



# Gluteus medius muscle decompression for buttock pain: a case-series analysis

Kyongsong Kim<sup>1,2</sup> · Toyohiko Isu<sup>1</sup> · Juntaro Matsumoto<sup>1</sup> · Koichi Miki<sup>1</sup> · Daijiro Morimoto<sup>3</sup> · Masanori Isobe<sup>1</sup> · Akio Morita<sup>3</sup>

Received: 16 January 2019 / Accepted: 18 April 2019 / Published online: 3 May 2019  
© Springer-Verlag GmbH Austria, part of Springer Nature 2019

## Abstract

**Background** The causes of low back and buttock pain are variable. Elsewhere, we presented a surgical technical note addressing the gluteus medius muscle (GMeM) pain that elicited buttock pain treatable by surgical decompression. Here, we report minimum 2-year surgical outcomes of GMeM decompression for intractable buttock pain.

**Methods** Between January 2014 and December 2015, we surgically treated 55 consecutive patients with a GMeM pain. Of these, 39 were followed for at least 2 years; they were included in this study. Their average age was 69.2 years; 17 were men and 22 were women. The affected side was unilateral in 24 patients and bilateral in the other 15 (total 54 sites). The mean follow-up period was 40.0 months (range 25–50 months). The severity of pre- and post-treatment pain was recorded on the numerical rating scale (NRS) and the Roland-Morris Disability Questionnaire (RDQ).

**Results** Of the 39 patients, 35 also presented with leg symptoms. They were exacerbated by walking in all 39 patients and by prolonged sitting in 33 patients; 19 had a past history of lumbar surgery and 4 manifested failed back surgery syndrome. Repeat surgery for wider decompression was performed in 5 patients due to pain recurrence 15.8 months after the first operation. At the last follow-up, the symptoms were significantly improved; the average NRS fell from 7.4 to 2.1 and the RDQ score from 10.5 to 3.3 ( $p < 0.05$ ).

**Conclusions** When diagnostic criteria are met, GMeM decompression under local anesthesia is a useful treatment for intractable buttock pain.

**Keywords** Buttock pain · Case series · Decompression surgery · Gluteus medius muscle · Surgical results

## Introduction

As the causes of low back and buttock pain are variable, a successful treatment outcome requires a precise differential diagnosis. The gluteus medius muscle (GMeM) is located in the buttock, partially under the gluteus maximus muscle

(GMaM). Elsewhere [9, 10, 12], we identified GMeM pain as a cause of buttock pain that can be treated by GMeM block and less-invasive surgical decompression under local anesthesia. In 2016, we presented our preliminary technical results of GMeM decompression in patients [10]. We now report the surgical results in 39 patients who were followed for at least 24 months after GMeM decompression for intractable buttock pain.

---

This article is part of the Topical Collection on *Spine*

---

✉ Kyongsong Kim  
kyongson@nms.ac.jp

<sup>1</sup> Department of Neurosurgery, Kushiro Rosai Hospital, Kushiro City, Hokkaido, Japan

<sup>2</sup> Department of Neurosurgery, Chiba Hokuso Hospital, Nippon Medical School, 1715, Kamagari, Inzai City, Chiba, Japan

<sup>3</sup> Department of Neurosurgery, Nippon Medical School, Bunkyo-ku, Tokyo, Japan

## Materials and methods

### Diagnostic criteria for GMeM pain

The diagnosis of GMeM pain was based on clinical symptoms [9, 10, 12]. We inspected radiograms to exclude patients with osteoarthritis of the hip joint. GMeM pain is defined as unilateral or bilateral buttock pain involving the area around the

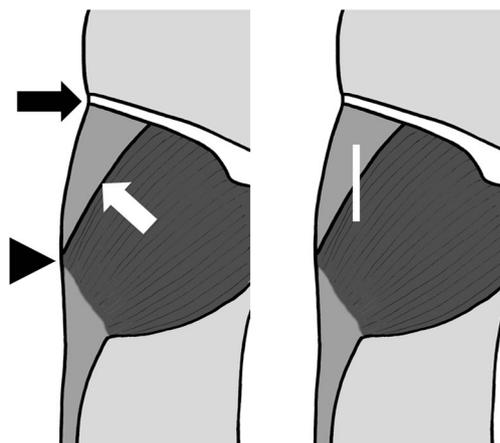
GMeM; the trigger point is located on the GMeM at the edge of the GMaM at the same distance from the iliac crest and the greater trochanter (Fig. 1). When local GMeM block with 5 ml of 1% lidocaine yields transient pain reduction of more than 50% on the numerical rating scale (NRS) within 2 h, our diagnosis is GMeM pain.

