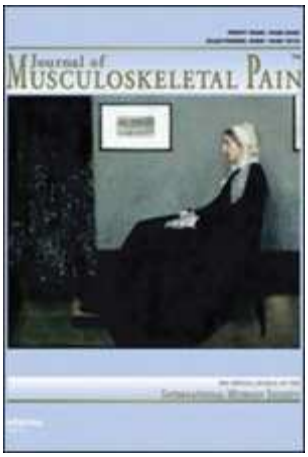

Treatment of attachment trigger points in the gluteal muscles to cure chronic gluteal pain – A case report

Journal:	*Journal Of Musculoskeletal Pain*
Manuscript ID:	WJMP-2010-0005.R1
Manuscript Type:	Case Report
Keywords:	gluteal muscles, lumbar spine, myofascial pain, trigger point

Treatment of Attachment Trigger Points in the Gluteal Muscles to Cure Chronic Gluteal Pain – A Case Report

Submitted: January 23, 2010

Resubmitted: September 12, 2010

ABSTRACT

Background: The purpose of this case report is to demonstrate a patient of chronic myofascial pain syndrome in the gluteal region secondary to lumbar facet joint lesion.

Findings: Initially, he was treated conservatively with medication and physical therapy and had only temporary pain relief for few days. Later, he was treated with lumbar facet joint injection (with kenacort) and myofascial trigger point injection (with 1% xylocaine) to bilateral L4-5 paraspinal muscle and gluteal muscles with a complete relief of low back pain but persistent pain in the gluteal region. Finally, he was found to have attachment trigger point in the origin and insertion sites of gluteus medius and gluteus minimus muscles. He then further received local injection with kenacort and 1% lidocaine to the attachment trigger points with immediate relief of gluteal pain. A follow-up confirmed that he has no pain recurrence up to six months.

Conclusion: Chronic myofascial pain syndrome in the gluteal region can be caused by both lumbar facet joint lesion and attachment trigger points of the involved muscles. It is necessary to eliminate the underlying etiological lesions appropriately in order to provide long-term relief of myofascial pain.

KEY WORDS: Gluteal muscles, lumbar spine, myofascial pain, trigger point

INTRODUCTION

Pain in the gluteal region involving one or more of the gluteal muscles [including gluteus maximus, gluteus medius, gluteus minimus, and piriformis] is a common pain complaint in clinical practice. It usually combines with pain in the low back. In many cases, myofascial trigger points [TrPs] can be found in the gluteal muscles. A TrP is a hypersensitive spot in a taut band of skeletal muscle fibers which can cause a typical referred pain and local twitch responses (1-3). Following the figures showing the location of TrPs in the *Trigger Point Manual* (2,3), one can identify the most tender spots, the latent TrPs [tender, but not painful spontaneously], in most of normal skeletal muscles in adults. Latent TrPs can be activated into active TrPs that are painful spontaneously and much tender than the latent ones.

The diagnosis of a TrP can be based on the existence of a most painful exquisite spot in a palpable taut band with the pain or discomfort similar to the patient's usual complaint [pain recognition]. It can be further confirmed by a referred pain in response to compression of this spot, and a local twitch response [a brief sudden contraction of the muscle fibers in the taut band] upon snapping palpation of the spot across the muscle fibers of the taut band (2,3).

The treatments frequently used for TrP release includes manual therapy [such as massage, stretch, mobilization, manipulation, etc.] (3-6), therapeutic modality [such as ultrasound, electrotherapy, laser therapy, etc.] (3,7), acupuncture or dry needling (3,7-12), and TrP injection (3,7,10,13,14). It has been suggested that the treatment of the etiological lesion is the most important strategy to inactivate TrPs, since most TrPs (3) are elicited or related to the underlying etiological lesion (1,7,14).

It has been found that lumbar facet joint lesion is one important cause of chronic low back pain with gluteal referral (15-17). In most cases, chronic pain in the low back and gluteal region can be eliminated completely and permanently by lumbar facet joint injection (16,17). However,

in this case report, we found a patient with lumbar facet lesion causing chronic pain in the lower back and bilateral gluteal regions had persistent pain in the gluteal regions after suppression of low back pain with lumbar facet joint injection. We finally decided to inject the attachment trigger points of the gluteus medius and gluteus minimus muscles in both sides, and had obtained an excellent result for a long period.

