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Intrapartum lesions to the lumbar portion of the lumbosacral plexus: an anatomical review

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SUMMARY

The lumbosacral plexus is formed by the ventral rami of L2-S3 and provides sensory and motor branches to the lower extremity. The spatial orientation of the lumbar portion of the plexus above the pelvic brim leaves it particularly susceptible to intrapartum injury by the fetal head. Such lesions are subdivided into two groups: upper lumbar plexus (L1-L4) and lumbosacral trunk (L4-L5). Given the root levels involved, upper lumbar plexus lesions produce symptoms suggestive of iliohypogastric, ilioinguinal, genitofemoral, femoral, and obturator neuropathies or L4 radiculopathies. Alternatively, involvement of the lumbosacral trunk can imitate a common fibular (peroneal) neuropathy or L5 radiculopathy. This symptomatic overlap with various neuropathies and radiculopathies, makes diagnosis of such lesions particularly challenging. To assist in the clinical diagnosis of intrapartum lumbosacral plexopathies, we provide an overview of the motor, sensory, and reflex deficits associated with such lesions and establish the clinical profile of such patients by presenting case studies from the literature of lumbosacral plexopathies. Only cases from the literature involving women who delivered via cesarean section are explored to isolate the presentation of these lesions from injuries related to birth trauma.

Based on this overview, we offer differential diagnostic tools which can be utilized to aid in the identification and subsequent treatment of intrapartum lesions to the lumbar portion of the lumbosacral plexus.

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INTRODUCTION

The lumbosacral plexus is formed by the ventral rami of the L2-S3 segments, with some contributions from L1 and S4 segments. It gives rise to six sensory nerves of the thigh and leg, and six major sensorimotor nerves responsible for innervating 43 muscles of the lower extremity (Van Alfen and Malessy, 2013). As the name would suggest, it consists of two components, the lumbar plexus and the sacral plexus, which are spatially separated. This anatomical separation results in a clinical division of lumbosacral plexus lesions into those affecting the lumbar plexus and those affecting the sacral plexus (Bademosi et al., 1980; Delarue et al., 1994; Katirji et al., 2002; Planner et al., 2006; Van Alfen and Malessy, 2013).

The morphology of the pelvic regions through which the nerves of the lumbosacral plexus traverse plays a crucial role in explaining this pattern of injury. The pelvis can be divided into two regions: the false pelvis and the true pelvis. The false pelvis is defined laterally by the broad ilium and has a relatively flat, planar surface on which the branches of the lumbar plexus (L1-4) are distributed (Gregory and Shoja, 2016). These branches provide sensory and motor innervation to the thigh and leg and are at risk of injury when a large fetal head is pressing down on the false pelvis from above (Pitman et al., 2016). Thus, damage to the branches of the lumbar plexus can occur intrapartum as well as during parturition.

More inferiorly, the narrow, rounded space of the true pelvis presents as a nearly vertical escarpment that dives deep into the pelvis to emerge at

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the inferior aperture, which is closed inferiorly by the pelvic and urogenital diaphragms. The roots of the sacral plexus (S1-S3) found in this region are distributed vertically and thus relatively protected by the vertical arrangement of the bones, although they may be at risk during parturition. Likewise, the use of forceps during vaginal delivery places these sacral nerve roots in jeopardy (Aranyi et al., 2016).

At the junctional zones between the false and true pelvis, the lumbosacral trunk carrying L4 and L5 roots appears, lying exposed on the anterior surface of the sacrum. Here the lumbosacral trunk is subject to compression injury by a large fetal head, particularly in cases of cephalopelvic disproportion, in which the size of the fetus head is much greater than the ability of the pelvis to accommodate (Maharaj and Teach, 2010). Compression of the lumbosacral trunk results commonly results in footdrop. This condition manifests primarily as weakness of dorsiflexion, and sensory changes in the L5 dermatome. Footdrop associated with cephalopelvic disproportion, is frequently seen after prolonged, difficult vaginal delivery. It is usually the brow of the fetal head that compresses the lumbosacral trunk, which is exposed as it passes over the pelvic brim to descend anterior to the sacral ala (Hill 1962; Murray 1964; Holdcroft et al., 1995; Holloway et al., 2000; Dar et al., 2002; Wong et al, 2003; Reynolds, 2009; Hinshaw and Arulkumaran, 2018).

