CASE REPORT

Management of Acute Myocardial Infarction in a Liver Transplant Recipient: A Rare Case Report

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Abstract

Liver transplantation (LT) improves outcomes and quality of life in patients with end-stage liver disease (ESLD). As a result of improved accessibility and recipient survival, transplant candidates are becoming increasingly older, have more comorbidities, and experience more long-term complications, all of which create new challenges in post-transplantation care.

In the post-transplant period, a multitude of factors can influence cardiovascular risk in transplant recipients due to aggravation in recipient populations from new-onset dyslipidaemia, hypertension, glucose intolerance, and nephrotoxicity as side effects of immunosuppressive agents.

Traditional cardiovascular risk factors are becoming increasingly prevalent in the ageing population of liver transplant candidates, and coronary artery disease (CAD) is considered to be more common than previously thought. Cardiovascular events are recognised as prominent causes of early and late mortality in liver transplant recipients.

The most common cardiovascular diseases in transplant candidates are ischaemic CAD and cardiomyopathy.

We describe a complex case of a liver transplant recipient in a 50-year-old male patient with no known history of CAD who developed progressive acute myocardial infarction within 6 months of liver transplant and was ultimately thrombolysed to optimise myocardial perfusion. Management of myocardial ischaemia is complicated by a high risk of bleeding in the setting of coagulopathy. Once thrombolysis and haemodynamic stability were achieved, the patient was immediately shifted for coronary angiography, and staged coronary angioplasty was performed for triple vessel coronary disease in the patient at the cardiac institute.

Keywords: Acute myocardial infarction, Case report, Coronary angioplasty, Liver transplant recipient, Thrombolysis.

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Introduction

Liver transplantation (LT) improves outcomes and quality of life in patients with end-stage liver disease (ESLD).[1] As a result of improved accessibility and recipient survival, transplant candidates are becoming increasingly older, have more comorbidities, and experience more long-term complications, all of which create new challenges in post-transplantation care. Liver transplant candidates have complex burdens of cardiovascular disease, at least partly attributed to specific characteristics of ESLD. The hyperdynamic circulation in ESLD that was once considered to lower cardiovascular risk, in combination with poor functional status, masks cardiac conditions during non-invasive cardiac evaluation.[2]

Traditional cardiovascular risk factors are becoming increasingly prevalent in the ageing population of liver transplant candidates, and coronary artery disease (CAD) is more common than previously thought.[3]

In the post-transplant period, a multitude of factors can influence the cardiovascular risk in transplant recipients due to aggravation in recipient populations from the new-onset dyslipidaemia, hypertension, glucose intolerance, and nephrotoxicity as side effects of immunosuppressive agents.[4–7]

Cardiovascular events are recognised as prominent causes of early and late-mortality in liver transplant recipients.[8–11] They are independent negative predictors of outcome following transplantation.

The most common cardiovascular diseases in transplant candidates are ischaemic CAD and cardiomyopathy. We came across such a liver transplant recipient, who developed acute myocardial infarction 6 months after liver transplant. The management of the patient is mentioned in detail in this case report. We searched the literature, but we could not find any such publications, so we decided to publish this case report.

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Case Description

Presentation

A 50-year-old male businessman with a known case of hypertension, recipient of liver transplant 6 months back for hepatocellular carcinoma and HBsAg-positive presented to our emergency department with complaint of sudden onset of chest pain radiating to the left upper arm for the last 45 minutes. The pain was severe in intensity (10/10 VAS) and was associated with 1 episode of vomiting, perspiration, and generalised weakness.

