High Transaminases Following Cardiac Surgery: A Narrative Review

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Abstract

Elevation of liver enzymes after cardiac surgery is encountered infrequently. Acute heart failure during and after surgery may be the culprit responsible for liver dysfunction. However, it may create clinical confusion whether acute liver dysfunction could induce some sort of cardiac dysfunction through mechanisms similar to those encountered in chronic liver disease. We searched through the Medline, Cochrane, and Embase databases up to January 2018. We included review articles, meta-analyses, and original trials on the elevation of liver enzymes after cardiac surgery, and combined the following MESH terms: "intensive care, "cardiac surgery," "high liver enzymes," "ischemia," "left ventricular dysfunction," and "critical illness." Case reports were excluded. Language restrictions were not applied. References were examined for other potentially useful articles. We did not find any articles that supported the cardiac decompensation phenomenon after acute liver injury. In contrast, low-hepatic flow, hypoxemia, or pump-induced inflammation could induce hepatic dysfunction in acute settings after cardiac surgery. In conclusion, a rise in the transaminases following cardiac surgery would favor an ischemic etiology for the condition.

Keywords
- transaminases
- cardiac surgery
- cardiac function
- high liver enzymes

Introduction

Elevation of liver function tests (LFT) is an infrequent complication after cardiac surgery with an incidence of less than 1%.[1] This complication plays a pivotal role in overall intensive care unit outcome, as it is associated with a series of hemodynamic, metabolic, inflammatory derangement that could affect the overall morbidity and mortality.[2] In acute settings, the cardiac causes of elevated LFTs included hypotension, low-cardiac output, right ventricular failure, intravascular volume depletion, and vasodilatation. The reason for the elevation in LFTs may not be clear.[3] Cardiac hepatopathy exists in two main forms which are acute cardiogenic liver injury and congestive hepatopathy. The former associates acute heart failure, while the latter comes about in the settings of right-sided heart failure as a consequence of passive venous congestion; the two situations may coexist and result in increase in the liver enzyme.[4,5]

Chronic liver disease is a state that may participate and accelerate impairment of cardiac functions and cardiac remodeling. Cirrhotic patients have 45 to 56% prevalence of diastolic dysfunction, while the latter is reflected in some degree by left atrial enlargement. Cardiomyopathy in patients with chronic liver disease could be boosted by many mechanisms, including β-adrenergic receptor pathway signaling inhibition,

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cytoplasmic as well as nuclear cardiomyocytes vacuolation and, finally, cardiomyocyte cellular membrane changes.[6]

The cardiac hepatic interaction is crucial for cardiologists, hepatologists, and intensivists.[7] Chronic liver diseases may induce a clinical picture of cardiomyopathy; in chronic hepatitis C, viral infection could stimulate proliferation of myocytes with subsequent hypertrophy and hypertrophic cardiomyopathy.[8] In patients with liver cirrhosis, the sympathetic nervous system activity is augmented and cardiac output is increased due to hyperdynamic circulation and reduced systemic vascular resistance. The latter changes could prompt left ventricular hypertrophy and myocardial remodeling that lead to disordered systolic and diastolic functions, and cardiomyopathy.[9] Chronic liver diseases may induce systolic and diastolic dysfunction in addition to prolongation of QT interval and electrophysiological alterations, given the changes in cirrhotic cardiomyopathy may recover completely following liver transplantations.

Causes of elevation of liver enzymes

1. Postsurgical
   Elevation in LFTs more than five-fold, which associates with increase in conjugated bilirubin, may reflect hepatocellular injury that occurs in hepatic hypoxia and drug-induced hepatitis.[2]
2. Myocardial dysfunction
   The low-cardiac output that follows heart failure, acute myocardial infarction, and pericarditis may result in liver dysfunction, owing to reduced blood supply to the liver and passive hepatic venous congestion. The latter is characterized by moderate elevation of LFTs < three-fold in 30 to 60% of cases, bilirubin elevation up to ten-fold in 25 to 80% of patients, and prothrombin time (2–6 seconds) in 80 to 90%.[10]