As a consequence of these aforementioned spatial differences, obstetric or intrapartum injuries to the lumbosacral plexus typically involve the lumbar portion of the plexus. Its position above the pelvic brim renders the lumbar plexus particularly susceptible to intrapartum compression by the fetal head, especially if the patient is of short status or cephalopelvic disproportion is present (Aranyi et al., 2016). The S1-S3 ventral rami, on the other hand, are protected in the lateral wall of the true pelvis, which seems to provide the sacral plexus some protection against intrapartum lumbosacral plexopathy.

In this paper we will discuss the following: (1) the anatomical basis of lesions to the lumbar portion of the lumbosacral plexus related to the intrapartum period; (2) typical clinical findings associated with these lesions; (3) actual patient presentations of lumbosacral plexopathies from the literature, and (4) the ways in which the anatomical manifestation of this plexopathy can be distinguished from peripheral neuropathies or radiculopathies. One difficulty in such an endeavor is that the traumas involving the birth process can produce confounding neuropathies or radiculopathies. In order to discuss lumbosacral plexopathy in its purest form, we have isolated our discussion to cases from the literature in which delivery occurred via cesarean section, thereby eliminating the effects of birth trauma in producing peripheral neuropathies or radiculopathies.

ANATOMY OF THE LUMBAR PORTION OF THE LUMBOSACRAL PLEXUS

Immediately after emerging from the intervertebral foramina, the L1-4 roots of the lumbar portion of the lumbosacral plexus become embedded in the psoas major muscle, where they split into anterior and posterior divisions. The anterior divisions give rise to the iliohypogastric (L1), ilioinguinal (L1), genitofemoral (L1-2), and obturator nerves (L2-4) (Fig. 1). These nerves provide sensory innervation to the lower abdomen, upper thigh and lateral genitalia, and motor innervation to the adductors and gracilis muscles, respectively. The posterior divisions form the lateral femoral cutaneous nerve (L2-3), which carries sensory fibers from the skin of the lateral thigh, and the femoral nerve (L2-4) (Fig. 1), which carries motor fibers to the quadriceps, sartorius, psoas and iliacus muscles and sensation to the anterior thigh and medial upper leg (Kaeckle, 2015). Table 1 summarizes the distribution of these peripheral nerves.

The lumbosacral trunk, meanwhile, is a portion of the lumbar plexus formed by the anterior rami of L4 and L5. It connects the lumbar plexus to the sacral plexus as it crosses the posterior brim of the pelvis (Fig. 1), medial to the psoas muscle (Ma, 2011). Once blended into the sacral plexus, the L4 -5 fibers originating from the lumbosacral trunk contribute to the tibial (L4-S3) and common fibular (peroneal) nerves (L4-S2), which provide motor innervation to the foot plantar flexors and dorsiflexors, and sensation to the heel, sole, lateral leg and dorsum of the foot. L4 and L5 also contribute to the superior (L4-S1) and inferior (L5-S2) gluteal nerves, which innervate the gluteal muscles (Table 1) (Ma, 2011; Kaeckle, 2015).

CLINICAL FEATURES OF LUMBOSACRAL PLEXOPATHY AND PATIENT PRESENTATIONS FOLLOWING A CESAREAN SECTION

Clinical patterns involving the lumbar portion of the lumbosacral plexus can be subdivided into the upper lumbar plexus (L1-L4) and the lumbosacral trunk (L4-L5). For diagnostic purposes, the clinical distinction between pathology involving these portions of the lumbosacral plexus is important, as the etiology and diagnostic approach (such as the choice of muscles for needle electromyography) varies accordingly (Van Alfen and Malessy, 2013).