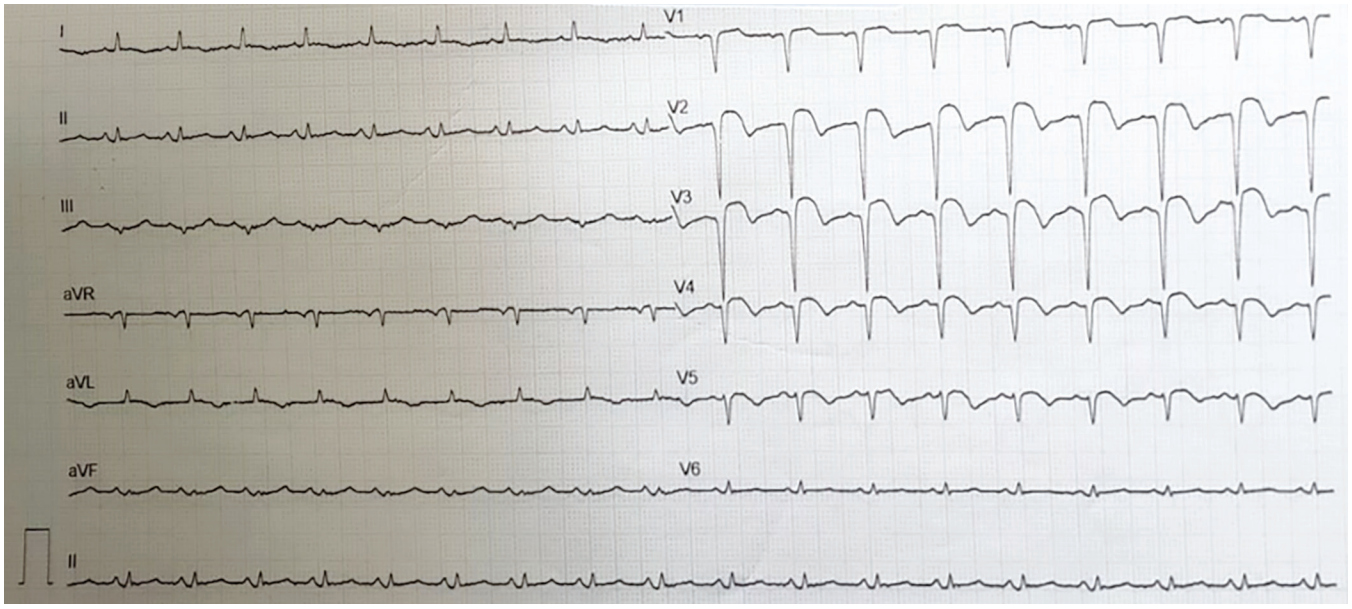

Fig. 1: Twelve-lead electrocardiogram (ECG) showing ST-elevation in V1–V4 suggestive of ST-elevation myocardial infarction (STEMI)

On Arrival in ED

Primary Survey

The airway was patent and clear with no secretion or vomitus in the airway, and the patient was speaking in full sentences.

His breathing was normal, bilateral equal breath sounds were present, and there was no additional breath sounds.

RR – 20/min regular with pulse oximetry showing SpO_2 96% on room air.

Circulation: HR: 100/min regular, BP: 110/80 mm Hg in the supine position in the right upper limb. No gallop or any additional signs of heart failure could be elicited. Mild perspiration was present, but no cyanosis or pallor was present.

His GCS was 15/15, with both pupils normal in size (3–4 mm) and reacting to light. The random blood sugar was 114 mg/dL.

Immediately, 12-lead electrocardiogram (ECG) was taken, which showed ST-segment elevation in chest leads from V1 to V4 (Fig. 1). Therefore, based on clinical symptoms and electrocardiographic monitoring, acute ST-elevation myocardial infarction (STEMI) was diagnosed.

Management

Immediately, the patient was kept on multipara cardiac monitoring and 6-lead ECG with SpO_2, and non-invasive BP monitoring at 10 min intervals was started. A large-bore IV cannula was inserted, and blood samples for CBC, S. Troponin, LFT, RFT, and coagulation profile were collected. A loading dose of chewable tab. aspirin (75 mg) 4 tablets, tab clopidogrel (75 mg) 4 tablets, and tab. atorvastatin (40 mg) 2 tablets were given to the patient. Inj. ondansetron 4 mg IV was given, and inj. tramadol 100 mg in 100 mL normal saline was given IV over 20 minutes in view of severe pain.

