Laparoscopic therapy for endometriosis and vascular entrapment of sacral plexus

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Deeply infiltrating endometriosis usually is associated with a multitude of symptoms and constitutes a complex treatment challenge. Whereas ureteral and intestinal involvements are well-known clinical forms of deeply infiltrating endometriosis, sacral plexus endometriosis is largely unknown and still considered rare in gynecology (1). The same low incidences are reported in the literature for endopelvic causes for sacral radiculopathies and sciatica (2). Our aim is to report our experience with pelvic pathologies of the sacral plexus and sciatic nerve and to draw gynecologists’, neurologists’, and neurosurgeons’ attention to such pathologies.

MATERIALS AND METHODS

Two hundred thirteen consecutive patients sent us between November 2004 and February 2010 for treatment of unilateral sciatica (→L5, S1-2) and/or pudendal or gluteal pain (→S2-4) of unknown genesis, after exclusion of any spinal cord cause, were included prospectively in this study. Patients with laparoscopically confirmed surgical (3) or traumatic damages to the sacral plexus were excluded from the study. All data were collected at the time of surgery on a standardized form used in the clinical care of all our patients.

Preoperative assessment not only focused on gynecologic aspects but also included neurologic symptoms for sacral radiculopathies. Careful anamnesis was performed with detailed information of pain involving the buttock, the pudendal area, and the lower extremities. Information also was obtained on possible motor deficits of hip adduction (L3–obturator nerve), knee extensors (L1-L4–femoral nerve), ankle dorsiflexion (foot drop–L5), and ankle plantar flexion (S1). Sphincter dysfunctions, motor or sensitive urinary urgency, or voiding difficulties were explored by urodynamic testing. Clinical examination included vaginal palpation (eventually anesthetic infiltrations) of pudendal nerves and the lower sacral nerve roots (S3, S4) and research for any hypoesthesia or hyperesthesia at the lumbosacral dermatomes and peripheral nerves. The same evaluation was performed for postoperative follow-up.

Our surgical principle is based on the full exposure of the nerves clinically suspected of being involved by a transperitoneal approach. Exposure of the sacral nerve roots below the cardinal ligament—“infracardinal level” of the sacral plexus—has been reported extensively elsewhere (4). Exposure of the distal portion of the sacral plexus and of the sciatic nerve—“supracardinal level”—can be obtained by starting dissection at the level of the lumbosacral fossa passing laterally to the external iliac vessels or by starting dissection at the level of the internal iliac artery. Full exposure of the sciatic nerve, of the gluteal nerves, and of the pudendal nerve before it leaves the pelvis is obtained by detaching interiliac lymph-fat-tissue from the pelvic sidewall and following the dorsal border of the obturator internus muscle in the direction of the sciatic spine.

Visual Analogue Scale (VAS) score was obtained before and after surgery. Surgical success was defined as 50% reduction of VAS score at the 6-month follow-up. Paired t-test was used to compare preoperative and postoperative VAS scores. Student’s t-test was used to compare the postoperative VAS scores according to each cause.

RESULTS

Two hundred thirteen consecutive patients were included in this study. The mean age of all patients was 28 years (range 17–79
years), and mean body mass index was 29.2 (range 25–32). All procedures were performed by laparoscopy without any need of conversion to laparotomy. Table 1 shows associated surgical procedures. No intraoperative complication occurred. One patient had during the first postoperative night an intima dissection of the right internal iliac artery that required an inguinal iliofemoral bypass. No further postoperative complications occurred.

Twenty-seven patients had an isolated endometrial cyst of the sciatic nerve (isolated sciatic endometriosis without identification of any further manifestation of endometriosis), 24 patients on the left side and 3 patients on the right (5). The lesions, with diameters varying from millimeters to centimeters, had built a true hole inside the nerve at the suprapyriformis level that contains the L5, S1, and S2 nerve fibers. Opening of the sciatic perineurium with intrafascicular neurolysis, excision of the lesion, and superficial coagulation of the entire sciatic hole were performed in all patients. Eleven required a further dissection of the nerve through the greater sciatic foramen downward to the gluteal region. Before surgery these patients had L5, S1, and S2 sciatica with corresponding cutaneous hypoesthesia in 13 of them and a partial foot drop in 4. None of them initially had bladder dysfunctions, hyperactivity, or hypersensitivity.

One hundred forty-eight further patients had parametric deeply infiltrating endometriosis with infiltration per continuum of the sacral plexus. Because of the anatomic relationship, deeply infiltrating endometriosis of the sacral uterine ligaments and/or the rectovaginal space was correlated with involvement of the sacral nerve roots S2, S3, and S4, whereas deeply infiltrating endometriosis starting higher from the ovarian fossa or directly from cardinal ligaments (ureteral endometriosis) involved preferentially the sacral nerve roots S1, S2, and S3, never L5. All patients initially were seen with S1-S4 sciatica, gluteal pain (buttock), and pudendal pain with troubles of the urinary system: urodynamic testing showed sensory urinary urgency in 49 patients, motor bladder hyperactivity in 38 patients, and detrusor hypotension in 37 patients.