## Patients

Between January 2014 and December 2015, we diagnosed and surgically treated GMeM-related pain in 55 consecutive patients. Our diagnostic criteria included the effectiveness of GMeM block. For pain control, we usually prescribed medication (acetaminophen and/or non-steroidal anti-inflammatory drugs (NSAIDs) and/or tramadol) and physiotherapy, and performed repeat local injections. Codeine or morphine was not used.

Only non-responders to drug treatment and patients with pain recurrence after the analgesic effect of muscle blockage wore off were considered surgical candidates. We did not include patients with a history of hip joint surgery, acute fractures, malignancy-related diseases, infection, or trauma to the affected area.

Of the 55 operated patients, 39 were followed for at least 2 years; they are the current study population. Informed consent was obtained from all participants included in the study. They were 17 men and 22 women; their average age was 69.2 years (range 33–90 years). The affected side was unilateral in 24 patients (left,  $n = 17$ ; right,  $n = 7$ ) and bilateral in 15 (total 54 sites). Before GMeM surgery, 19 patients had undergone lumbar surgery (fusion surgery,  $n = 3$ ); 4 presented with failed back surgery syndrome (FBSS). The number of blocks delivered before GMeM decompression surgery depended on the patients' condition and the blocks' effect; on average, we administered 5.1 blocks (range 2–16 blocks). The mean follow-up period was 40.0 months (range 25–50 months).



**Fig. 1** Trigger point (white arrow) of gluteus medius muscle (GMeM) pain (left). Iliac crest (arrow), greater trochanter (arrow head). Skin incision for GMeM decompression surgery. A 5-cm skin incision (vertical bar) is made across the trigger point on the buttock

## Surgical treatment for GMeM pain

With the patient in the prone position and under local anesthesia, the senior author (T.I.) performed microscopic GMeM decompression. A 5-cm linear skin incision was placed across the trigger point on the buttock (Fig. 1). For wide GMeM exposure, the tight fibrous tissue and fascial attachment to the GMaM was dissected and the muscle was retracted medioinferiorly (Fig. 2a). After wide exposure of the gluteal aponeurosis over the GMeM, it was cut and opened for sufficient GMeM decompression. A stellate incision was placed rostral to the iliac crest, medial to the sacroiliac joint, caudal to near the greater trochanter, and lateral to the tensor fascia latae muscle (Fig. 2b, c). After surgery, patient movements were not restricted. A subcutaneous drain was removed 1 day after surgery.

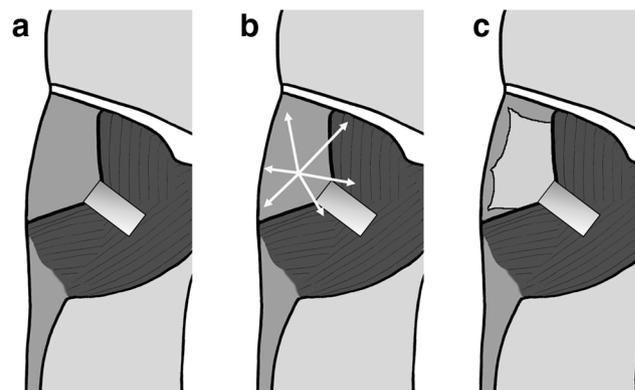
## Evaluation of clinical outcomes

The severity of buttock pain was evaluated on the NRS for low back pain (LBP) including buttock pain and on the Roland-Morris Disability Questionnaire (RDQ) before treatment and at the last follow-up [4, 6]. For statistical analysis, we subjected our data to the paired  $t$  test using Statmate III software (ATMS Co., Ltd.). Differences of  $p < 0.05$  were considered statistically significant.

