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

Samer Sheikh Ismael, Gerard Amarenco, Béatrice Bayle, Jacques Kerdraon

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 outpatient's 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, dyskasia 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 (bulbocavernous 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 SEPPNs in 1/19, and normal PNTMLs in all the patients. Urodynamical 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.

Keywords: postpartum, lumbosacral plexopathy, electrophysiological investigations.
Electrophysiological findings in 19 patients with postpartum lumbosacral plexopathy

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<th>Result</th>
<th>EMG</th>
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<td>Denervation potentials</td>
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<td>Abnormal</td>
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EMG=bulbocavernous muscle electromyography; BCRL= bulbocavernous reflex latency; PNTML=pudendal nerve terminal motor latency; SEPPN=somatosensory evoked potential of the pudendal nerve.

Discussion

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

Direct obstetric trauma is rare, but pressure from the gravid uterus may cause plexitis to develop in pregnancy. 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.

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.

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.

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, Snooks et al 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.

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.
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.