MEDICAL HISTORY

A 45-year-old male patient had chronic pain in the low back and bilateral gluteal regions off and on for many years. The pain in bilateral gluteal regions was usually elicited during extension of low back with simultaneous rotation to the painful side, but no pain during trunk flexion. He had been a basketball player since at high school. He was initially treated with oral nonsteroidal anti-inflammatory drugs with poor results. X-ray films of the lumbar spine and pelvis showed evidence of degenerative changes with marginal osteophytes and reduced disc space at L4-5 and L5-S1 levels of lumbar spine. He then received physical therapy including thermotherapy followed by pelvic traction to the lumbar spine. The pain intensity was temporarily reduced after therapy for few days, but never had complete pain relief. He then received three local injections to the lumbar facet joints at bilateral L4-5 and L5-S1 levels with kenacort 1 ml (triamcinolone 40 mg) + 1% lidocaine 1ml for each site, and also received TrP injections (1% lidocaine 1-2 ml for each site) at bilateral L4-S1 lumbar paraspinal muscles and bilateral gluteal muscles immediately after each facet joint injection. For L5-S1 facet injection, the patient was in a comfortable supine position with the trunk in full extension, and the needle was inserted perpendicularly through the skin at the mid-point of the line connected from L5 spinous process to the ipsilateral posterior superior iliac spine, and then deeply penetrated to contact with the bony structure. The needle then was moved in-and-out (penetration and withdrawal) with a very small angle for each movement in different direction for locating the facet joint region. For any needle penetrating

movement, a sudden deeper needle movement before reaching the bony structure with bony boundary around the sides of the needle suggested that the needle was in the facet joint. For L4-5 facet injection, the needle was inserted through the skin at a point in the line through the site of L5-S1 injection and parallel to the line connecting the spinous process of L4 and L5, but above the site of L5-S1 injection for a distance equal to the distance between L4 and L5 spinous process. The other procedures are similar to that for L5-S1 facet injection. The pain in the low back and bilateral gluteal regions subsided immediately after injection. However, bilateral gluteal pain recurred two to three weeks later after each injection. He then was referred to our pain clinic.

PHYSICAL EXAMINATION

On examination, he had nearly normal ranges of motion in lumbar spine but mild paraspinal muscle spasm. Facet joint provoking sign [ipsilateral rotation followed by hyperextension of lumbar spine] was unremarkable in both sides. Straight leg raise with 90 degrees of hip flexion induced no radicular symptoms in both sides. There were active TrPs in bilateral gluteus medius and bilateral gluteus minimus muscles. The confirmation of TrPs was based on the existence of most tender spot in a palpable taut band with elicited referred pain (3). Resistive contraction of the gluteus medius or the gluteus minimus muscle induced TrP pain and pain in the origin and insertion sites of the tested muscle in either side. Compression of the muscle attachment region [either the origin or insertion site] can elicit pain in the TrP regions of the corresponding muscle. Neurological examination was unremarkable.

METHOD OF TREATMENT

Under the impression of attachment TrPs in the gluteal muscles, both the original sites and insertion sites of bilateral gluteus medius and gluteus minimus were injected with Kenacort-lidocaine solution [with a ratio of 1 ml (40 mg) Kenacort to 4 ml of 1 percent

lidocaine]. The amounts of solution injected were approximately 3 ml at the origin site and 2 ml at the insertion site for each muscle.

OUTCOME OF TREATMENT

Immediately after the injection, the gluteal pain subsided. All follow-up examinations one month, three months, and six months later revealed no evidence of active TrPs in the gluteal muscles in both sides. Resistive contraction of any gluteal muscle induced no pain. Lumbar facet provoking test was normal. Neurological examination was also normal.

DISCUSSION

This case report has demonstrated that chronic gluteal pain due to persistent TrP in gluteal muscles can be related to the persistent existence of attachment TrPs at both origin and insertion sites of the corresponding muscle, and appropriate treatment to the attachment TrP can provide a long-term relief of gluteal pain.

Diagnosis of Lumbar Facet Lesion Related to Gluteal Myofascial Trigger Points

Based on this patient's complaint of gluteal pain elicited by extension and ipsilateral rotation of low back, the diagnosis of lumbar facet lesion causing gluteal TrPs was suggested. In other lesions such as spondylolisthesis or lumbar spinal stenosis, extension and ipsilateral rotation of low back may also elicit similar pain. However, flexion of trunk can also induce pain in such cases. Since no pain was elicited during trunk flexion in our case, the diagnosis of lumbar facet lesion was much acceptable. A TrP in a specific muscle can be associated with a specific level of a facet joint lesion (7). This can be confirmed by provoking tests that irritate the facet joint to reproduce or aggravate pain in that TrP. The provoking test of a lumbar facet joint lesion can be performed by extension with rotation of lumbar spine to the painful side [facet sign] (7,16-18). With this maneuver, gluteal pain can be elicited. This finding can be further confirmed during facet joint injection. When the injecting needle encounters the corresponding facet joint

[usually L5-S1 or L4-5], sharp pain in the TrP region of gluteal muscles can usually be provoked. However, immediately after injection of local anesthetic (Lidocaine), this can be suppressed completely.

Correlation between Lumbar Facet Lesion and Gluteal Myofascial Trigger Points

Bogduk and Simons (19) suggested that a TrP in the upper trapezius or levator scapula muscle is frequently related to a C4-5 facet joint lesion, and a TrP in the rhomboid minor muscle is often related to a C5-6 facet joint lesion. In the lower limb, a TrP in the gluteus minimus muscle is often related to a lesion in the L4-5 facet joint, and a TrP in the piriformis muscle is frequently related to a lesion in the L5-S1 facet joint (7,17). It has been suggested that the TrP and the corresponding level of facet joint share the same neural pathway to the dorsal horn of spinal cord (19), so that injection of facet joint can relieve pain of TrP. However, if this is true, then injection of TrP can also suppress the pain in the facet joint. Clinically, we have never observed such phenomenon. It is most likely that TrPs are activated from the facet joint lesion. It is important to eliminate the etiological lesion, facet joint lesion, in order to avoid recurrence of peripheral TrPs.

