

Can Coronary Vein Size Predict Hemodynamic Instability During Inferior Vena Cava Clamping in Liver Transplantation?

C.-E. Huang, S.-C. Yang, C.-L. Chen, Y.-F. Cheng, K.-W. Cheng, C.-H. Wang, S.-C. Wu, C.-J. Huang, B. Jawan, and T.-H. Shih

ABSTRACT

Objective. This study aimed to determine whether coronary vein size can serve as a predictor of hemodynamic instability during inferior vena cava clamping in living-donor liver transplantations.

Methods. Fifty-two patients' hemodynamic data before and after clamping were retrospectively analyzed and compared with the use of linear regression and repeated measurement. Data included arterial blood pressure, heart rate, central venous pressure, cardiac output, cardiac index, stroke volume, stroke volume variation, and systemic vascular resistance.

Results. The values of hemodynamic parameters at 1, 3, 10, and 30 minutes after clamping were compared with baseline data. All changes were found to be significant when the presence of the coronary vein was not considered. When the coronary vein was taken into consideration, linear regression analysis showed that only the percentage changes of cardiac index; stroke volume at 1, 3, and 10 minutes; and systemic vascular resistance at 1 minute after portal and inferior vena cava clamping were significantly correlated with the presence of the coronary vein.

Conclusions. Coronary vein size is a weak predictor of hemodynamic tolerability and instability during portal vein and inferior vena cava clamping in this kind of surgery.

TOTAL CLAMPING of the portal vein and inferior vena cava (IVC) during the anhepatic phase in liver transplantation (LT) without venovenous bypass (VVB) may cause significant hemodynamic changes due to sudden decrease of venous return [1,2]. In our LT program, VVB is not used in pediatric or adult LT. We have observed that the tolerability or degree of severity in hemodynamic changes caused by IVC clamping varies widely among patients and is therefore not predictable. It has been reported that even baseline cardiac index does not predict hemodynamic instability during LT [3]. The mechanisms behind these unpredictable changes remain unclear and need to be further studied.

Portal hypertension is common in patients with chronic liver disease who require LT. High portal pressure results in the formation of portosystemic collaterals that divert portal blood into the systemic circulation [4]. The utility of portosystemic collateral presence as a predictor of hemodynamic stability or tolerability for IVC clamping in LT is not currently known. The present study aimed to evaluate

0041-1345/14/\$-see front matter http://dx.doi.org/10.1016/j.transproceed.2013.11.024 whether the size of the coronary vein can act as a predictor of hemodynamic instability after total clamping of the IVC and portal vein without VVB in adult living-donor LT.

PATIENTS AND METHODS

Approval to review patient charts was obtained from the Institutional Review Board (99-2234B). The anesthesia records of adult patients who underwent living-donor LT from May 2010 to October 2010 in Kaohsiung Chang Gung Memorial Hospital, Taiwan, were

© 2014 by Elsevier Inc. All rights reserved. 360 Park Avenue South, New York, NY 10010-1710

From the Department of Anesthesiology (C.-E.H., S.-C.Y., K.-W.C., C.-H.W., S.-C.W., C.-J.H., B.J., T.-H.S.), Department of Surgery (C.-L.C.), and Department of Radiology (Y.-F.C.), Kaohsiung Chang Gung Memorial Hospital and Chang Gung University College of Medicine, Kaohsiung, Taiwan.

The first two authors contributed equally in this work.

Address reprint requests to Tsung-Hsiao Shih, Department of Anesthesiology, Kaohsiung Chang Gung Memorial Hospital, Taiwan. E-mail: 8802057@adm.cgmh.org.tw

	Preclamp	1 min	3 min	10 min	30 min	60 min	P value			
CI	4.0 ± 0.9	$\textbf{3.3}\pm\textbf{0.8}$	$\textbf{3.2}\pm\textbf{0.7}$	$\textbf{3.3}\pm\textbf{0.7}$	3.3 ± 0.6	$\textbf{3.2}\pm\textbf{0.6}$.0001			
SV	40 ± 10.2	$\textbf{31.4} \pm \textbf{8.3}$	$\textbf{30.1} \pm \textbf{7.2}$	$\textbf{31.8} \pm \textbf{7.3}$	$\textbf{31.4} \pm \textbf{7.0}$	$\textbf{30.9} \pm \textbf{7.3}$.0001			
SVV	13.1 ± 6.5	$\textbf{16.4} \pm \textbf{7.1}$	15.5 ± 6.5	$\textbf{16.1} \pm \textbf{6.6}$	$\textbf{16.9} \pm \textbf{6.9}$	18.0 ± 6.9	.0001			
HR	102.9 ± 14.9	106.8 ± 16.6	107.5 ± 16.4	108.0 ± 16.0	108.3 \pm 16.3	107.9 ± 15.4	.0001			
MAP	$\textbf{74.3} \pm \textbf{7.8}$	69.0 ± 7.2	67.4 ± 7.0	68.4 ± 5.7	69.4 ± 5.4	69.1 ± 5.3	.0001			
CVP	10.7 ± 2.5	$\textbf{9.5} \pm \textbf{2.4}$	$\textbf{9.7} \pm \textbf{2.5}$	10.1 ± 2.5	10.5 ± 2.5	10.8 ± 2.5	.0041			
SVR	761 ± 118	$\textbf{876.5} \pm \textbf{227.0}$	$\textbf{885.3} \pm \textbf{280.9}$	836.7 ± 212.9	850.9 ± 181.2	854.8 ± 173.7	.0001			

Table 1. Changes in Measured CI, SV, SVV, HR, MAP, CVP, and SVR Using Repeated Measurement Before and After Clamping of the IVC, Without Considering the Presence or Size of Coronary Vein

Abbreviations: CI, cardiac index; L/min/m²; SV, stroke volume, mL; SWV, stroke volume variation, %; HR, heart rate, beats/min; MAP, mean arterial pressure, mm Hg; CVP, central venous pressure, mm Hg; SVR, systemic vascular resistance, dyn*s/cm⁵.

reviewed retrospectively. Patients with body weight ${<}40~{\rm kg}$ and age ${<}12~{\rm years}$ were excluded.