   Cardiac arrest or arrhythmia-induced hypotension can result in hypoxic hepatitis, with striking elevation in LFTs.[5] Cardiopulmonary bypass (CPB) surgery may also induce liver dysfunction.[11] Off pump coronary bypass decreases the possibility of hepatocellular damage induced by CBP.[12]
3. Sepsis
   Reperfusion injury and hepatic ischemia may follow sepsis.[9] Septic shock is associated with major metabolic demand increment, where tissue oxygen consumption and oxygen extraction are affected.[13] Oxygen consumption increment could also be associated with cytokine production and the deviation of oxygen to the generation of reactive oxygen species.[14] In addition, hepatic flow may be reduced due to the effect of mechanical ventilation from increased intrathoracic pressure.[15]

   Hepatic microcirculation is affected and the LFTs deteriorate gradually after sepsis.[16] Complex interaction does occur between pro- and anti-inflammatory mediators, aiming to maintain normal arterial pressure and protect liver tissue; the severity of hepatic injury is determined by Kupffer cells in shock situations.[17]
4. Hypoxic hepatitis
   The dual liver blood supply form the hepatic artery and portal vein attempt to maintain constant hepatic flow even in shock status. Dilatation of hepatic artery occurs in response to reduced portal venous flow (in the range of 20–30%).[18] The oxygen content in the hepatic artery is higher than the portal vein, which made oxygen compensation substantially better.[19] Cellular ischemic is produced if hepatic microcirculation ceases under a critical level, resulting in hepatic injury and dysfunction. At the same time, nitric oxide (NO) seeks to maintain endothelial integrity and hepatic microcirculation, which is the result of the insult.[20] Pericentral necrosis and ATP depletion is induced by extended low-flow with hypoxia, where regeneration of oxygen and ATP supplies after shorter-term, low-flow ischemia reproduces programmed cell death.[21] In heart failure, liver hypoxia is referred to as reduced hepatic blood flow in left-sided heart failure, and as venous congestion in right-sided heart failure. The rapid increase in LFTs that accompany hypoxia usually leads to recovery within a few days.[22]
5. Acute respiratory distress syndrome (ARDS)
   Liver dysfunction may accompany ARDS; however, on the other hand, hepatic injury association significantly impairs outcome in critically ill adults.[23]
6. Intervention induced liver dysfunction
   Medical and surgical procedures in critically ill patients may be associated with abnormalities in LFTs. Ultrafiltration and hemodialysis could be associated with hepatic hypoxia from reduced splanchnic, femoral, and systemic blood flows.[24] The critically ill have commonly tied physiological reserve with altered variation in drug metabolism and higher chances of adverse events. ICU medication induced by hepatitis is common and may be due to inotropes. Hepatic injury form systemic arterial blood flow changes may include but not be limited to granulomatous hepatitis (hydralazine and quinidine), acute hepatitis (amiodarone, diltiazem, b-blockers, lisinopril, and hydralazine), fibrosis (amiodarone), cholestasis (nifedipin and disopyramide), or mixed changes (procainamide).[2]

Results

We did not find any literature to support the cardiac decompensation nature, following acute liver insults. In contrary, low-hepatic flow, hypoxemia, or pump-induced inflammation could induce hepatic dysfunction in acute settings after cardiac surgery.

Discussion

An argument exists as to whether the liver dysfunction may aggravate the cardiac dysfunction as a part of systemic affection in acute settings. In sepsis-induced cardiomyopathy, model dilatation of both ventricles could occur with associated reduction in ejection fraction, which is reflected in fluid resuscitation and poor response of catecholamines.[25] Sepsis-induced cardiomyopathies have led physicians to hypothesize about the possible systemic nature of myocardial dysfunction in acute hepatic events. Raman et al (►**Table 1**) described liver injury after cardiac surgery and called it severe ischemic early liver injury (SIELI).[26] The authors noted