Diagnosis of Attachment Trigger Points

Attachment TrP was initially defined by Fischer (20) and further explained by Simons (3). Simons also defined the TrP in the endplate zone of a muscle as a central TrP (3). Compression of an attachment TrP of a certain muscle can elicit pain locally and referred pain in the central TrP of this muscle (3). When we examined our patient, referred pain to the central TrPs could be elicited during compression of the origin or insertion site of gluteus medius or gluteus minimus muscle. Therefore, this spot in the origin or insertion site could be a typical attachment TrP. Resistive contraction of the gluteal muscle could also cause pain in the corresponding gluteal muscle. Resistive contraction can stretch the tendon attachment sites and cause pain in the

central TrP. If the pain was limited in these attachment sites, the diagnosis was just tendinopathy or enthesopathy (21,22). When the pain referred to the central TrP region, it should be an attachment TrP.

Pathogenesis of Attachment Trigger Points

Simons suggested that the strong tension in the taut band can cause prolonged stretch to the attachment site of tendon [enthesis (22)] and subsequently develop enthesopathy [attachment TrP] at this site (3). The tension in the taut band is elicited by the sustained contracture [shortening] of sarcomeres in the endplate zone [central portion of muscle fibers] as demonstrated both morphologically (23) and electrophysiologically (3,24). ~~with relatively lengthening of the sarcomeres in the two ends of muscle fibers (3). This phenomenon can be supported by two important findings in previous studies. The first evidence is a morphological finding of a contraction knot observed in the central portion of canine muscle fibers due to shortened sarcomeres and also evidence of lengthened sarcomeres in the two ends (23). The second evidence is an electrophysiological finding of endplate noise recorded in the TrP region [endplate zone] (3,24). Endplate noise is non-propagated potentials so that only the sarcomeres in the endplate zone contract, but not in the sarcomeres in the peripheral portion in both ends of the muscle fibers.~~ Based on reviewing previous literature, Simons ~~concluded that endplate noise is a consequence of excessive leakage of acetylcholine from the endplate. Simons~~ has further developed an integrated hypothesis of TrP (3,25,26), and has been supported by recent biochemical studies by Shah (27) who found ~~There are three essential features of TrPs: excessive acetylcholine release, sarcomere shortening, and release of sensitizing substances. These three essential features relate to one another in a positive feedback cycle that is self-perpetuating once it is started (3,25,26). An increased acetylcholine release in the motor endplates can cause an increase of the muscle fiber tension [taut band formation]. This can cause~~

~~"energy crisis" with increased metabolism and local ischemia with hypoxia, which can then induce secretion of sensitizing substances to cause pain. The sensitizing substances can further cause abnormal ACh release so that a vicious cycle is activated. Recent studies by Shah (27) has demonstrated~~ increased concentrations of pain- and inflammation-related substances in the TrP region.~~, and has further support Simons' integrated hypothesis of TrP. In summary,~~ Simons' integrated hypothesis of TrP can explain the formation of attachment TrPs.

Correlation of Attachment Trigger Points and Enthesopathy

Enthesopathy is usually caused by over contraction or persistent contraction of the tendon with subsequent injury to the attachment sites [entheses] (22). Persistent tightness of the taut band due to TrP can cause such damage. Therefore, enthesopathy can occur secondary to a long-standing attachment TrPs. On the other hand, a musculoskeletal lesion can activate TrPs [conversion of latent TrP into active TrP via central sensitization]. The association of active TrPs with cervical disc lesions (28), cervical facet lesions (19), cervical radiculopathy (29), lumbar disc lesions (30), osteoarthritis of knee (31), teres minor tendinitis (32), lateral epicondylitis (33), floating kidney (34), or herpes zoster (35) has been demonstrated. In summary, a TrP can cause enthesopathy due to the tension from the taut band, and an enthesopathy can also activate a latent TrP via central sensitization. Therefore, in any case, a vicious cycle can be initiated.

CONCLUSION

Chronic myofascial pain syndrome in the gluteal region can be caused by a lumbar facet joint lesion, or an attachment TrP, or both. It is necessary to treat both underlying etiological lesions appropriately in order to avoid the recurrence of myofascial pain.

