Title

Myocardial injury in patients with hemodynamic derangements during and/or after liver transplantation

Journal

Clinical Transplantation, 30(12)

ISSN

0902-0063

Authors

Huang, Shun
Apinyachon, Worapot
Agopian, Vatche G
et al.

Publication Date

2016-12-01

DOI

10.1111/ctr.12855

Peer reviewed

Accepted: 18 September 2016

DOI: 10.1111/ctr.12855

ORIGINAL ARTICLE

WILEY Clinical TRANSPLANTATION
The Journal of Clinical and Translational Research

Myocardial injury in patients with hemodynamic derangements during and/or after liver transplantation

Shun Huang[1,2] | **Worapot Apinyachon**[3] | **Vatche G. Agopian**[4] | **Christopher L. Wray**[2] | **Ronald W. Busuttil**[4] | **Randolph H. Steadman**[2] | **Victor W. Xia**[2]

[1]Department of Anesthesiology, Beijing Hospital, National Center of Gerontology, Beijing, China

[2]Department of Anesthesiology, Ronald Reagan UCLA Medical Center, Los Angeles, CA, USA

[3]Department of Anesthesiology, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand

[4]Department of Surgery, David Geffen School of Medicine at UCLA, Los Angeles, CA, USA

Correspondence
Victor W. Xia, Department of Anesthesiology, Ronald Reagan UCLA Medical Center, David Geffen School of Medicine at UCLA, Los Angeles, CA, USA.
Email: vxia@mednet.ucla.edu

Funding Information
Support was provided solely from departmental sources.

Abstract

Myocardial injury, defined as an elevation of cardiac troponin (cTn) resulting from ischemia, is associated with substantial mortality in surgical patients, and its incidence, risk factors, and impact on patients undergoing liver transplantation (LT) are poorly understood. In this study, adult patients who experienced perioperative hemodynamic derangements and had cTn measurements within 30 days after LT between 2006 and 2013 were studied. Of 502 patients, 203 (40.4%) met the diagnostic criteria (cTn I ≥0.1 ng/mL) of myocardial injury. The majority of myocardial injury occurred within the first three postoperative days and presented without clinical signs or symptoms of myocardial infarction. Thirty-day mortality in patients with myocardial injury was 11.4%, significantly higher compared with that in patients without myocardial injury (3.4%, $P<.01$). Cox analysis indicated the peak cTn was significantly associated with 30-day mortality. Multivariable logistic analysis identified three independent risk factors: requirement of ventilation before transplant (odds ratios (OR) 1.6, $P=.006$), RBC≥15 units (OR 1.7, $P=.006$), and the presence of PRS (OR 2.0, $P=.028$). We concluded that post-LT myocardial injury in this high-risk population was common and associated with mortality. Our findings may be used in pretransplant stratification. Further studies to investigate this postoperative cardiac complication in all LT patients are warranted.

KEYWORDS
liver transplantation, myocardial injury, perioperative management, postoperative complication, retrospective study, risk factors, troponin

1 | INTRODUCTION

Cardiovascular complications including myocardial ischemia occur frequently after liver transplant (LT) and are a leading cause of postoperative mortality and morbidity.[1,2] The high incidence of postoperative cardiovascular complications in LT is contributed by many preoperative and intra-operative factors.[3-8] Studies on postoperative cardiovascular complications have traditionally concentrated on myocardial infarction (MI).[9] Such an approach has limitations. First, the clinical diagnosis of MI in the postoperative period is difficult to discern. This is, in major part, due to the fact that cardiac symptoms and electrocardiographic evidence are often absent in the postoperative setting.[10,11] Second, although MI is serious, it only represents a portion of postoperative cardiovascular complications. Many patients may sustain myocardial injury without a formal diagnosis of MI. Emerging data suggest that these patients do poorly postoperatively.[10] Finally, interventional strategies designed to address myocardial injury that is beyond the definition of MI may reduce postoperative cardiovascular complications and improve patient outcomes.[10]

