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REVIEW ARTICLE

Meralgia Paresthetica: Diagnosis and Management Strategies

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ABSTRACT

Meralgia paresthetica (MP), coined from the Greek words *meros* (thigh and algos), meaning pain, is a neurological disorder characterized by a localized area of paresthesia and numbness on the anterolateral aspect of the thigh. The incidence of MP is more common than often reported in the literature. The etiology of MP includes mechanical factors such as obesity, pregnancy, and other conditions associated with increased intrabdominal pressure, surgery of the spine, and pelvic osteotomy. A coherent history and pertinent physical examination is essential in making the diagnosis; however, red flags such as tumor and lumbar disk herniations must be recognized and appropriately treated. While the diagnosis of MP is essentially a clinical diagnosis, sensory nerve conduction velocity studies are a useful adjunctive diagnostic tool. The management of MP includes treating the underlying cause (if any) and conservative management. Surgery should only be adopted when all nonoperative therapies have failed to manage the condition in an effective manner.

Key Words. Assessment; Diagnosis; Meralgia Paresthetica; Treatment Options

Introduction

Meralgia paresthetica (MP) is a neurological disorder characterized by a localized area of paresthesia and numbness on the anterolateral aspect of the thigh.

The aim of this article is to increase awareness of this condition and to aid clinicians in its diagnosis and subsequent management.

Meralgia paresthetica was first described by Bernhardt in 1878 [1], and in 1885 Hager described hip pain secondary to lateral cutaneous nerve injury following trauma [2]. In 1895 both Bernhardt and Roth published independent articles on MP [3,4]. The syndrome was initially known as Bernhardt–Roth syndrome. Roth is credited with coining the clinical entity "Meralgia Paresthetica," and this word was derived from the Greek word *meros* (thigh and algos), meaning pain. Sigmund Freud even published an article about his own affliction with "Bernhardt's syndrome" and initially ascribed the clinical symptomatology to psychosomatic factors [5,6].

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Epidemiology

Although spontaneous MP can occur in any age group [7], it is most frequently noted in 30- to 40-year-olds. Its incidence in children may be higher than previously recognized [8]. One-third of all children treated for osteoid osteoma developed MP [9]. Only recently has the incidence of MP been quantified in a Dutch population study (all patients with a diagnosis of MP between 1990 and 1998 in a computerized registration network of patients years (173,375) from general practitioners in Rotterdam). The incidence rate was 0.43 per 10,000 person years [10]. Prior to this, an incidence of three cases in 10,000 general-clinic patients was noted [11]. There is as of yet no consensus whether there is sex predominance [11]. But in one study that evaluated 150 cases of MP,

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there was a higher incidence in men [12]; one series has evaluated a family with MP in four generations, suggesting an autosomal dominant trait [13].

Meralgia paresthetica has typically shown a preponderance in patients who are obese [14] or diabetic [15] as well as in pregnant women [16]. However, a series has been reported in children with a relatively slim body habitus [17].

Etiology

Meralgia paresthetica is one of the peripheral nerve disorders that is commonly associated with and can be categorized as an entrapment neuropathy with a long list of differential diagnoses, some of which are general and applicable to most of the peripheral nerves while others address a more specific lateral femoral cutaneous nerve (LFCN) dysfunction.

Meralgia paresthetica has many etiologies upward of 80, and different causes of MP have been reported in the literature [18]. It can be categorized as spontaneous or iatrogenic.

Spontaneous MP occurs in the absence of any prior surgical procedure and can be divided into idiopathic or metabolic. The iatrogenic form of MP is due to many common orthopedic and indeed nonorthopedic procedures. Mechanical and metabolic factors may be involved [19].

Spontaneous Causes

Spontaneous causes include mechanical factors such as obesity, pregnancy, and other conditions associated with increased intrabdominal pressure [20]. According to one study, anatomical variations include LFCN types A, B, and C [21]. The wearing of belts, corset, and tight trousers can also result in direct pressure on the LFCN [22]; even wearing a tight belt with an accompanying holster for a pistol has been described as a cause for bilateral MP [23]. Pelvic benign masses such as uterine fibroids have been reported as presenting as MP [24]. In addition, a tumor in the iliac crest has presented as MP [25].

Other causes of spontaneous MP include radiological degenerative pubic symphysis [15]. Limb length discrepancy has also been implicated as a cause of MP [26]. In children, MP presentation has been associated with osteoid sarcoma in more than one-third of patients following treatment. Pelvic osteotomy and pelvic crush fractures have been described in one case series in children as being associated with MP [8].

