Case report

Iatrogenic nerve lesion following laparoscopic surgery. A case report

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1. Introduction

Neurological deficit in the postoperative period is an under recognized potential untoward event in surgery. Peripheral neurological injuries, either sensory, motor or autonomic, during surgery may be due to pressure-compression, entrapment, traction, direct trauma, including crushing or laceration injuries, and indirect trauma secondary to hematoma formation, last but not least the intraoperative nerve injury by anaesthetic blocks and electrosurgical burns should also be mentioned.

The proportion of neurologic complications by nerve trauma is related to the types of operations performed, for instance, it is higher in vascular surgery, both abdominal and thoracic.

The overall incidence of iatrogenic nerve injuries in surgery is about 1.9%, while the risk of peroneal nerve damage due to only compression-stretching is much lower, around 0.1–0.2% [1,2].

The severity of these lesions depends on multiple factors, which include among others: age, glucose control, length of surgery, comorbidities, use of certain medications. Endothelial health in these patients may be compromised by altered glycosylation, hypertension, excessive platelet activity, reduced nitric oxide tissue availability, and excessive generation of reactive oxygen species. In addition, the immune regenerative process may be compromised when the patient has also peripheral vascular disease, and improvement in metabolic and tissue functionality is less likely. All the above factors, singularly or in connection between each other, influence the degree of the nerve lesion and its outcome by ultimately decreasing blood flow into the peripheral nerves tissues, depriving them of nutrients and oxygen supply. We focused only on a specific nerve injury due to compression-stretching related to patient’s positioning and following gynaecologic laparoscopic surgery [3,4].

The main cause of the above type of nerve lesions is the patient’s unconsciousness and decreased muscle tone, secondary to general anaesthesia in connection with a prolonged patient’s station in non-physiological postures, that in certain cases may generate nerve...
compression. Nerve branching abnormalities, pre-existing peripheral neuropathy and morphotype (patient’s obesity) may worsen the consequences of a prolonged nerve pressure [5,6].

Regarding the pathogenesis of this pathology, it is well known that nerve compression causes a demyelinating lesion focus, while a prolonged stretching along with the compression may cause axonal loss. Only sensory symptoms usually indicate a transitional impairment, while the association of sensitive and motor symptoms indicates a more severe damage [7,8].

Since iatrogenic nerve injuries from intraoperative patient’s positioning, although preventable, still occur with some frequency in spite of seemingly stringent prophylactic measures, early recognition of individuals at risk, provision of correct information to the patient, and adequate treatments are of paramount importance in order to avoid permanent nerve damage [5].

2. Case report

A 25-year-old woman undergoes a laparoscopic gynaecologic surgery for multiple bilateral dermoid cysts, after being placed in dorsal lithotomy position with both legs supported by two adjustable stirrups; the surgery lasts more than 3 h. On postoperative day one the patient reports paraesthesia of the lateral aspect of the left leg below the knee and dorsum of the left foot and a left foot motor impairment with inability to walk for the feeling of instability of the right ankle. Robotic assisted procedures and advanced laparoscopic procedures requiring patients to stay in prolonged lithotomy position (on adjustable stirrups used to support legs and allow access to the vagina) and steep Trendelenburg may increase the risk of nerve injury by compression-stretching [9]. She had no history of autoimmune disease, diabetes, neuropathies, myopathies, and malignancies. The evaluation of nutrient status did not show deficiency. At the clinic we performed the following laboratory tests: fasting blood glucose, a complete blood count, erythrocytes sedimentation rates, serum protein immunofixation electrophoresis, renal liver and thyroid function, blood levels of vitamin B12, homocysteine, folate, glycate haemoglobin, thyrotropin; all of them were normal. Ultrasound examination of the knee was performed and the results were reportedly normal [10,11].

