CASE REPORT

Brachial Plexus Injury After Right Hepatectomy

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Abstract Iatrogenic nerve palsies due to faulty positioning on the operating table are commonly seen over the elbow and popliteal fossa. However, injury to the brachial plexus (BP) is rarely encountered and is a recently reported phenomenon due to the increasing number of complex surgeries including hepatobiliary surgical procedures. Brachial plexus injury (BPI) needs to be recognized as a potential complication of prolonged abdominal surgery. The present case report highlights the potential for BPI and its early recognition, management, and prevention in complex prolonged abdominal surgical procedures. BPI has been described in a 64-year-old patient following a prolonged right hepatectomy.

Keywords Brachial plexus injury · Hepatectomy · Compression · Stretch

Introduction

Iatrogenic peripheral nerve injuries are a well-recognized occurrence in clinical practice and are common cause of post-procedural morbidity. Of all peripheral nerve injuries, brachial plexopathies have been

described more concretely. The incidence of brachial plexus injury (BPI) is 0.2% of all patients receiving general anesthesia [1]. Iatrogenic BPI accounts for 5 to 10% of all and has been mostly described after cardiac, liver, breast, and orthopedic surgeries [2–4]. The mechanism of injury in the majority of cases is due to stretching or compression of the nerve tissue and treatment is mainly supportive. Prevention is the key in minimizing this form of patient morbidity. It is the surgeon's responsibility to mitigate this risk by taking care of proper positioning techniques and communicating closely with the anesthesia team wherever applicable. We present in this paper our experience of BPI in a patient who underwent right hepatectomy. The aim is to highlight the relevance of positioning of the patient and monitoring during surgery.

Case Report

A right hepatectomy for hepatocellular carcinoma was done in a 64-year-old male. During the procedure, the patient was placed with arms abducted to about 90°. The Thompson retractors were used for retraction of costal margins. The surgery was prolonged due to dense adhesions of the tumor with surrounding adjacent structures and adhesions due to previous surgical procedures. Trendelenburg position and right lateral tilts were used during hepatic parenchymal transection and porta dissection, respectively. The surgery lasted for a total of 6 h. On the first post-operative day, the patient complained of tingling and numbness along the outer aspect of the right arm and, initially, this was thought to be due to the prolonged use of a non-invasive blood pressure cuff. The next day, the patient had difficulty abducting the

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right shoulder and dorsiflexing the right wrist. There was numbness along the thumb and index finger and the radial border of the forearm. Biceps flexion was absent with weakness of the triceps extension. The patient was assessed by a neurologist, who confirmed the clinical diagnosis of upper brachial plexopathy. Magnetic resonance imaging (MRI) scan of the brachial plexus (BP) and spine showed osteophytes of the C3/4 to C6/7 levels, patchy areas of T2 hyperintensity in the right trapezius muscle, the right serratus anterior muscle, and the right coracobrachialis muscle and there was no cervical rib. There were edematous changes in the right axilla, likely to be secondary to traction injury at the Erb's point. The vertebrae, spine and the proximal nerve roots and trunks of BP were normal. Nerve conduction studies were not done. The patient was started on a course of physiotherapy with active and passive range of motion. He was also given steroids (prednisolone 50 mg/day for 5 days and tapered within following 5 days) along with non-steroidal analgesics (acetaminophen 325 mg three times a day) for initial 2 weeks. Oral gabapentin (75 mg twice a day) was given for a total of 4 weeks. A protective broad arm sling was used to protect the flail limb. After 6 weeks of starting this regime, the nerve palsy recovered. The patient started dorsiflexing the right wrist and had lesser difficulty in abducting the right shoulder with grade 4/5 power and by the 10th week, he was back to using the limb normally. There was no sensory loss.

Discussion

Iatrogenic peripheral nerve injuries and specifically brachial plexopathies are a well-recognized occurrence in clinical practice and possibility in most operative or procedural environments. Since the majority of them occur in the perioperative period, they are mostly attributed to surgeons and anesthesiologists [5]. Myelinated axons of the peripheral nerves are very limited in their ability to withstand injury. Iatrogenic nerve injuries generally occur when the nerve is acutely subjected to stretch, compression, or ischemia leading to rupture of endoneurial capillaries with small hematoma formation, endoneurial edema, and ischemic injury. Spectrum of severity ranges from less severe demyelinating injuries to more severe, axon loss with different variable levels of nerve conduction problems ranging from conduction slowing to failure. Iatrogenic nerve injury usually presents with abrupt onset of symptoms. From a physical

exam standpoint, it is difficult to differentiate axonal loss from demyelinating conduction block immediately after onset of symptoms. Both injury types result in symptoms of motor and sensory loss in the area of distribution of damaged nerve. In terms of recovery, demyelinating injuries recover earlier than axonal loss. In the most severe cases of nerve disruption, axonal regeneration may be impossible [6, 7].