Metabolic factors that have been implicated in MP include lead poisoning, alcoholism, and diabetes mellitus [19]. Leprosy has also been associated with MP, which often presents with a clinical syndrome akin to ischialgia [27]. With respect to diabetes mellitus, two theories have evolved as to why diabetics get MP. A myo-inositol-related defect in nerve Na(+)-K(+)-ATPase in experimental diabetes has been invoked in the pathogenesis of diabetic neuropathy. A study explored the relationship between the myo-inositol-sensitive and protein kinase C-agonist-sensitive Na(+)-K(+)-ATPase defects in diabetic rat nerve. The nonadditivity and implied equivalence of the Na(+)-K(+)-ATPase defect corrected by myo-inositol *in vivo* and by protein kinase C agonists *in vitro* are consistent with the postulated existence of a deficient myo-inositol-dependent phospholipid-derived protein kinase C agonist (potentially diacylglycerol) in diabetic nerve that regulates nerve Na(+)-K(+)-ATPase either directly or via a protein kinase C mechanism [28]. Another theory postulated that in diabetics there is increased swelling of the LFCN due to decreased axoplasmic transport, rendering it more susceptible to compression; however, optimization of blood glucose does not ameliorate the situation [29].

Iatrogenic Causes

Iatrogenic causes of MP include orthopedic procedures, including pelvic osteotomy [30] and spine surgery. In a prospective analysis of 105 spine surgeries and subsequent follow-up 1 year post surgery, 20% of patients suffered an injury to the LFCN. In six of these patients, the injury was bilateral. The patients had all undergone surgery on the Hall–Reiton frame. The authors in this study advised that the frame should be padded, kept posterior to anterior superior iliac spine (ASIS), and that retraction be minimized when taking a bone graft. They also advised caution with traction on psoas muscle during retroperitoneal dissection [31]. Neurotemesis of the LFCN while coring for iliac crest bone grafts has also been described by other authors as being associated with MP, and they advise caution because of the various courses that the LFCN takes, including crossing over the iliac crest, which increases the risk of injury during coring for cancellous bone graft [32]. Total hip replacement has also been linked with MP [33].

Various other surgical techniques have resulted in injury to the LFCN, including laparoscopic cholecystectomy [34], laparoscopic myomectomy

[35], coronary artery bypass grafting [36], aortic valve surgery [37], and gastric reduction surgery for morbid obesity [14,38,39]. Laparoscopic inguinal hernia repair has also been well documented in the literature as being associated with MP [40–45].

Diagnosis

The symptoms of MP consist of unpleasant paresthesias in the upper and lateral thigh. In most instances, the condition is unilateral; however, 20% of patients present with bilateral complaints [12]. Patients typically describe a burning, stinging, or tingling sensation in the thigh and can usually localize the sensations to the skin itself. Some describe allodynia over the distribution of the nerve. Most patients, however, do not refer to these sensations as overt pain but more of a dysesthesia. Some patients note that paresthesias in the thigh can be initiated by tapping on the inguinal ligament; extending the thigh posteriorly, which stretches the nerve, can aggravate the condition; in addition, erect posture and prolonged standing have also been identified as associated aggravating factors [46]. Symptoms are relieved by sitting, but on occasion symptoms aggravated by sitting have been reported [17]. Other neurological, urogenital, and gastrointestinal symptoms and signs do not fit the picture of MP and should indicate to the attending physician that the leg pain is due to another condition. Physical examination usually reveals tenderness over the lateral inguinal ligament at the point where the nerve crosses the inguinal ligament. Patients may also have an area of hair loss on the anterior thigh due to constant rubbing of the region by the patient, and this is an important diagnostic marker.

While the diagnosis of MP is usually diagnosed on a coherent history and anatomically pertinent physical examination, one must have a clinically relevant differential diagnosis when evaluating patients. Red flags—metastasis in iliac crest [25] and lumbar disk herniations [47,48] masquerading as MP—must be ruled out in patients with a clinical diagnosis of MP. Avulsion fracture of the ASIS has been reported to present as MP [49]. Even chronic appendicitis has presented as a clinical MP-like syndrome [50]. Any patient with a motor deficit or reflex changes or sensory deficits not specific to LFCN should be completely evaluated [49]. MP has been reported as mimicking lumbar radiculopathy [51]. While the name MP refers only to painful dysaesthetic cases of LFCN neuropathy, the pathology may only cause numbness within the distal distribution of the nerve without any pain. Under rare circumstances, the entrapment-related pain may radiate proximally toward the spine, much akin to that observed in carpal tunnel syndrome, which can make diagnosis quite a challenge.

Plain X-ray of the pelvis in addition to CT of lumbar spines should be performed to eliminate disk herniations or pelvic tumors as etiologies.

Ultrasound and magnetic resonance imaging can be employed to evaluate the retroperitoneal regions. On physical examination, there should be no tenderness over the sciatic notch and straight leg raising should be negative. A full blood analysis should be performed, including thyroid function testing, as MP has been associated with hypothyroidism [52].

