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Neuropraxia: An Underappreciated Morbidity of Liver Transplantation

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Abstract

Background: Peripheral nerve injuries can be devastating complications of surgery, potentially resulting in severe functional disability and decreased quality of life. Long surgeries with considerable tissue manipulation, for example, liver transplantation, may present increased risk; however, neuropraxia in transplantation has not been well investigated.

Materials and methods: This is a retrospective study of all adult patients undergoing liver transplantation at a large academic center between January 2013 and December 2015. Descriptive analyses, logistic regressions, and forward selection procedures were used to determine the odds of developing neuropraxia and associated factors.

Results: Of the 283 liver recipients, the mean age was 55.8 y, 35.1% were female, 65.6% were Caucasian, 8.9% were African American, 16.7% were Hispanic, and mean model for end-stage liver disease sodium score at transplant was 24.2 ± 10.9 . The underlying etiology was alcohol (26.2%), hepatitis C (34.8%), nonalcoholic steatohepatitis (13.1%), and other (14.2%). The incidence of neuropraxia after liver transplantation was 8.3% ($n = 25$), with 60% ($n = 16$) upper extremities, 82% left sided, and 84% male. There was no difference in age, race, body mass index, hypertension, diabetes, hyperlipidemia, or smoking in those with neuropraxia versus those without. In multivariate analysis, neuropraxia was significantly associated with male gender, lower model for end-stage liver disease score, and longer duration of surgery ($P < 0.05$). Symptoms lasted median 5 d, with a wide range up to 187 d. Neuropraxia-specific treatment (physical therapy or medications) was required in 32% ($n = 9$).

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Disclosure

The authors report no proprietary or commercial interest in any product mentioned or concept discussed in this article.

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Conclusions: Peripheral nerve injuries are an unexplored complication of liver transplantation. Although transient, a high number (8.2%) of patients developed neuropraxia, negatively affecting their ability for recovery. Exploration of mechanisms for minimizing risk and intraoperative detection and prevention should be considered to mitigate this complication.

Keywords

Liver transplantation; Neuropraxia; Postoperative complications; Peripheral nerve

Introduction

Peripheral nerve injuries are common and potentially devastating complications of anesthesia and surgery. Indeed, nerve injury is the second most common litigation claim in the American Society of Anesthesiologists' Closed Claims Database.¹ The morbidity of peripheral nerve injury can range from transient numbness and tingling to severe permanent disability. Most injuries are thought to be the result of stretching, compression, ischemia, and/or patient malpositioning for extended periods during surgery.^{1,2} Operative factors unique to major liver surgery may place these patients at particularly increased risk of peripheral nerve injury, such as the routine use of large bore venous catheters, veno-veno bypass, lengthy operative times, potential for significant intraoperative hypotension, and retractors that cause the cephalad displacement of the rib cage toward the clavicle thus compressing the brachial plexus.³ Although anecdotally described, the incidence of peripheral nerve injury in liver transplantation has not been well investigated. Perhaps, the largest series is in the living donor liver population, the Adult-to-Adult Living Donor Liver Transplantation or A2ALL cohort, in which neuropraxia is reported in 3%–4% of living donors, taking up to 3 y to resolve, and resulting in permanent work disability in some.^{4,5} Studies from the United Kingdom and Spain from 20 to 30 y ago suggest that perioperative neuro-muscular injuries occur in a similar number of liver transplant recipients.^{6,7} However, the incidence of neuropraxia in recipients in the current era of liver transplantation is unknown. Furthermore, information regarding risk factors associated with the development of peripheral nerve injury in liver transplantation is lacking. In this study, we sought to assess the incidence of neuropraxia in a contemporary series of liver transplant recipients and to determine patient-level characteristics associated with injury. These findings may allow for the preoperative identification of patients at high risk of developing neuropraxia and for targeted interventions.