Secondary Survey

During further evaluation, it was found that the patient was a known case of hypertension. He had a history of liver transplant (recipient) 6 months back for hepatocellular carcinoma at some transplant institutes, and he was diagnosed as HBsAg-positive 5 months before his liver transplant during his hospital visit for renal stones. Preoperatively, before LT, his cardiac evaluation was not significant, with an EF of 55% in 2D echo. He also had H/o jaundice in childhood and underwent upper GI surgery for large oesophageal varices and PCNL for right renal pelvis stones, but details could not be obtained. He was taking tab. tacrolimus 1 mg BD, tab. aspirin 75 mg OD, tab. metformin 500 mg TDS and tab. dapagliflozin 10 mg OD as post-transplant medications when presented to our department with chest pain.

Point-of-care ultrasound (POCUS) performed in the ED showed severe left ventricular dysfunction (LVD), 30% ejection fraction (EF), regional wall motion abnormalities (RWMA) and anteroseptal wall hypokinesia.

We immediately contacted his liver transplant surgeon on phone, and after thorough discussion, the details and risk of thrombolysis were explained to relatives, and the decision of thrombolysis with inj. streptokinase was taken after obtaining high-risk informed consent from relatives. Thrombolysis was performed with inj. streptokinase 1.5 lakh IU in 100 mL NS glass pint IV over 1 hour considering various factors. During thrombolysis, the patient remained stable.

Troponin-I: >50,000 pg/mL, CPK-MB: 968 U/L, Pro BNP: 498 pg/mL, Na+/K+/Cl–: 144/4.7/108 mEq/L, serum creatinine: 0.87 mg/dL, serum bilirubin: 1.3/0.49/0.81 mg/dL, SGPT/SGOT/ALP: 83/368/130 U/L, total protein/albumin/globulin: 7/4/3 gm/dL, HBsAg-positive status, Hb: 13.2, TLC: 16390, PC: 174000.

After thrombolysis, the patient was sent to the cardiac institute for further management in stable haemodynamic conditions. At that place, the patient underwent coronary angiography on the same day as s/o CAD-TVD in the LCA, LMCA, and RCA, and the patient was advised to undergo CABG. However, relatives refused the same treatment and took leave against medical advice (LAMA). After 5 days, the patient was readmitted to the same cardiac institute, and the decision for double vessel coronary angioplasty was made. Coronary angioplasty was performed with a drug-eluting stent in the left anterior descending (LAD) vessel after 8 days of acute MI. After that, the patient developed an acute kidney injury with increased S. creatinine; hence, percutaneous

transluminal coronary angioplasty (PTCA) to RCA was kept on hold in view of it, and tablet torsemide was kept on hold. After recovery from acute kidney injury, S. creatinine improved, and PTCA in the RCA was performed after 2 months of acute MI.

The patient was discharged in a stable condition with tab. aspirin 75 mg, tab. clopidogrel 75 mg, metformin 500 mg TDS, tab. dapagliflozin 10 mg OD and tab. torsemide 10 mg OD. 2D Echo after 2 months of acute MI showed EF 45%, trivial AR, MR and TR, mild LAD territory hypokinesia, akinetic and thin apical septum and layered LV apical clot of 12 × 12 mm.

On a recent visit to his home after 6 months of angioplasty, the patient was doing well and performing his routine task without any problems.

Discussion

Organ transplantation is growing in frequency, with the most common transplanted organ being the kidney, followed by the liver, heart, and lung.

Most transplant patients require lifelong immunosuppression. Transplant patients can develop several acute to life-threatening emergencies, including transplant-related infection, medication side effects, rejection, graft-vs-host disease and postoperative complications or complications of altered physiology secondary to the transplanted organ.