Diagnosed cases of intrapartum maternal lumbosacral plexopathy in the literature are relatively rare, and those experienced following a cesarean section are even rarer. A PubMed literature search using the words “lumbosacral plexopathy” and “pregnancy” identified nine original articles. When these articles were examined for discussion of case reports and cesarean section, the results narrowed to two publications reporting on ten cases (Delarue et al., 1994; Katiriji et al., 2002). We then organized and discussed the ten case reports relative to upper lumbar and lumbosacral trunk lesions. Motor and sensory findings for each patient are summarized in Table 2.

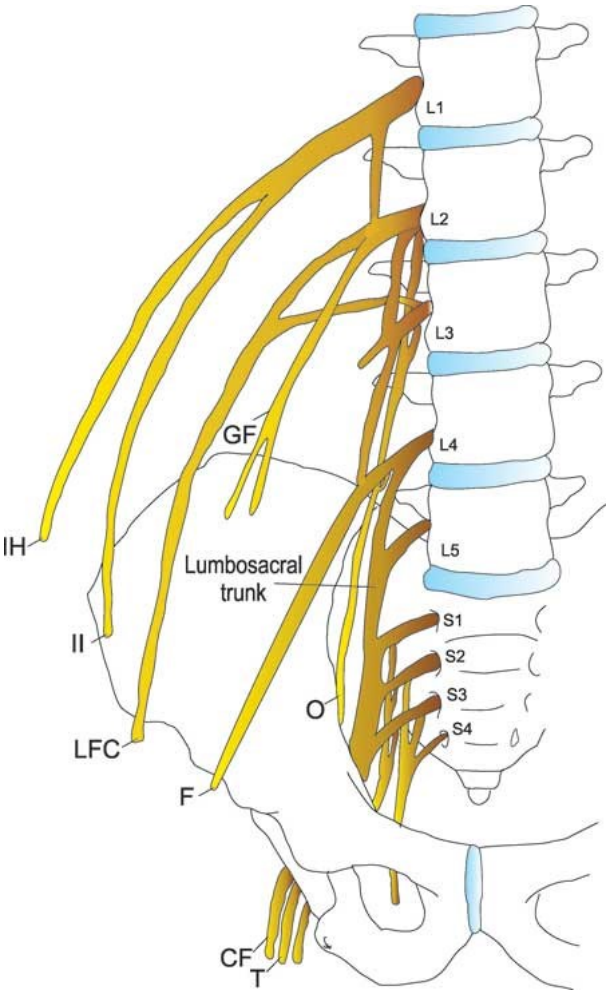

Fig 1. Drawing of lumbosacral plexus and relevant derivative nerves: iliohypogastric nerve (IH), ilioinguinal nerve (II), lateral femoral cutaneous nerve (LFC), genitofemoral nerve (GF), lumbosacral trunk (LST), femoral nerve (F), obturator nerve (O), common fibular nerve (CF), tibial nerve (T).

Upper Lumbar Lesions

Upper lumbar plexus lesions produce symptoms in the iliohypogastric, ilioinguinal, genitofemoral, femoral, and obturator nerve territories due to the involvement of the L1-4 segments. Consequently, this may manifest in reduced hip flexion and knee extension (i.e., L2-4 nerve roots) associated with psoas, iliacus, pectineus, sartorius, and quadriceps weakness (Bademosi et al., 1980; Delarue et al., 1994). An alternate presentation can involve the same lumbar segments (i.e., L2-4), but presents as loss of function or weakness in thigh adduction (adductor longus, brevis, anterior half of magnus, and gracilis (Bademosi et al., 1980). Sensory loss is often seen in the lower abdomen, inguinal region, over the entire thigh (excluding the posterior portion), and the lower medial leg. The knee jerk reflex, which is mainly supplied by L3 and L4, may be decreased or absent (Yii et al., 2017). Lesions in this portion of the lumbosacral plexus are commonly confused with a femoral or obturator neuropathy or a L4 radiculopathy due to the presence of quadriceps weakness and/or anterior thigh hypoesthesia or hyperalgesia. Symptoms consistent with lesion to the upper lumbar portion of the lumbosacral plexus were identified in two of the ten case reports. Both cases, identified as Patient B and Patient C, came from the work of Delarue et al. (1994).

