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Inferior Gluteal Nerve Injury Due to Intramuscular Injection

İntramüsküler Enjeksiyona Bağlı İnferior Gluteal Sinir Yaralanması

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ABSTRACT

Aim: The purpose of this study was to determine the clinical features of the inferior gluteal nerve (IGN) injury due to intramuscular (IM) injection.
Material and Methods: Patients with clinical and electrodiagnostic features of the sciatic nerve (SN) and possible IGN injuries due to IM injection were included in this retrospective study. The presence of an IGN injury was considered in patients with weakness in the gluteus maximus (GM) muscle or in those who demonstrated needle electromyography (EMG) abnormality in the GM muscle.
Results: There were 44 (95.6%) patients with an SN injury only, 1 (2.2%) patient with both an SN and an IGN injury, and 1 (2.2%) patient with an IGN injury only. The complaints of the patient with an IGN injury only occurred within hours to days after the IM injection; this patient had no muscle weakness. The complaints of the patient with both IGN and SN injuries occurred minutes to hours after IM injection; this patient had mild weakness in the plantar flexion of the foot. In 40 of the patients with only an SN injury, complaints occurred immediately after or within a few seconds following the IM injection, while complaints occurred within minutes to hours in the remaining 4 patients.
Conclusion: Although rare when compared to SN injury, the IGN can be injured by IM injection. Therefore, the GM muscle should be examined with needle EMG in patients with complaints associated with IM injection. Muscle weakness may not occur in nerve injuries due to IM injections.
Keywords: Electromyography; inferior gluteal nerve; intramuscular injection; sciatic nerve.

ÖZ

Amaç: Bu çalışmanın amacı intramüsküler (İM) enjeksiyona bağlı gelişen inferior gluteal sinir (İGS) yaralanmasının klinik özelliklerinin belirlenmesidir.
Gereç ve Yöntemler: Klinik ve elektrodiagnostik özellikleri İM enjeksiyona bağlı gelişen siyatik sinir (SS) ve olası İGS yaralanmaları ile uyumlu olan hastalar bu geriye dönük çalışmaya dahil edildi. Gluteus maksimus (GM) kasında güçsüzlük ya da GM kasında iğne elektromiyografi (EMG) anormalliği olan hastalarda İGS yaralanması olduğu kabul edildi.
Bulgular: Sadece SS yaralanması olan 44 (%95,6) hasta, hem SS hem İGS yaralanması olan 1 (%2,2) hasta ve sadece İGS yaralanması olan 1 (%2,2) hasta mevcuttu. Sadece İGS yaralanması olan hastanın şikayetleri İM enjeksiyondan sonra saatler ile günler içinde oluşmuştu ve bu hastanın kas güçsüzlüğü yoktu. Hem İGN hem SS yaralanması olan hastanın şikayetleri İM enjeksiyondan sonra dakikalar ile saatler içinde oluşmuştu ve bu hastanın ayak plantar fleksiyonunda hafif derecede güçsüzlük mevcuttu. Sadece SS yaralanması olan hastaların 40'ında şikayetler İM enjeksiyonu takiben hemen ya da saniyeler içinde ortaya çıkarken geri kalan 4 hastada ise şikayetler IM enjeksiyonu takiben dakikalar ya da saatler içinde oluşmuştu.
Sonuç: SS yaralanması ile karşılaştırıldığında nadir olsa da, İM enjeksiyon ile İGS yaralanabilir. Bu nedenle İM enjeksiyon ile ilişkili şikayetleri olan hastalarda GM kası iğne EMG ile değerlendirilmelidir. İM enjeksiyonlara bağlı gelişen sinir yaralanmalarında kas güçsüzlüğü oluşmayabilir.
Anahtar kelimeler: Elektromiyografi; inferior gluteal sinir; intramüsküler enjeksiyon; siyatik sinir.

INTRODUCTION

The sciatic nerve (SN) injury can occur as a result of hip surgery, intramuscular (IM) injection, or trauma (1-4). The degree of injury can range from mild to severe and can even result in disability (3). The SN is formed from the ventral rami of the L4, L5, S1, S2, and S3 spinal nerves. The SN leaves the pelvis through the greater sciatic foramen inferior to the piriformis muscle (5,6). The inferior gluteal nerve (IGN) originates from the ventral rami of the spinal nerves of L5, S1, and S2 and exits the pelvis along a similar course to that of the SN. Along this course, the IGN runs medial and very close to the SN (6). Because the superior gluteal, posterior femoral cutaneous, and pudendal nerves also pass through the greater sciatic foramen, they can be injured, along with the IGN and SN, whether by IM injection or other conditions (2,7). There are also reports that the IGN specifically can be injured by conditions such as schwannoma or colorectal carcinoma (8,9). The aim of the study was to find the clinical and electrodiagnostic features of an SN and/or possible IGN injury due to an IM injection.

