SHORT REPORT

Postpartum lumbosacral plexopathy limited to autonomic and perineal manifestations: clinical and electrophysiological study of 19 patients

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Abstract

The objective was to describe perineal electrophysiological findings and to determine their diagnostic value in a type of lumbosacral plexopathy after vaginal delivery, which only involves the lower part of the plexus (S2-S4).

Consecutive female patients referred to an outpatients' urodynamic clinic were the source. Nineteen previously healthy women, 13 multiparae and six para 1, were investigated. Mean age was 33.7 (SD 5.4) (range 28-41) years. All of them presented with urinary (stress incontinence 14, dysuria five), anorectal (faecal incontinence eight, dyskesia one), or sexual dysfunctions (hypoorgasmia or anorgasmia six) after vaginal delivery. No associated lower limb sensory or motor deficits were noted. All the patients had electrophysiological recordings (bulbocavernosus muscle EMG, measurements of the bulbocavernosus reflex latencies (BCRLs), somatosensory evoked potentials of the pudendal nerve (SEPPNs), and pudendal nerve terminal motor latencies (PNTMLs)). Cystometry and urethral pressure profile (UPP) were performed in the 14 patients with stress urinary incontinence.

Perineal electrophysiological examination disclosed signs of denervation in the perineal muscles in all the cases, prolonged BCRLs in 17/19, and abolished BCRLs in 2/19, abnormal SEPPN in 1/19, and normal PNTMLs in all the patients. Urodynamic investigations disclosed low urethral closure pressure for age (< 50 cm H₂O) in half of the patients.

In conclusion, Lower postpartum lumbosacral plexopathy is evoked when perineal sensory disturbances whether or not associated with urinary or faecal incontinence persist after a history of a difficult vaginal delivery. Electrophysiological investigations precisely identify the site of the lesion and demonstrate distal innervation integrity.

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Lumbosacral plexopathy occurring during the third part of pregnancy, intrapartum, or postpartum is reported in the literature as a rare obstetric complication.^{1,2} It usually involves the upper plexus (L2-S1) causing obturator, femoral, or sciatic nerve palsies.^{3,4} Symptoms in the lower limbs often are unilateral involving more than one nerve, sensory and motor. Autonomic involvement is rare. Urinary, anorectal, or sexual dysfunctions due to autonomic involvement are always reported to be associated with motor or sensory disorders in the lower limbs. No specific study of autonomic involvement was found in the literature. Prognosis of the lumbosacral plexopathy is reported to be excellent and recovery usually occurs within 4 to 6 months.^{1,5-8}

We describe here 19 patients with postpartum urinary, anorectal, or sexual dysfunctions due to lumbosacral plexopathy. Contrary to the literature, motor deficit in the lower limbs was absent and prognosis seemed to be different. Electrophysiological data, available for all the patients, showed proximal plexic lesions.

Patients and methods

Nineteen patients, 13 multiparae and six para 1, consecutively referred for urinary, anorectal, or sexual disorders after vaginal delivery were examined in a urodynamic outpatient clinic between 1992 and 1999. These patients were reviewed retrospectively. The mean age was 33.7 (SD 5.4) (range 28 to 41) years, The mean interval between delivery and consultation was 12.8 (SD 13.2) (range 1 to 48) months. All patients were reported to have been previously healthy except for one who had a history of sciatica with no neurological sequelae. History of low back pain during pregnancy was not recorded in any of them. History of forceps delivery was found in 10 patients. Urinary disorders were found in 18 patients, 14 with stress incontinence and five with dysuria with urinary retention (bladder residual volume > 100 ml). The urinary desire to void and urethral urine sensation were abolished in two patients and normal in the others. Anorectal disorders were found in nine patients (five incontinent to gas, two to liquid, one to solid, and one dyskesic). Sexual disorders were noted in six patients, hypo-orgasmia or anor-

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Distribution and associations of symptoms in 19 patients with postpartum lumbosacral plexopathy: 1=urinary disorders only; 2=urinary disorders and anorectal disorders; 3=urinary disorders, anorectal disorders, and sexual disorders; 4=urinary disorders and sexual disorders; 5=sexual disorders only.

gasmia associated in five of them with vaginal numbness. Distribution and associations of these disorders are presented in the figure.

On physical examination perineal hypoaesthesia was noted in seven patients, three unilateral right, three unilateral left, and one bilateral. In two patients perineal anaesthesia was noted.

All patients had electrophysiological studies including perineal muscle EMG, measurements of bulbocavernosus reflex latencies (BCRLs) obtained by clitoral stimulation (normal value <44 ms), somatosensory evoked potentials of the pudendal nerve (SEPPNs), and pudendal nerve terminal motor latencies (PNTMLs).

