J Anesth (2008) 22:308–311
DOI 10.1007/s00540-008-0636-0

Journal of
Anesthesia

Bilateral brachial plexus injury after liver transplantation

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Abstract
We report a case of bilateral brachial plexus injury following living-donor liver transplantation. A 35-year-old man with hepatitis C cirrhosis underwent liver transplantation under general anesthesia, performed in the supine position with 90° arm abduction. The surgery lasted for 14 h, and the anesthesia for 16 h. On postoperative day 1, it was noticed that he had brachial plexus injuries. We investigated the cause of the nerve injuries, in particular, the possible involvement of stretching, compression, or nerve ischemia, which can often result from excessive abduction, the use of shoulder braces, compression by the poles used in the operating theater or compression caused by surgeons leaning on the patient, or serious general status (e.g., hypotension or hypoxemia). Our findings were inconclusive, but we postulated that 90° abduction of the arms per se may have resulted in excessive stretching of the brachial nerves, causing his injuries.

Key words Brachial plexus injury · Living-donor liver transplantation · General anesthesia

Introduction

Peripheral nerve injury is a well-known complication associated with anesthesia. According to a 1999 analysis from the American Society of Anesthesiologists (ASA) closed claims project database [1], there were 670 claims for nerve damage (16% of all 4183 claims), and these were the second most common claims, with death being the most common (32%). In these nerve injuries, 137 claims (20%) were brachial plexus injuries, 12% of which were bilateral [1,2].

Brachial plexus injuries associated with anesthesia are likely to occur in cardiothoracic procedures requiring median sternotomy [3–5]. Also, brachial plexus injuries are usually attributed to stretching or compression of the plexus in relation to the use of shoulder braces for head-down position [3,4] or excessive abduction of the arms during anesthesia.

We report a bilateral brachial plexus injury, which occurred despite the provision of definitive preventive care, in a liver transplantation recipient.

Case report

A 35-year-old man (height, 175 cm; weight, 81 kg) with hepatic insufficiency due to hepatitis C cirrhosis underwent a living-donor liver transplantation. Preoperatively, he was in a state of decompensated cirrhosis with intractable ascites, although he had no symptoms of hepatic encephalopathy or peripheral neurological abnormalities. Laboratory studies demonstrated a low platelet count (platelets, $3.8 \times 10^4 \cdot \mu l^{-1}$), hypoalbuminemia (serum albumin, $2.8\ g \cdot dl^{-1}$), coagulopathy (prothrombin time, INR 1.77), hyperbilirubinemia (total bilirubin, $5.9\ mg \cdot dl^{-1}$), and hyperammonemia (NH_3, $80\ \mu g \cdot dl^{-1}$).

Anesthesia was induced and maintained with propofol, fentanyl, isoflurane, and vecuronium bromide. The patient was placed in the supine position with his arms abducted to 90° on soft-pad-covered arm boards and fixed loosely with cloth bands. Cannulations in the bilateral upper limbs, right internal jugular vein, and left radial artery were performed uneventfully. When the poles for the cradle and rib retractor were placed at the sides of the operating table, the anesthesiologists and nurses confirmed that the poles were not in contact with the patient's shoulders or arms.

The surgery lasted for 14 h, and anesthesia for 16 h. Hepatic flow occlusion (ahepatic period) lasted for 3 h and 40 min. Venovenous bypass was not placed. Intraoperative blood loss was 7450 ml, urine output 1200 ml, and gastric drainage 600 ml (total output, 9250 ml).

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Received: November 8, 2006 / Accepted: April 10, 2008

Intraoperative fluid infusion consisted of 10800 ml of crystalloid solution, 4500 ml of colloidal solution, and 3720 ml of blood including packed red cells, 2240 ml; fresh-frozen plasma, 1280 ml; and platelet concentrate, 200 ml (total infusion, 19020 ml). The patient remained hemodynamically stable throughout the surgery. The anesthesiologists and nurses periodically checked the patient's arm positions, noting that the arms were not extended beyond 90°. At the end of the surgery, we noticed that the soles of the patient's feet protruded from the operating table. The patient was not in the Fowler or Trendelenburg position during the surgery. As the soles of his feet had been placed just at the edge of the bed before the incision was made, it was apparent that his body had moved about 5 cm caudally. Postoperatively, the patient was transferred to the intensive care unit, under sedation and mechanical ventilation.

The following day, after extubation, the patient complained of numbness and weakness in his right hand and forearm. Hand grasp and release, and dorsal and palmar flexion of the wrist and elbow were difficult. Sensory impairments included anesthesia on the ulnar side (C8-T1) and hypoesthesia on the radial side (C6-C7) of the right forearm, accompanied by pain. His left hand and forearm were injured as well (Fig. 1a). An orthopedist examined him and diagnosed bilateral brachial plexus injury.