REFERENCES

1. Hong C-Z, Simons DG: Pathophysiologic and electrophysiologic mechanism of myofascial trigger points. Arch Phys Med Rehabil 79: 863-872, 1998.
2. Travell JG, Simons DG: Myofascial pain and dysfunction: The trigger point manual. Vol. 2. Baltimore: Williams & Wilkins, 1992.
3. Simons DG: Diagnostic criteria of myofascial pain caused by trigger points. J Musculoske Pain 7: 111-120, 1999.
4. Hsieh CYJ, Adams AH, Tobis J, Hong C-Z, Danielson C, Platt K, Hoehler F, Reinsch S, Rubel A: Effectiveness of four conservative treatments for subacute low back pain: A randomized clinical trial. Spine 27: 1142-1148, 2002.
5. Lewit K: Manipulative therapy in rehabilitation of the locomotor system. Ed.2. Butterworth Heinemann. Oxford, 1991.
6. Kostopoulos D, Rizopoulos K: The manual of trigger point and myofascial therapy. SLACK Incorporated, Thorofare, New Jersey 2001.
7. Hong C-Z: Myofascial pain therapy. J Musculoske Pain 12: 37-43, 2004.
8. Chu J, Yuen K, Wang B, Chan R, Schwartz I, Neuhauser D: Electrical twitch-obtaining intramuscular stimulation in lower back pain: a pilot study. Am J Phy Med Rehabil 83:104-111, 2004.
9. Lewit K: The needle effect in the relief of myofascial pain. Pain 6: 83-90, 1979.
10. Hong C-Z: Lidocaine injection versus dry needling to myofascial trigger point: The importance of the local twitch response. Am J Phy Med Rehabil 73(4): 256-263, 1994.
11. Gunn C, Milbrandt W: Tenderness at motor points - A diagnostic and prognostic aid for low-back injury. J Bone Joint Surg Am 58: 815-825, 1976.
12. Cummings T, White A: Needling therapies in the management of myofascial trigger point

pain: A systemic review. Arch Phys Med Rehabil 82: 986-992, 2001.

13. Fischer AA: Treatment of Myofascial Pain. J Musculoske Pain 7: 131-142, 1999.
14. Hong C-Z: Considerations and recommendations regarding myofascial trigger point injection. J Musculoske Pain 2: 29-59, 1994.
15. Badgley CE: The articular facet in relation to low back pain and sciatic radiation. J Bone Joint Surg 23: 481, 1941.
16. Mooney V, Robertson J: The facet syndrome. Clin Orthop Relat Res. Mar-Apr: 149-156, 1976.
17. Kao M-J, Kuan T-S, Hsieh Y-L, Yang J-F, Hong C-Z: Myofascial Low Back Pain. Tw J Phy Med Rehabil 36: 1-14, 2008.
18. Chou L-W, Hong J-Y, Hong C-Z: A New Technique for Acupuncture Therapy and Its Effectiveness in Treating Fibromyalgia Syndrome: A Case Report. J Musculoske Pain 16: 193-198, 2008.
19. Bogduk N, Simons DG: Neck pain: joint pain or trigger points? In: Progress in Fibromyalgia and Myofascial Pain, Chapter 20, pp267-73, Vol 6 of Pain research and clinical management. Ed. Vaeroy H, Mersky H. Elsevier, Amsterdam 1993.
20. Fischer AA: New approaches in treatment of myofascial pain. Phy Med Rehab Clinic North America 8(1): 153-169, 1997.
21. Birrell F, Arden NK: A view on the pathogenesis of osteoarthritis from the shoulders of giants. Rheumatology 47: 1263-1264, 2008.
22. McGonagle D, Tan AL, Grainger AJ, Benjamin M: Heberden's nodes and what Heberden could not see: the pivotal role of ligaments in the pathogenesis of early nodal osteoarthritis and beyond. Rheumatology 47: 1278-1285, 2008.
23. Simons DG, Stolov WC: Microscopic features and transient contraction of palpable bands in

canine muscle. Am J Phys Med Rehabil 55: 65-88, 1976.

24. Simons DG, Hong C-Z, Simons LS: Endplate potentials are common to midfiber myofacial trigger points. Am J Phys Med Rehabil 81: 212-222, 2002.
25. Mense S, Simons DG: Muscle pain. Understanding its nature, diagnosis, and treatment. Philadelphia: Lippincott Williams & Wilkins, 2001.
26. Simons DG: New aspects of myofascial trigger points: etiological and clinical. J Musculoske Pain 12: 15-21, 2004.
27. Shah JP, Danoff JV, Desai MJ, Parikh S, Nakamura LY, Phillips TM, Gerber LH: Biochemicals associated with pain and inflammation are elevated in sites near to and remote from active myofascial trigger points. Arch Phys Med Rehabil 89: 16-23, 2008.
28. Hsueh T-C, Yu S, Kuan T-S, Hong C-Z: Association of active myofascial trigger points and cervical disc lesions. J Formos Med Assoc 97: 174-180, 1998.
29. Cannon DE, Dillingham TR, Miao H, Andary MT, Pezzin LE: Musculoskeletal disorders in referrals for suspected cervical radiculopathy. Arch Phys Med Rehabil 88: 1256-1259, 2007.
30. Samuel AS, Peter AA, Ramanathan K: The association of active trigger points with lumbar disc lesions. J Musculoske Pain 15: 11-18, 2007.
31. Bajaj P, Bajaj P, Graven-Nielsen T, Arendt-Nielsen L: Trigger Points in Patients with Lower Limb Osteoarthritis. J Musculoske Pain 9: 17-33, 2001.
32. Wang C-F, Chen M, Lin M-T, Kuan T-S, Hong C-Z: Teres minor tendinitis manifested with chronic myofascial pain syndrome in the scapular muscles: A case report. J Musculoske Pain 14: 39-43, 2006.
33. Fernández-Carnero J, Fernández-de-Las-Peñas C, de la Llave-Rincón AI, Ge HY, Arendt-Nielsen L: Prevalence of and referred pain from myofascial trigger points in the forearm muscles in patients with lateral epicondylalgia. Clin J Pain 23: 353-360, 2007.

