: Administration of steroids after RFN does not reduce the incidence of post-neurotomy neuritis. Concurrently administering neuropathic medications does not protect against neuritis.

Key Words: Radiofrequency neurotomy, radiofrequency ablation, neuritis, corticosteroid, lumbar facet pain, post neurotomy neuritis.

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Pain arising from the lumbar facet joints is a common cause of axial back pain in adults. Facet pain was originally described by Goldthwaite in 1911, and was described as a "facet syndrome" in 1933 by Ghormley (1-2). Facet pain is defined as pain that arises from any part of the facet joint: the capsule, synovial membrane, hyaline cartilage, and bone (3-4).

The reported prevalence of lumbar facet pain varies widely; however, a conservative estimate based on well-performed studies indicates a prevalence between 5 and 15% (5-7). The diagnosis of facet pain is controversial and may be made with single, double, or placebo-controlled blocks of the medial branches of the spinal dorsal rami. Two medial branches of adjacent spinal dorsal rami innervate each facet joint. In general, definitive treatment with radiofrequency neurotomy (RFN) is pursued if the patient experiences at least 50% pain relief within a time period consistent with the anesthetic given.

In the mid-1970s, Shealy was the first to report using RFN to treat facet pain (8-9). RFN involves stimulation of the medial branch continuously with a probe, generating heat and ablating the nerve. The temperature is monitored to avoid damage to nearby structures. The most common side effects of RFN include local post-procedural pain and a transient, localized, burning, neuritic-type pain (10). The neuritic-type pain has been termed post-neurotomy neuritis (PNN) and is often described as a "sunburnt" type sensation. It is uncommon and usually of short duration (11). The cause of this phenomenon is unknown, but possible etiologies include incomplete neurotomy of the medial branch and or incidental neurotomy of a nearby branch such as the lateral branch of the dorsal ramus (12). Some clinicians administer corticosteroids through the RF cannula after neurotomy to prevent PNN; however, to the knowledge of the authors, no study has examined the effect of steroid administration on the prevalence of PNN.

Methods

This retrospective study protocol (IRB # 1801018898) was approved by the local Institutional Review Board and was conducted at a single-site interventional pain management practice in an urban tertiary academic medical center. All patients treated with lumbar facet RFN between January 2017 and December 2017 were included in this study. Patients were between the ages of 18–99 years, presented with low back pain from lumbar facet syndrome, had physical examination, and imaging studies consistent with facet arthropathy. In order to be considered for lumbar RFN, all patients had to report > 75% reduction in back pain following 2 sets of diagnostic medial branch blocks (MBBs) in accordance with Spine Intervention Society (SIS) guidelines (13).

Baseline demographic information was obtained from the medical records including: age, gender, BMI, duration of pain, and baseline numerical rating scale (NRS) pain scores. Concurrent use of neuropathic agents (gabapentin, pregablin, topiramate, and amitriptyline) was also recorded. Our primary outcome measurement was the incidence of PNN within the first 6 weeks following lumbar RFN. PNN was defined as a localized, burning, neuritic-type pain which was new following the RFN. The incidence of PNN was directly compared in those patients who received and did not receive corticosteroid following neurotomy.

Procedures

Based on history, physical examination, and imaging studies, the treating physician (JRS) selected the facet joints to be diagnostically blocked. Two rounds of diagnostic MBBs were conducted, and those with > 75% relief of pain were scheduled for lumbar facet RFN. At the time of the neurotomy procedure, patients were positioned prone on a fluoroscopy table and the lumbar region was prepped with chlorhexidine and draped in a standard sterile manner. The skin and subcutaneous tissues were anesthetized with 1% preservative-free lidocaine (McKesson Xylocaine® - MPF). Once the tissues superficial to the target sites were adequately anesthetized, a 20 gauge 10cm RF electrode with a 10mm curved active tip (Epimed International Inc, Farmers Branch, TX), was positioned. The neurotomy technique followed SIS guidelines (13). For the L1-4 medial branches, using intermittent fluoroscopic guidance, the active tip was positioned at the concavity of the junction between the superior articular process and the transverse process at each level. For the L5 dorsal ramus, the active tip was positioned to lie parallel to the nerve at the junction of the S1 superior articulate process and the sacral ala. Proper needle placement was confirmed using anterior-posterior, lateral and oblique views. Once the RF needle was properly positioned, motor testing was performed at 2 Hz to confirm placement over the medial branch with no twitch response observed in the extremities. After appropriate electrode positioning, 1 mL of 2% lidocaine was injected through the introducer needle to provide anesthesia during the neurotomy. The RFN lesion was then performed at each target site at 80°C for 90 seconds. The needles target-

ing the L1-4 medial branches were then rotated 120 degrees and the needle targeting the L5 dorsal ramus was withdrawn slightly. A second lesion was then performed at 80°C for 90 seconds.

