Abstract: P3466

Cardiac output in end-stage liver disease increases with the severity of liver dysfunction

Authors:
AN Koshy¹, B Cailes¹, P Gow², A Testro², JK Sajeev¹, H Han¹, J Ko¹, L Weinberg³, H Lim¹, A Teh¹, O Farouque¹, ¹Austin Health Hospital, Department of Cardiology - Melbourne - Australia, ²Austin Health Hospital, Victorian Liver Transplant Unit - Melbourne - Australia, ³Austin Health Hospital, Department of Anaesthesia - Melbourne - Australia,

Topic(s): Non-cardiac Surgery/Pre-surgical Assessment

Citation:

Background:

End-stage liver disease is associated with significant systemic and haemodynamic alterations that affect cardiac function. Cirrhotic cardiomyopathy remains an ill-defined entity among cardiologists. Understanding the complex interplay between liver dysfunction and cardiac function can lead to a better understanding of the compensatory mechanisms of the heart in liver failure.

Purpose:

To investigate whether severity of liver disease affects baseline cardiac output in a large contemporary cohort of patients undergoing liver transplant work-up.

Methods:

Consecutive patients that underwent pre-liver transplant (LT) workup between 2010-2017 were included. All patients underwent a resting echocardiogram. Cardiac output (CO) was prospectively recorded at baseline by pulsed-wave Doppler examination of the left ventricular outflow tract from the apical window and systemic vascular resistance (SVR) was calculated as 80 x (mean arterial pressure (MAP)/CO). Severity of liver disease was characterized by the model of end-stage liver disease (MELD) and Child-Pugh scores.

Results:

560 patients were included (mean age 57.5 ± 7.7, 74.8% male). Mean MELD score was 19 ±7 and Child-Pugh Score was 9 ±3. There was an inverse linear relationship between the severity of liver disease by the MELD score and baseline SVR (rho 0.40, P<0.001). As SVR reduced, there was also a significant rise in baseline CO with a strong inverse correlation between the two variables (rho 0.86, p< 0.001). There was a significant linear correlation between the severity of liver disease and baseline CO with both the scores (MELD Score rho 0.42, p<0.001; Child Pugh rho 0.44, p<0.001) (Figure).

Conclusions:

Baseline CO increased with the severity of liver dysfunction due to a reduced afterload. A higher resting CO may lead to patients encroaching on their cardiac reserve at rest. This provides a pathophysiological insight suggesting a limited role for beta-blockers, particularly in patients with advanced liver cirrhosis.
Abstract: P3466
Cardiac output in end-stage liver disease increases with the severity of liver dysfunction
Authors: AN Koshy 1, B Cailes 1, P Gow 2, A Testro 2, JK Sajeev 1, H Han 1, J Ko 1, L Weinberg 3, H Lim 1, A Teh 1, O Farouque 1
1 Austin Health Hospital, Department of Cardiology - Melbourne - Australia, 2 Austin Health Hospital, Victorian Liver Transplant Unit - Melbourne - Australia, 3 Austin Health Hospital, Department of Anaesthesia - Melbourne - Australia,
Topic(s): Non-cardiac Surgery/Pre-surgical Assessment
Citation: Background: End-stage liver disease is associated with significant systemic and haemodynamic alterations that affect cardiac function. Cirrhotic cardiomyopathy remains an ill-defined entity among cardiologists. Understanding the complex interplay between liver dysfunction and cardiac function can lead to a better understanding of the compensatory mechanisms of the heart in liver failure.
Purpose: To investigate whether severity of liver disease affects baseline cardiac output in a large contemporary cohort of patients undergoing liver transplant work-up.
Methods: Consecutive patients that underwent pre-liver transplant (LT) work-up between 2010-2017 were included. All patients underwent a resting echocardiogram. Cardiac output (CO) was prospectively recorded at baseline by pulsed-wave Doppler examination of the left ventricular outflow tract from the apical window and systemic vascular resistance (SVR) was calculated as 80 × (mean arterial pressure (MAP)/CO). Severity of liver disease was characterized by the model of end-stage liver disease (MELD) and Child-Pugh scores.
Results: 560 patients were included (mean age 57.5 ± 7.7, 74.8% male). Mean MELD score was 19 ± 7 and Child-Pugh Score was 9 ± 3. There was an inverse linear relationship between the severity of liver disease by the MELD score and baseline SVR (ρ 0.40, P<0.001). As SVR reduced, there was also a significant rise in baseline CO with a strong inverse correlation between the two variables (ρ 0.86, p<0.001). There was a significant linear correlation between the severity of liver disease and baseline CO with both the scores (MELD Score ρ 0.42, p<0.001; Child Pugh ρ 0.44, p<0.001) (Figure).
Conclusions: Baseline CO increased with the severity of liver dysfunction due to a reduced afterload. A higher resting CO may lead to patients encroaching on their cardiac reserve at rest. This provides a pathophysiological insight suggesting a limited role for beta-blockers, particularly in patients with advanced liver cirrhosis.