34. Hsu H-C, Hong C-Z: Floating Kidney with Chronic Myofascial Pain Syndrome in the Abdominal Muscles as the Major Clinical Manifestation: A Case Report. J Musculoske Pain 16: 199-204, 2008.

35. Chen S-M, Chen J-T, Kuan T-S, Hong C-Z: Myofascial trigger points in intercostal muscles secondary to herpes zoster infection of the intercostal nerve. Arch Phys Med Rehabil 79: 336-338, 1998.

Treatment of Attachment Trigger Points in the Gluteal Muscles to Cure Chronic Gluteal Pain – A Case Report

Submitted: January 23, 2010

Resubmitted: September 12, 2010

ABSTRACT

Background: The purpose of this case report is to demonstrate a patient of chronic myofascial pain syndrome in the gluteal region secondary to lumbar facet joint lesion.

Findings: Initially, he was treated conservatively with medication and physical therapy and had only temporary pain relief for few days. Later, he was treated with lumbar facet joint injection (with kenacort) and myofascial trigger point injection (with 1% xylocaine) to bilateral L4-5 paraspinal muscle and gluteal muscles with a complete relief of low back pain but persistent pain in the gluteal region. Finally, he was found to have attachment trigger point in the origin and insertion sites of gluteus medius and gluteus minimus muscles. He then further received local injection with kenacort and 1% lidocaine to the attachment trigger points with immediate relief of gluteal pain. A follow-up confirmed that he has no pain recurrence up to six months.

Conclusion: Chronic myofascial pain syndrome in the gluteal region can be caused by both lumbar facet joint lesion and attachment trigger points of the involved muscles. It is necessary to eliminate the underlying etiological lesions appropriately in order to provide long-term relief of myofascial pain.

KEY WORDS: Gluteal muscles, lumbar spine, myofascial pain, trigger point

INTRODUCTION

Pain in the gluteal region involving one or more of the gluteal muscles [including gluteus maximus, gluteus medius, gluteus minimus, and piriformis] is a common pain complaint in clinical practice. It usually combines with pain in the low back. In many cases, myofascial trigger points [TrPs] can be found in the gluteal muscles. A TrP is a hypersensitive spot in a taut band of skeletal muscle fibers which can cause a typical referred pain and local twitch responses (1-3). Following the figures showing the location of TrPs in the *Trigger Point Manual* (2,3), one can identify the most tender spots, the latent TrPs [tender, but not painful spontaneously], in most of normal skeletal muscles in adults. Latent TrPs can be activated into active TrPs that are painful spontaneously and much tender than the latent ones.

The diagnosis of a TrP can be based on the existence of a most painful exquisite spot in a palpable taut band with the pain or discomfort similar to the patient's usual complaint [pain recognition]. It can be further confirmed by a referred pain in response to compression of this spot, and a local twitch response [a brief sudden contraction of the muscle fibers in the taut band] upon snapping palpation of the spot across the muscle fibers of the taut band (2,3).

The treatments frequently used for TrP release includes manual therapy [such as massage, stretch, mobilization, manipulation, etc.] (3-6), therapeutic modality [such as ultrasound, electrotherapy, laser therapy, etc.] (3,7), acupuncture or dry needling (3,7-12), and TrP injection (3,7,10,13,14). It has been suggested that the treatment of the etiological lesion is the most important strategy to inactivate TrPs, since most TrPs (3) are elicited or related to the underlying etiological lesion (1,7,14).

It has been found that lumbar facet joint lesion is one important cause of chronic low back pain with gluteal referral (15-17). In most cases, chronic pain in the low back and gluteal region can be eliminated completely and permanently by lumbar facet joint injection (16,17). However,

in this case report, we found a patient with lumbar facet lesion causing chronic pain in the lower back and bilateral gluteal regions had persistent pain in the gluteal regions after suppression of low back pain with lumbar facet joint injection. We finally decided to inject the attachment trigger points of the gluteus medius and gluteus minimus muscles in both sides, and had obtained an excellent result for a long period.