When there is doubt about the diagnosis following history and physical examination, then electrodiagnostic testing can be used [53]. The electrophysiologic test routinely performed to confirm the diagnosis of LFCN neuropathy includes sensory nerve conduction velocity (SNCV) and somatosensory-evoked potentials (SSEP) recorded on the scalp in following LFCN stimulation [54]. However, the value of SSEP in making the diagnosis of MP has been consistently debated. SSEP were evaluated in 20 healthy volunteers as compared with 22 patients and led to the conclusion that SSEP is useful as a diagnostic aid [55]. Another study found that SSEP were useful in determining whether MP was caused by an injury in a region proximal to the ASIS [56]. On the contrary, Seror evaluated SSEP in MP and concluded that only in very serious nerve damage was there an abnormal SSEP recorded, and did not recommend them for routine electrodiagnosis of MP [57].

Furthermore, Seror illustrated that SNCV was abnormal in all patients with MP [57]. He concluded that SNCV has a role in the diagnosis of MP.

Recently, the same author evaluated the diagnostic utility of SSEP studies of the LFCN in assessing patients with MP. Twenty-one consecutive patients with unilateral MP, as defined clinically (sensory impairment of the lateral aspect of the thigh) and electrodiagnostically (abnormal sensory nerve conduction), and 21 control subjects were studied with two SSEP methods. SSEP were elicited by stimulation of the LFCN below the ASIS stimulation and by cutaneous stimulation of the lateral aspect of the distal third of the thigh

(thigh stimulation). Overall, SSEP after ASIS stimulation had no diagnostic value.

Seror concluded that recording of SSEP after thigh stimulation was only of benefit in obese patients where SNCV cannot be measured [58]. In patients where a clinical diagnosis of MP has been made, a diagnostic block may be made with 8 mL of bupivicaine 0.25% [59]. Utilizing a nerve stimulator is superior to the fan technique (100% success vs 40% in one study) [60].

Pathophysiology

Variant anatomy has been evaluated as a clinical predictor of whether an individual will develop MP. In a study of 52 cadavers to investigate the anatomy of the LFCN, the variability of its course and locations as it exits the pelvis was described and related to soft-tissue and bony landmarks. Five different types (Figure 1) were identified: type A, posterior to the ASIS, across the iliac crest (4%);