Materials and methods

Study population

This is a retrospective review of 283 adult patients undergoing liver transplantation between January 2013 and December 2015 at the Northwestern Medicine Organ Transplantation Center. The Northwestern Institutional Review Board reviewed and approved this study with a waiver of informed consent (STU00105002). Patients were excluded if they died during the hospitalization for transplantation or before their first clinic follow-up appointment. Patient characteristics were ascertained from the electronic medical record (age, sex, race, body mass index [BMI], coronary artery disease, hypertension, diabetes, hyperlipidemia,

smoking history, dialysis within 6 mo before transplant, indication for liver transplantation, calculated model for end-stage liver disease [MELD] score, and hepatocellular carcinoma status). Operative and postoperative characteristics included living *versus* deceased donor, liver alone *versus* simultaneous liver-kidney transplant, duration of surgery (total time on the operating table), number of packed red blood cells transfused intra-operatively, length of hospitalization, and discharge disposition (home, inpatient rehabilitation, or long-term care/nursing facility). Patient intraoperative positioning practices were uniform among all surgeons, involving supine positioning and placement of both arms on armboards at <90° abduction.

All patients were evaluated for pretransplant neuropraxia by determination of (1) any prior history of neuropathy diagnosis, (2) presence of self-reported neurologic symptoms, and (3) neurologic examination during Hepatology and Transplant Surgery pretransplant evaluation. Postoperative neuropraxia was classified as numbness, weakness, tingling, and/or burning pain localized to an extremity. Patients were considered to have neuropraxia if they reported the development of these symptoms during their postoperative hospitalization or in outpatient records up to 1 mo postoperatively. Electronic medical records were reviewed by a clinician, and a second clinician independently reviewed all charts and confirmed all diagnoses. Equivocal diagnoses were adjudicated by a third physician. For patients who developed neuropraxia, duration was determined by a review of all electronic medical records for 1 y posttransplant. Specifically, postliver transplantation, patients follow up at least weekly with a transplant surgeon for the first month and at least monthly with a transplant hepatologist for the first 6 mo. Neuropraxia-specific therapy was defined as initiation of gabapentin or amitriptyline for symptoms or targeted physical or occupational therapy.

Statistical methods

Demographic, clinical, and surgical characteristics were compared between individuals with neuropraxia and those without using t-tests for pseudonormally distributed continuous variables and chi-squared tests for categorical variables. Logistic regression was used to calculate the unadjusted odds of developing neuropraxia for each factor individually. Using a forward selection procedure with an alpha threshold of 0.05, factors were selected for the multivariable model, and adjusted odds ratios were calculated. All analyses were performed using SAS version 9.4 (SAS Institute Inc, Cary, NC) and R version 3.3.2 (R Core Team, Boston, MA).

Results

Study population

Between January 2013 and December 2015, 283 adult patients underwent liver transplantation. Of these, 43 (15.2%) underwent simultaneous liver and kidney transplantation, and 40 (14.1%) underwent a living donor liver transplant. The mean age was 55.8 y, 35.1% were female, 65.6% were Caucasian, 8.9% were African American, and 16.7% were Hispanic (Table). The mean calculated model for end-stage liver disease sodium (MELD-Na) at transplantation was 24.2 ± 10.9 . The underlying etiology of liver disease was

hepatitis C (34.8%), alcohol (26.2%), nonalcoholic steatohepatitis (13.1%), autoimmune (11.7%), and other (14.2%; Table).

No patients were determined to have neuropraxia before transplantation. The incidence of neuropraxia within 1-mo posttransplantation was 8.3% ($n = 25$). Comparing patients who developed neuropraxia *versus* those who did not, there was no significant difference in age, race, and BMI or in incidence of comorbid conditions including hypertension, diabetes, hyperlipidemia, coronary artery disease, and smoking history. Patients with neuropraxia were more likely to be male (84% *versus* 62.4%; $P = 0.03$), to have hepatocellular carcinoma (56% *versus* 36.4%; $P = 0.05$), and to have a lower calculated MELD-Na (20.0 ± 8.1 *versus* 24.2 ± 9.1 ; $P = 0.02$; Table). The mean duration of surgery was longer in those who developed neuropraxia ($10.5 \text{ h} \pm 2.5$ *versus* 9.3 ± 2.2 ; $P = 0.02$). There was no difference in length of hospital stay or in discharge disposition (home *versus* rehabilitation *versus* nursing facility).