Table 1 Outcome of high LFT after cardiac surgeries

Reference	Wang et al[28]	Kurian and Paddikkala[29]	UJ and MI[30]	Shahbazi et al[31]	Sabzi and Faraji[39]	Raman et al[26]
Study aims	AST level after CABG is associated with increased mortality and morbidity	effect of oxidative stress on organs as liver and possible role of magnesium	Assess effects of CPB and ACC time on postoperative LFTs	Assess factors influencing LFTs on-pump CABG.	Find out the incidence and significance of high LFTs after open heart cardiac surgery.	Assess the clinical implication, incidence and prognosis of postcardiac surgery SIELI
Study Population	804 isolated CABG patients	92 CABG patients	100 CABG patients	146 on-pump CABG	200 CABG patients	1800 cardiac surgeries analyzed, 20 patients identified
Setting	The prognostic utility of 2× upper limit of normal of AST (N90 U/L) prespecified for adverse outcomes was assessed	Total revascularization was expected	Patients were divided into two groups according to ACC time	Quasiexperimental clinical trial	Patient's characteristics with bilirubin value (1.5 mg or > 1.5 mg) were compared	Group I included 20 patients with ALT > 500 IU/L, Group II included 20 control cases with matched Parsonnet score
Analyzed	AST measurement after surgery within 48 hours	The erythrocytes thiobarbituric acid reactive substances which is a product of lipid peroxidation	Perioperative demographic factors, plus a questionnaire was used as a research tool	Assay of LFTS pre- and postoperative, then define preoperative and intraoperative risk factors for elevation:	LFTs preoperative and postoperative at 24, 48, and 72 hours,	LFTs and organ functions
Period	Two years July 2010–June 2012	Two years from January 2003 to December 2004	1 year	1 year, during 2011	1 year	Data of 4 years was analyzed
Results	Multivariable analysis showed that AST N90 U/L is independently associated with increased 30-day mortality, long term mortality and composite morbidity (OR 12.0, 95% CI 2.99–47.9, $p = 0.001$), (OR 12.0, 95% CI 1.69–34.8, $p = 0.001$) and (OR 3.31, 95% CI 1.56–7.02, $p = 0.002$) respectively	In patients undergoing CABG, a reduced level of antioxidant enzyme activities of erythrocytes in patients during ischemic reperfusion. The thiobarbituric acid reactive substances) in erythrocytes is increased significantly in Mg treated patients.	There was difference between pre- and postoperative ALP and bilirubin showed significant from pre to post-operative levels ($p < 0.05$), but no significant changes observed in other LFTS related to ACC time.	Use of IABP and ACC time were significantly related to the changes in the LFTs except for ALT and ALP. Preoperative central venous pressure had a significant relationship with the changes in AST and ALT.	A significant increase of total bilirubin, AST, and ALP were noted in the 3rd postoperative day. Significant association was found between hypotension and AST and ALP changes.	In SIELI patients, acute renal failure, a low-CI state, and mortality were common. Analysis with linear regression showed that peak ALT predictors included only low CI and diabetes association ($p < 0.05$). Females were more likely to be affected by SIELI in multivariate regression analysis (odds ratio: 6.13; 95% CI 1.08 to 34.82)
Conclusion	AST levels increase within 48 hours of CABG was a strong independent predictor of short- and long-term mortality and morbidity	These results reveal an increase in oxidative stress after CPB in erythrocytes; thereby, it can adversely affect distant organs like liver and kidney. Magnesium treatment enhance the cellular response to ischemia therefore offer cardiac protection	ACC time had a significant effect on postoperative bilirubin and ALP but not on AST and ALT levels. However,pre and post operative values of bilirubin and ALP showed a significant difference.	Reducing ACC and CPB time techniques may be advantageous in protecting the liver function.	Transient alterations of LFTs after CABG surgery presumably due to the decreased hepatic flow, hypoxia, or pump-induced inflammation	A high-mortality is attributed to SIELI after cardiac surgery, the latter is associated with low CO and increased filling pressures

Abbreviations: ACC, aortic cross clamp; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase; CABG, coronary artery bypass grafting; CI, cardiac index; CO, cardiac output; CPB, cardiopulmonary bypass; IABP, intra-aortic balloon pump; LFTs, liver function tests; SIELI, severe ischemic early liver injury.

the high-mortality rate in this group and associated morbidity, including acute kidney injury and low-cardiac index. The hemodynamic profile included higher rates of peak postoperative pulmonary artery occlusion (PAOP) and central venous pressure (CVP) with higher demands of norepinephrine and milrinone. Liver ischemia following cardiac surgery could be driven by an integration of congestion and decreased perfusion.[26] The liver's unique dual blood supply offers a proportional guard against ischemic injury; however, ischemia and necrosis are more common in hepatocytes in zone 3 due to low-oxygen tension in the microcirculation (sinusoidal blood of the hepatic acinus).[27]