Following completion of the neurotomy lesions, 0.5 mL of 0.5% bupivacaine (Hospira, Lake Forest, IL) was injected at each site to provide post-procedure analgesia. Prior to June 2017, the treating physician would routinely administer corticosteroid (40 mg of triamcinolone, Bristol-Myers Squibb, New York, NY) mixed with bupivacaine following lumbar RFN. However, following a discussion at an annual pain meeting in July 2017, this practice pattern changed. Thus, patients treated between January and June 2017, were administered local steroid post-RFN at each lesion site and those treated between July and December 2017 were not given post-RFN steroids.

Following the procedure, patients were observed for approximately 15-20 minutes and were then discharged if clinically stable. Patients were asked to follow up in 6 weeks after the RFN procedure and were instructed to call if there were any complications.

Data Analysis

All data extracted from the medical charts was entered into a password-protected database. Neuropathic medication and dosages were obtained and recorded. The number of individuals reporting PNN was collected and analyzed. This was done by looking at the follow-up encounter post-neurotomy. If the patient did not report any periods of transient burning at the RFN sites during the routine 6-week follow-up period, then that patient was considered negative for occurrence of PNN. Those patients that came in prior to 6 weeks or had telephone encounters with the physician or nurse practitioner were examined more closely. If the patient did report increased burning or neuropathic-type pain at the procedural sites during the 6-week follow-up period, they were recorded as having developed PNN.

Statistical Analysis

Descriptive statistics (including mean, standard deviation, range and percent) were calculated to characterize the steroid and non-steroid cohorts. The 2-sample t-test, χ^2 test, and Fisher exact test were used to compare age, gender, and symptom duration, respectively, between the 2 groups. Statistical software was used to analyze the data (SPSS, Version 22; Chicago, IL). Proportions and 95% confidence intervals (CI) were calculated and groups were compared with Chi Square or Fisher Exact Tests for categorical variables. The level of significance was set at 0.05. Two-sided testing was used for all hypothesis testing.

Primary Outcome: Effect of Corticosteroid on Incidence of Neuritis

The Fisher exact test was used to compare the incidence of neuritis between the steroid cohort and non-steroid cohort.

Secondary Outcome: Effect of Neuropathic Agents on Incidence of Neuritis

The Fisher exact test was used to compare the incidence of neuritis between patients concurrently taking neuropathic agents and those not on these medications.

Results

The study included 164 individuals. Seventy-seven patients were given corticosteroid (40 mg of triamcinolone) mixed with local anesthetic (0.5% bupivacaine) following RFN, while 87 patients were given 0.5% bupivacaine alone. A comparison of demographic characteristics between the steroid and non-steroid group revealed no significant difference in age, gender, baseline pain, and duration of pain. The average age in years of the steroid group vs. the non-steroid group was 65.5 ± 14.9 and 64.4 ± 15.3 (P = 0.64, 95% CI -3.47 to 5.68), respectively. Baseline NRS pain scores in the steroid and non-steroid groups were 5.8 ± 1.9 vs. 5.2 ± 2.6 (P = 0.09, 95% CI -0.10 to 1.30) and duration of pain in months was 20.0 ± 15.4 vs. 21.0 ± 11.8 (P = 0.64, 95% CI -5.21 to 3.21), respectively. There was no statistical significant difference between the steroid and non-steroid cohorts (Table 1).

The proportion of patients in the steroid group who reported symptoms consistent with PNN were 5 out of 77 (6.4%) while those patients in the non-steroid group reporting PNN-like symptoms were 6 out of 87 (6.9%). The incidence of PNN is shown in Table 2 and was not statistically different among the 2 groups (P = 0.99).

The incidence of PNN was compared between patients taking and not taking neuropathic agents. Out of 164 patients, 48 were concurrently taking neuropathic agents. Of these, 3 patients (6.3%) reported symptoms of PNN. Eight out of the 116 patients not taking neuropathic medications (6.9%) reported symptoms of PNN.

Table 1. *Patient demographics.*

	Steroid n (n = 77)	Standard Deviation	No Steroid (n = 87)	Standard Deviation	*P* value	95% Conf Interval
Age	65.5	14.9	64.4	15.3	0.64	.3.47 to 5.68
Gender					0.14	
Male	25		35			
Female	52		42			
Side					0.27	
Right	41		54			
Left	36		33			
Baseline Pain	5.8	1.9	5.2	2.6	0.09	-0.10 to 1.30
Duration of Pain (months)	20.0	15.4	21.0	11.8	0.64	-5.21 to 3.21
BMI	28.7	5.1	27.1	5.6	0.06	-0.06 to 3.26

Unpaired t test for categorical data
Fishers test for gender

Table 2. *Neuritis.*

	Steroid n = 77 N (%)	No Steroid n = 87 N (%)	*P* value
Incidence of Neuritis	5 (6.4%)	6 (6.9%)	0.99

Fishers Exact T Test

Table 3. *Neuritis and neuropathic agents.*

	Neuropathic Agent n = 48 N (%)	No Neuropathic Agent n = 116 N (%)	*P* value
Neuritis	3 (6.3%)	8 (6.9%)	0.99

Fishers Exact T Test

Table 4. *Neuritis and neuropathic agents.*

Neuritis n = 3	No Neuritis n = 45	*P* value
6.3%	93.7%	

Fishers Exact T Test

There was no statistically significant protective effect observed for the development of PNN in patients taking neuropathic medications (*P* = 0.99, Tables 3,4).