Risk factors for neuropraxia

In univariate analysis, a longer duration of surgery was associated with an increased incidence of neuropraxia (odds ratio [OR]: 1.23, 95% confidence interval [CI]: 1.04–1.46; $P = 0.017$; Fig. 1). Female gender (OR: 0.32, 95% CI: 0.10–0.95; $P = 0.04$) and MELD-Na (OR: 0.94, 95% CI: 0.089–0.99; $P = 0.027$) were negatively associated with neuropraxia (Fig. 1).

In the forward selected model, a longer duration of surgery (adjusted OR [aOR]: 1.27, 95% CI: 1.05–1.55; $P = 0.014$) was associated with neuropraxia, whereas female gender (aOR 0.26, 95% CI: 0.07–0.89; $P = 0.032$) and MELD-Na (aOR 0.94, 95% CI: 0.88–0.99; $P = 0.026$) remained negatively associated (Fig. 2).

Effects of neuropraxia

In the patients who developed neuropraxia, the upper extremities were affected in 60% ($n = 16$). A unilateral distribution was found in 69% ($n = 11$) of these patients, with the majority being left sided (82%). The lower extremities alone were affected in 16% ($n = 4$), whereas upper and lower extremity symptoms were exhibited in 24% ($n = 6$). Symptoms lasted a median of 5 d, but with a wide range of up to 187 d. Neurology consultation was performed for 24% ($n = 6$), and two severe cases were further evaluated by electromyography (EMG). These two patients had high symptom burden with severe impairment of their hands for up to 60 d postoperatively, with EMG showing multiple severe neuropathies including left brachial plexopathy. Neuropraxia-specific treatment was implemented for 32% of patients, including the use of gabapentin and targeted physical therapy.

Discussion

Postoperative peripheral nerve injuries can handicap a patient's ability for recovery after transplantation, potentially resulting in severe functional disability and tedious litigation claims. This is the first study to characterize the incidence, risk factors, and outcomes of peripheral nerve injuries after liver transplantation. Our incidence of 8.3% in liver recipients is higher than the previously reported 3%–4% in living liver donors.^{4,5} The true incidence

may be unknown, as it is likely underreported; the symptoms of neuropraxia may appear minor when compared with the severity of liver disease and the patient's presurgical condition and thus may be dismissed. Furthermore, symptoms may not be addressed, with the assumption that the condition is short lived and self-limited, although in our patient cohort, up to one-third of patients required neuropraxia-specific intervention. Clearly, the incidence of these injuries is sufficiently great to warrant further study into their causation and prevention.

Studies in large, nontransplant populations have previously identified patient risk factors thought to predispose to perioperative peripheral nerve injury. For example, diabetes, hypertension, and smoking cause microvascular changes that increase susceptibility to peripheral nerve injury.² Although we saw an increased incidence of hypertension, diabetes, hyperlipidemia, and smoking in transplant recipients who developed neuropraxia, this did not reach statistical significance. It may be that larger studies are needed for sufficient power to detect such differences.

Extremes in weight have been associated with perioperative neuropathies⁸; however, in this study, no association was found between BMI and neuropraxia after liver transplantation. The vast majority (84%) of liver transplant patients who developed neuropraxia were male. This has been described before in the closed claims database of the American Society of Anesthesiologists for ulnar neuropathy, which has a 75% male predominance.⁹ This has been attributed to sex-specific factors that predispose men to injury, including anatomical differences such as a larger tubercle of the coronoid with less surrounding fat, and increased susceptibility to direct pressure on unmyelinated nerve fibers.^{10,11} These results would suggest that male patients may merit particular attention during operative positioning, padding, and retractor placement.