Of all iatrogenic peripheral nerve injuries, damage to the BP is the most common and will be the focus of the present case report. Anatomically, the BP consists of five nerve roots (C5–T1). They combine to form three trunks (upper, middle, and lower), followed by six divisions (three anterior and three posterior), three cords (lateral, medial, and posterior), and subsequently terminal nerves (axillary, musculocutaneous, median, ulnar, and radial). Clinically, BP distribution is categorized into supraclavicular and infraclavicular components, where the former refers to roots and trunks and the latter to cords and terminal nerves [6, 8].

BP is particularly prone to injury due to its superficial location, as well as its relative immobilization proximally by the vertebrae and prevertebral fascia and distally by the axillary fascia. Various mechanisms of injury to BP have been described in the literature most common being the direct compression, stretch, or repetitive trauma. The degree of injury may vary with the severity and duration of compression [9]. Comorbidities like diabetes, anatomical variations, positioning of the patient, use of automated blood pressure cuffs, surgical retraction, and intraoperative hypotension or vascular compromise in the limb have all been associated with BPI [10]. Compression between the ribs and the clavicle at the level of scalene muscles and stretching across the humeral head has been implicated [7]. Several position-related factors predispose patients to BPI, including arm abduction of 90° or greater, extension of the upper extremity, external rotation of the upper extremity, contralateral head rotation, and sometimes the use of steep Trendelenburg position. The former three factors are thought to result in injury by the humeral head acting as a fulcrum across which the plexus stretches. Contralateral head rotation stretches the plexus at the level of the vertebrae and adjacent fascia. Trendelenburg position results in injury by caudal force on the humeral head by either shoulder or wrist restraints resulting in downward stretch on the plexus [7–9]. Injuries to the supraclavicular component of the BP are more common than infraclavicular. These typically present with motor and sensory loss with or without paresthesias. The upper trunk is the most commonly affected rather than all the three trunks. When the latter is the

case, mid and lower trunk injuries resolve quickly, whereas involvement of the upper trunk persists. Sometimes along with compression of nerves, there can compression of the right subclavian vessels at the level of the clavicle leading to a combined neurovascular compromise with impending compartment syndrome [8, 9]. BPI during prolonged liver surgeries has been attributed to prolonged malpositioning of the arm or prolonged use of the Thompson retractors [11, 12]. BPI has been reported in liver surgeries with an incidence of 5.8% [12]. In our patient, there was a unilateral involvement of the right upper limb. The Thompson retractor was used for better exposure which may have caused excessive pressure on the right BP trunks and divisions between the first rib and middle part of the clavicle.

The most crucial component of diagnosing BP as well as other peripheral nerve injuries is physician awareness of these entities. Diagnosis is usually delayed because of potent analgesia that is common in post-procedural care, incisional pain, and lack of patient awareness. A detailed history and physical examination is imperative for diagnosis. If a neurologic deficit is discovered, then neurologic consultation is indicated. Electromyography serves as an important adjunct in evaluation of these injuries and can be helpful in evaluating whether an injury is chronic or acute [13].

Prevention is the primary goal with these injuries. Several strategies are followed to minimize risk: reduction in arm abduction, extension, and external rotation; frequent limb repositioning; maintaining the head in neutral position; and proper shoulder padding in the Trendelenburg position [7, 13]. Using these recommendations, one group was able to reduce the incidence of BPI to 0.02% over 15,000 procedures [14]. With all these measures, BPI can be avoided in most of the cases.

If injury occurs despite best practices, management of peripheral nerve injuries, including brachial plexopathy, is largely supportive: an initial period of conservative therapy for 3 weeks, except where vascular issues or compartment syndrome is suspected, or there is suspicion that the nerve itself is inadvertently cut. After 3 weeks, electrodiagnostics (Edx) should be performed. An Edx performed prior to 3 weeks yields very poor information since Wallerian degeneration is not complete and information is not reliable. In case of Sunderland type I injury, nothing further needs to be done as recovery is usually spontaneous and full [13, 15]. If axonotmesis is suspected, it is best to refer the patient to a hand surgeon dealing with BPI for further management. Early referral can potentially result in timely treatment and better recovery.

Conclusion

Injury to the BP and peripheral nerves can be a potential complication of prolonged abdominal surgeries including hepatobiliary procedures and is associated with significant morbidity. Its awareness, preventive measures, early recognition, and timely remedial measures can prevent the associated short- and long-term morbidity.

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Contribution Details

	Author 1	Author 2
Concepts	✓	✓
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Definition of intellectual content	✓	✓
Literature search	✓	
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Conflict of Interest The authors declare that they have no conflict of interest.

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