Recent advances have greatly extended our understanding of the cardiovascular diseases and the ability to diagnose them.[12] With current technology, a trace amount of biomarkers, particularly cardiac

troponin (cTn) released from damaged myocardial cells, can be detected in a highly sensitive and specific fashion.[13] After evolution in the last few decades, measurement of cTn has become a part of the standard in diagnosing and quantifying myocardial damage. In fact, the latest definition of myocardial ischemia has embraced the central diagnostic role of cTn measurement.[11,14] Recently, a study proposes a term for myocardial injury after noncardiac surgery, which is defined by mildly elevated cTn level. Myocardial injury is broader than the definition of MI and yet is associated with substantial mortality.[10]

In this retrospective study, we attempted to determine a level of cTn that can be used in diagnosis for myocardial injury after LT. In addition, we attempted to identify the incidence, risk factors, and impact of myocardial injury after LT. We hypothesized that myocardial injury was common after LT and was associated with increased mortality.

2 | MATERIALS AND METHODS

After approval by the University of California, Los Angeles (UCLA) Institutional Review Board, a retrospective study was carried out. Medical records of patients who underwent LT at UCLA Medical Center between January 2006 and August 2013 were reviewed. The study included all consecutive cases of adult patients (age≥18). Preoperative and intra-operative variables were prospectively collected and stored in the UCLA transplant database. Postoperative clinical cardiac symptoms, electrocardiographic findings, cardiac diagnosis, cardiology consultations, and cTn levels were collected retrospectively.

Liver transplantation selection criteria and techniques at our institution have been described elsewhere.[15] In brief, all recipients underwent multidisciplinary selection. Preoperative cardiac evaluation included history and physical examination, echocardiography, stress tests, or cardiac catheterization, depending on cardiac risk stratification. A balanced anesthetic technique with intravenous and inhalational anesthetics combined with opioids and the muscle relaxants was used for anesthetic induction and maintenance. In addition to the American Society of Anesthesiologists standard monitors, intra-arterial and pulmonary arterial catheters were employed and transesophageal echocardiography was used if there were no contraindications. Intraoperative fluid therapy included crystalloids, albumin, and blood products with red blood cells (RBC), fresh frozen plasma (FFP), platelets, and cryoprecipitate. Blood products were administered via a rapid transfusion device. Intra-operative vasopressors were either in continuous infusion or in bolus.[16] Postreperfusion syndrome (PRS) was defined by a 30% decrease in blood pressure from prereperfusion baseline that lasted more than 1 minute in 5 minutes after reperfusion of the liver graft.[17]

Postoperatively, LT patients were transferred to the intensive care unit and managed by a multidisciplinary team. Cardiology was consulted for possible cardiac complications or management. Postoperative cTn I was measured in selected patients with hemodynamic derangements without a standard protocol during the study period. Hemodynamic derangements that prompts cTn measurement included persistent perioperative hypotension (mean blood pressure ≤55 mm Hg for ≥10 minutes), hemodynamic instability, requirements of large amount of blood products (packed red blood cells >15 units), vasopressors, electrocardiographic changes, or ischemic symptoms. cTn levels within 30 days after LT were included in the study. cTn I was the standard cardiac biomarker used by UCLA during the study period. The lowest detectable level of cTn I was 0.04 ng/mL during the study period. When the cTn measurement was initiated, a series of measurements was typically performed until the cTn levels trended downward. The peak (highest) value of the series of measurements for each patient was used in analysis.

Thirty-day all-causes mortality was assessed in patients with various postoperative cTn I levels. The lowest cTn I level that was significantly associated with 30-day mortality was determined and used to define myocardial injury. Diagnosis of myocardial injury was made after excluding conditions in nonischemic nature using criteria modified from The Vascular Events in Noncardiac Surgery Patients Cohort Evaluation study.[10] Patients with MI were also identified using the Third Universal Definition.[18] The incidence and risk factors that were associated with myocardial injury after LT were determined.