In liver transplant recipients, we demonstrated significant associations between neuropraxia and a longer time spent on the operating room table. Longer surgery duration may contribute to prolonged ischemic compression and/or stretch of nerves, emphasizing the importance of careful, thoughtful patient positioning by nursing, surgical, and anesthesia teams. This concept has already gained attention in the general surgery literature, with studies demonstrating that longer positioning in lithotomy, for example, results in increased risk of lower extremity neuropraxia.^{12,13} To achieve adequate surgical exposure during liver surgery, metal retractors that pull the rib cage in an anterior and cephalad direction are frequently used.³ In this study, the extent of retraction was routinely monitored by using the loss or dampening of a right radial arterial waveline pressure tracing as a surrogate of compression of the neurovascular bundle between the clavicle and first rib because of retractor use. Interestingly, we found that 82% of neuropathies were left sided, suggesting that attention should also be placed toward the leftward pull of retractors for exposure. Central venous cannulas were routinely placed on the right side, and fewer than 1% of patients underwent left axillary cannulation for veno-venous bypass; thus, these cannot explain the finding of a left extremity predominance. Furthermore, 36% of those patients who developed neuropraxia had lower extremity symptoms, suggesting that focus on prevention in the upper extremities alone is shortsighted. Potential causes for peripheral

nerve injury of the lower extremity include femoral arterial catheter placement, cannulas for veno-venous bypass, and inappropriate padding during extended cases.

Because alcohol-induced toxic neuropathy is relatively common in patients with alcoholic cirrhosis,^{14,15} we speculated that these patients may demonstrate a higher incidence of postoperative neuropraxia. However, this was not the case, and indeed neuropraxia was not associated with any specific etiology for liver disease. Interestingly, a lower calculated MELD-Na score was significantly associated with the development of neuropraxia. The MELD score, as a liver disease severity index, is a predictor of short-term mortality in patients with cirrhosis.¹⁶ It was, therefore, surprising that 52% of those patients who developed neuropraxia were in the lowest MELD tertile (MELD-Na <19). This association merits further investigation in future studies. One potential hypothesis is that hepatic encephalopathy before transplant, with its theorized gamma aminobutyric acid mechanism of action, may mask the detection of short-term neuropathy in higher presurgical MELD-Na patients.

Although frequently transient, we saw symptoms for up to 187 d in our cohort, with two patients demonstrating months of severe hand impairment, and one-third of affected patients receiving neuropraxia-specific interventions. The potential burden of these injuries may warrant the implementation of intraoperative neuromonitoring tools to identify and mitigate the occurrence of neuropraxia. Intraoperative nerve monitoring of peripheral and/or central nerves is a well-established practice in spinal, cerebral, and vascular surgery, during which sensory- or motor-evoked potentials are used to identify nerve compromise.¹⁷ Historically intraoperative monitoring has involved large, cumbersome systems requiring expensive consoles and trained technicians. However, recent advances have yielded simple and inexpensive neuromonitoring tools, and some of these newer devices have been explored to monitor upper extremity nerves in orthopedic procedures.^{18–20} A clinical feasibility study using a Food and Drug Administration-approved device consisting of adhesive electrodes and a tablet computer to continuously monitor somatosensory-evoked potentials in cardiac surgery patients demonstrated promising ability to detect intraoperative nerve compromise.²¹ Given the preponderance of neuropraxia in our cohort, prospective randomized trials looking at the ability of these sticker-like electrodes on the upper and lower extremities to prevent nerve injury during liver transplantation can be explored.

Several limitations of this study must be considered, including the inherent drawbacks of a retrospective analysis. Registry data are unable to capture more granular clinical details, and thus, single-center studies such as this one are useful for identifying postoperative complications not recorded in large national United Network for Organ Sharing or Scientific Registry of Transplant Recipients databases. This study is an important contribution to the field, as it is the first focused investigation of this complication and its associated factors in a cohort of liver transplant recipients. We anticipate that this study will elicit an awareness of the problem within the transplant community that will incite further multi-institutional study. The diagnosis of neuropraxia is usually clinical, and EMG and nerve conduction studies, while helping with possible localization of peripheral nerve injuries, are not necessary for diagnosis.^{22,23} The majority of our patients did not have these adjunctive tests performed, but future studies could consider implementing them to gain further details about the level of

nervous system injury. To address the robustness of the definition of neuropraxia in this study, the electronic medical records were reviewed by two independent clinicians, with any disagreements adjudicated by a third physician. However, it is possible that the incidence of neuropraxia was underreported, in particular, in patients who had complicated hospital postoperative courses.