MEDICAL HISTORY

A 45-year-old male patient had chronic pain in the low back and bilateral gluteal regions off and on for many years. The pain in bilateral gluteal regions was usually elicited during extension of low back with simultaneous rotation to the painful side, but no pain during trunk flexion. He had been a basketball player since at high school. He was initially treated with oral nonsteroidal anti-inflammatory drugs with poor results. X-ray films of the lumbar spine and pelvis showed evidence of degenerative changes with marginal osteophytes and reduced disc space at L4-5 and L5-S1 levels of lumbar spine. He then received physical therapy including thermotherapy followed by pelvic traction to the lumbar spine. The pain intensity was temporarily reduced after therapy for few days, but never had complete pain relief. He then received three local injections to the lumbar facet joints at bilateral L4-5 and L5-S1 levels with kenacort 1 ml (triamcinolone 40 mg) + 1% lidocaine 1ml for each site, and also received TrP injections (1% lidocaine 1-2 ml for each site) at bilateral L4-S1 lumbar paraspinal muscles and bilateral gluteal muscles immediately after each facet joint injection. For L5-S1 facet injection, the patient was in a comfortable supine position with the trunk in full extension, and the needle was inserted perpendicularly through the skin at the mid-point of the line connected from L5 spinous process to the ipsilateral posterior superior iliac spine, and then deeply penetrated to contact with the bony structure. The needle then was moved in-and-out (penetration and withdrawal) with a very small angle for each movement in different direction for locating the facet joint region. For any needle penetrating

movement, a sudden deeper needle movement before reaching the bony structure with bony boundary around the sides of the needle suggested that the needle was in the facet joint. For L4-5 facet injection, the needle was inserted through the skin at a point in the line through the site of L5-S1 injection and parallel to the line connecting the spinous process of L4 and L5, but above the site of L5-S1 injection for a distance equal to the distance between L4 and L5 spinous process. The other procedures are similar to that for L5-S1 facet injection. The pain in the low back and bilateral gluteal regions subsided immediately after injection. However, bilateral gluteal pain recurred two to three weeks later after each injection. He then was referred to our pain clinic.

PHYSICAL EXAMINATION

On examination, he had nearly normal ranges of motion in lumbar spine but mild paraspinal muscle spasm. Facet joint provoking sign [ipsilateral rotation followed by hyperextension of lumbar spine] was unremarkable in both sides. Straight leg raise with 90 degrees of hip flexion induced no radicular symptoms in both sides. There were active TrPs in bilateral gluteus medius and bilateral gluteus minimus muscles. The confirmation of TrPs was based on the existence of most tender spot in a palpable taut band with elicited referred pain (3). Resistive contraction of the gluteus medius or the gluteus minimus muscle induced TrP pain and pain in the origin and insertion sites of the tested muscle in either side. Compression of the muscle attachment region [either the origin or insertion site] can elicit pain in the TrP regions of the corresponding muscle. Neurological examination was unremarkable.

METHOD OF TREATMENT

Under the impression of attachment TrPs in the gluteal muscles, both the original sites and insertion sites of bilateral gluteus medius and gluteus minimus were injected with Kenacort-lidocaine solution [with a ratio of 1 ml (40 mg) Kenacort to 4 ml of 1 percent

lidocaine]. The amounts of solution injected were approximately 3 ml at the origin site and 2 ml at the insertion site for each muscle.

OUTCOME OF TREATMENT

Immediately after the injection, the gluteal pain subsided. All follow-up examinations one month, three months, and six months later revealed no evidence of active TrPs in the gluteal muscles in both sides. Resistive contraction of any gluteal muscle induced no pain. Lumbar facet provoking test was normal. Neurological examination was also normal.

DISCUSSION

This case report has demonstrated that chronic gluteal pain due to persistent TrP in gluteal muscles can be related to the persistent existence of attachment TrPs at both origin and insertion sites of the corresponding muscle, and appropriate treatment to the attachment TrP can provide a long-term relief of gluteal pain.

Diagnosis of Lumbar Facet Lesion Related to Gluteal Myofascial Trigger Points

Based on this patient's complaint of gluteal pain elicited by extension and ipsilateral rotation of low back, the diagnosis of lumbar facet lesion causing gluteal TrPs was suggested. In other lesions such as spondylolisthesis or lumbar spinal stenosis, extension and ipsilateral rotation of low back may also elicit similar pain. However, flexion of trunk can also induce pain in such cases. Since no pain was elicited during trunk flexion in our case, the diagnosis of lumbar facet lesion was much acceptable. A TrP in a specific muscle can be associated with a specific level of a facet joint lesion (7). This can be confirmed by provoking tests that irritate the facet joint to reproduce or aggravate pain in that TrP. The provoking test of a lumbar facet joint lesion can be performed by extension with rotation of lumbar spine to the painful side [facet sign] (7,16-18). With this maneuver, gluteal pain can be elicited. This finding can be further confirmed during facet joint injection. When the injecting needle encounters the corresponding facet joint

[usually L5-S1 or L4-5], sharp pain in the TrP region of gluteal muscles can usually be provoked. However, immediately after injection of local anesthetic (Lidocaine), this can be suppressed completely.