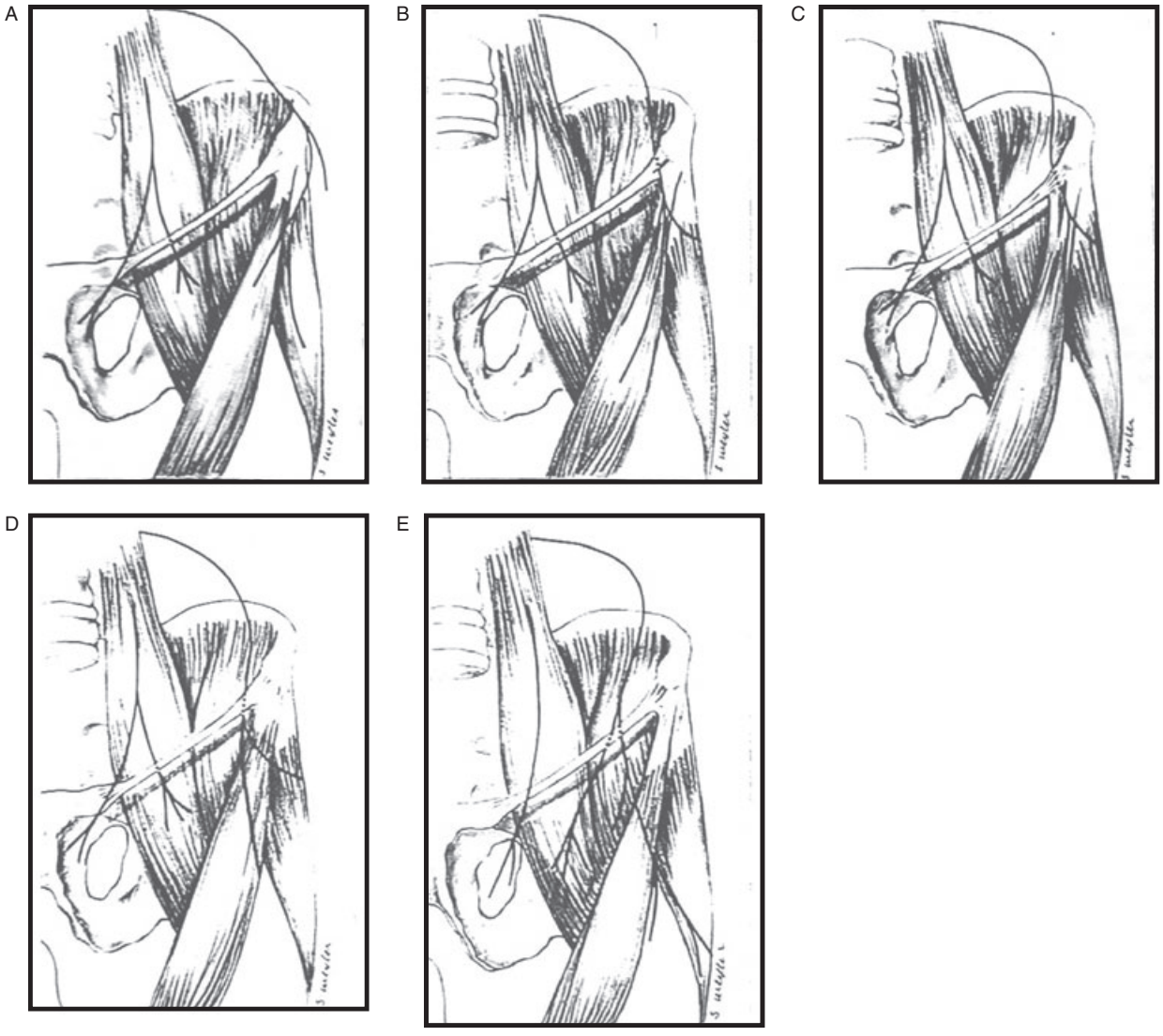

Figure 1 The variations in the course of the lateral femoral cutaneous nerve are illustrated as it exits the abdomen. The nerve may course across the iliac crest posterior to the anterior superior iliac spine (A), may be encapsulated by the inguinal ligament just medial to the anterior superior iliac spine (B), may be ensheathed in the tendinous origin of sartorius muscle medial to the anterior superior iliac spine (C), may be located in a groove between sartorius muscle and the iliopsoas muscle deep to the inguinal ligament (D), or may be found in the most medial position on top of the iliopsoas muscle contributing the femoral branch to the genitofemoral nerve (E). (Reproduced with permission from Aszmann OC, Dellon ES, Dellon AL. Anatomical course of the lateral femoral cutaneous nerve and its susceptibility to compression and injury. Plast Reconstr Surg 1997;100:600–4. Lippincott Williams and Wilkins.)

type B, anterior to the ASIS and superficial to the origin of the sartorius muscle but within the substance of the inguinal ligament (27%); type C, medial to the ASIS, ensheathed in the tendinous origin of the sartorius muscle (23%); type D, medial to the origin of the sartorius muscle located in an interval between the tendon of the sartorius muscle and thick fascia of the iliopsoas muscle deep to the inguinal ligament (26%); and type E, most medial and embedded in loose connective tissue, deep to the inguinal ligament, overlying the thin fascia of the iliopsoas muscle, and contributing the femoral branch of the genitofemoral nerve (20%). Aszmann and colleagues concluded that nerve types A, B, or C were the most susceptible to trauma [21]. In another study to evaluate the anatomical variation of LFCN and the consequences for surgery, 200 cadavers were dissected and the course of the LFCN was described as normal (151 sections)—the nerve emerges beneath the lateral margin of the psoas and continues obliquely downward and outward across the iliac fossa and at the ASIS to the thigh beneath the fascia lata (type 1). Variations included passage via a slitlike aponeurosis in the inguinal ligament (type 2); another course was for the nerve to cross over, under, through, or medial to the tendinous fibers of sartorius (type 3); and more rarely it crossed just behind the ASIS (type 5). De Ridder and colleagues classified 151 as normal (type 1) and 49 as abnormal (types 2–5). When they evaluated a patient series of 82 patients who experienced LFCN lesion as a consequence of having pelvic surgery following an ilioinguinal approach, they found that 37 of these patients experienced altered sensation for several years postoperatively. The authors concluded from their work that an anatomical variation is of the LFCN of the thigh in 25% of the patient population. In addition, when an operative procedure is carried out in the vicinity of the ASIS, the patient should be informed that there is as a 35% chance of loss of sensation and a 5% risk of developing MP [61]. In a routine autopsy study, five out of 12 LFCNs showed pathologic changes in myelinated nerve fibers in the vicinity of the inguinal ligament. These changes included both local demyelination and Wallerian degeneration affecting the larger fibers in particular. The presence of polarized internodal swellings on single nerve fibers from two specimens suggested that mechanical factors were involved in the pathogenesis. In the region of the inguinal ligament, endoneurial vascular thickening was seen, which could be responsible for symptoms of MP [62]. The course of the LFCN in another anatomical study of 26 cadavers was evaluated. The authors concluded from their observations that the nerve, as it passed from the pelvis into the thigh, ran through an "aponeuroticofascial tunnel," beginning at the iliopubic tract and ending at the inguinal ligament. As it traverses the tunnel there is an enlargement of its side-to-side diameter, which suggests that structures proximal to the inguinal ligament may be responsible for development of MP [63].