Patient intraoperative positioning plays a critical role in the development of nerve damage, and positioning practices are not uniform across institutions. In this study, patients' arms were routinely placed on an armboard at <90° abduction to allow for anesthesia access, whereas some centers tuck arms at the patient's side. Variations in patient positioning may affect results across transplant centers. We do know, however, from studies in cardiac surgery, which has a relatively high incidence of nerve injury, that the incidence of brachial plexus injuries is similar whether the arms are placed at the side or on an armboard at 90° abduction.³

In conclusion, neuropraxia constitutes a relatively unexplored but significant source of morbidity after liver transplantation. Little is known regarding risk factors and patient outcomes, and for the first time, here, we characterized risk factors, neuropraxia symptom distribution, duration, and interventions in liver transplant recipients. Further studies into means for intraoperative detection and prevention are critical to reduce the incidence of nerve injury after liver transplantation.

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Fig. 1 -
Unadjusted odds of neuropraxia by cohort characteristics.

Fig. 2 -
Adjusted odds ratio of neuropraxia from forward selection model.

Table -

Demographic, clinical, and surgical characteristics.

Variable	No neuropraxia (n = 258)	Neuropraxia (n = 25)	P value
Age (y), median (IQR)	58.0 (51–64)	59.0 (45–63)	0.5
Female, %	37.6	16.0	0.03
Race, %			
White	65.5	68.0	0.8
Black	8.5	12.0	
Hispanic	16.7	16.0	
Other	9.3	4.0	
BMI (kg/m ²), median (IQR)	26.9 (24.0–31.9)	28.8 (25.1–32.5)	0.5
Underweight (<18.5)	2.3	0.0	0.8
Normal (18.5 to <25)	29.5	24.0	
Overweight (25 to <30)	36.4	36.0	
Obese (>30)	31.8	40.0	
BMI <25, %	31.8	24.0	0.5
BMI >25, %	68.2	76.0	
Cause of ESLD			
Hepatitis C, %	28.7	32.0	0.8
Alcohol, %	16.7	20.0	0.6
NASH, %	12.8	8.0	0.7
Autoimmune, %	12.4	24.0	0.1
HCC, %	36.4	56.0	0.05
Coronary artery disease, %	9.7	8.0	0.8
Hypertension, %	32.9	44.0	0.3
Diabetes, %	27.1	32.0	0.6
Hyperlipidemia, %	13.2	16.0	0.7
Dialysis, %	6.2	8.0	0.7
Smoking history, %	26.0	36.0	0.3
MELD, mean ± SD	22.8 ± 9.3	18.7 ± 8.6	0.03
MELD-Na, mean ± SD	24.2 ± 9.1	20.0 ± 8.1	0.02

Variable	No neuropraxia (<i>n</i> = 258)	Neuropraxia (<i>n</i> = 25)	<i>P</i> value
Living donor, %	13.9	16.0	0.8
SLK, %	14.7	20.0	0.6
Duration of surgery, mean hours $\pm$ SD	9.3 $\pm$ 2.2	10.5 $\pm$ 2.5	0.02
Red blood cell transfusion, median (IQR)	11.0 (3.0–19.0)	15.0 (6.0–23.5)	0.5
Reoperation, %	20.9	24.0	0.8
Length of hospital stay (d), median (IQR)	9 (5–17)	6 (5–17)	0.7
Discharge home, %	65.1	68.0	0.5
Discharge to inpatient rehabilitation, %	22.9	28.0	
Discharge to long-term care or nursing facility, %	12.0	4.0	

ESLD = end-stage liver disease; HCC = hepatocellular carcinoma; IQR = interquartile range; MELD = model for end-stage liver disease score; NASH = nonalcoholic steatohepatitis; SLK = simultaneous liver-kidney; SD = standard deviation.