All analyses were performed using the Statistical Package for Social Sciences, version 22.0 (IBM, Armonk, NY, USA). Reported *P* values are two sided. The categorical data were analyzed with Pearson's chi-square test. Student's *t* test or analysis of variance was used to compare the preoperative and intra-operative quantitative variables. The categorical variables were presented as percentages, and the quantitative variables were presented as mean±SD. Quantitative variables were dichotomized by the median, mean, quartiles, or a clinically meaningful value. Variables with a statistically significant relationship to myocardial injury in univariate analysis were entered into binary logistic regression. Statistical significance was defined as *P* less than .05. Patient survival was compared between patients with and without myocardial injury by Kaplan-Meier survival analysis. Cox survival analysis was performed to identify risk factors for recipient mortality.

3 | RESULTS

During the study period, 1386 adult patients underwent LT. Of 1386 patients, 502 (36.2%) had hemodynamic derangements during and/or after LT which prompted cTn I measurements within 30 days after LT (Figure 1). Mean age of patients with hemodynamic derangements was 56.0 years. There were more male patients (62.5%) than female patients. The most common indication for LT was hepatitis C cirrhosis. Mean MELD score was 33.4 in this patient group (Table 1).

Overall 30-day mortality after LT in studied patients was 6.8%. To assess a relationship between the peak cTn levels and 30-day mortality, patients were divided into four groups according to quartiles of the peak cTn level. As the peak cTn levels increased, 30-day mortality rates increased. Specifically, patients with cTn groups at ≤0.04, 0.05-0.09, 0.1-0.25, and ≥0.25 ng/mL had 30-day mortality at 1.9%, 4.0%, 7.8%, and 18.4%, respectively, *P*<.001 (Figure 2). Although higher peak cTn I levels were associated with higher 30-day mortality, the

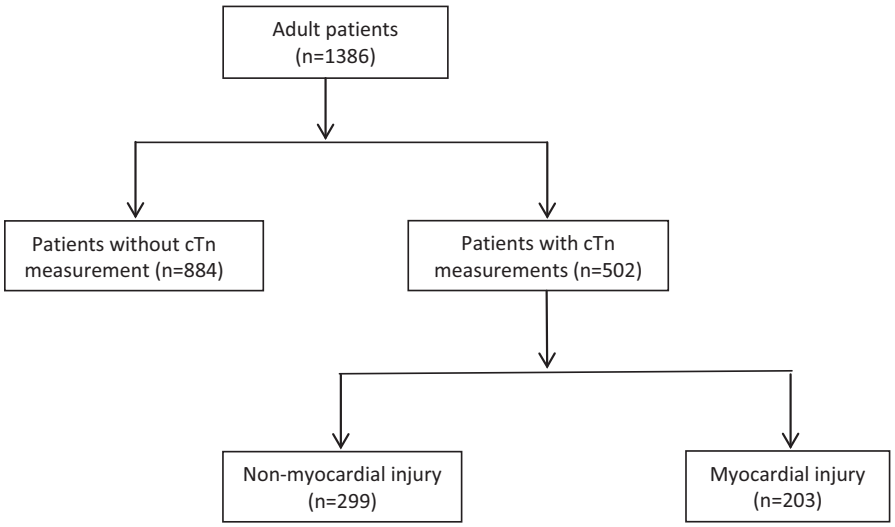

FIGURE 1 Of 502 patients whose cTn I levels were measured within 30 d after LT prompted by hemodynamic derangement, 203 met the diagnostic criteria of myocardial injury