In 37 further patients, laparoscopic exploration showed an isolated sacral plexus vascular entrapment that meant the compression tying of a nerve by an atypical enlarged vein or less frequently an artery. The treatment consisted of the primary exposure of the nerves with bipolar coagulation and resection of the atypical vessels. The most frequent entrapped nerves in this series were S1, S2, and the sciatic nerve by atypical sacral and inferior gluteal veins (Fig. 1) and the lumbosacral trunk by enlarged superior gluteal veins.

One patient had a compression of S2 right by a hypertrophic pyriformis muscle (pyriformis syndrome). Partial pyriformis muscle resection with decompression of S2 was performed.

Reductions of VAS pain scores at 6-month follow-up are shown in Table 2 (mean follow-up 21 months, range 7–76 months). In patients with foot drop because of massive isolated sciatic endometriosis, surgical treatment did not result in recovery of foot flexion, but surgery did not cause the functional disorders to deteriorate.

**DISCUSSION**

Even if numerous causes have been reported in the literature (6–10), the incidence of sacral radiculopathies and other pelvic neuropathies is still underestimated in the literature. Endometriosis, one of the most prevalent gynecologic disorders affecting millions of women around the world, has been reported only twice in the literature as a possible cause of sacral radiculopathy (1, 11, 12). Only a few case reports are available on acute pelvic nerve damage resulting from pelvic surgeries (3, 13). This is in absolute contradiction to the reality if one considers that neurogenic dysfunctions of the lower urinary or intestinal tract (14, 15) and chronic neural pelvic pain (3, 16, 17) resulting from pelvic surgery are common complaints in many medical offices. Probably the main reasons for omission of pelvic nerve pathologies are not only difficulty of diagnosis and treatment but also lack of awareness that such lesions may exist (2). In our series, the finding that most patients had undergone several laparoscopies previously (four on average)

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<th>Table 1: Associated surgical procedures.</th>
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<td>Procedure</td>
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<tr>
<td>Ureterolysis or ureteral resection</td>
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<td>Rectum resection (discoid or segmental)</td>
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**FIGURE 1**

Vascular entrapment covering the entire distal sacral plexus and the sciatic nerve (right side) (A) Before decompression. (B) After decompression and full exposure of the sciatic nerve.
before suspicion of sacral plexus pathology was raised once corroborates this hypothesis. A further dilemma for patients affected by pelvic nerve pathologies is that causes, diagnosis, and treatments are dispersed into completely different specialties, which usually have nothing in common.

In the present article, we report our experience with sacral plexus endometriosis. Isolated sciatic endometriosis and sacral nerve root endometriosis are apparently two different entities, with distinguishable clinical and surgical findings. Sacral nerve root endometriosis is systematically a part of a parametric deeply infiltrating endometriosis: because of close anatomic relationships, sacrucerine ligament deeply infiltrating endometriosis is a risky situation for S3 and S4 involvement, whereas infiltrations of cardinal ligaments and ovarian fossa correlate with S2 and S3 involvements. Typical neurologic symptoms are then pudendal and gluteal pains (S3, S4), S2 sciatica, and troubles of sensibility and functions of pelvic organs (e.g., bladder hyperactivity or sensitivity, troubles of continence, detrusor hypercontractility), but never problems with locomotion.

In contrast, isolated sciatic endometriosis always is located at the suprapyriform portion of the sciatic nerve (L5, S1, ±S2) and never part of parametric deeply infiltrating endometriosis. Symptoms are L5 and S1 sciatica, gluteal pain, and, sometimes, troubles of locomotion (foot drop), but never bladder dysfunctions; pudendal pain is also absent. Isolated sciatic endometriosis seems to develop and grow inside the sciatic nerve itself and to expand caudally through the greater sciatic foramen. Therefore, a translumbar approach for sciatic nerve decompression exposes the patients to the risk of incomplete surgery by missing the endopelvic part—the main part—of the lesion. Isolated sciatic endometriosis requires intraspinal neurolysis of the sciatic nerve with resection of destroyed or involved parts of the nerve, whereas in sacral nerve root endometriosis exposure or decompression of the sacral nerve roots is usually sufficient.

This article is the first report about vascular entrapment as a potential cause of pelvic nerve pain, an absolutely unknown pathology in gynecology. This pathology is on the contrary well known by neurosurgeons as a frequent etiology for facial neuralgia. The vascular entrapment of the pelvic nerves seems to be a special clinical form of a pelvic congestion syndrome that emphasizes the necessity of a neurologic examination and anamnesis in patients with chronic pelvic pain.

This study emphasizes the anatomic advantage of laparoscopy with the possibility of exposing by this method all somatic pelvic nerves. Development of videolaparoscopy and instruments enables good access to all areas in the pelvis, providing the necessary visibility of the nerves and the technical feasibility for neurosurgical procedures (18, 19). However, preoperative assessment is the most important part of management because it not only justifies indication for surgery but also orients the dissection to expose only the damaged nerves, avoiding unnecessary dissection.

## REFERENCES