JTI
JOURNAL OF
TRAUMA AND INJURY

ORIGINAL ARTICLE
J Trauma Inj 2020;33(1):38-42
https://doi.org/10.20408/jti.2020.014

Immediate Post-laparotomy Hypotension in Patients with Severe Traumatic Hemoperitoneum

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Received: March 20, 2020
Revised: March 23, 2020
Accepted: March 24, 2020

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This paper was presented at 2017 5th Pan Pacific Trauma Congress.

Purpose: Immediate post-laparotomy hypotension (PLH) is a precipitous drop in blood pressure caused by a sudden release of abdominal tamponade after laparotomy in cases of severe hemoperitoneum. The effect of laparotomy on blood pressure in patients with significant hemoperitoneum is unknown.

Methods: In total, 163 patients underwent laparotomy for trauma from January 1, 2013 to December 31, 2015. Exclusion criteria included the following: negative laparotomy, only a hollow viscous injury, and hemoperitoneum <1,000 mL. After applying those criteria, 62 patients were enrolled in this retrospective review. PLH was defined as a decrease in the mean arterial pressure (MAP) ≥10 mmHg within 10 minutes after laparotomy.

Results: The mean estimated hemoperitoneum was 3,516 mL. The incidence of PLH was 23% (14 of 62 patients). The MAP did not show significant differences before and after laparotomy (5 minutes post-laparotomy, 67.5±16.5 vs. 68.3±18.8 mmHg; p=0.7; 10 minutes post-laparotomy, 67.5±16.5 vs. 70.4±18.8 mmHg; p=0.193). The overall in-hospital mortality was 24% (15 of 62 patients). Mortality was not significantly higher in the PLH group (two of 14 [14.3%] vs. 13 of 48 [27.1%]; p=0.33). No statistically significant between-group differences were observed in the intensive care unit and hospital stay.

Conclusions: PLH may be less frequent and less devastating than it is often considered. Surgical hemostasis during laparotomy is important. Laparotomy with adequate resuscitation may explain the equivalent outcomes in the two groups.

Keywords: Hypotension; Wounds and injuries; Shock; Intra-abdominal hypertension; Hemorrhage

http://www.jtraumainj.org
eISSN 2287-1683
pISSN 1738-8767

INTRODUCTION

Blood pressure management is crucial during surgery, as intraoperative hypotension can cause severe complications, such as myocardial infarction and stroke [1-4]. Immediate post-laparotomy hypotension (PLH) is a precipitous drop in blood pressure caused by the sudden release of abdominal tamponade after laparotomy in cases of severe hemoperitoneum. It is a feared phenomenon by anesthesiologists, and the concerns of some anesthesiologists regarding PLH may delay emergency laparotomy until they are able to prepare more blood for transfusion and other equipment. Adequate preoperative preparation and resuscitation require time and effort. In these cases, we must decide whether to open the abdomen immediately to stop the bleeding or to wait for the blood to be transfused. The most common cause of shock in trauma is hemorrhage [5-11]. Therefore, controlling the source of bleeding is an essential and urgent component of trauma care. However, the effect of laparotomy on blood pressure in patients with significant hemoperitoneum is unknown. This study aimed to elucidate the frequency of PLH and to determine the effect of laparotomy to blood pressure in cases of significant hemoperitoneum.

METHODS

A retrospective cohort study was performed, involving a total of 163 patients who underwent laparotomy for trauma at the Gil Medical Center which is the regional trauma center of Incheon, Korea from January 1, 2013 to December 31, 2015. Patients with a negative laparotomy, hollow viscous injury, and hemoperitoneum <1,000 mL were excluded from this study because these conditions do not lead to any significant increase in intra-abdominal pressure. After applying the above criteria, 62 patients were ultimately enrolled in the current study. PLH was defined as a decrease in the mean arterial pressure (MAP) ≥10 mmHg within 10 minutes after laparotomy. The MAP at 5 minutes before laparotomy was compared to the MAP at 5 and 10 minutes after laparotomy. We divided the enrolled patients into two groups (PLH and non-PLH) and compared variables between the groups. The primary outcome of the study was in-hospital mortality, and the secondary outcome was intensive care unit (ICU) and hospital stay.