Conus medullaris MRI was done on all the patients to rule out other potential mechanisms of prolonged or abolished BCRLs (compression by a space occupying lesion, demyelination, ischaemic lesions).

Plexus lesion was defined as a BCRL more than 44 ms associated with normal PNTML and unilateral or bilateral hypoaesthesia at S2-S4, with no objective conus medullaris lesion on MRI.

Urodynamic investigations (cystometry and urethral pressure profile) were performed according to the International Continence Society recommendations in the 14 patients with stress urinary incontinence.

Results

The electrophysiological study showed denervation potentials in the perineal muscles in all the patients, prolonged sacral latencies (BCRLs) in 17 patients, and abolished potentials in the remaining two (with perineal anaesthesia on physical examination). Pudendal nerve terminal motor latencies were normal in all the patients. Somatosensory evoked potentials of the pudendal nerve were abnormal only in one patient (table).

Electrophysiological findings in 19 patients with postpartum lumbosacral plexopathy

Result	EMG	BCRL	PNTML	SEPPN
Denervation potentials	19/19			
Abnormal		17/19		1/19
Normal			19/19	18/19
Abolished		2/19		

EMG=bulbocavernosus muscle electromyography; BCRL=bulbocavernosus reflex latency; PNTML=pudendal nerve terminal motor latency; SEPPN=somatosensory evoked potential of the pudendal nerve.

Conus medullaris MRI was normal in all the patients.

Urodynamic investigations in the 14 incontinent patients showed low urethral closure pressure <50 cm H₂O in seven. In the five patients presenting with dysuria, cystometry revealed underactive bladders.

No significant associations were found between the clinical findings and the electrophysiological measurements and the urodynamic findings.

Discussion

Beatty³ in 1833 and Bianchi,⁴ in 1867 described traumatic paralysis in lower limbs in the postpartum period.

Direct obstetric trauma is rare,⁷ but pressure from the gravid uterus may cause plexitis to develop in pregnancy.⁸ Symptoms usually develop around 32 to 34 weeks of gestation and involve the upper plexus. Pain is usually felt in the groin area, the lateral aspect of the calf, and over the dorsum of the foot; and weakness can involve the hip flexors, leg extensors, or feet. Electromyographic evaluation shows denervation of the involved muscles without paraspinal changes. Risk factors are short primigravida carrying a relatively large baby (>3750 g), with prolonged labour, malpresentation, and midforceps rotation after a transverse arrest.⁹

Neurological deficit might occur by damage to lumbosacral roots from an epidural anaesthetic catheter. This is extremely rare and would be unlikely to cause damage to multiple roots.¹⁰

Lumbosacral plexopathy must be differentiated from peroneal nerve compression against the fibular head by leg holders, and also from sciatic pain due to a concomitant discal herniation.¹¹

We found no literature about urinary or anorectal incontinence, or sexual disorders occurring without an associated lower limb deficit in the lumbosacral plexopathy. However, faecal and urinary incontinence are well known complications of vaginal delivery. Classically thought to be due to direct sphincter division or stretch or damage to the perineal floor musculature,^{12,13} Snooks *et al*¹⁴ suggested that it resulted also from damage to the innervation of the pelvic floor sphincter muscles which seems to be initiated by childbirth. The PNTMLs after vaginal delivery were significantly delayed.

Our patients had neither deficit in the lower limbs nor delayed PNTMLs after vaginal deliveries. They had urinary, anorectal, or sexual symptoms, perineal sensory disturbance, and, always, denervation on EMG associated with delayed BCRLs and normal PNTMLs. We describe this as a lumbosacral plexopathy involving the lower part of the plexus (S2-S4).

The urodynamic investigations permitted the demonstration of low urethral closure pressure in half of the patients. This lack of urethral pressure may be contributory to the incontinence and is perhaps due to urethral sphincter denervation.

Classic risk factors found in our patients were multiparity in 13/19 and midforceps rotation in 10/19. Birth weight >3750 g was found in only 2/19 patients. Follow up gave a poor prognosis, as symptoms were present at a mean interval of 12.8 months.

In conclusion, persistent perineal unilateral or bilateral hypoesthesia after vaginal delivery must evoke the diagnosis of lower lumbosacral plexus injury. Electrophysiological examinations confirm the diagnosis and allow the localisation of its territory and severity. Risk factors are probably the same as in postpartum upper plexus injury. Prognosis might be worse. Preventive measures are speculative.

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