Correlation between Lumbar Facet Lesion and Gluteal Myofascial Trigger Points

Bogduk and Simons (19) suggested that a TrP in the upper trapezius or levator scapula muscle is frequently related to a C4-5 facet joint lesion, and a TrP in the rhomboid minor muscle is often related to a C5-6 facet joint lesion. In the lower limb, a TrP in the gluteus minimus muscle is often related to a lesion in the L4-5 facet joint, and a TrP in the piriformis muscle is frequently related to a lesion in the L5-S1 facet joint (7,17). It has been suggested that the TrP and the corresponding level of facet joint share the same neural pathway to the dorsal horn of spinal cord (19), so that injection of facet joint can relieve pain of TrP. However, if this is true, then injection of TrP can also suppress the pain in the facet joint. Clinically, we have never observed such phenomenon. It is most likely that TrPs are activated from the facet joint lesion. It is important to eliminate the etiological lesion, facet joint lesion, in order to avoid recurrence of peripheral TrPs.

Diagnosis of Attachment Trigger Points

Attachment TrP was initially defined by Fischer (20) and further explained by Simons (3). Simons also defined the TrP in the endplate zone of a muscle as a central TrP (3). Compression of an attachment TrP of a certain muscle can elicit pain locally and referred pain in the central TrP of this muscle (3). When we examined our patient, referred pain to the central TrPs could be elicited during compression of the origin or insertion site of gluteus medius or gluteus minimus muscle. Therefore, this spot in the origin or insertion site could be a typical attachment TrP. Resistive contraction of the gluteal muscle could also cause pain in the corresponding gluteal muscle. Resistive contraction can stretch the tendon attachment sites and cause pain in the

central TrP. If the pain was limited in these attachment sites, the diagnosis was just tendinopathy or enthesopathy (21,22). When the pain referred to the central TrP region, it should be an attachment TrP.

Pathogenesis of Attachment Trigger Points

Simons suggested that the strong tension in the taut band can cause prolonged stretch to the attachment site of tendon [enthesis (22)] and subsequently develop enthesopathy [attachment TrP] at this site (3). The tension in the taut band is elicited by the sustained contracture [shortening] of sarcomeres in the endplate zone [central portion of muscle fibers] as demonstrated both morphologically (23) and electrophysiologically (3,24). Based on reviewing previous literature, Simons has further developed an integrated hypothesis of TrP (3,25,26), and has been supported by recent biochemical studies by Shah (27) who found increased concentrations of pain- and inflammation-related substances in the TrP region. Simons' integrated hypothesis of TrP can explain the formation of attachment TrPs.

Correlation of Attachment Trigger Points and Enthesopathy

Enthesopathy is usually caused by over contraction or persistent contraction of the tendon with subsequent injury to the attachment sites [entheses] (22). Persistent tightness of the taut band due to TrP can cause such damage. Therefore, enthesopathy can occur secondary to a long-standing attachment TrPs. On the other hand, a musculoskeletal lesion can activate TrPs [conversion of latent TrP into active TrP via central sensitization]. The association of active TrPs with cervical disc lesions (28), cervical facet lesions (19), cervical radiculopathy (29), lumbar disc lesions (30), osteoarthritis of knee (31), teres minor tendinitis (32), lateral epicondylitis (33), floating kidney (34), or herpes zoster (35) has been demonstrated. In summary, a TrP can cause enthesopathy due to the tension from the taut band, and an enthesopathy can also activate a latent TrP via central sensitization. Therefore, in any case, a vicious cycle can be initiated.

CONCLUSION

Chronic myofascial pain syndrome in the gluteal region can be caused by a lumbar facet joint lesion, or an attachment TrP, or both. It is necessary to treat both underlying etiological lesions appropriately in order to avoid the recurrence of myofascial pain.

REFERENCES

1. Hong C-Z, Simons DG: Pathophysiologic and electrophysiologic mechanism of myofascial trigger points. Arch Phys Med Rehabil 79: 863-872, 1998.
2. Travell JG, Simons DG: Myofascial pain and dysfunction: The trigger point manual. Vol. 2. Baltimore: Williams & Wilkins, 1992.
3. Simons DG: Diagnostic criteria of myofascial pain caused by trigger points. J Musculoske Pain 7: 111-120, 1999.
4. Hsieh CYJ, Adams AH, Tobis J, Hong C-Z, Danielson C, Platt K, Hoehler F, Reinsch S, Rubel A: Effectiveness of four conservative treatments for subacute low back pain: A randomized clinical trial. Spine 27: 1142-1148, 2002.
5. Lewit K: Manipulative therapy in rehabilitation of the locomotor system. Ed.2. Butterworth Heinemann. Oxford, 1991.
6. Kostopoulos D, Rizopoulos K: The manual of trigger point and myofascial therapy. SLACK Incorporated, Thorofare, New Jersey 2001.
7. Hong C-Z: Myofascial pain therapy. J Musculoske Pain 12: 37-43, 2004.
8. Chu J, Yuen K, Wang B, Chan R, Schwartz I, Neuhauser D: Electrical twitch-obtaining intramuscular stimulation in lower back pain: a pilot study. Am J Phy Med Rehabil 83:104-111, 2004.
9. Lewit K: The needle effect in the relief of myofascial pain. Pain 6: 83-90, 1979.
10. Hong C-Z: Lidocaine injection versus dry needling to myofascial trigger point: The importance of the local twitch response. Am J Phy Med Rehabil 73(4): 256-263, 1994.
11. Gunn C, Milbrandt W: Tenderness at motor points - A diagnostic and prognostic aid for low-back injury. J Bone Joint Surg Am 58: 815-825, 1976.
12. Cummings T, White A: Needling therapies in the management of myofascial trigger point

pain: A systemic review. Arch Phys Med Rehabil 82: 986-992, 2001.