TABLE 1 Preoperative characteristics of patients

	All patients (n=502)
Age (y)	56.0±10.7
Weight (kg)	79.0±21.2
Height (cm)	169.5±11.0
Gender (male, %)	62.5
Etiology of liver disease (%)	
Hepatitis C	37.1
Hepatitis B	6.0
Alcoholic cirrhosis	22.4
NASH	6.9
Acute	4.4
MELD score	33.4±7.5
Hypertension (%)	36.1
History of coronary artery disease (%)	12.1
Variceal bleed (%)	37.2
Preoperative renal replacement therapy (%)	43.2
Preoperative endotracheal intubation (%)	28.5
Preoperative pressors (%)	21.4
Baseline hematocrit (%)	29.0±5.8
Baseline INR	1.8±0.6
Baseline creatinine (mg/dL)	1.8±1.4

NASH, nonalcoholic steatohepatitis; MELD, model for end-stage liver disease.

statistical significance was only achieved when cTn I level was 0.1 ng/mL or higher. There were 214 patients who had cTn I ≥0.1 ng/mL. After exclusion of 11 patients with the cTn elevation due to nonischemic etiologies (eight with sepsis and three with cardioversion), 203 patients met the myocardial injury diagnostic criteria. The incidence of myocardial injury in this patient group was 40.4%.

Thirty-day mortality in patients with myocardial injury was 11.8%, significantly higher compared with those without myocardial injury (3.3%, all $P<.001$). Cox survival analysis indicated that cTn I ≥0.1 ng/mL was an independent risk factor for patient 30-day mortality (hazard ratios 3.5, 95% CI: 2.04-6.20, $P<.001$). The multivariate predictive model for 30-day mortality improved when cTn was included (the area under the curve increased from 0.691 to 0.738). Other two risk factors for 30-day mortality were the requirement of preoperative pressors and intra-operative blood transfusion.

Myocardial injury occurred most frequently on the first postoperative day (43.3%), and the majority occurred on the first three days after LT (70.9%, Figure 3). The incidence of myocardial injury decreased significantly after the first three days and remained relatively constant afterward. Of 203 myocardial injury patients, only 3.6% met the definition of MI, and 96.4% of the patients presented with no clinical symptoms or signs of MI.

Comparison between patients with and without myocardial injury is presented in Table 2. Patients with myocardial injury differed significantly from those without myocardial injury. Patients with myocardial injury had higher MELD score, lower baseline hematocrit, higher requirement of intra-operative red blood cell transfusion, and higher incidence of postreperfusion syndrome compared with those without myocardial injury. In addition, more patients in the myocardial injury required preoperative intubation, intra-operative pressors, and intra-operative venovenous bypass. Multivariable logistic analysis showed three variables (preoperative intubation, OR 1.6, 95% CI: 1.05-2.51, $P=.006$; intra-operative blood transfusion ≥15 units, OR 1.8, 95% CI: 1.17-2.67 $P=.006$; and the presence of postreperfusion syndrome, OR 2.0, 95% CI: 1.14-3.36, $P=.028$) were independent risk factors for myocardial injury (Table 3).

4 | DISCUSSION

In this retrospective study, we found that myocardial injury defined as peak cTn I ≥0.1 ng/mL occurred at 40.4% in adult LT patients who experienced perioperative hemodynamic derangements and was associated with substantial 30-day mortality. Most of myocardial