Patient B was a 31-year-old gravida 2 who suffered from acute pain in her right groin at 32 weeks of amenorrhea. The pain was increased upon bending and by the Valsalva maneuver. Signs of radiation of the pain were absent. She complained of progressive muscular weakness. Her clinical history revealed that she had experienced the same pain and weakness beginning at 34 weeks of amenorrhea. In the previous case, the symptoms resolved spontaneously several weeks after an uncomplicated delivery. During the second pregnancy, neurological examination revealed a grade IV paresis of the iliopsoas, quadriceps femoris, and biceps femoris muscles. Electromyographic examination revealed denervation activity in the adductors, iliopsoas, and rectus femoris muscles, with no abnormalities detected in the paravertebral muscles. Lumbar plexopathy was diagnosed, most likely involving the L2-L4 segments. The patient experienced a normal delivery. The neurological examination in the puerperium was identical.

Following therapy, three months later the patient had fewer complaints, and the strength in her right leg was normal (Delarue et al., 1994).

Patient C was a 31-year-old gravida 3, para 2 woman who developed lower back pain at 34 weeks of amenorrhea. This pain evolved into a progressive pain located in the groin and on the anterior side of the right thigh. After delivery of a healthy baby, she complained of progressive weakness of the muscles in the right thigh.

Neurological examination demonstrated a grade IV motor weakness of the iliopsoas, quadriceps and adductor muscles. The spine X-ray, hematological and biochemical investigations were normal. Myelography was normal.

Electromyography demonstrated denervation activity in the quadriceps femoris, biceps femoris, semitendinosus, sartorius, and iliopsoas muscles. No denervation activity was found in the paravertebral muscles. Neurological examination 6 months later demonstrated normal muscle strength (Delarue et al., 1994).

Lumbosacral Trunk Lesions

The second and most common pattern of intrapartum lumbosacral plexopathy involves the lumbosacral trunk. Unlike the other derivatives of the lumbar portion of the lumbosacral plexus, the lumbosacral trunk is not protected by the psoas muscle, leaving it particularly susceptible to compression by the fetus at the pelvic brim. Lesion of the lumbosacral trunk produces a foot drop involving the muscles of the anterior crural compartment (tibialis anterior, extensor hallucis longus, and ex-

Table 1. Peripheral nerve and lumbar root distribution

Root Level	Peripheral nerve	Sensory Distribution	Muscles Innervation	Lower Extremity Action
L1	Iliohypogastric	skin of lower abdomen and hip	internal oblique and transversus abdominis	N/A
L1	Ilioinguinal	skin of external genitalia and proximal medial thigh	inferior abdominal muscles	N/A
L1-2	Genitofemoral	skin of scrotum in males, labia majora in females, anterior thigh inferior to middle portion of inguinal region	remaster muscle in males	N/A
L2-3	Lateral femoral cutaneous	skin of lateral thigh, some sensory branches to perineum	N/A	N/A
L2-4	Obturator	skin of medial thigh	adductor magnus (anterior half), adductor longus, adductor brevis, gracilis, obturator externus	Hip adduction
L2-4	Femoral	skin to anterior and medial thigh	quadriceps femoris, sartorius, pectineus, iliacus	Knee extension
L4-S3	Tibial	skin of posterior leg and sole of foot	hamstrings (except short head of biceps femoris, posterior part of adductor magnus), triceps surae, tibialis posterior, popliteus, flexor digitorum longus, flexor hallucis longus, intrinsic muscles of foot	Knee flexion(primary), foot plantar flexion, toe flexion
L4-S2	Common fibular	skin of anterior and lateral leg and dorsum of foot	short head of biceps femoris, fibularis longus and brevis, tibialis anterior, extensor hallucis longus, extensor digitorum longus and brevis	knee flexion (short head of biceps femoris), foot dorsiflexion and eversion, toe extension
L4-S1	Superior gluteal	buttock pain	gluteus medius, gluteus minimus, tensor fasciae latae	hip abduction
L5-S2	Inferior gluteal	N/A	gluteus maximus	hip external rotation and power extension
L4	L4 radiculopathy	medial calf and foot (pain in anterior thigh)	quadriceps femoris, adductors, iliopsoas	ankle dorsiflexion
L5	L5 radiculopathy	dorsum of foot, big toe, lateral calf	tibialis anterior and posterior, extensor hallucis longus, gluteus medius, tensor fascia latae	big toe extension