## Results

### Symptoms

As show in Table 1, all 39 patients experienced tenderness on palpation of the gluteus medius region. All reported buttock pain; 35 also suffered lateral and posterior femoral



**Fig. 2** Surgical procedure of gluteus medius muscle (GMeM) decompression. For wide GMeM exposure, the tight fibrous tissue and fascial attachment to the gluteus maximus muscle are dissected and the muscle is retracted medioinferiorly (a). The gluteal aponeurosis of the GMeM is cut for wide GMeM exposure (b). Arrows indicate the incision and direction. Completed GMeM decompression (c)

**Table 1.** Symptoms in 39 patients with gluteus medius muscle pain.

Tenderness on palpation of the gluteus medius region	<i>n</i> = 39
Buttock pain	<i>n</i> = 39
Lateral and posterior femoral pain	<i>n</i> = 35 (89.7%)
Symptoms exacerbated by	
Walking	<i>n</i> = 39
Prolonged sitting	<i>n</i> = 33 (84.6%)
Prolonged standing	<i>n</i> = 11 (28.2%)
Half-rising posture	<i>n</i> = 4 (10.3%)
Standing	<i>n</i> = 3 (7.7%)

pain. The symptoms were exacerbated by walking (39 patients), prolonged sitting (33 patients) or prolonged standing (11 patients), standing (3 patients), and in the half-rising posture (4 patients). Buttock pain was elicited and increased by compression of the trigger point. The preoperative NRS and RDQ scores were 7.4 (range 4–10) and 10.5 (range 2–20), respectively.

### Surgical results

We encountered no surgical complications. Surgery was not effective in three females with bilateral involvement; their NRS fell from 8.3 to 6.7 and their RDQ score rose from 15.3 to 15.5. In the course of GMeM decompression follow-up, 16 patients underwent 20 additional procedures (lumbar surgery *n* = 7, SCN neurolysis for superior cluneal (SCN) entrapment neuropathy *n* = 6, middle cluneal nerve (MCN) neurolysis for MCN entrapment, *n* = 7). Repeat surgery for wider decompression was required in 5 sites of the all 54 sites due to buttock pain recurrence after 15.8 months (range 7–38 months); the mean follow-up period of these 5 cases after the second procedure was 24.0 months (range 5–33 months). At the last follow-up, the NRS and RDQ scores improved to 2.1 (range 0–7) and 3.3 (range 0–18), respectively ( $p < 0.05$ ).

## Discussion

### GMeM pain characteristics and symptoms

The GMeM is located in the buttock and partially covered by the GMaM. It helps to stabilize the pelvis in the raised leg position and when standing, walking, or running, and it plays a role in stabilizing the femur and pelvis during these activities. Although the GMeM is most highly active during walking, considering its size, it generates an exceptionally large force [19, 22, 25]. Excessive loading can elicit GMeM pain.

Symptoms of GMeM pain involve the buttock without paresis. Although 35 of our 39 patients also suffered leg symptoms, the main pain region was located in the anatomical area harboring the GMeM. Clinical features are diagnostically important. All of our patients reported buttock pain that was exacerbated by walking. Pain elicited by prolonged sitting was also a characteristic symptom; standing, prolonged standing, and a half-rising posture worsened their symptoms. As such, symptoms are also reported by patients with lumbar disease, a specific differential diagnosis is needed. In our 4 patients with FBSS, GMeM pain treatment was effective [7, 9, 15, 26].

There is no objective method for the diagnosis of GMeM and clinical symptoms are of diagnostic importance. Although magnetic resonance imaging (MRI) may be useful, we did not obtain MRI scans of the GMeM. The MRI characteristics in patients with GMeM pain require further elucidation. For the diagnosis and treatment of GMeM pain, muscle blocking is useful [10, 12, 15]. When GMeM blockage results in pain reduction, our diagnosis is buttock pain attributable to GMeM pain. We do not know why 3 of our 39 patients failed to experience sufficient improvement after GMeM decompression. In patients with suspected GMeM pain, we consider GMeM decompression surgery only when the effect of muscle blockage is transient and conservative therapy with medication, physiotherapy, and repeat local injections is ineffective. Our current diagnostic criteria for GMeM pain include transient pain reduction by more than 50% after GMeM blockage [9, 10, 12]; changing this criterion to a pain reduction by more than 75% may lower the number of patients with unsatisfactory surgical outcomes.