Wang et al emphasized the predictive power of aspartate aminotransferase (AST) in predicting morbidity and mortality after cardiac surgery.[28] There is an increase in oxidative stress after CPB in erythrocytes; therefore, it can adversely affect distant organs like liver.[29] Aortic cross-clamp (ACC) may have a significant effect on postoperative bilirubin and alkaline phosphatase (ALP).[30] Shahbazi et al[31] stated that reducing the CPP and ACC times may abort the raise in liver functions (►**Table 1**).

There is no published research to support the theory that acute liver dysfunction induces acute changes in cardiac muscle function. On the contrary, acute heart failure emerges in settings of marked systemic hypotension which is associated with acute cardiopulmonary collapse.[32] Despite the obscurity of profound hypotension, severe hypoxemia could be the culprit behind ischemic hepatitis.[33] Centrilobular necrosis of zone 3 hepatocytes characterizes ischemic liver injury. Histological evidence of inflammation indicative of viral hepatitis is absent.[34] Reduced hepatic blood flow is associated with increased oxygen consumption, but when tissue hypoxia is persistent and end-organ perfusion is inappropriate or when acute shock evolves, reduced hepatic flow exhausts the protecting mechanism against hypoxic liver damage. Liver injury supervenes, which is associated with a sharp elevation of serum AST, alanine aminotransferase (ALT), lactic dehydrogenase (LDH), and prothrombin time prolongation. These changes peak within 1 to 3 days and revert subsequently. The unique dual liver blood supply may be restored back to normal within 5 to 10 days following cardiogenic ischemic hepatitis.[35] Ischemic liver injuries can be differentiated from other forms of acute hepatitis by a marked increase in LDH levels, associated with ALT/LDH ratio of less than 1.5.[36]

Assuming that impaired cardiac function, which is secondary to liver affection after cardiac surgery, does not hold true position unlike in sepsis model where sepsis-induced cardiomyopathy produces impairment of left and right ventricular functions without being selective to one chamber.[37] However, any cause of right ventricular dysfunction can be associated with severe hepatic congestion; patients with hepatic congestion are usually asymptomatic and congestion may be indicated only by abnormal LFTs during routine laboratory analysis. The primary pathophysiology involved in hepatic dysfunction is either passive congestion from increased filling pressures or low-cardiac output and the consequences of impaired perfusion.[37] Interestingly, in the Raman study, the peak postoperative ALT levels correlated significantly with pulmonary artery occlusion and central venous pressures, and negatively with cardiac index.[11] The factors that raise a propensity for liver injury after cardiac surgery include the amount of blood transfused, number of valves replaced, existing chronic hepatic congestion, hypothermia, low-blood supply, hemolysis, sepsis, and renal failure.[2] The LFTs could be affected also by the duration of the ACC and use of an intra-aortic balloon pump (IABP).[31] The possibility of statin-induced acute elevation of LFTs after cardiac surgery is remote.[38] Finally, the transient elevation of LFTs following a coronary artery bypass grafting could be attributed to the decreased hepatic flow, hypoxia, or pump-induced inflammation.[39]

Conclusion

Introducing transaminases for postoperative cardiac surgeries risk assessments represent an important paradigm in perioperative medicine. Assessments of transaminases provide risk prediction and help to allocate resources in the right direction of patient care. Hepatic dysfunction-induced cardiac dysfunction is known in chronic liver conditions, but its value in acute settings needs better understanding. We concluded that s rise in the transaminases after cardiac surgery would favor the ischemic nature of the condition. We anticipate that ongoing research on risk stratifying and treating patients with high transaminases will further improve outcomes for patients undergoing cardiac surgery.

Authors' Contributions

ASO initiated the idea, wrote the manuscript, designed the study, and submitted the manuscript; AKT, as chair of the intensive care department, offered critical revision provided general support. All authors read and approved the final manuscript.

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Conflicts of Interest

None declared.

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