The anesthesia and monitoring of the patients were the same as previously reported [5] except for cardiac output monitoring, for which an additional, minimally invasive, monitor was used (Flotrac Sensor; Edwards Lifesciences, Irvine, California; and Vigileo monitor; Edwards Lifesciences).

Because hemodynamic data varied drastically upward and downward during the IVC clamping period, the means of the data during the last 5 minutes before IVC clamping were considered to be baseline, and the mean data from 0–1, 1–3, 3–10, and 10–30 minutes were used as representative data for 1, 3, 10, and 30 minutes, respectively. The percentage changes in the parameters were derived from the calculation of measured values at baseline and each time point after IVC clamping. The size of the coronary vein diameter was routinely measured by computerized tomographic angiography (CTA) before the LT operation [6].

The changes in hemodynamic data, such as mean arterial blood pressure (BP), heart rate (HR), central venous pressure (CVP), cardiac output (CO), cardiac index (CI), stroke volume (SV), stroke volume variation (SVV), and systemic vascular resistance (SVR), from baseline were analyzed by repeated measurements. These data were further analyzed by linear regression, in which they were all treated as dependent factors, with the size of the coronary vein diameter as the only independent factor. A *P* value of <.05 was considered to be significant.

RESULTS

Fifty-two patients were included in this study. The anhepatic phase time was 1.16 ± 0.28 hours, ie, only 8.7% of the total anesthesia time, but 32% of the red blood cells and 21% of the fresh frozen plasma transfusion were given during that time. The patients also received during that time 29% and 28%, respectively, of the total 5% albumin and crystalloids received. In the anhepatic phase, 43% of the sodium bicarbonate and 37% of the calcium gluconate also were given.

Table 1 presents the changes in measured CI, SV, SVV, HR, MAP, CVP, and SVR before and after clamping of the portal vein and IVC, with the presence and size of the coronary vein not taken into consideration. These data points were significantly different for as long as the portal vein and IVC were clamped. When the presence of the coronary vein was taken into account, linear regression analysis showed that only some of the changes remained significant. Specifically, percentage changes of CI at 1 and 3 minutes; SV at 1, 3, and 10 minutes; and SVR at 1 minute after portal and IVC clamping were significantly correlated with the presence of the coronary vein. No significant change was noted for the other parameters or other time points (Table 2). The R^2 value was relatively small, indicating that the coronary vein is a weak predictor for the tolerability of IVC clamping.

Note that most parameters tended to change by a smaller amount as the size of the coronary vein increased, except CVP. This value decreased initially, but increased to a value even greater than that its preclamp level after fluid resuscitation (Table 1).

DISCUSSION

The results of repeated-measurement analysis showed that the final values in all the measured and monitored

Table 2. Results of Linear Regression Analysis of the Percentage Changes in Hemodynamic Data With the Size of the Coronary Vein Diameter (cm)

Ven Diameter (Chi)								
	Time	Change, %	R ²	P value				
CI	1 min	-17.0 ± 16.8	0.152	.004*				
	3 min	-19.2 ± 17.1	0.097	.022*				
	10 min	-15.5 ± 13.7	0.060	.073				
	30 min	-16.0 ± 14.4	0.042	.139				
SV	1 min	-19.7 ± 17.1	0.139	.005*				
	3 min	-22.7 ± 17.0	0.092	.026*				
	10 min	-18.7 ± 14.1	0.073	.048*				
	30 min	-19.5 ± 14.6	0.054	.091				
HR	1 min	$\textbf{3.8} \pm \textbf{8.5}$	0.005	.608				
	3 min	4.6 ± 8.9	0.006	.575				
	10 min	5.1 ± 8.2	0.006	.573				
	30 min	$\textbf{5.4} \pm \textbf{9.5}$	0.006	.577				
MAP	1 min	-6.4 ± 10.8	0.030	.216				
	3 min	-8.6 ± 10.6	0.052	.101				
	10 min	-7.2 ± 9.3	0.042	.140				
	30 min	-5.8 ± 9.5	0.016	.356				
CVP	1 min	-8.8 ± 18.1	0.002	.754				
	3 min	-7.4 ± 19.6	0.001	.804				
	10 min	-3.5 ± 18.9	0.008	.527				
	30 min	-0.1 ± 20.7	0.010	.474				
SVR	1 min	17.0 ± 24.7	0.083	.036*				
	3 min	17.1 ± 24.7	0.043	.136				
	10 min	11.0 ± 15.8	0.019	.329				
	30 min	14.1 ± 19.7	0.019	.323				

Abbreviations as in Table 1.

*P < .05.

Download English Version:

https://daneshyari.com/en/article/4258372

Download Persian Version:

https://daneshyari.com/article/4258372

Daneshyari.com