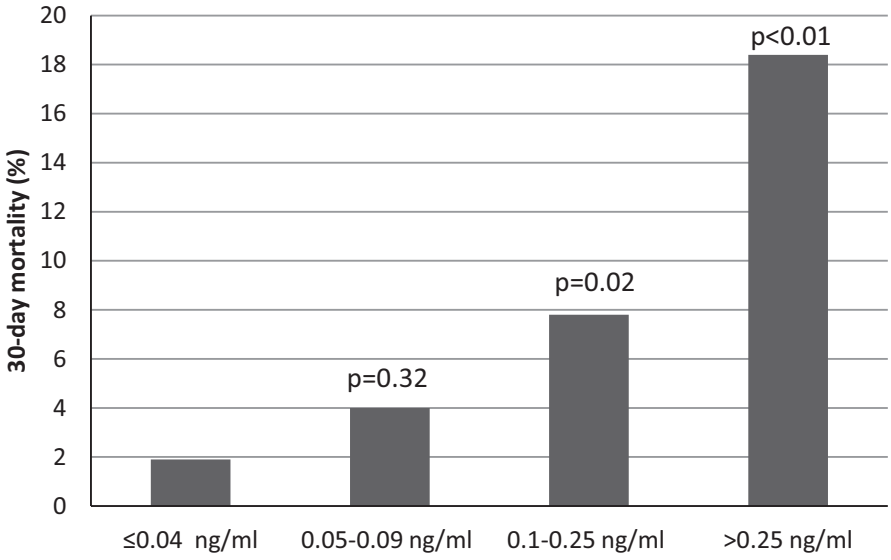

FIGURE 2 Peak postoperative cTn levels were positively associated with 30-d mortality. When compared with the group of cTn ≤0.04 ng/mL, groups with higher cTn levels had higher 30-d mortality. However, the statistical significance could be only reached when cTn level was 0.1 ng/mL or higher

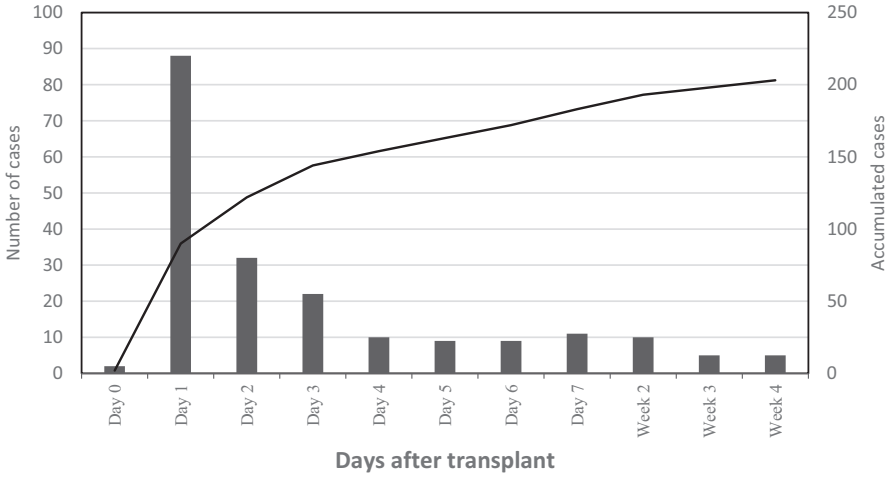

FIGURE 3 Myocardial injury occurred most frequently on the first postoperative day. The majority of the cases occurred on the first three postoperative days. After the first week, myocardial injury occurred significantly less

injury occurred in the immediate postoperative period and presented without classic symptoms or signs of MI. Risk factors for myocardial injury in this patient population were preoperative intubation, intraoperative red blood cell transfusion, and the presence of postreperfusion syndrome.

Postoperative cTn elevation in noncardiac surgery is common, but the reported incidence varies in different studies.[19,20] This discrepancy is largely due to different patient population types of surgery and cTn cutoff values used in the studies.[21,22] The VISION study found that slightly elevated cTn T (≥0.04 ng/mL) was associated with substantial mortality.[10] In this study of LT patients, we found cTn I ≥0.1 ng/mL was the lowest value that was associated with mortality. Using these criteria, the incidence of myocardial injury in our study was 40.4%, which is higher compared with other studies in non-LT patients.[10,23] The high incidence of myocardial injury in our study could be potentially caused by the following: (i) Patients undergoing LT surgery are at high risk for cardiovascular complications including myocardial injury, and (ii) our patients were those who had hemodynamic derangements, which render them a higher risk.

