

Misdiagnosis of an Early Postoperative Upper Limb Deficit

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Abstract

Transient left upper limb deficit was diagnosed on the first postoperative day following a long surgery procedure and was assumed to have been caused by a positioning injury. Three months after the operation, multiple sclerosis was diagnosed. Importantly, this Case Report emphasizes the importance of carefully considering a differential diagnosis of perioperative nerve injury when observed in the early postoperative period. Also, the question arises as to whether the onset of multiple sclerosis was directly related to anesthesia and/or surgery and whether the early postoperative upper limb deficit was the initial sign of malignant sclerosis.

Keywords: Anesthetic techniques; General anesthesia; Complications; Neurological; Multiple sclerosis; Surgical positioning

Introduction

Left upper limb sensory and motor deficits involving the distal distribution of the radial, ulnar and median nerves in the left hand were observed shortly after a surgical procedure and were assumed to be related to a positioning injury. However, subsequently this patient was diagnosed with Multiple Sclerosis (MS) and the early postoperative neurologic deficits were possibly the first clinical signs of MS [1-3].

Case Report

A 35-year old, 164-cm, 57-kg woman underwent rectosigmoidectomy, hysterectomy and ovariectomy for deeply infiltrating endometriosis. Her preoperative medical examination was totally unremarkable. Premedication consisted of oral hydroxyzine 100 mg, ~120 min prior to induction of General Anesthesia (GA) with propofol. Tracheal intubation was facilitated using atracrium and GA was maintained using desflurane in combination with sufentanil. During the operation, the patient was in the supine position on a vacuum mattress (Vacuform, Schmidt Manufacture, Grabsen, Germany) filled with pellets which mold to the contour of the patient. The right arm was positioned along the side of the patient's body. The left arm was abduction <80° and shoulder braces were not used. The patient's legs were placed in stirrups and elevated ~15°. Standard padding was provided at all pressure points.

The surgical procedure lasted 540 min and required frequent 'head-up' and 'head-down' repositioning. The patient's trachea remained intubated for 30 min in the Postoperative Care Unit (PACU). The patient received multi-modal analgesia with a combination of paracetamol, ketoprofen and IV patient-controlled morphine administration. After recovering for two hours in the PACU, the patient was discharged to the postsurgical ward. The following morning, the patient reported an inability to move her left hand, including all five fingers, as well as numbness and tingling of the palmar surface of the hand. The patient was completely oriented, with intact speech and cognitive functioning. Neurological examination disclosed a loss of sensation to light touch and pin-prick at the palmar surface of all five fingers of the left hand. The plantar flexion reflex was intact bilaterally and anal sphincter tone was reportedly normal. Both knee and ankle joint reflexes and upper extremity reflexes were intact. Bladder function could not be investigated since a catheter had been placed in the bladder at the time of surgery.

Due to the lengthy duration of the surgical procedure with frequent head-up and head-down repositioning, an intraoperative positioning injury was presumed to have caused this neurologic deficit [4]. According to the neurologist, no immediate electromyography was required. The patient's sensory and motor function had completely returned to the hand five days after surgery. However, five weeks later the patient began complaining about the recent onset of dysesthesias, numbness, tingling and burning sensations irregularly distributed on her left hand and forearm. A repeat neurological assessment failed to ascertain any abnormalities and electromyographic testing was normal. Gabapentin and amitriptyline were prescribed and the patient's neurologic symptoms disappeared within three weeks. Approximately six weeks later, the patient again contacted the neurologist complaining about the reoccurrence of upper left limb symptoms, as well as a new area of hyperesthesia on the inner side of the left thigh. Asthenia, diffuse myalgias and arthralgias were present. The patient was hospitalized in a neurology unit. She confirmed that she had never experienced any symptoms or sensations of neurologic dysfunction prior to the operation. Decreased reflexes were recorded in the left knee and ankle joints, and in the left upper limb compared with all joint reflexes recorded on the right side. The remainder of her neurologic examination was completely normal. An urodynamic assessment was also within normal limits.

Blood screening for infectious, systemic, inflammatory, autoimmune and collagen vascular diseases was negative [5]. Cerebrospinal fluid examination showed normal albumin levels and no cells [5]. Immunofixation detected one IgG-kappa monoclonal band and two IgG-lambda monoclonal bands in the light chains which were

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not detected in serum [5]. Similarly, isoelectric focusing detected three oligoclonal IgG bands, which were not detected in serum [5].