MATERIAL AND METHODS

Subjects

Patients who applied to our neurology outpatient clinic and laboratory of clinical neurophysiology between July 2018 and January 2020 were analyzed retrospectively. Patients with clinical and electrodiagnostic findings of an SN and/or an IGN injury due to IM injection were included in the study, provided that their complaints started after an IM injection. An SN injury due to an IM injection (SNIII) was considered in patients with sensory abnormalities or muscle weakness, or, alternately, in those who exhibited electrodiagnostic findings consistent with an SN injury. The inclusion criteria for an IGN injury were weakness in the gluteus maximus (GM) muscle; abnormal needle electromyography (EMG) findings in the GM muscle; or sensory abnormality over the lower outer quadrant (the greater trochanter) as the IGN may have sensory branches (10). The patients with an IGN injury had to have normal needle EMG findings in the gluteus medius, as well as in the L3, L4, L5, and S1 paraspinal muscles. Lumbosacral magnetic resonance imaging (MRI) findings of these patients should not be compatible with radiculopathy. Individuals with polyneuropathy (or a disease that may cause polyneuropathy such as diabetes mellitus), neurodegenerative disease, lumbosacral radiculopathy, and low back pain were excluded from the study. Muscle strength was analyzed using the Medical Research Council (MRC) scale (11). The Turkish version of the Leeds assessment of neuropathic symptoms and signs (LANSS) was used to evaluate neuropathic complaints (12).

This study was approved by the Ethics Committee of Adana City Training and Research Hospital (number: 45/620, date. December 4, 2019).

Electrodiagnostic Tests

The Cadwell Sierra Summit EMG unit (Cadwell laboratories, Kennewick, Washington, USA) was used for nerve conduction studies and needle EMG. Electrodiagnostic tests were performed if the temperature of the limb was ≥32 ºC, otherwise, cold limbs were heated. Low-high band filters for sensory and motor nerve conduction were set at 20Hz-2kHz and 20Hz-10kHZ, respectively. Stimulation and recordings were performed with surface electrodes. Nerve stimulation was performed supramaximally. The sweep speeds for sensory and motor nerve conduction studies were set as 1 ms/division and 5 ms/division, respectively. Sensitivity levels for sensory and motor nerve conduction studies were 10 μV/division and 2 mV/division, respectively. Reference values for nerve conduction studies were obtained from previous studies (13-15). Peroneal, tibial, superficial peroneal, peroneal, and sural nerve conduction studies were performed bilaterally (1). Compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes were measured from peak to peak. To exclude peroneal neuropathy, peroneal nerve points were stimulated at the ankle, fibula head, and popliteal fossa. The peroneal nerve CMAP was recorded from both the extensor digitorum brevis (EDB) and the tibialis anterior (TA) muscles. The recording electrode was placed over the abductor hallucis muscle to obtain the tibial nerve CMAP. Superficial peroneal and sural sensory nerve conduction studies were performed antidromically. Sensory nerve conduction velocity was calculated using onset latency. The reference lower limits for the CMAP amplitudes of the tibial nerve and the peroneal nerve recorded from the EDB/TA muscle were 4.4 mV, 2.6 mV / 1.7 mV, respectively. The reference lower limit for amplitudes of both the superficial peroneal and sural nerves was 5 μV (13-15). The amplitude of CMAP or SNAP was considered abnormal if the CMAP or SNAP amplitude was lower than the reference lower limit, or lower than 50% of the CMAP or SNAP amplitude of the intact extremity nerve. Needle EMG was performed using a concentric needle electrode (length=50mm, diameter=0.46mm, Bionen Medical Devices, Florence, Italy). Low-high band filters for needle EMG were 10Hz-10kHz. The sweep speed for active denervation and motor unit action potential (MUAP) analysis was 10 ms/division. Sensitivity levels for active denervation and MUAP analysis were 50-100 μV and 500-1000 μV, respectively. Needle EMG was performed visually. Positive sharp wave (PSW) and fibrillation potentials were carefully analyzed. The MUAP was analyzed during mild muscle contraction. If the MUAP amplitude was >4 mV and duration was >15 ms, the MUAP was considered neurogenic. Needle EMG was applied to the medial gastrocnemius, TA, peroneus longus, biceps femoris short head, vastus lateralis and GM muscles of the patients. To exclude lumbosacral plexopathy or lumbosacral radiculopathy, needle EMG was also performed on the gluteus medius and L3, L4, L5 and S1 paraspinal muscles of some patients. Further, hip and lumbosacral MRIs were analyzed in some patients to exclude lumbosacral radiculopathy and masses in the gluteal region.