Although our observation therapy included muscle stretching and muscle-strength training, surgery was required in 39 patients. The development of specialized physiotherapy may help to reduce their number. Botulinum toxin treatment, effective in patients with piriformis syndrome [3, 8], may be useful; however, it has not been approved in Japan.

### Relationship between GMeM pain and lumbar- and para-lumbar spine diseases

Of our 39 patients, 19 had a past history of lumbar surgery. After GMeM decompression, 7 patients required lumbar surgery, 6 underwent SCN decompression for SCN-entrapment (SCN-E), and 7 were additionally treated by MCN decompression. This indicates that patients with GMeM pain may manifest FBSS and that GMeM pain might be attributable to other lumbar and para-lumbar spine disorders.

The relationship between LBP and GMeM activation has been documented [2, 19, 25], suggesting that the GMeM may play a significant role in LBP. An increase in the paravertebral muscle tonus due to several lumbar diseases affects the tension in the posterior layer of the thoracolumbar fascia [1, 20]. As a large portion of the fascia is attached to the tight gluteal aponeurosis over the GMeM, the tenor fascia latae muscle and the iliotibial tract [24] force are transmitted to the buttocks and lower limbs. Entrapment of the SCN or MCN by the thoracolumbar fascia can elicit LBP and buttock pain [7, 11, 13, 14, 16, 17]. An increase in muscle-loading and in the fascial tension tonus may lead to the concurrent elicitation of pain attributable to different diseases. Consequently, the treatment of only one such disease may not resolve all LBP and some patients may require combination treatments. This was the case in 16 of our patient who underwent surgery for co-existing lumbar or para-lumbar spine diseases after GMeM decompression.

### Etiology of GMeM pain

The etiology of GMeM pain remains unknown. Patients who underwent fasciotomy and GMeM decompression reported improvement in their buttock pain, an observation that sheds some light on the etiology of GMeM pain. With respect to the paraspinal muscle, patients with chronic compartment syndrome can be asymptomatic at rest while their LBP is exacerbated by muscle loading; chronic loading of the muscle can be addressed by its decompression via fasciotomy [18, 23].

The GMeM is located in a relatively rigid, closed space between the iliac bone and the gluteal aponeurosis and is prone to burdening [19, 25]. Gluteal compartment syndrome (GCS) is one cause of gluteal pain and tenderness and it can progress to rhabdomyolysis [5, 21, 23]. Although rare, GCS can be due to prolonged gluteal muscle compression and tends to progress rapidly.

None of our patients manifested rapid disease progression or rhabdomyolysis. Intraoperatively, we observed GMeM expansion upon GMeM decompression. However, 5 of our patients suffered GMeM pain recurrence due to insufficient GMeM decompression; subsequent additional GMeM decompression yielded satisfactory results. As GMeM pain may be attributable to chronic compartment syndrome involving the GMeM, intramuscular pressure measurements may be of diagnostic value.

As it remains unclear whether GMeM irritation is the driver of pain or whether it is the result of another process, it is necessary to rule out other potential causative factors of LBP before GMeM decompression alone can be considered successful.

### Limitations

First, ours is a retrospective, single-center study of a relatively small number of patients who underwent GMeM decompression after muscle blocks failed to yield long-lasting relief from intractable buttock pain. Second, we did not evaluate symptoms elicited by hip and pelvis movement. A prospective cohort study involving several institutions is underway to shed more light on the etiology of GMeM pain and to evaluate the clinical outcome after GMeM decompression.

### Conclusions

When diagnostic criteria are met, GMeM decompression under local anesthesia is an acceptable treatment in patients with intractable buttock pain.

### Compliance with ethical standards

All procedures involving human subjects were in compliance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Conflict of interest** The authors declare that they have no conflict of interest.