On the fifth postoperative day she refers a light improvement in her paraesthetic symptoms of the left leg, but the motor-impairment is still present. On the 12th postoperative day an electromyography shows: reduction of amplitude of sensory action potentials (SAPs) of the left superficial peroneal nerve (calf-ankle), reduction of amplitude of left motor action potential (MAPs) of the common peroneal nerve of tibialis anterior muscle (TA) and of extensor digitorum brevis muscle (EDB) with conduction block at the head of the left fibula (Fig. 1), and motor conduction velocity reduced below the compressed nerve segment, denervation changes (axonal loss) in all peroneal muscles below the fibula head with severe reduced interference pattern (no evidence of more proximal involvement) in comparison with those registered on the right leg (Fig. 2). [12].

In conclusion the EMG result is consistent with a severe compression lesion of the left common peroneal nerve (mixed pattern of conduction block and axonal loss) at the fibular head.

The patient undergoes four months to left ankle intensive rehabilitation therapy, consisting of ankle proprioceptive exercises, passive physiotherapy, electrical stimulation for denervated muscles and transcutaneous electrical nerve stimulation (TENS) to keep under control algic-paresthesia symptoms, isometric and isotonic exercises and use of a brace AFO (Ankle Foot Orthosis) to avoid steppage gait.

Rehabilitation exercise and physical therapy had been made daily for the first three months and 3 times in a week for the following 3 months. AFO brace had been used for the first four months and then it was abandoned because the ankle dorsiflexion were sufficient also for an appropriate deambulation.

After 6 months from the occurrence of the lesion, the EMG exam showed an effective reinnervation of the muscles below the lesion, but reduced SAPs and reduced MAPs of the left superficial peroneal nerve (Fig. 3) with persistent mild reduced interference pattern of EDB muscle.

After 2 years from the lesion, the patient refers a complete resolution of the symptomatology and normal MAPs of the left superficial peroneal nerve (Fig. 4).

3. Discussion

The majority of neuropathies occurring as a result of prolonged duration of surgery are associated with faulty body position during the operation. One of the most interesting features of these disorders is that they are truly new illnesses, pathological conditions which essentially arise after surgery. Common causes of iatrogenic injuries of peripheral nerves can be divided into non operative and operative. Common non operative causes include: injection or needle injury, compression of hematoma secondary to drawing blood or through anticoagulation,
dressing, and casts of orthotic devices. Operative causes can be divided into pressure and traction injuries due to positioning during anaesthesia and direct intraoperative damage.

Primary prevention of nerve damage by compression-stretching due to patient malposition during gynaecological surgery, although not always feasible, is certainly possible in the majority of cases by strictly adhering to the following rules:

1) Choose stirrups of Allen-type, which allow the weight of patient’s legs to rest on the feet
2) Do not flex the knees and hips more than 90°, and align the knees and ankles to the contralateral shoulder with minimal abduction and external hip rotation.
3) Protect hips, lateral fibulas, posterior thighs and heels by placing pads between them and stirrups.
4) Use shoulder blocks to prevent stretching of the brachial plexus due to patient placed in extreme Trendelenburg, with arms fixed to her sides, sliding down, no forgetting that shoulder blocks should not be placed too medially in order to avoid direct pressure on the nerves that should lie on the acromial processes.
5) Change patient position even a little bit if surgery lasts more than 4 hours.
6) Avoid prolonged dorsolithotomy position especially in those patients with predisposing factors such as obesity, arthritis of hip and lower extremities, diabetes
7) Avoid exaggerated dorsolithotomy position with iperflexion of the hips, which can cause compression stretching of the femoral nerve below the inguinal ligament [13-15].

Secondary prevention of this type of compression neuropathy following gynaecological laparoscopic surgery is based on early recognition of the lesion and adequate treatment. Often the patient appears fairly normal during recovery from anaesthesia though even at this stage careful examination sometimes reveals pupillary abnormalities, failure to move one side of the body, or an unexpected delay in the restoration of clear consciousness. Such appearances are usually too equivocal to cause alarm until the next day after surgery, when persistent somnolence, bed-wetting, unexpected fever with or without focal or generalized convulsion, limb paraesthesia, motor impairments signal the onset of neurologic complications. When faced with a postoperative case of suspected neuropraxia, the first thing is to make sure that the sign encountered has occurred postoperatively for the first time [16,17].