Chronic regional pain syndrome (CRPS) type 2 has been defined as a burning pain allodynia and hyperpathia usually in the hand or foot after partial injury of a nerve or one of its major branches [64]. The diagnostic criteria include the following:

1. The presence of continuing pain, allodynia, or hyperalgesia after a nerve injury, not necessarily limited to the distribution of the injured nerve.
2. Evidence at some time of edema, changes in skin blood flow, or some abnormal sudomotor activity in the region of the pain.
3. This diagnosis is excluded by the existence of a condition that would otherwise account for the degree of pain and dysfunction.

All three criteria must be satisfied to make the diagnosis. MP almost never evolves into CRPS type 2.

Treatment Options

In another study, the majority of cases of MP follow a benign course and will respond to conservative treatment, with resolution of the vast majority (85%) within 4–6 months of presentation [59]. A conservative treatment regime involved the use of a local anesthetic block with 0.25% bupivicaine to confirm the diagnosis.

After this, repeat blocks were administered with methylprednisolone on alternate days in divided doses of 20–120 mg each. All patients were administered a minimum of five blocks each. All patients were administered daily therapy of diphenylhydantoin (100–300 mg in divided doses) for 10–12 weeks [59].

Dureja and colleagues attributed their high success rate to the repetitive blocking of the LFCN—which breaks an afferent efferent loop at the spinal segmental level. However, there are limitations in the study just mentioned in that it was a case series without a control group so results have to be interpreted cautiously. In addition, local steroids reduce the hyperexcitability of neurons and c

fibers [65]. While the membrane stabilizing properties (prolonging recovery time of activated sodium channels) of diphenylhydantoin thus reduces neuronal firing [66]. The benefits of tricylic antidepressants, antiarrhythmic agents, and anticonvulsants to treat the effects of neuropathic pain have been documented [67]. Capsaicin (selective excitation of C-polymodal nociceptors) has also been used to treat itch and surface hypersensitivity in MP [68]. Topical lidocaine has also been employed in resistant neuropathic pain of MP with good effect [69].

Globally nonoperative treatment has yielded excellent results in the management of MP. Most clinical series have utilized many different treatment modalities in the management of MP. The benefit of one treatment regime over another is therefore not possible to quantify. Conservative treatment in two case series yielded the following results in a series with 277 patients over a 25-year period. Williams and Trzil reported symptoms relief in 91% of patients [20]. Ecker and Woltmann showed that 67% improved with nonoperative management at 2-year follow-up [12]. Pulsed radio frequency of the LFCN has shown to be of benefit, but this therapy warrants further investigation [70].

Surgical Intervention for MP

Surgical intervention for MP is generally reserved for patients who are resistant to conservative management [20]. The operative techniques for surgical management include neurolysis of the constricting tissue, neurolysis and transposition of the LFCN, and transection with excision of a portion of the LFCN. Six series have been published to date on operative intervention for MP.

MacNichol and Thompson evaluated 29 patients with refractory MP. Exploration and decompression of the LFCN was successful in 11 (44%) of these patients at follow-up of an average of 5.5 years [71]. Nahabedian and Dellon also noted that there was complete relief of symptoms in 18 of 23 patients after surgical decompression of the nerve [29]. Edelson and Stevens reported on their case series of 21 cases of MP in 13 children aged 1–17 years who were treated with decompression of the nerve. At mean follow-up of 38 months (range 25–60 months), there was complete relief in 14 cases and significant improvement in five cases, and two cases with persistent pain only when doing exercise/sport activities [8].

Keegan and Holyoke described two cases where LFCN release and medial transposition provided good results [72]. Aldrich and van den Heever described a suprainguinal ligament approach for release and transposition [73]. It should be noted that n both of these studies, the nerve was appearing as a single trunk at the ASIS. Both of these series are small and this approach has not been tested in a clinical trial. However, the presence of a neurinoma and the frequency of anatomical variation render neurolysis and transposition difficult. Williams and Trzil reported on 24 cases where they resected the nerve in refractory MP and reported excellent relief in 23 of these patients. The authors in this study advised patients that sectioning of the LFCN offers uniformly good results and should be easily reproducible once adequate identification of the nerve is accomplished, and patients are willing to accept permanent anesthesia on an area of the anterolateral thigh in exchange for relief of their symptoms [20].

Ivins, in another series of 14 adult patients with follow-up over 3–6 years, initially treated them conservatively; however, six of these 14 patients required operative treatment. Ivins initially performed decompression (four cases) in three patients with good initial results, including prompt relief of symptoms; however, within 2–24 months of the initial decompression, there was recurrence, and re-exploration with resection produced long-lasting relief at follow-up. A further two patients had primary resection with similar results [74]. Van Eerten evaluated transection and neurolysis in 21 patients after the failure of conservative management and found that transection (9/11 patients complete relief of symptoms) was superior to neurolysis (3/10 complete relief of symptoms). It should be noted that in this study the procedures were performed by different neurosurgeons [75].