Postoperative cardiovascular complications in LT are common and have been extensively studied. However, myocardial injury after LT has not been reported. A previous report studying the relationship between postoperative cTn levels and mortality in LT could not meet the definition of myocardial injury proposed by the VISION study due to a high cTn cutoff and inclusion of nonischemic patients.[24] In addition, this report was severely underpowered by including a small number of patients. Our study was the first attempting to use the lowest meaningful cTn cutoff and excluded patients with nonischemic causes for cTn elevation. The exclusion of nonischemic etiology is necessary for diagnosis of myocardial injury. This is because cTn can be released as a result of many nonischemic etiologies, and prophylactic and therapeutic interventions for myocardial ischemia are very different from nonischemic injuries.

Consistent with the VISION study, the majority of myocardial injuries were silent and occurred in the first 3 days after LT. If the traditional diagnostic criteria of MI were used, the incidence of myocardial injury would have significantly been underestimated. Although we reviewed cTn data 30 days after LT, myocardial injury was infrequent

TABLE 2 Comparison between patients with and without myocardial injury

	Nonmyocardial injury (n=299)	Myocardial injury (n=203)	*P*
Age (y)	56.3±10.5	55.4±11.1	.384
Weight (kg)	79.6±20.5	77.8±22.3	.381
Height (cm)	169.7±10.8	169.2±11.1	.723
Gender (male, %)	63.0	61.6	.083
Etiology of liver disease (%)			
Hepatitis C	42.8	27.6	.001
Hepatitis B	4.8	8.1	.138
Alcoholic cirrhosis	21.5	23.8	.563
NASH	6.8	7.0	.907
Acute	3.2	6.5	.087
MELD score	32.5±7.5	34.4±7.5	.008
Hypertension (%)	38.7	31.7	.120
History of coronary artery disease (%)	10.6	14.6	.193
Variceal bleed (%)	32.9	44.5	.012
Preoperative renal replacement therapy (%)	39.7	49.2	.043
Preoperative endotracheal intubation (%)	24.9	34.6	.022
Preoperative pressors (%)	18.6	26.0	.057
Baseline hematocrit (%)	29.4±6.1	28.2±5.3	.041
Baseline INR	1.8±0.6	1.8±0.5	.145
Baseline creatinine (mg/dL)	1.8±1.4	1.8±1.3	.736
Intra-operative dialysis (%)	13.5	14.0	.871
Pressor infusion (%)	69.9	87.6	<.001
Pressor bolus (in large quantity, %)	35.2	49.7	.002
Postreperfusion syndrome (%)	12.3	19.1	.046
Venovenous bypass (%)	44.6	56.6	.013
Donor from donation after cardiac death (%)	4.2	5.6	.490
Cold ischemia time (min)	400.3±140.5	397.8±146.5	.855
Warm ischemia time (min)	44.4±12.3	42.7±10.9	.134
Surgery time (min)	357.6±142.3	363.4±148.5	.776
Red blood cell transfusion (in unit)	20.7±19.3	24.5±18.9	.032
Fresh frozen plasma (in unit)	25.5±21.9	28.0±19.4	.213

NASH, nonalcoholic steatohepatitis; MELD, model for end-stage liver disease.

TABLE 3 Risk factors for postoperative myocardial injury

Risk factors	OR	95% CI	*P*
Preoperative intubation	1.6	1.05-2.51	.006
Red blood cells ≥15 units	1.8	1.17-2.67	.006
Presence of postreperfusion syndrome	2.0	1.14-3.36	.028

after the first postoperative week. Despite the similarities, there were significant differences between our study and the VISION study. First, types of cTn used in studies were different. cTn I has been clinically used at our hospital and therefore was used in our study. In contrast, cTn T was used in the VISION study. Studies show that both cTns are sensitive and selective.[25] Second, the cutoffs used in the two studies were not identical (≥0.1 ng/mL in our study vs ≥0.04 ng/mL in the VISION). We are not sure if the difference is resulted from the different types of cTn or from different patient populations. Third, the VISION study was prospective, and all patients were included. Ours was retrospective, and only patients who experience hemodynamic derangements were selected for cTn testing. Therefore, the incidence of myocardial injury in our study only represents this complication in this particular patient population, and the overall incidence of postoperative myocardial injury for LT patients cannot be known until all patients are studied. We are currently enrolling all patients after LT for cTn measurement and should be able answer this question in the near future.