tensor digitorum longus). Weak ankle dorsiflexion and eversion are often noted, as well as weakness in ankle inversion and toe flexion. Variable buttock pain and gluteal and hamstring weakness may also manifest. Numbness in the lateral leg and dorsum of the foot, following a L5 dermatomal distribution is common. The ankle jerk reflex (i.e., Achilles tendon reflex), which is mediated primarily by S1, is usually normal, as is plantarflexion at the foot (Katiriji et al., 2002; Yii et al., 2017). These lesions can be diagnostically challenging, as they imitate a common fibular (peroneal) neuropathy or an L5 radiculopathy with the presence of footdrop and L5 myotome involvement. We identified symptoms consistent with lumbosacral trunk lesions in eight of the ten case reports. One patient, Patient A, came from the work of Delarue, Vles and Hasaart (1994), while the remaining seven, Patients 1 through 7, were described by Katriji and colleagues (2002).

Patient A a 32-year-old primagravida with twin pregnancy, complained of acute back pain starting at 33 weeks of amenorrhea. Several days later she noted sensory abnormalities in and weakness of the left foot. Neurological examination demonstrated a grade IV motor weakness of the extensor hallucis longus, anterior tibial and peroneal muscles, as well as a sensory loss on the dorsum of the foot.

Following a normal delivery, neurological examination on the third day of the puerperium demonstrated denervation activity in muscles supplied by the L4 and L5 myotomes, but no abnormalities in the paravertebral muscles. Three months later, the patient had no complaints and the neurological examination was normal (Delarue et al., 1994).

Patient 1 was admitted at 40 weeks of gestation for irregular uterine contractions and induction of labor. She had previously delivered two girls (9 pounds 6 ounces; 11 pounds and 11 ounces) by

Table 2. Summary of lumbosacral plexopathy patient symptoms from the litera-

Lesion to Upper Lumbar Portion				
Patient	**Motor Deficit**	**Sensory Deficit**	**Ankle Jerk**	**Foot Drop**
Patient B	weakness in iliopsoas, quadriceps femoris, biceps femoris, adductors, iliopsoas, and rectus femoris	acute groin pain (increased by Valsalva maneuver)	Not provided	No
Patient C	weakness in iliopsoas, quadriceps femoris, biceps femoris, adductors, semitendinosus, and sartorius	lower back, groin, and anterior upper leg pain	Not provided	No
Lesion to Lumbosacral Trunk				
Patient	**Motor Deficit**	**Sensory Deficit**	**Ankle Jerk**	**Foot Drop**
Patient A	weakness in anterior leg muscles; denervation in L4-5 myotomes	hypoesthesia on dorsum of foot	Not provided	Yes
Patient 1	initial presentation: absent ankle dorsiflexion, eversion, inversion, toe dorsiflexion presentation at 4 weeks: weak hamstrings & gluteal muscles	hypoesthesia on right lateral leg and dorsum of foot, less involvement of sole of foot	Normal	Yes
Patient 2	weak dorsiflexion, inversion, eversion of ankle, weak toe extension and flexion, mild weakness for plantar flexion, knee flexion, and hip extension	hypoesthesia along lateral leg and dorsum of foot, severe pain in right buttock and leg	Normal	Yes
Patient 3	no ankle flexion, eversion, inversion, or toe dorsiflexion	hypoesthesia along dorsum of right foot, including big toe	Normal	Yes
Patient 4	weakness in ankle dorsiflexion, inversion, and eversion; toe extension	hypoesthesia on dorsolateral foot and along L5 dermatome	Normal	Yes
Patient 5	absent ankle dorsiflexion, inversion, eversion, toe extension	hypoesthesia anterolateral leg, dorsum of foot	Normal	Yes
Patient 6	weakness in ankle dorsiflexion, inversion, and eversion; toe dorsiflexsion; knee flexion; hip abduction	back pain, radiating into right buttock and posterior thigh; hypoesthesia over dorsolateral distal leg and dorsum of foot	Normal	Yes
Patient 7	weakness in ankle dorsiflexion, inversion, and eversion; toe dorsiflexsion; knee flexion; hip abduction and extension	hypoesthesia over lateral calf, dorsum and sole of foot	Normal	Yes