On postoperative day 5, the patient was discharged from the intensive care unit, although the neurological disorders showed no improvement and the pain had increased. Treatment for the pain was restricted due to liver dysfunction and immunosuppressive therapy. Acetaminophen (150 mg, as needed) and mexiletine hydrochloride (300 mg·day^{-1}) did not give sufficient

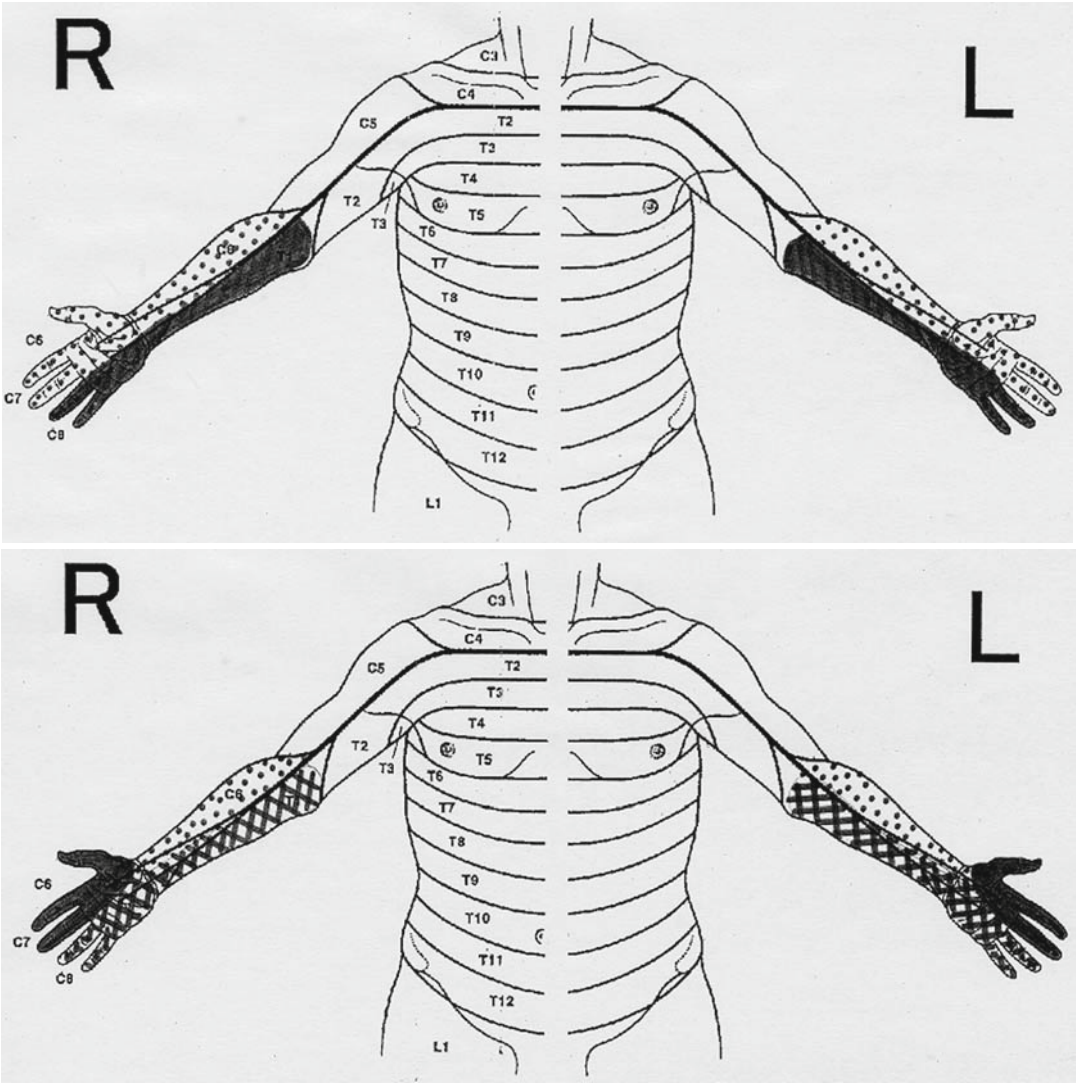

Fig. 1. **a** Areas of sensory impairment 2 days after the operation. **b** Areas of sensory impairment 3 months after the operation. *Dots*, Hypoesthesia; *black areas*, anesthesia; *crossed lines*, allodynia and hyperalgesia

pain relief, nor did linearly polarized near-infrared irradiation or stellate ganglion block with lidocaine. Amitriptyline hydrochloride (20 mg·day^{-1}) showed the adverse effects of liver dysfunction and drowsiness. Physical therapies, such as massage, hot packs, and acupuncture, were thus the mainstay for pain relief.