Continuous data were presented as means±standard deviation. Differences in nominal and categorical variables were compared using the Fisher's exact test, while the Mann-Whitney *U* test was utilized to compare differences between continuous variables. The MAP readings before and after laparotomy were compared using the paired *t*-test. Binary logistic regression was used in the multivariate analysis. A two-tailed *p*-value <0.05 was considered to indicate statistical significance. All statistical analyses were performed with SPSS version 19.0 for Windows (IBM Corp., Armonk, NY, USA).

RESULTS

The mean estimated hemoperitoneum was 3,516 mL. The incidence of PLH was 23% (14 of 62 patients) when the preoperative MAP was compared with the MAP at 5 minutes after laparotomy. When we compared the preoperative MAP with the MAP at 10 minutes after laparotomy, the incidence was 26% (16 of 62 patients). The incidence of MAP elevation was 60% (37 of 62 patients) at 5 minutes and 63% (39 of 62 patients) at 10 minutes after laparotomy.

When we defined PLH as a decrease in MAP at 5 minutes after laparotomy, among the variables that we investigated, the initial MAP of the PLH group was significantly higher than that of the non-PLH group (82.0±18.4 vs. 64.6±17.6 mmHg; p=0.006), as illustrated in Table 1. The initial lactate level of the PLH group was also higher than that of the non-PLH group (7.8±5.0 vs. 4.9±3.5 mmol/L; p=0.046), as presented in Table 1.

The MAP before laparotomy was not significantly different from the MAP after laparotomy at either time point (5 minutes after laparotomy, 67.5±16.5 vs. 68.3±18.8 mmHg; p=0.7; 10 minutes after laparotomy, 67.5±16.5 vs. 70.4±18.8 mmHg; p=0.193).

The overall in-hospital mortality rate was 24% (15 of 62 patients). PLH did not significantly increase mortality (two of 14 [14.3%] with PLH vs. 13 of 48 [27.1%] without PLH; p=0.33) (Table 2). No statistically significant be-

tween-group differences were observed in ICU and hospital stay (Table 2). The multivariate analysis showed that PLH was not an adverse factor associated with in-hospital mortality (Table 3).

DISCUSSION

The current study was designed to examine the incidence of PLH and the effect of laparotomy to patients with significant intra-abdominal bleeding. Depending on one's point of view on PLH, there may be a conflict of opinion between the anesthesiologist and the surgeon, but little evidence exists regarding this issue. To the best of our knowledge, this is first study to explore the incidence of PLH.

We had to define PLH, due to the lack of consensus in the literature. We chose a definition of PLH in this study as a decrease in the MAP of ≥10 mmHg within 10 minutes after laparotomy, because the MAP within 10 minutes after laparotomy would reflect its effect better than other possible definitions.

Although no statistical significance was observed, in the present study, it was found that the MAP did not decrease but rather increased at both 5 minutes and 10 minutes after laparotomy. This observation is counterintuitive, but a plausible explanation draws upon the pathophysiology and management of abdominal compartment syndrome (ACS) [12,13]. A variety of factors, ranging from medical to surgical, can cause intra-abdominal hypertension (IAH), but one of the most important causes observed in trauma patients is intra-abdominal hemorrhage [12]. In particular, in cases of hemoperitoneum caused by arterial bleeding, IAH can soon become aggravated, progressing to ACS. Once a patient develops ACS, more complex events occur, including a sudden collapse of venous return that aggravates hypotension, ultimately resulting in a vicious cycle [12]. It is likely that a patient whose abdomen is distended due to hemoperitoneum has already fallen into ACS, in which case the

Table 1. Comparison of demographic and physiologic variables between the PLH and non-PLH groups