Patients A-C taken from Delarue, Vles, and Hassart, 1994; Patients 1-7 taken from Katirji, Wilbourn, Scarberry, and Preston, 2002

uneventful vaginal delivery. During labor the patient noted intermittent numbness of the right foot. Following 2.5 hours of labor with the baby in a persistent vertex position, the patient then underwent a low transverse cesarean section under local anesthesia, delivering a 12-pound 12-ounce baby boy. On the first attempt to stand eight hours following delivery, the patient exhibited a complete right foot drop, with numbness of the entire foot and lateral aspect of the right leg. She noted mild pain in the right buttock with no bruises, back pain, or radicular symptoms. Neurological testing revealed no detectable function of dorsiflexion, eversion, and inversion the right ankle, or of dorsiflexion of the toes of the right foot. Deep tendon reflexes, including ankle jerk reflexes, were normal and symmetric. Decreased sensation was noted on the right lateral leg and dorsum of the right foot with less involvement of the sole.

When the patient was seen four weeks later, she was still unable to dorsiflex, invert, or evert the right foot at the ankle or dorsiflex the toes. Plantar flexion was normal, but toe flexion was weak. The right gluteus maximus was weak, as were the hamstrings and gluteus medius. The right quadriceps and iliopsoas were normal. There was an area of hypoesthesia to touch and pinprick over the lateral aspect of the right leg and dorsum of the right foot. Sensation over the plantar surface of the right foot had become normal. Eight weeks after delivery, the patient experienced a rapid and dramatic improvement of ankle dorsiflexion, and the sensory deficits had nearly disappeared (Katiriji et al., 2002).

Patient 2 was admitted for onset of contractions and spontaneous rupture of membranes at 39.5 weeks of gestation. An epidural catheter was placed for labor pain control, although the patient did not experience buttock or leg pain. After 2.5 hours of labor, forceps delivery was unsuccessfully attempted. A low transverse cesarean section was performed, resulting in a healthy 8-pound 11-ounce baby boy. When the effects of the epidural anesthesia had disappeared, the patient noticed weakness of the right foot and numbness along the lateral leg and dorsum of the foot, along with

severe pain in the right buttock and leg. Neurological examination performed two weeks later demonstrated weakness of dorsiflexion and of inversion and eversion of the right ankle, accompanied by weakness of toe extension and flexion. Mild weakness was observed for plantar flexion, knee flexion, and hip extension. Knee extension and hip flexion were normal.

Hypoesthesia for pinprick and light touch sensation was noted over the dorsum of the right foot. The deep tendon reflexes were all normal and symmetric.

Examination at 13.5 weeks following delivery demonstrated improvement of the condition, with minimal hypoesthesias and mild residual weakness of ankle dorsiflexion and weakness of toe flexion and ankle inversion (Katiriji et al., 2002).