Three months after the operation, the muscles of the bilateral forearm had atrophied and the patient was unable to pick up objects using his first and second fingers. Paresthesia persisted and allodynia and hyperalgesia appeared in some areas of the forearms (Fig. 1b). The patient complained of cold sensation and occasional unbearable pain, and his hands and forearms looked pale. Only codeine phosphate (80–160 mg·day^{-1}) alleviated the pain, but this treatment had to be terminated because his serum bilirubin level tended to be elevated at this time.

Neurological examination performed 5 months postoperatively showed normal nerve conduction velocities in the ulnar nerves, but it was impracticable to carry out such examination on the bilateral median nerves because of allodynia. Electromyography in the bilateral first dorsal interosseous muscles and adductor pollicis muscles demonstrated a neuropathic pattern of high amplitude, long duration, and poor recruitment. Head and neck computed tomography and magnetic resonance imaging showed no abnormalities. In consideration of the areas of the injuries, central neurological and spinal disorders or more peripheral nerve damage (carpal-tunnel syndrome or cubital tunnel syndrome) were unlikely. The ulnar and median nerve lesions, led to the final diagnosis of bilateral median cord injury of the brachial plexus. The patient left the hospital 6 months after the surgery.

Ten months postoperatively, the patient's pain was slightly relieved. He could extend the fingers of both hands fairly well, while the areas of hypoesthesia in the left forearm and hyperalgesia and allodynia in the right forearm were reduced. Eighteen months postoperatively, although the pain had decreased considerably, paresthesia in the hands and forearms, and disturbance of skilled motor action in the fingers remained bilaterally as aftereffects.

Discussion

In 83 brachial plexus injuries reported between 1990 and 1995, 8 cases were obviously related to patient positioning intraoperatively, 4 to the use of shoulder braces in the head-down position, 3 to improper positioning of the arm, and 1 to sustained neck extension [1].

Several reports have indicated a relatively high incidence of neurological complications during liver transplantations; brachial plexus injury was seen in 5.8% of cases (7 of 120 patients) [6], and neuromuscular complications in 3.8% (24 of 627 patients including 6 cases of brachial plexus neuropathies) [7], in two separate reports of patients undergoing liver transplantation. It was suggested that these neuropathies were probably related to prolonged anesthesia, invasive central venous monitoring catheters, and surgical procedures such as venovenous bypass. Another case report noted brachial plexus injury caused by compression of the plexus between the first rib and clavicle, resulting from rib cage retraction for surgical exposure [8].

The cause of our patient's injuries was difficult to identify. We did not use either shoulder braces or venovenous-bypass, and all cannulations were smooth. No significant hypotension, ischemia, or hypoxemia occurred intraoperatively. Direct stresses on the nerves caused by the placement of poles or by surgeons, who sometimes lean over the arms of patients, were unlikely, under our vigilance.

Excessive abduction of the arms could be the most probable cause of the neuropathies in our patient. With the arm abducted, the brachial plexus would not only be stretched with the caput humeri as a fulcrum, but it would be compressed by tension of the pectoralis minor muscle or by the coracoid process [9]. This stretching and compression occurs in the cord portion of the brachial plexus, and would be compatible with the brachial plexus injury in our patient. It is generally recommended that arm abduction in the supine position should not exceed 90° [3,10]. In our patient, the only unusual incident was that the patient's body had moved 5 cm caudally by the end of the surgery. We examined whether caudal displacement of the body by 5 cm would have caused further abduction of the arms, in a similar-sized volunteer on the same operating table, using the same arm boards and cloth bands for fixing. We found that the caudal displacement was likely to have caused greater abduction, but this was so slight as to be hardly noticeable.

We also questioned whether 90° abduction per se could result in brachial plexus injury, as there are some people who feel tingling in their fingertips if they lie supine with their arms abducted to 90°. This means that 90° abduction, which has so far been considered safe, would already represent excessive stretching of the brachial plexus in some patients. In the supine position, the arms may be pulled posteriorly (toward the ground) by gravity, causing additional stretch of the brachial plexus.

Immobilization of a patient for many hours may be an additional contributory factor. Thus, slight pressure or stretching, which normally does not have adverse effects, may cause nerve dysfunction during prolonged surgeries.

These factors led us to suspect that the bilateral brachial plexus injury in our patient resulted from slightly excessive stretching of the nerves for a long time. We would like to emphasize that 90° abduction of the arms may cause excessive stretching of the brachial nerves in some patients undergoing prolonged surgery. Before the induction of anesthesia, anesthesiologists should confirm with the patient that there is no discomfort with abduction, to prevent excessive stretching of the brachial plexus. And the staff in the operating room should know the potential for neuropathy and pay careful attention to patient position intraoperatively.

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