Statistical Analysis

The Shapiro-Wilk test was used to determine the distribution of the data. Mean±standard deviation was calculated for descriptive statistics. Categorical variables were summarized as percentage and frequency. Statistical Package for the Social Sciences (SPSS IBM Corp; Armonk, NY, USA) v.22.0 was used to perform the statistical analysis.

RESULTS

Forty-six patients (34 male, 12 female) were included in the study. Apart from these forty-six patients, three patients had clinical findings compatible with SNIII. However, these three patients had needle EMG abnormality in the L4, L5, and S1 paraspinal muscles in addition to the GM muscle, and also had lumbosacral MRI findings compatible with lumbosacral radiculopathy. The mean age of the patients was 39.9±14.7 (range, 19-69) years. The mean body mass index (BMI) of the patients was 21.8±3.4 (range, 15.6-31.9) kg/m^2, respectively. Twelve (26.1%) patients had a BMI <18.5 kg/m^2 while 4 (8.7%) had a BMI >25 kg/m^2. The mean interval between the time of IM injection and the time of electrodiagnostic test was 9.7±9.8 (range, 0.7-36.1) months. The IM gluteal injection was applied by a nurse or a paramedic to the upper outer quadrant of the gluteal region of all patients. The reasons for IM injections were upper respiratory tract infection in 14 (30.4%) patients, muscle or joint pain in 8 (17.4%), generalized pain in 7 (15.2%), abdominal pain in 5 (10.9%), headache in 4 (8.7%), urinary tract infection in 4 (8.7%), toothache in 3 (6.5%), and allergy in 1 (2.2%). IM agents were analgesics in 35 (76.1%) patients, antibiotics in 5 (10.9%) patients, antibiotics+analgesics in 3 (6.5%) patients, and an allergy drug in 1 (2.2%) patient. IM agents were unknown in two patients.

The mean LANSS score of the patients was 14.1±5.8 (range, 3-24). The LANSS score was ≥12 in 30 (65.2%) of the patients. The right lower extremity was affected in 16 (34.8%) patients. The neurological examination findings of the patients are shown in Table 1. Sensory abnormality over the sole of the foot only was observed in 3 (6.5%) patients, while sensory abnormality in the skin area supplied by the peroneal nerve only was observed in 14 (30.4%) patients. Five patients had weakness in only peroneal nerve innervated muscles. None of the patients exhibited weakness in only tibial nerve innervated muscles. Table 2 shows abnormalities of nerve conduction studies in patients. Needle EMG abnormalities of patients are shown in Table 3. SN injury only was present in 44 (95.6%) patients (33 male, 11 female), both SN and IGN injuries in 1 (2.2%) patient (male), and only an IGN injury in 1 (2.2%) patient (female). The complaints of 40 (87.0%) patients with SN injuries occurred immediately following IM injection, while 5 (10.9%) patients occurred within minutes to hours following IM injection.

The patient suffering exclusively with an IGN injury -a thirty-year old woman- applied to our EMG laboratory four weeks after IM injection. Diclofenac was applied to the patient intramuscularly due to generalized pain. The complaint of this patient started within hours to days following IM injection, and progressed over the following days. Within two to three weeks, the pain and paresthesia severity of the patient increased and reached its peak. The patient had hip pain, paresthesia (pins and needles), and sensory loss over the lateral gluteal region, but had no weakness. Bilateral tibial, peroneal, superficial peroneal, and sural nerve conduction studies were normal. The patient had PSW and fibrillation potentials in the GM muscle, but there were no needle EMG abnormalities in other muscles including the MG, TA, PL, biceps femoris short head, vastus lateralis, gluteus medius, L3, L4, L5, and S1 paraspinal muscles. The complaints of this patient improved within two months. She had normal hip and lumbosacral MRIs.

The patient with both SN and IGN injuries -a fifty-eight year old man- expressed complaints within minutes following the IM injection. Diclofenac was given intramuscularly due to an upper respiratory infection. This patient was referred to our EMG laboratory thirty days after IM injection. The MRC score of his foot plantar flexion was four. There was sensory loss in the dorsal region and sole of the foot. The tibial nerve CMAP and sural SNAP amplitudes were reduced. There were PSW and fibrillation potentials in GM and MG muscles, but no neurogenic MUAPs were present in these muscles. Needle EMG was normal in the TA, PL, biceps femoris short head, gluteus medius, vastus lateralis, and L3, L4, L5 and S1 paraspinal muscles. He had normal hip and lumbosacral MRIs. After two months, sensory complaints of the patient decreased, but muscle weakness persisted.