	PLH (n=14)	Non-PLH (n=48)	*p*-value
Age (years)	50.7±20.3	51.6±17.2	0.86
Male/female ratio	0.64 (9/14)	0.89 (41/48)	0.008
Blunt mechanism	81.2	76.1	0.745
Inotropic agent	100 (14/14)	82.6 (38/48)	0.08
Injury severity score	23.3±10.8	22.1±11.1	0.73
RTS	7.1±1.2	6.3±1.3	0.12
RBC transfusion (pack)	3.9±2.1	4.3±3.5	0.61
Initial MAP (mmHg)	82.0±18.4	64.6±17.6	0.006
Initial Hb (g/dL)	9.7±3.4	11.2±3.0	0.1
pH	7.0	7.0±0.1	0.55
Lactate (mmol/L)	7.8±5.0	4.9±3.5	0.046

Values are presented as mean±standard deviation or number (%).
PLH: post-laparotomy hypotension, RTS: Revised Trauma Score, RBC: red blood cell, MAP: mean arterial pressure, Hb: hemoglobin.

Table 2. Comparison of outcomes between the PLH group (at 5 minutes after the laparotomy) and the non-PLH group

	PLH (n=14)	Non-PLH (n=48)	*p*-value
Mortality rate	14.3 (2/14)	27.1 (13/48)	0.33
ICU stay (day)	9.0±11.0	10.9±19.7	0.725
Hospital stay (day)	18.0±13.3	30.4±36.2	0.053

Values are presented as mean±standard deviation unless otherwise indicated.
PLH: post-laparotomy hypotension, ICU: intensive care unit.

Table 3. Multivariate analysis of in-hospital mortality after laparotomy

	OR	95% CI	*p*-value
PLH	2.4	0.2–36.2	0.51
EBL	1.0	0.9–1.0	0.17
Mechanism	0.6	0.04–10.6	0.78
Initial MAP	1.0	0.97–1.08	0.34
Hb	1.2	0.8–1.7	0.24
Lactate	1.0	0.6–1.6	0.88
ISS	0.9	0.8–1.0	0.18
Transfusion	1.0	0.6–1.6	0.78

OR: odds ratio, CI: confidence interval, PLH: post-laparotomy hypotension, EBL: estimated blood loss, MAP: mean arterial pressure, Hb: hemoglobin, ISS: Injury Severity Score.

https://doi.org/10.20408/jti.2020.014

appropriate management strategy is laparotomy [12,13]. Once laparotomy is performed, the restored venous return compensates for the sudden release of tamponade [14,15].

The serum lactate level of the PLH group was significantly higher than that of non-PLH group. Furthermore, although the difference was not statistically significant, the PLH group had lower initial hemoglobin levels than the non-PLH group. This might suggest that the physiological condition of the PLH group was poorer than that of the non-PLH group. The initial MAP of the PLH group was higher than that of the non-PLH group, which might have been caused by more the frequent use of inotropic agents in the PLH group than in the non-PLH group. Interestingly, even though there were no significant differences, the outcomes analyzed in this study—including mortality rate, ICU stay, and hospital stay—were better in the PLH group than in the non-PLH group. We could not determine whether this result was caused by coincidence, so a follow-up study should be conducted with a larger number of patients.

There are limitations of this study. First, this study was retrospective in nature. There could not be a thorough evaluation of changes in the amount of inotrophic agent use and fluid/blood infusion during the peri-laparotomy period in the current study. Second, the intra-abdominal pressure could not be estimated. Routinely checking the intra-abdominal pressure in patients with hemoperitoneum would be very helpful for evaluating our hypothesis.

CONCLUSION

In conclusion, PLH may be less frequent and less devastating than it is often considered. Surgical hemostasis duri ng laparotomy is important, as is preparation to manage PLH in patients with significant hemoperitoneum. The effect of this preparation on outcomes requires further study. Laparotomy with adequate resuscitation may explain the equivalent outcomes in the two groups.

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