Patient 3 went into labor at 40 weeks of gestation. An epidural anesthesia was initiated because of prolonged labor and pain. A forceps delivery was unsuccessful. The patient ultimately delivered a healthy 7-pound 15-ounce baby boy by cesarean section under epidural anesthesia. When the patient attempted walking the next day, she noted that she was dragging her right foot and was unable to “wiggle” her toes. Neurological examination performed three days later demonstrated complete right foot drop with no flexion of the ankle and toe dorsiflexors, and of the evertors and inverters of the right ankle. Plantar flexors and proximal right leg musculature all demonstrated normal activity. Deep tendon reflexes were normal. Mild hypoesthesia for pinprick and touch sensations on the dorsum of the right foot, including the big toe was observed. Gradual improvement of symptoms was noted, with a complete resolution of symptoms was observed at 5 months (Katiriji et al., 2002).

Patient 4 was admitted in active labor at 41 weeks of gestation. Eighteen hours later, she complained of cramping pain in her right leg. Labor was prolonged, with the presenting fetal head in the left occipital anterior position. The patient was given epidural anesthesia. At 22 hours she underwent a cesarean section under epidural anesthesia because of an arrested descent. Soon after delivery, the patient complained of right leg weakness and numbness. She exhibited a right foot drop, normal plantar flexion, and sensory loss in the right dorsolateral foot. The patient’s condition was unchanged two weeks later. Neurological examination revealed severe weakness of dorsiflexion the right ankle, as well as weakness of inversion and eversion of the right foot at the ankle, and extension of the toes. The following tests on the right side were normal: plantar flexion, knee flexion and extension, hip adduction, and hip flexion. Deep tendon reflexes were all normal. Hypoesthesia for pain and touch were noted on the distribution of the L5 dermatome (Katiriji et al., 2002.

Patient 5 underwent a cesarean section under epidural anesthesia because of arrested labor. She delivered a healthy boy. The patient noted a complete left foot drop when she attempted to walk 24 hours later. She noted the absence of pain in the back or leg. No sensory disturbances were noted. Neurological examination revealed loss of left ankle dorsiflexion, ankle inversion or eversion, or extension of the toes of the left foot. Plantar flexion was normal. Deep tendon reflexes were normal. Hypoesthesia was noted over the left distal anterolateral leg and dorsum of the left foot. Magnetic resonance imaging revealed a normal lumbar spine. Six weeks later the patient began to experience gradual improvement of function of the foot at the ankle. A neurological examination eight weeks after delivery revealed severe weakness of dorsiflexion and inversion of the left foot at the ankle (Katiriji et al., 2002).

Patient 6 complained of back pain radiating into her right buttock and posterior thigh after 16 hours of labor. Cephalopelvic disproportion was present, resulting in a cesarean section under general anesthesia. When the patient awoke the next day, she noted leg weakness, foot drop, and leg numbness on the right side. No improvement was noted at 3.5 weeks later, when a neurological examination revealed weakness of the following on the right side: ankle and toe dorsiflexion, ankle inversion, and eversion, knee flexion and hip abduction. Right hip flexion, hip extension, knee extension, and plantar flexion were all normal. Hypoesthesia was noted over the right dorsolateral distal leg and dorsum of the foot. Knee and ankle tendon reflexes were normal. Partial recovery was noted at 2 months, with complete recovery present at 5 months (Katiriji et al., 2002).

Patient 7 was admitted in active labor at 41 weeks of gestation. Despite a fully dilated cervix, no further progression of labor was noted. Persistent late fetal decelerations developed and cephalopelvic disproportion was noted. The patient did not report buttock or leg pain. She underwent a cesarean section under general anesthesia and delivered a normal girl. On the first postpartum day, the patient complained of numbness and weakness of the right foot, but no pain was noted. The patient exhibited a complete right foot drop. Pain was noted over the right lateral calf and foot. Neurological examination at six weeks revealed the presence of complete right foot drop, with weakness of the following on the right side: ankle and toe dorsiflexion, ankle eversion, ankle inversion, knee flexion, hip abduction, and hip extension. Right hip flexion, knee extension, and plantar flexion were normal. Hypoesthesia was present over the lateral right calf and along the dorsum and sole of the right foot. Deep tendon reflexes were normal and symmetrical. When seen at three months, the patient exhibited complete resolution of symptoms (Katiriji et al., 2002).