Of all the diagnostic tools that can be employed for establishing the diagnosis of nerve damage, electromyography (EMG) is the most important and often can reveal even a subclinical damage. EMG conduction studies (VCS-VCM) provide quantitative data of the velocity and the amplitudes of individual nerves, revealing the nature of the damage (e.g. axonal rather than demyelinating disease). EMG is also able to detect the presence of muscle reinnervation phenomena distinguishing it from an apparent muscle contraction during a neurological exam.

The results of conduction studies may be difficult to interpret during the first 10 days after lesion because wallerian degeneration may not be entirely presented; however, the assessment of the amplitude of motor action potentials (MAPs) allows us to evaluate the axonal loss usually after about 12 days after injury.

The examination with needle electrode in resting muscle allows highlighting the presence of denervation potentials indicating axonal loss; these findings generally become evident after about three weeks after the injury. The presence of residual motor unit potentials (P.U.M.) in a clinically paralyzed muscle indicates that the nerve lesion is partial.

Beside the EMG with a correct identification of the muscles tested, other diagnostic techniques may be used to perfect the diagnosis, for instance, ultrasound is useful in assessing continuity and size of the nervous trunk examined, the computed tomography (CT), and the magnetic resonance imaging (MRI), may give further information on size of lesion and concomitant problems. In special cases, other means are employed such as somatic-sensitive evoked potentials, neurography, and study of reflexes. Once a diagnosis is made, hopefully as early as possible, the therapeutic objective should not be just to improve symptoms, but to normalize or optimize all factors involved in nerve healing to prevent neuropathy progression. Correction of metabolic abnormalities and personalized physiotherapy can either slow or stop the disease progression, reversing nerve damage.

Therefore a special attention should be given to low levels of nutrients that fall between the Recommended Daily Allowance and the levels that produce recognized deficiency diseases. Alpha-lipoic acid (ALA), Acetyl-L-Carnitine (ALCAR), Methylcobalamin (Methyl vitamin B12), Folic Acid (FA), and Pyridoxal -5 phosphate (PSP) have a demonstrated efficacy in achieving and maintaining control of symptoms [18-20]. Metabolic correction of the biochemical derangements is extremely safe and goes beyond symptom control to also provide improvement in nerve blood supply, contributing to restoration of nerve function and fibres density. Regarding the applicable physical therapy, it is well known that the rules on what is and what is not a suitable physiotherapeutic approach to a given patient sometimes may vary from one therapist to another, depending on the individual’s experience or expertise. Recovery from a postoperative neuropathy can be very slow and incomplete, leading to anxiety and frustration, and potentially become a cause of litigation. It is clear that except in the hands of expert practitioners, physical therapy is an important part of the rehabilitation program especially in cases in which neither the history nor the physical signs are definite enough to permit a precise diagnosis, or to encourage other form of treatment. Surgeon and anaesthetist should be trained to perform operations and positioning of patients on the operative table in order to avoid such injuries and complications. It would be useful a validate questionnaires such as a neurological improvement score and pain score at regular intervals from the recorded onset of patients symptoms and in the follow-up.

4. Conclusion

Iatrogenic nerve injuries tend to be sporadic and various in natures, they are difficult to study as a group. Almost 40% of such complications are classified as accidental and almost one quarter are due to faulty techniques or failure to adhere to routines. This type of complications may be quite devastating, especially if not recognized on time, even because the postoperative patient is more difficult to evaluate for de novo neuropathies. Such lesions are sometimes so bizarre and unexpected that diagnosis is delayed to a point when the damage is difficult or impossible to repair.

Primary prevention is of paramount importance and is realized by adhering to specific protocols in patient’s positioning. In the occurrence of the lesion a prompt diagnosis is highly recommendable and a comprehensive therapeutic plan is necessary to correctly address the specific pathology. Physicians should aim to improve all factors, vascular and metabolic, involved in the restoration of nerve function to its maximum potential. However, in order to design an effective
therapeutic regimen capable of achieving the best clinical response, specific physiotherapeutic expertise is mandatory. It means going beyond a general protocol or guideline and formulating treatment also in relation to the genetic variations of the population that sometimes produces biochemical derangements associated with specific health risks.

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