Ivins in his study classified patients into three groups (following failure of nonoperative treatment) based on their symptoms for operative intervention:

1. Adults with symptoms for less than 1 year and all pediatric patients should undergo simple decompression.
2. Patients from group 1 who have persistent or recurrent symptoms should be considered for resection.
3. Adult patients with symptoms for more than 1 year should be considered for primary resection.

In another interesting case, a 40-year-old woman with MP after malignant tumor resection

in the right inguinal region underwent a novel procedure including neurolysis and the use of a deep inferior epigastric perforator adiposal flap wrapping as a prophylactic procedure against re-entrapment [76].

Conclusion

The incidence of MP is more common than often reported in the literature. A coherent history and pertinent physical examination is essential in making the diagnosis; however, red flags such as tumor and lumbar disk herniations must be recognized and appropriately treated. SNCV has role in the diagnosis of MP, but SSEP have no role to play in the diagnosis of MP. In orthopedic procedures, particularly iliac crest harvesting and pelvic osteotomy, an in-depth knowledge of the anatomy must be appreciated and, in particular, that 25% of the patient population have LFCN that is "abnormal." First-line management is always to treat the underlying cause (if any), i.e., simple measures such as losing weight, not wearing tight belts, etc. A series of corticosteroid blocks in combination with pharmacological therapy should form the mainstay of conservative management. Corticosteroids appear to be less efficacious in children than adults. Neurosurgical management is only reserved for resistant cases of MP and first-line therapy should be neurolysis and decompression followed by transection. Once MP is readily recognized, it responds favorably to adequate treatment.

References

1 Bernhardt M. Neuropathologische Beobachtungen. I. periphere Lahmungen. D Arch Klin Med 1878;22:362–93.
2 Hager W. Neuralgia femoris. Resection des Nerv. cutan. femoris anterior externus. Heilung. Deutsch Med Wochenschr 1885;11:218–9.
3 Bernhardt M. Ueber Isolirt im Gebiete des N. cutaneous femoris externus vorkommende Parasthesien. Neurol Centrabl 1895;14:242–4.
4 Roth V. [Meralgia Paresthetica]. Med Obozr 1895;43:678.
5 Freud S. Ueber die Bernhardt'sche Sensibilitatsstorung am oberschenkel. Neurol Centralbl 1895; 14:491–2.
6 Schiller F. Sigmund Freud's meralgia paresthetica. Neurology 1985;35:557–8.
7 Massey EW, Pellock JM. Meralgia paraesthetica in a child. J Pediatr 1978;93:325–6.
8 Edelson R, Stevens P. Meralgia paresthetica in children. J Bone Joint Surg Am 1994;76:993–9.
9 Goldberg V, Jacobs B. Osteoid osteoma of the hip in children. Clin Orthop 1975;106:41–7.
10 van Slobbe AM, Bohnen AM, Bernsen RM, Koes BW, Bierma-Zeinstra SM. Incidence rates and determinants in meralgia paresthetica in general practice. J Neurol 2004;251:294–7.
11 Ecker AD. Diagnosis of meralgia paresthetica. JAMA 1985;253:976.
12 Ecker A, Woltmann HW. Meralgia paresthetica: A report of one hundred and fifty cases. JAMA 1938;110:1650–2.
13 Malin JP. Familial meralgia paresthetica with an autosomal dominant trait. J Neurol 1979;221:133–6.
14 Abarbanel JM, Berginer VM, Osimani A, Solomon H, Charuzi I. Neurologic complications after gastric restriction surgery for morbid obesity. Neurology 1987;37:196–200.
15 Bierma-Zeinstra S, Ginai A, Prins A, et al. Meralgia paresthetica is related to degenerative pubic symphysis. J Rheumatol 2000;27:2242–5.
16 Kitchen C, Simpson J. Meralgia paresthetica. A review of 67 patients. Acta Neurol Scand 1972;48:547–55.
17 Richer LP, Shevell MI, Stewart J, Poulin C. Pediatric meralgia paresthetica. Pediatr Neurol 2002;26:321–3.
18 Edelson JG, Nathan H. Meralgia paresthetica. An anatomical interpretation. Clin Orthop 1977;122:255–62.
19 Grossman MG, Ducey SA, Nadler SS, Levy AS. Meralgia paresthetica: Diagnosis and treatment. J Am Acad Orthop Surg 2001;9:336–44.
20 Williams PH, Trzil KP. Management of meralgia paresthetica. J Neurosurg 1991;74:76–80.
21 Aszmann OC, Dellon ES, Dellon AL. Anatomical course of the lateral femoral cutaneous nerve and its susceptibility to compression and injury. Plast Reconstr Surg 1997;100:600–4.
22 Boyce JR. Meralgia paresthetica and tight trousers. JAMA 1984;251:1553.
23 Korkmaz N, Ozcakar L. Meralgia paresthetica in a policeman: The belt or the gun. Plast Reconstr Surg 2004;114:1012–3.
24 Suber DA, Massey EW. Pelvic mass presenting as meralgia paresthetica. Obstet Gynecol 1979;53:257–8.
25 Tharion G, Bhattacharji S. Malignant secondary deposit in the iliac crest masquerading as meralgia paresthetica. Arch Phys Med Rehabil 1997; 78:1010–1.
26 Goel A. Meralgia paresthetica secondary to limb length discrepancy: Case report. Arch Phys Med Rehabil 1999;80:348–9.
27 Theuvenet WJ, Finlay K, Roche P, Soares D, Kauer JM. Neuritis of the lateral femoral cutaneous nerve in leprosy. Int J Lepr Other Mycobact Dis 1993;61:592–6.
28 Kim J, Kyriazi H, Greene DA. Normalization of Na(+)-K(+)-ATPase activity in isolated membrane fraction from sciatic nerves of streptozocin-induced