### References

1. Cloward RB (1963) Lesions of the intervertebral disks and their treatment by interbody fusion methods. *The painful disk. Clin Orthop Relat Res* 27:51–77
2. Cooper NA, Scavo KM, Strickland KJ, Tipayamongkol N, Nicholson JD, Bewyer DC, Sluka KA (2016) Prevalence of gluteus medius weakness in people with chronic low back pain compared to healthy controls. *Eur Spine J* 25:1258–1265
3. Fishman LM, Wilkins AN, Rosner B (2017) Electrophysiologically identified piriformis syndrome is successfully treated with incobotulinum toxin a and physical therapy. *Muscle Nerve* 56:258–263
4. Fujiwara A, Kobayashi N, Saiki K, Kitagawa T, Tamai K, Saotome K (2003) Association of the Japanese Orthopaedic Association score with the Oswestry Disability Index, Roland-Morris Disability Questionnaire, and Short-Form 36. *Spine* 28:1601–1607
5. Hill SL, Bianchi J (1997) The gluteal compartment syndrome. *Am Surg* 63:823–826
6. Hjerstad MJ, Fayers PM, Haugen DF, Caraceni A, Hanks GW, Loge JH, Fainsinger R, Aass N, Kaasa S, European Palliative Care Research Collaborative (EPCRC) (2011) Studies comparing numerical rating scales, verbal rating scales, and visual analogue scales for assessment of pain intensity in adults: a systematic literature review. *J Pain Symptom Manag* 41:1073–1093
7. Isu T, Kim K, Morimoto D, Iwamoto N (2018) Superior and middle cluneal nerve entrapment as a cause of the low back pain. *Clinical review. Neurospine* 15:25–32

8. Jankovic D, Peng P, van Zundert A (2013) Brief review: piriformis syndrome: etiology, diagnosis, and management. *Can J Anaesth* 60: 1003–1012
9. Kim K, Isu T, Morimoto D, Iwamoto N, Kokubo R, Matsumoto J, Kitamura T, Sugawara A, Morita A (2017) Common diseases mimicking lumbar disc herniation and their treatment. *Mini-Invas Surg* 1:43–51
10. Kim K, Isu T, Chiba Y, Iwamoto N, Morimoto D, Isobe M (2016) Decompression of the gluteus medius muscle as a new treatment for buttock pain: technical note. *Eur Spine J* 25:1282–1288
11. Kim K, Isu T, Matsumoto J, Yamazaki K, Isobe M (2018) Low back pain due to middle cluneal nerve entrapment neuropathy. *Eur Spine J* 27:S309–S313
12. Kokubo R, Kim K, Isu T, Morimoto D, Iwamoto N, Kobayashi S, Morita A (2017) Superior cluneal nerve entrapment neuropathy and gluteus medius muscle pain: their effect on very old patients with low back pain. *World Neurosurg* 98:132–139
13. Maigne JY, Doursounian L (1997) Entrapment neuropathy of the medial superior cluneal nerve. Nineteen cases surgically treated, with a minimum of 2 years' follow-up. *Spine* 22:1156–1159
14. Matsumoto J, Isu T, Kim K, Iwamoto N, Morimoto D, Isobe M (2018) Surgical treatment of middle cluneal nerve entrapment neuropathy. Technical note. *J Neurosurg Spine* 18:1–6
15. Matsumoto J, Isu T, Kim K, Iwamoto N, Yamazaki K, Morimoto D, Isobe M (2018) Impact of additional treatment of para-lumbar- and peripheral nerve diseases after lumbar spine surgery. *World Neurosurg* 112:e778–e782
16. Morimoto D, Isu T, Kim K, Chiba Y, Iwamoto N, Isobe M, Morita A (2017) Long-term outcome of surgical treatment for superior cluneal nerve entrapment neuropathy. *Spine* 42:783–788
17. Morimoto D, Isu T, Kim K, Imai T, Yamazaki K, Matsumoto R, Isobe M (2013) Surgical treatment of superior cluneal nerve entrapment neuropathy. *J Neurosurg Spine* 19:71–75
18. Nathan ST, Roberts CS, Deliberato D (2012) Lumbar paraspinal compartment syndrome. *Int Orthop* 36:1221–1227
19. Nelson-Wong E, Callaghan JP (2010) Is muscle co-activation a predisposing factor for low back pain development during standing? A multifactorial approach for early identification of at risk individuals. *J Electromyogr Kinesiol* 20:256–263
20. Pedersen HE, Blunck CF, Gardner E (1956) The anatomy of lumbosacral posterior rami and meningeal branches of spinal nerves (sinu-vertebral nerves) with an experimental study of their functions. *J Bone Jt Surg Am* 38:377–391
21. Rudolph T, Løkebø JE, Andreassen L (2011) Bilateral gluteal compartment syndrome and severe rhabdomyolysis after lumbar spine surgery. *Eur Spine J* 20:S180–S182
22. Semciw A, Neate R, Pizzari T (2016) Running related gluteus medius function in health and injury: a systematic review with meta-analysis. *J Electromyogr Kinesiol* 30:98–110
23. Styf J, Lysell E (1987) Chronic compartment syndrome in the erector spinae muscle. *Spine* 12:680–682
24. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, van Wingerden JP, Snijders CJ (1995) The posterior layer of the thoracolumbar fascia. Its function in load transfer from spine to legs. *Spine* 20:753–758
25. Ward SR, Winters TM, Blemker SS (2010) The architectural design of the gluteal muscle group: implications for movement and rehabilitation. *J Orthop Sports Phys Ther* 40:95–102
26. Yamauchi T, Kim K, Isu T, Iwamoto N, Yamazaki K, Matsumoto J, Isobe M (2018) Additional treatments on undiagnosed peripheral nerve diseases as a possible solution for failed lumbar disc surgery. *Asian Spine J* 12:720–725