The most common acute disorders prompting ED visits are infection (39%), followed by non-infectious GI/GU pathology (15%), dehydration (15%), electrolyte disturbances (10%), cardiopulmonary pathology (10%) or injury (8%), and rejection (6%).[12]

Coronary artery disease, sudden cardiac death, and heart failure are the result of premature cardiovascular disease in solid-organ recipients due to underlying comorbidities and metabolic effects of immunosuppression. Hence, when such patients come to the ED, immediate diagnosis and management saves life and reduces mortality and morbidity, which occurred in our case.

Management of coagulopathy in liver transplant recipients is complex in the setting of ongoing myocardial ischaemia due to opposing concerns: correction of coagulopathy may cause plaque-associated thrombus propagation, but not correcting coagulopathy leads to the cascade of bleeding, hypotension, reduced preload, and myocardial perfusion pressure, as well as exacerbation of ischaemia. The use of antifibrinolytics to achieve haemostasis had to be made cautiously in the setting of myocardial ischaemia, with the understanding that it could worsen myocardial blood supply via thrombosis propagation. The decision to correct coagulopathy lies in balancing these opposing priorities and should be goal-directed using clinical findings and point-of-care laboratory tests.

The successful outcome of this medically challenging patient in the emergency department largely depended on the coordinated effort of a multidisciplinary team, which included transplant surgeons, interventional cardiologists, and critical care physicians. Shared decision-making among the different disciplines was of utmost importance to ensure the smooth and successful delivery of care for this patient.[13]

Dr Sheryl S Ang et al. published an article on "Intra-operative Type I Acute Myocardial Infarction During Liver Transplant Requiring Intra-Aortic Balloon Pump: A Case Report" in 2022 describing a complex case of liver transplant who developed progressive intra-operative left ventricular myocardial dysfunction secondary to class I acute myocardial infarction, ultimately requiring intra-operative intra-aortic balloon pump insertion to optimise myocardial perfusion. The management of MI was complicated by bleeding due to coagulopathy, which was corrected, and after stabilisation of homeostasis, the patient underwent coronary angiography followed by angioplasty. The patient had a normal preoperative cardiac workup with a negative history of CAD.

In our case, MI developed after 6 months of liver transplant, and the patient was already on antiplatelets. Furthermore, due to coagulopathy and a recent history of major surgery, the risk of bleeding was high.

Sharma et al. published a case series on "acute heart failure after orthotopic liver transplantation" in which they described three cases of nonischaemic acute HF developing shortly after LT in patients who had a normal preoperative cardiac evaluation. The challenges associated with both the diagnosis and management of acute HF in the setting of a newly implanted graft were discussed.

Manish Tandon, Sunaina Tejpal Karna, Chandra Kant Pandey, and Ravindra Chaturvedi published a case series on the "Diagnostic and therapeutic challenge of heart failure after liver transplant: Case series" in 2017. The study concluded that a retrospective analysis of 360 recipients who underwent LT at their tertiary care institute from 6 years of data identified six patients who developed heart failure in the immediate postoperative period. They diagnosed heart failure by severely decreased left ventricle ejection fraction (LVEF) on echocardiography. Clinical presentation was similar in all six recipients, and only two survived.[14]

Conclusion

Myocardial infarction in a liver transplant recipient is a very rare condition. When such patients come to the ED, it is a major challenge for on-duty emergency physicians to proceed, specifically when the patient is already on anticoagulants. Except for tertiary care, the ED cardiologist and Cath lab is not available everywhere, and one has to take care of the heart as well as the liver graft. Therefore, one should be very careful to manage such cases.

Take-home Message

When such patients come to the ED through examination and in time, discussion and decisions regarding the line of treatment can prevent morbidity and mortality and maintain quality of life.

Disclaimer

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