However, cerebral, cervical and thoraco-lumbar magnetic resonance imaging (MRI) showed several left posterior ovoid lesions extending over more than two segments, at C2, C3-C4, C6, T1-T2-T3-T4, and T8-T9 [6]. Medulla morphology was unaltered. Hyperintense signals from the white matter of the supratentorial region of the brain were observed. An inflammatory disease of the Central Nervous System (CNS) was diagnosed [5]. All the neurological symptoms spontaneously resolved in three weeks. Five months later, the neurologic symptoms recurred and continued to worsen, including tingling, heaviness and fatigability in the left upper limb, tingling and numbness in the fingertips of the right hand, and insomnia. Several episodes of nagging pain were reported in the right eye. The MRI was repeated and detected eight additional periventricular lesions of increased signal intensity and one subcortical lesion in the right frontal lobe [6]. Post contrast images revealed annular enhancement of the left-posterior periventricular lesion. MS was diagnosed [5-7].

Discussion

MS was not part of the initial differential diagnosis when the patient developed early relapsing postoperative peripheral deficit of the left upper limb because it was assumed to be a result of an intraoperative positioning injury [4]. Indeed, according to the American Society of Anesthesiologists Closed Claims Project database lists, nerve injury is the second most common class of intraoperative injury [4]. Long-duration surgery associated with frequent head-up and head-down repositioning can contribute to positioning injuries [4]. The pathological mechanisms underlying nerve injuries include stretching, compression, generalized ischemia, and metabolic derangements [4]. The brachial plexus lies in close proximity to the first rib, clavicle and humerus and is predisposed to compression against these fixed structures [4]. These injuries can be asymptomatic for several days after surgery [4]. Under such circumstances neither an immediate electrophysiologic study nor other diagnostic procedures were deemed necessary. Importantly, the first neurologic signs in this case resolved within five days.

MS is a rare autoimmune demyelinating disorder of the CNS [7]. There is no convincing evidence that non-traumatic stressful events or even physical trauma alter the progression of MS [7]. Although exacerbations and relapses of MS have been reported following surgery and/or anesthesia, [1-3,7,8] most of the reports of postoperative MS exacerbation are anecdotal or consistent with baseline and exacerbation rates occurring in non-surgical "at risk" patient populations [7]. In a large prospective study, Confavreux et al. reported that epidural analgesia did not increase the risk of neurologic deficits in parturients with MS [9]. In another study, 139 patients with CNS disorders including MS (75% had active neurologic symptoms), underwent neuraxial anesthesia or analgesia and there was no evidence of new or worsening postoperative neurologic deficits [2]. On the other hand, GA has never been reported to be associated with the onset of MS.

Consequently, recommendations are that anesthetic care for patients with MS should mainly focus on identifying preoperative motor deficits and respiratory impairments because they are more likely to alter the patient's postoperative rehabilitation [1,10]. Succinylcholine should probably be avoided because it may lead to hyperkalemia in patients with motor deficits [1,10]. Lower concentrations of local

anesthetics are recommended due to the susceptibility of demyelinated neurons, to local anesthetic neurotoxicity [1,10]. The response to nondepolarizing muscle relaxants has also been reported to be highly variable [1,10]. In light of the frequency of autonomic nervous system dysfunction, use of invasive hemodynamic monitoring may reduce the risk of intraoperative hemodynamic instability [1,10]. Hyperthermia can also aggravate pre-existing motor block and should be aggressively prevented [7,10].

In retrospect, the early relapse of the upper left limb symptoms in the same distribution would not have been expected if the nerve injury was related to the operation [4,7]. Furthermore, clinical features of MS could have been recorded earlier in that patient [7]. MS is a relapsing and remitting degenerative disease of the CNS [7]. Women are more commonly affected than men [7]. In most patients it begins at about 30 years of age [7].

Finally, it is still unclear whether the early postoperative upper limb deficit was related to a positioning injury which preceded the onset of MS or was the first sign of MS [1-3,7,10]. Whether GA contributed to the onset of MS symptoms on the first postoperative day in this patient, or whether it was merely coincidental with the onset of the patient's MS remains also unclear [1-3,7,10]. Of interest, ovarian hormonal changes associated with pregnancy have been reported to have caused exacerbations of MS up to three months after delivery [9], and major ovarian hormonal imbalance has been reported in women with endometriosis [11]. Analogous to the post-partum situation, one might speculate that the hormonal changes following the removal of endometrial tissue caused the onset of MS symptoms or a relapse of subclinical MS, in this patient [9,11]. Importantly, this report highlights an important challenge in the clinical approach to the differential diagnosis of postoperative peripheral neuropathic symptoms [4]. Simply assuming that the onset of a neurological deficit in the early postoperative period resulted from an intraoperative injury can result in a serious misdiagnosis with potential medical-legal implications for the anesthesiologist, surgeon and surgical facility.

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