Needle EMG was applied to the L3, L4, L5 and S1 paraspinal muscles in 37 (80.4%) patients with an SN injury only; there was no active denervation in the paraspinal muscles of these patients. Thirty-nine (84.8%) of the patients with an SN injury only had lumbosacral MRIs and their findings were not compatible with lumbosacral radiculopathy.

Table 1. Clinical features of the patients

Neurologic examination	n (%)
Sensory abnormality	
Dorsum of foot or lateral of foot (peroneal)	40 (87.0%)
Sole of foot (tibial)	29 (63.0%)
Posterolateral leg (sural)	28 (60.9%)
Lateral aspect of the GM muscle	1 (2.2%)
None	2 (4.3%)
Weakness	
Peroneal nerve innervated muscles	38 (82.6%)
Tibial nerve innervated muscles	24 (52.2%)
Knee flexors	31 (67.4%)
GM muscle	0 (0.0%)
None	3 (6.5%)

GM: Gluteus maximus

Table 2. Nerve conduction studies of the patients

CMAP and SNAP amplitude abnormality	n (%)
Peroneal nerve EDB	26 (56.5%)
Peroneal nerve TA	16 (34.8%)
Peroneal nerve EDB or TA	34 (73.9%)
Tibial nerve	28 (60.9%)
Superficial peroneal nerve	30 (65.2%)
Sural nerve	35 (76.1%)
None	2 (4.3%)

CMAP: Compound muscle action potential, SNAP: Sensory nerve action potential, EDB: Extensor digitorum brevis, TA: Tibialis anterior

Table 3. Needle electromyography abnormalities of the patients, n (%)

Muscle	Active Denervation	Neurogenic MUAPs	Active Denervation or Neurogenic MUAPs
TA	24 (52.2%)	18 (39.1%)	30 (65.2%)
PL* (n=43)	16 (37.2%)	18 (41.9%)	24 (55.8%)
TA or PL	25 (54.3%)	23 (50.0%)	31 (67.4%)
MG	23 (50%)	19 (41.3%)	31 (67.4%)
Biceps femoris - short head* (n=44)	22 (50%)	19 (43.2%)	31 (70.5%)
GM	2 (4.3%)	0 (0.0%)	2 (4.3%)
None	7 (15.2%)	21 (45.7%)	1 (2.2%)

MUAP: Motor unit action potential, TA: Tibialis anterior, PL: Peroneus longus, MG: Medial gastrocnemius, GM: Gluteus maximus, *: Note that the short head of the biceps femoris and the peroneus muscles cannot be examined by needle electromyography in 3 and 2 patients, respectively

DISCUSSION

SNIII is associated with factors such as the angle of the injector, the amount of gluteal protective tissue, and the position of the patient (3,16-18). Thin individuals are more likely to have less gluteal protective tissue, so these individuals may be at risk for SNIII (3,4). The fact that 12 patients in this study had a BMI <18.5 kg/m^2 may support this observation. In addition, there were only 4 (8.7%) patients with BMI >25 kg/m^2. In this study, it was found that analgesics were among the IM agents associated with SNIII. This may be related to frequent use of analgesics. Many drugs that are administered intramuscularly, such as vitamins and antibiotics, can cause SNIII (3). Peripheral nerve injury is associated with the neurotoxicity of the drug. Some neurotoxic drugs such as benzylpenicillin, chlorpromazine, and diazepam can cause peripheral nerve injury even if they are injected extra-fascicularly (19).

It is known that the peroneal part of the SN is more affected than the tibial part in SNIII (1,3,4). This can be explained by the fact that the peroneal part is more lateral and has less connective tissue than the tibial part (20,21). In this study, weaknesses were more prominent in peroneal nerve innervated muscles. Also, sensory abnormalities were higher in the skin area innervated by the peroneal nerve. Yuen et al. (1) found that only the peroneal part was affected in approximately 10.0% of patients with an SN injury, and there were no patients with only the tibial part affected. In our study, five patients had weakness in peroneal innervated muscles only and no patients had weakness in tibial innervated muscles exclusively. Therefore, it should be noted that while more than one branch of the SN is affected in SNIII, the peroneal part is more severely affected (1,3,4). Although patients often complain of muscle weakness and pain, sensory abnormalities can be found in most patients with a careful neurological examination. In this study, the sensory examination of 44 (95.6%) patients was abnormal. Neuropathic pain is an important symptom in patients with nerve injury due to IM injection. The high number of patients with LANSS scores ≥12 in our study supports this situation.