Three risk factors for myocardial injury found in this study may aid in identification of patients at risk of developing this serious postoperative complication. Requirement of preoperative intubation and ventilation is generally considered as a marker for severity of disease and has been found to be a risk factor for a number of worse postoperative outcomes.[26] Intra-operative RBC requirement has been associated with a variety of adverse postoperative outcomes after LT[27] and it was not a surprise to find a link with myocardial injury in our study as well. Massive bleeding can lead to low perfusion to myocardial tissue and result in the imbalance of the supply and demand of myocardial oxygenation. The presence of PRS usually indicates severe ischemia and reperfusion injury related to quality of donor organ and can also lead to a difficult postreperfusion and postoperative course. The presence of PRS is unique to LT patients and cannot be found in patients undergoing other surgery.

Our study had limitations. First, this was a retrospective study with many known inherent limitations. Second, the elevation of cTn in the preoperative period is not measured in this study. There is evidence suggesting that some patients have elevated cTn levels before LT that are also associated with postoperative outcomes. Third, a possibility of other unmeasured confounding factors should be considered when our results are interpreted. Finally, the results of our study were based on data from one center and need to be confirmed by other studies.

In conclusion, in this retrospective study of adult patients undergoing LT, we found that myocardial injury defined as cTn I ≥0.1 ng/mL was common in patients with perioperative hemodynamic derangements

and was associated with increased 30-day mortality. Most of myocardial injury occurred immediately after LT and presented without symptoms of MI. Several preoperative and intra-operative risk factors were identified for this important postoperative event. Future studies to assess the role of elevated cTn in postoperative myocardial injury in all LT patients are warranted.

CONFLICT OF INTEREST

All authors have no conflict of interest to disclose.