Discussion

Radiofrequency neurotomy (RFN) has been shown to be a successful treatment option for patients diagnosed with lumbar facet pain who have had positive responses to diagnostic medial branch blocks (14). Although RFN is well tolerated, some patients may develop PNN, a burning, neuritic-type pain involving the paravertebral skin of the lumbar spine. The severity and duration is quite variable, ranging from transient symptoms to symptoms lasting up to 6 months and requiring medical treatment (10,15). The incidence of PNN after lumbar RFN has been reported to be approximately 1% to 10% (3-6).

The use of steroid to reduce post-operative pain and neuritis is controversial. Some data advocate for its use while others recommend RFN with anesthetic only (13,16). One prospective study conducted by Dobrogowski et al (17) assessed 45 patients for pain using the visual analog scale (VAS) at 4 time points after a RFN procedure: 1 week, 1 month, 3 months, and 6 months. The patients were randomly divided into 3 treatment groups, receiving pentoxifylline, methylprednisolone, or saline (placebo) after RFN. The patients in all 3 groups reported significant reduction in pain at all time points, with no statistically significant difference among the groups. To assess for postoperative inflammation, local tenderness was determined at each post-operative time point by inquiring if the patient experienced pain to palpation in comparison to the contralateral side.

Analysis demonstrated that post-procedural pentoxifylline or methylprednisolone reduced post-procedural pain; however not significantly so when compared to saline. The group that received saline resulted in the highest number of patients with severe local tenderness. According to this study, it appears that administration of methylprednisolone or pentoxifylline reduces the occurrence of postoperative pain (17).

While this study examines post-RFN pain, it did not specifically address the phenomenon of PNN.

The present study demonstrates that administration of steroids after RFN does not significantly reduce the incidence of PNN. There was no statistical difference in the incidence of PNN in those that did (6.4%) and did not (6.9%) receive steroids. Our incidence of PNN is comparable to previous estimates. Several RFN studies performed without steroids reported having no side effects (6,18-21). It is unclear why steroids do not reduce the incidence of PNN, as steroid administration has shown to be effective in decreasing post procedure pain in animal models (22). In addition, there is evidence to show that steroid, when added to a local anesthetic, prolongs the duration of nerve blockade (23). It may be the case that steroid delays the onset of PNN; however, this was not expressly examined in this study.

It is plausible that neuropathic pain medications could be protective against PNN. A retrospective review by Welsh et al (24) observed a lower incidence of PNN in patients taking gabapentin for more than 2 weeks prior to RFN (7.1%) versus those not taking gabapentin (13.2%). Contrary to Welsh et al (24), we found no statistical difference in the incidence of neuritis in those patients taking neuropathic agents (6.3%) and those not taking neuropathic agents (6.9%). The deafferentation pain caused by a radiofrequency lesion may require higher dosages of neuropathic medication than the patient takes at baseline. In addition, as much as two-thirds of all patients might not be adherent to medications for neuropathic pain (25).

Our findings are relevant to clinical practice, as steroids have been shown to have various metabolic side effects, including an increased risk of low bone density and vertebral fractures (26). The adverse effects of steroids are dose-dependent, therefore any opportunity to minimize dose will benefit the patient.

PNN may be attributable to an incomplete neurotomy of cutaneous branches of the dorsal ramus (6). This is plausible; however, PNN seems to be independent of the specific needle placement technique utilized (10,15). The RF needles used in this study were 20 gauge needles. It is possible that larger-diameter needles would cause a lower incidence of incomplete ablations and therefore PNN.

Limitations

This study has several limitations. The relatively small sample size and the retrospective study design make it difficult to draw strong conclusions. Patients were required to self-report episodes of PNN and were not directly asked about symptoms. Thus, we likely underestimated the overall prevalence of PNN. RFN of the medial branches was conducted as per Spine Intervention Society guidelines; however, variations in technique and individual anatomy are inevitable. The time of initiation, duration of treatment, and adherence to neuropathic pain medications were not recorded. The onset and duration of PNN was not measured – it is possible that there is a correlation between steroid use and these parameters. This study only examined the incidence of PNN following lumbar RFN. Our conclusions may not be generalizable to other neural structures treated with RFN.

Conclusion

In conclusion, administration of local steroids post RFN does not reduce the incidence of PNN. In addition, this study suggests that concomitantly administered neuropathic pain medications are not protective against the development of PNN. In the future, a follow-up randomized control trial can be conducted to further investigate PNN. This study would allow for better standardization in protocol and increase the level of evidence. Areas for future study include investigating the incidence of PNN after RFN of other neural structures such as the genicular or occipital nerves. In addition, the effect of steroid administration on the onset and duration of PNN should be quantified.

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