13. Fischer AA: Treatment of Myofascial Pain. J Musculoske Pain 7: 131-142, 1999.
14. Hong C-Z: Considerations and recommendations regarding myofascial trigger point injection. J Musculoske Pain 2: 29-59, 1994.
15. Badgley CE: The articular facet in relation to low back pain and sciatic radiation. J Bone Joint Surg 23: 481, 1941.
16. Mooney V, Robertson J: The facet syndrome. Clin Orthop Relat Res. Mar-Apr: 149-156, 1976.
17. Kao M-J, Kuan T-S, Hsieh Y-L, Yang J-F, Hong C-Z: Myofascial Low Back Pain. Tw J Phy Med Rehabil 36: 1-14, 2008.
18. Chou L-W, Hong J-Y, Hong C-Z: A New Technique for Acupuncture Therapy and Its Effectiveness in Treating Fibromyalgia Syndrome: A Case Report. J Musculoske Pain 16: 193-198, 2008.
19. Bogduk N, Simons DG: Neck pain: joint pain or trigger points? In: Progress in Fibromyalgia and Myofascial Pain, Chapter 20, pp267-73, Vol 6 of Pain research and clinical management. Ed. Vaeroy H, Mersky H. Elsevier, Amsterdam 1993.
20. Fischer AA: New approaches in treatment of myofascial pain. Phy Med Rehab Clinic North America 8(1): 153-169, 1997.
21. Birrell F, Arden NK: A view on the pathogenesis of osteoarthritis from the shoulders of giants. Rheumatology 47: 1263-1264, 2008.
22. McGonagle D, Tan AL, Grainger AJ, Benjamin M: Heberden's nodes and what Heberden could not see: the pivotal role of ligaments in the pathogenesis of early nodal osteoarthritis and beyond. Rheumatology 47: 1278-1285, 2008.
23. Simons DG, Stolov WC: Microscopic features and transient contraction of palpable bands in

canine muscle. Am J Phys Med Rehabil 55: 65-88, 1976.

24. Simons DG, Hong C-Z, Simons LS: Endplate potentials are common to midfiber myofacial trigger points. Am J Phys Med Rehabil 81: 212-222, 2002.
25. Mense S, Simons DG: Muscle pain. Understanding its nature, diagnosis, and treatment. Philadelphia: Lippincott Williams & Wilkins, 2001.
26. Simons DG: New aspects of myofascial trigger points: etiological and clinical. J Musculoske Pain 12: 15-21, 2004.
27. Shah JP, Danoff JV, Desai MJ, Parikh S, Nakamura LY, Phillips TM, Gerber LH: Biochemicals associated with pain and inflammation are elevated in sites near to and remote from active myofascial trigger points. Arch Phys Med Rehabil 89: 16-23, 2008.
28. Hsueh T-C, Yu S, Kuan T-S, Hong C-Z: Association of active myofascial trigger points and cervical disc lesions. J Formos Med Assoc 97: 174-180, 1998.
29. Cannon DE, Dillingham TR, Miao H, Andary MT, Pezzin LE: Musculoskeletal disorders in referrals for suspected cervical radiculopathy. Arch Phys Med Rehabil 88: 1256-1259, 2007.
30. Samuel AS, Peter AA, Ramanathan K: The association of active trigger points with lumbar disc lesions. J Musculoske Pain 15: 11-18, 2007.
31. Bajaj P, Bajaj P, Graven-Nielsen T, Arendt-Nielsen L: Trigger Points in Patients with Lower Limb Osteoarthritis. J Musculoske Pain 9: 17-33, 2001.
32. Wang C-F, Chen M, Lin M-T, Kuan T-S, Hong C-Z: Teres minor tendinitis manifested with chronic myofascial pain syndrome in the scapular muscles: A case report. J Musculoske Pain 14: 39-43, 2006.
33. Fernández-Carnero J, Fernández-de-Las-Peñas C, de la Llave-Rincón AI, Ge HY, Arendt-Nielsen L: Prevalence of and referred pain from myofascial trigger points in the forearm muscles in patients with lateral epicondylalgia. Clin J Pain 23: 353-360, 2007.

34. Hsu H-C, Hong C-Z: Floating Kidney with Chronic Myofascial Pain Syndrome in the Abdominal Muscles as the Major Clinical Manifestation: A Case Report. J Musculoske Pain 16: 199-204, 2008.

35. Chen S-M, Chen J-T, Kuan T-S, Hong C-Z: Myofascial trigger points in intercostal muscles secondary to herpes zoster infection of the intercostal nerve. Arch Phys Med Rehabil 79: 336-338, 1998.