DIFFERENTIAL DIAGNOSTIC ASPECTS OF INTRAPARTUM LUMBOSACRAL PLEXOPATHY

Compression of the lumbosacral plexus can occur as the fetal head crosses the ala of the sacrum. As evident in the patient presentations provided above, lumbosacral injuries oftentimes pose a diagnostic challenge because such lesions have features which overlap with various neuropathies and radiculopathies. For instance, femoral and obturator neuropathies have also been described as compression injuries following delivery (Massey and Cefalo, 1979; Montag and Mead, 1981; Vargo et al., 1990). However, patterns of weakness involving the iliopsoas, quadriceps femoris, and the adductors, can be used to distinguish a lumbosacral plexopathy from a femoral or obturator mononeuropathy (Schwartzman, 2006).

Lesions of the lumbosacral trunk commonly present with footdrop, variable buttock pain, and numbness in the lateral leg and the dorsum of the foot. While a common fibular neuropathy may initially be suspected, detection of weakness in ankle inversion (tibialis posterior) or toe flexion (flexor digitorum longus) eliminates this as a diagnosis, as both actions are mediated by the tibial nerve (Katirji, 2007). Furthermore, compression of the lumbosacral trunk portion of the plexus oftentimes produces clinical findings which imitate a severe L5 radiculopathy (Planner et al., 2006; Van Alfen and Malessy, 2013). This is because the L5 root fibers travel solely through the lumbosacral trunk. However, in contrast to L5 radiculopathy, lumbosacral plexopathy patients also present with foot drop. Tibialis anterior is the primary ankle dorsiflexor and is innervated solely by the lumbosacral trunk (L4-5). Only modest weakness in ankle dorsiflexion would be noted with a L5 radiculopathy due to the dual innervation of tibialis anterior, in contrast to pronounced foot drop with a lumbosacral lesion (Katirji, 2007).

Location and timing of symptoms is also informative in diagnoses of lumbosacral plexopathy. These lesions often begin with pain in the groin. The onset of symptoms during pregnancy rather than after delivery is very informative, as it rules out neurological damage due to the delivery process. In particular, prolonged use of the lithotomy position, or squatting, are known to produce peripheral neuropathies.

Magnetic resonance imaging, of course, can be helpful in establishing the correct diagnosis.

Lumbar degenerative disease of the spine and spinal stenosis can lead to compression of multiple nerve roots, which can cause pain, sensory symptoms, and paresis in a pattern that resembles that of a lumbosacral plexopathy. However, lumbar pain and paravertebral muscle spasm offer clues as to the clinical causation of this type of presentation. In addition, symptoms will be provoked by walking and other motor activities (Van Alfen and Malessy, 2013).

CONCLUSION

While obstetric or intrapartum lumbosacral plexopathy is a relatively rare condition occurring in 1.5 to 5/10,000 deliveries, it can raise medico-legal and disability problems. It is a consequence of compression of the lumbosacral plexus (L2-S3) by the fetal head during the third trimester of gestation. Risk factors include small prima gravida, large infant, or both (Bucklin et al., 2016). As this type of lesion typically involves multiple nerve roots, it can mimic femoral, obturator, common fibular neuropathies or L5 radiculopathies. While some motor features can be persistent, pain usually resolves after delivery and most patients fully recover in a few weeks to months. Careful consideration of the clinical findings, coupled with utilization of computed tomography, magnetic resonance imaging, and electromyopathic nerve conduction studies can aid identifying the location and thus type of malfunction.

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