Three (6.5%) patients had no weakness and 2 (4.3%) patients had normal nerve conduction studies, while all patients except one had needle EMG abnormalities. These findings indicate that electrodiagnostic tests are important in patients with SN and/or IGN injuries due to IM injection, and that needle EMG should be applied to these patients. Needle EMG also plays an important role in differential diagnosis. Three patients who were excluded from the study had needle EMG abnormalities compatible with lumbosacral radiculopathy. The patient with an IGN injury only had normal nerve conduction study results and needle EMG of the muscles innervated by the SN and its branches. Therefore, even if the nerve conduction studies are normal in patients with complaints associated with IM injection, needle EMG should be applied to the GM muscle to exclude an IGN injury. It should be noted that nerve conduction studies, including those of the sural sensory nerve, may have normal results in an SN injury. The sural SNAP amplitude abnormality found in this study (76.1%) was close to that found in another study (1).

IGN paresis is a rare condition. It can be damaged alone or along with other nerves. Inferior gluteal neuropathy has been reported as a result of colorectal carcinoma, schwannoma, IM injection or inadequate stabilization of the back due to lumbar lordosis (2,7-9). Obach et al. (2) reported 137 cases with nerve injuries due to gluteal IM injection. In 2 of these patients, the IGN was injured along with other nerves. The IGN is located very close and medial to the SN during part of its course. This indicates that the SN and the IGN can be injured together (2,6). In this study, the SN and IGN were injured together in 1 patient. IM injection can damage the nerve directly or damage the nerve through diffusion as a result of IM injection very close to the nerve or to the epineurium. While complaints begin immediately following IM injection directly to the nerve, complaints begin within minutes-hours following IM injection very close to the nerve (3,18,20). In this study, in most patients, complaints began immediately following IM injection, similar to previous studies (3,18). However, the complaints of the patient with an IGN injury only started within hours and the complaints intensified within days. While the IGN is more superficial in the medial part of GM muscle, it is slightly deeper in the lateral part (5). Since the nerve is located deeper in the region where the IM injection was performed, the possibility of intraneural injection appears to be difficult. However, there may still be a possibility of an IGN injury due to IM injection. If the IM injection is made into the fatty tissue around the nerve, complaints may start later due to toxic swelling, vascular lesions, necrosis, or fibrosis (2). This may be one of the reasons for the late onset of the complaints of the patient with the IGN injury only. To protect from the nerve injury, IM injection should not be administered deep into the GM muscle, especially when applied to the medial part of this muscle.

There is a study stating that most of the IGN (75.0%) has a sensory branch (10). It was found that these sensory branches originate from the terminal motor branches of the IGN, and are located mostly in the lower outer quadrant of the GM muscle and rarely in its upper outer quadrant. In addition to the superior and inferior cluneal nerves, the sensory branch or branches of the IGN was thought to supply the skin area over the posterior of the greater trochanter (10). Paresthesia in the lateral of the hip in the patient with the IGN injury can only be explained by the injury of the sensory branch of the IGN. In addition, severe hip pain in some patients may be due to the injury of the IGN and its sensory branches.

There were some limitations of this study. It could be noted that the only cause in patients with needle EMG abnormality in the GM muscle is not an IGN injury. However the hip and the lumbosacral MRIs of the patients with IGN injuries were normal. In addition, needle EMG was normal in the gluteus medius and paraspinal muscles of these two patients. Electrodiagnostic tests were not performed for the posterior femoral cutaneous and pudendal nerves. This was another limitation. It has been considered that further studies including electrodiagnostic tests for these nerves in patients with symptoms after IM injection could be important and interesting.

CONCLUSION

The IGN can be injured by IM injection. Therefore, the GM muscle should be analyzed for an IGN injury by needle EMG in patients with complaints following IM injection. If there are abnormal needle EMG findings in the GM muscle, it is useful to examine the lumbosacral paraspinal and the gluteus medius muscles (and other additional muscles) with needle EMG to exclude lumbosacral radiculopathy or plexopathy, as well as nerve injuries such as to the superior gluteal nerve. It should also be noted that muscle weakness may not occur in nerve injuries due to IM injections.

Ethics Committee Approval: The study was approved by the Ethics Committee of Adana City Training and Research Hospital (04.12.2019, 45/620).

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