diabetic rats by dietary myo-inositol supplementation in vivo or protein kinase C agonists in vitro. Diabetes 1991;40:558–67.
29 Nahabedian MY, Dellon AL. Meralgia paresthetica: Etiology, diagnosis, and outcome of surgical decompression. Ann Plast Surg 1995;35:590–4.
30 Hogh J, Macnicol MF. The Chiari pelvic osteotomy. A long-term review of clinical and radiographic results. J Bone Joint Surg Br 1987;69:365–73.
31 Mirovsky Y, Neuwirth M. Injuries to the lateral femoral cutaneous nerve during spine surgery. Spine 2000;25:1266–9.
32 van den Broecke DG, Schuurman AH, Borg ED, Kon M. Neurotmesis of the lateral femoral cutaneous nerve when coring for iliac crest bone grafts. Plast Reconstr Surg 1998;102:1163–6.
33 Ahsan MR, Curtin J. Meralgia paresthetica following total hip replacement. Ir J Med Sci 2001;170:149.
34 Yamout B, Tayyim A, Farhat W. Meralgia paresthetica as a complication of laparoscopic cholecystectomy. Clin Neurol Neurosurg 1994;96:143–4.
35 Hutchins FL Jr, Huggins J, Delaney ML. Laparoscopic myomectomy–an unusual cause of meralgia paresthetica. J Am Assoc Gynecol Laparosc 1998;5:309–11.
36 Parsonnet V, Karasakalides A, Gielchinsky I, Hochberg M, Hussain SM. Meralgia paresthetica after coronary bypass surgery. J Thorac Cardiovasc Surg 1991;101:219–21.
37 Antunes PE, Antunes MJ. Meralgia paresthetica after aortic valve surgery. J Heart Valve Dis 1997;6:589–90.
38 Grace DM. Meralgia paresthetica after gastroplasty for morbid obesity. Can J Surg 1987;30:64–5.
39 Macgregor AM, Thoburn EK. Meralgia paresthetica following bariatric surgery. Obes Surg 1999;9:364–8.
40 Broin EO, Horner C, Mealy K, et al. Meralgia paraesthetica following laparoscopic inguinal hernia repair. An anatomical analysis. Surg Endosc 1995;9:76–8.
41 Kraus MA. Nerve injury during laparoscopic inguinal hernia repair. Surg Laparosc Endosc 1993;3:342–5.
42 Van Hee R, Goverde P, Hendrickx L, Van der Schelling G, Totte E. Laparoscopic transperitoneal versus extraperitoneal inguinal hernia repair: A prospective clinical trial. Acta Chir Belg 1998;98:132–5.
43 Marks SC Jr, Gilroy AM, Page DW. The clinical anatomy of laparoscopic inguinal hernia repair. Singapore Med J 1996;37:519–21.
44 Dibenedetto LM, Lei Q, Gilroy AM, et al. Variations in the inferior pelvic pathway of the lateral femoral cutaneous nerve: Implications for laparoscopic hernia repair. Clin Anat 1996;9:232–6.
45 Ger R, Mishrick A, Hurwitz J, Romero C, Oddsen R. Management of groin hernias by laparoscopy. World J Surg 1993;17:46–50.
46 Stookey B. Meralgia paresthetica: Etiology and surgical treatment. JAMA 1928;90:1705–7.
47 Cedoz ME, Larbre JP, Lequin C, Fischer G, Llorca G. Upper lumbar disk herniations. Rev Rhum Engl Ed 1996;63:421–6.
48 Trummer M, Flaschka G, Unger F, Eustacchio S. Lumbar disc herniation mimicking meralgia paresthetica: Case report. Surg Neurol 2000;54:80–1.
49 Thanikachalam M, Petros JG, O'Donnell S. Avulsion fracture of the anterior superior iliac spine presenting as acute-onset meralgia paresthetica. Ann Emerg Med 1995;26:515–7.
50 Ghavanini MR, Ghavanini AA. Meralgia paresthetica as the presenting feature of chronic appendicitis. Am J Phys Med Rehabil 2001;80:703–5.
51 Kallgren MA, Tingle LJ. Meralgia paresthetica mimicking lumbar radiculopathy. Anesth Analg 1993;76:1367–8.
52 Suarez G, Sabin TD. Meralgia paresthetica and hypothyroidism. Ann Intern Med 1990;112:149.
53 Esteban A. Lateral femoral cutaneous neuropathy: Paresthetic meralgia. Neurophysiological diagnosis. Rev Neurol 1998;26:414–5.
54 Lagueny A, Deliac MM, Deliac P, Durandeau A. Diagnostic and prognostic value of electrophysiologic tests in meralgia paresthetica. Muscle Nerve 1991;14:51–6.
55 Po HL, Mei SN. Meralgia paresthetica: The diagnostic value of somatosensory evoked potentials. Arch Phys Med Rehabil 1992;73:70–2.
56 Wiezer MJ, Franssen H, Rinkel GJ, Wokke JH. Meralgia Paraesthetica: Differential diagnosis and follow-up. Muscle Nerve 1996;19:522–4.
57 Seror P. Lateral femoral cutaneous nerve conduction vs. somatosensory evoked potentials for electrodiagnosis of meralgia paresthetica. Am J Phys Med Rehabil 1999;78:313–6.
58 Seror P. Somatosensory evoked potentials for the electrodiagnosis of meralgia paresthetica. Muscle Nerve 2004;29:309–12.
59 Dureja GP, Gulaya V, Jayalakshmi TS, Mandal P. Management of meralgia paresthetica: A multimodality regimen. Anesth Analg 1995;80:1060–1.
60 Shannon J, Lang SA, Yip RW, Gerard M. Lateral femoral cutaneous nerve block revisited. A nerve stimulator technique. Reg Anesth 1995; 20:100–4.
61 de Ridder VA, de Lange S, Popta JV. Anatomical variations of the lateral femoral cutaneous nerve and the consequences for surgery. J Orthop Trauma 1999;13:207–11.
62 Jefferson D, Eames RA. Subclinical entrapment of the lateral femoral cutaneous nerve: An autopsy study. Muscle Nerve 1979;2:145–54.
63 Filho L, Valenca MM, Guimares Filho FAV, et al. Lateral femoral cutaneous neuralgia: An anatomical insight. Clin Anat 2003;16:309–16.
64 Merskey H, Bogduk N. Classification of chronic pain: Description of chronic pain syndromes and