REFERENCES

1. Fouad TR, Abdel-Razek WM, Burak KW, Bain VG, Lee SS. Prediction of cardiac complications after liver transplantation. *Transplantation*. 2009;87:763–770.
2. Fussner LA, Heimbach JK, Fan C, et al. Cardiovascular disease after liver transplantation: when, what, and who is at risk. *Liver Transpl*. 2015;21:889–896.
3. Coss E, Watt KD, Pedersen R, Dierkhising R, Heimbach JK, Charlton MR. Predictors of cardiovascular events after liver transplantation: a role for pretransplant serum troponin levels. *Liver Transpl*. 2011;17:23–31.
4. Dec GW, Kondo N, Farrell ML, Dienstag J, Cosimi AB, Semigran MJ. Cardiovascular complications following liver transplantation. *Clin Transplant*. 1995;9:463–471.
5. Desai S, Hong JC, Saab S. Cardiovascular risk factors following orthotopic liver transplantation: predisposing factors, incidence and management. *Liver Int*. 2010;30:948–957.
6. Therapondos G, Flapan AD, Plevris JN, Hayes PC. Cardiac morbidity and mortality related to orthotopic liver transplantation. *Liver Transpl*. 2004;10:1441–1453.
7. Carey WD, Dumot JA, Pimentel RR, et al. The prevalence of coronary artery disease in liver transplant candidates over age 50. *Transplantation*. 1995;59:859–864.
8. Johnston SD, Morris JK, Cramb R, Gunson BK, Neuberger J. Cardiovascular morbidity and mortality after orthotopic liver transplantation. *Transplantation*. 2002;73:901–906.
9. Biccard BM, Devereaux PJ, Rodseth RN. Cardiac biomarkers in the prediction of risk in the non-cardiac surgery setting. *Anaesthesia*. 2014;69:484–493.
10. Botto F, Alonso-Coello P, Chan MT, et al. Myocardial injury after noncardiac surgery: a large, international, prospective cohort study establishing diagnostic criteria, characteristics, predictors, and 30-day outcomes. *Anesthesiology*. 2014;120:564–578.
11. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. *J Am Coll Cardiol*. 2012;60:1581–1598.
12. Agewall S, Giannitsis E. Troponin elevation in coronary ischemia and necrosis. *Curr Atheroscler Rep*. 2014;16:396.
13. Sherwood MW, Kristin Newby L. High-sensitivity troponin assays: evidence, indications, and reasonable use. *J Am Heart Assoc*. 2014;3:e000403.
14. Mitka M. New definition of myocardial infarction puts biomarkers front and center. *JAMA*. 2012;308:1511–1512.
15. Agopian VG, Petrowsky H, Kaldas FM, et al. The evolution of liver transplantation during 3 decades: analysis of 5347 consecutive liver transplants at a single center. *Ann Surg*. 2013;258:409–421.
16. Xia VW, Du B, Braunfeld M, et al. Preoperative characteristics and intraoperative transfusion and vasopressor requirements in patients with low vs. high MELD scores. *Liver Transpl*. 2006;12:614–620.
17. Aggarwal S, Kang Y, Freeman JA, Fortunato FL, Pinsky MR. Postreperfusion syndrome: cardiovascular collapse following hepatic reperfusion during liver transplantation. *Transplant Proc*. 1987;19(Suppl 3):54–55.
18. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. *Circulation*. 2012;126:2020–2035.
19. Adams JE 3rd, Sicard GA, Allen BT, et al. Diagnosis of perioperative myocardial infarction with measurement of cardiac troponin I. *N Engl J Med*. 1994;330:670–674.
20. Kim LJ, Martinez EA, Faraday N, et al. Cardiac troponin I predicts short-term mortality in vascular surgery patients. *Circulation*. 2002;106:2366–2371.
21. Redfern G, Rodseth RN, Biccard BM. Outcomes in vascular surgical patients with isolated postoperative troponin leak: a meta-analysis. *Anaesthesia*. 2011;66:604–610.
22. Levy M, Heels-Ansdell D, Hiralal R, et al. Prognostic value of troponin and creatine kinase muscle and brain isoenzyme measurement after noncardiac surgery: a systematic review and meta-analysis. *Anesthesiology*. 2011;114:796–806.
23. Le Manach Y, Perel A, Coriat P, Godet G, Bertrand M, Riou B. Early and delayed myocardial infarction after abdominal aortic surgery. *Anesthesiology*. 2005;102:885–891.
24. Snipelisky D, Donovan S, Levy M, Satyanarayana R, Shapiro B. Cardiac troponin elevation predicts mortality in patients undergoing orthotopic liver transplantation. *J Transplant*. 2013;2013:252838.
25. Sethi A, Bajaj A, Malhotra G, Arora RR, Khosla S. Diagnostic accuracy of sensitive or high-sensitive troponin on presentation for myocardial infarction: a meta-analysis and systematic review. *Vasc Health Risk Manag*. 2014;10:435–450.
26. Zhao W, Ge X, Sun K, et al. Acute respiratory distress syndrome after orthotopic liver transplantation. *J Crit Care*. 2016;31:163–167.
27. Massicotte L, Sassine MP, Lenis S, Roy A. Transfusion predictors in liver transplant. *Anesth Analg*. 2004;98:1245–1251.

How to cite this article: Huang, S., Apinyachon, W., Agopian, V. G., Wray, C. L., Busuttil, R. W., Steadman, R. H. and Xia, V. W. (2016), Myocardial injury in patients with hemodynamic derangements during and/or after liver transplantation. Clinical Transplantation, 30: 1552–1557. doi: 10.1111/ctr.12855