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

## Comments

Dr Kim and his colleagues report their two-year experience surgically treating patients who they diagnosed as having gluteus medius syndrome. There are a number of articles describing this syndrome that appear outside the neurosurgical literature. The diagnosis seems to be defined by a trigger point pain over the muscle, exacerbation of pain with rising from a seated position, and relief of symptoms with injection of local anesthetic into the muscle. Other symptoms appear non-specific and overlap with a number of other pain syndromes. Dr. Kim and his colleagues report relief at two-year follow-up in a cohort of patients with this syndrome who underwent stellate laceration of the fascia over lining the muscle. 15 of the 39 had relief following the first procedure, and an additional five patients had a lasting relief after a second procedure. As a practicing neurosurgeon, my main problem is differentiating patients in which the gluteus medius is the primary generator of the patient's pain. Most patients I have encountered with trigger point pain over this muscle can be treated with stretching and massage therapy (there are at least three videos on YouTube demonstrating the appropriate maneuvers). Other patients have gotten symptomatic relief from simple trigger point injections.

Allan Friedman  
NC, USA

The authors present a series of 55 consecutive patients that underwent gluteus medius muscle decompression and analyzed 39 after 2 years (71%). Overall outcome was quite good with a reduction in NRS from 7.4 to 2.1 and RDQ from 10.5 to 3.3, but 5 required a reoperation (13%), 16 another procedure (41%) and 3 failed treatment (8%). The authors are to be congratulated for the attempt to identify a new cause of LBP and adding a procedure to our armamentarium. It has often been argued that “non-specific” low back pain is in reality more specific, but we simply don't know or can't identify the causative factors yet. The paper by Kim et al. follows this path by adding a rather novel cause of LBP: Gluteus medius muscle compression or chronic compartment syndrome. Parts of the patient cohort additionally underwent surgery for superior cluneal (SCN) entrapment neuropathy or middle cluneal (MCN) entrapment neuropathy. The authors have published the technical note on gluteus medius decompression in the *European Spine Journal* in 2016 and include piriformis syndrome and sacroiliac pain as well as chronic compartment syndrome of the paraspinal muscle in the differential diagnosis and the treatable causes of “non-specific” LBP. In another series published in *World Neurosurgery* the group also performed common peroneal nerve neurolysis (for peroneal nerve entrapment neuropathy) and decompression surgery for tarsal tunnel syndrome after failure of gluteus medius muscle decompression. The manuscript addresses a critical question in treating low back pain: Are there other identifiable specific causes in addition to those we commonly treat today? I like their endeavor to help patients failing common modes of therapy and their detailed approach with multiple diagnostic injections makes sense. On the other hand, however, the placebo effect of injections is enormous and their reliability limited, as it has been shown for facet joint injections. Additionally, the high rate of gluteus medius compression syndrome (and of SCN entrapment neuropathy) in the study suggest a “If you have a hammer, everything looks like a nail”-problem. The high rate of various other “alternative” treatment concepts gives the impression that in a “trial and error” concept the authors kept on performing invasive procedures until something helped. However, maybe this is what we have to do for some of our patients. I am looking forward to more prospective data by these distinguished and other groups. Sensitive MRI techniques may also be helpful in identifying this syndrome.

Cludius Thome  
Tirol, Austria