definition of pain terms. 2nd edition. Seattle: IASP Press; 1994:42–3.
65 Johnson A, Hao J, Sjolund B. Local corticosteroid application blocks transmission in normal nociceptive fibers. Acta Anaesthesiol Scand 1990;34:355–8.
66 Yaari Y, Selzer ME, Pincus JH. Phenytoin: Mechanism of its anticonvulsant action. Ann Neurol 1986;20:171–84.
67 Massey EW. Sensory mononeuropathies. Semin Neurol 1998;18:177–83.
68 Puig L, Alegre M, de Moragas JM. Treatment of meralgia paraesthetica with topical capsaicin. Dermatology 1995;191:73–4.
69 Devers A, Galer BS. Topical lidocaine patch relieves a variety of neuropathic pain conditions: An open-label study. Clin J Pain 2000;16:205–8.
70 Gofeld MN, Niv D. Pulsed radio frequency treatment for intractable meralgia paresthetica. International Neuromodulationsociety (Madrid 2003) 2003: Abstract 22 p. 58.
71 MacNichol MF, Thompson WJ. Idiopathic meralgia paresthetica. Clin Orthop 1990;254:270–4.
72 Keegan JJ, Holyoke EA. Meralgia paresthetica. An anatomical and surgical study. J Neurosurg 1962;19:341–5.
73 Aldrich EF, van den Heever CM. Suprainguinal ligament approach for surgical treatment of meralgia paresthetica. Technical note. J Neurosurg 1989; 70:492–4.
74 Ivins GK. Meralgia paresthetica, the elusive diagnosis: Clinical experience with 14 adult patients. Ann Surg 2000;232:281–6.
75 van Eerten PV, Polder TW, Broere CA. Operative treatment of meralgia paresthetica: Transection versus neurolysis. Neurosurgery 1995;37:63–5.
76 Namba Y, Ito S, Tsutsui T, Koshima I. Treatment of meralgia paresthetica with a deep inferior epigastric perforator adiposal flap: Case report. J Reconstr Microsurg 2003;19:69–72.