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ORIGINAL ARTICLE

Clinical and echocardiographic predictors for post-induction hypotension during general anesthesia

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ABSTRACT

Background: Post-induction hypotension (PIH) is a frequent complication in patients undergoing surgery under general anaesthesia in daily anaesthetic practice. We hypothesized that the previous complication might be linked to the preoperative cardiac status. We conducted this investigation to estimate the incidence and predictors (clinical and echocardiographic) of PIH.

Methods: We included 150 patients who underwent preoperative echocardiography before undergoing surgery under general anaesthesia. Their data were retrospectively reviewed. The incidence of PIH was estimated, and the patients were divided into two groups: No-PIH and PIH groups.

Results: PIH was detected in 56 cases (37.33%). The female gender showed a higher prevalence in the PIH group. However, the reported medications and systemic comorbidities did not have a significant effect on that complication, apart from heart failure, which increased in association with PIH. The same group had significantly lower haemoglobin levels. Regarding echocardiographic findings, the PIH group expressed higher end-systolic and diastolic left ventricular diameters, deceleration time, and E/e' ratio, while the same group showed lower ejection fraction, E/A ratio, and inspiratory IVC diameter. The presence of valvular pathology was not associated with PIH. Although anaesthetic drugs were comparable between the two groups, epidural analgesia was significantly associated with PIH.

Conclusion: Female gender, heart failure, low haemoglobin levels, high end-systolic and diastolic ventricular diameter, low ejection fraction, low E/A ratio, prolonged deceleration time, increased E/e', decreased inspiratory IVC diameter, and epidural analgesia were significant risk factors for PIH.

Keywords: Post-induction hypotension; Predictors; Echocardiography.

INTRODUCTION

Post-induction hypotension (PIH) is a frequent complication that is encountered in about 30% of patients undergoing surgical procedures under general anaesthesia **[1, 2]**. This drop in arterial pressure may cause harm to organs with high blood flow leading to serious health consequences like stroke, acute kidney injury, or myocardial ischemia **[3-6]**. Post-operative mortality rates have been linked to this entity **[1]**. Contrarily, maintaining blood pressure within the normal ranges during surgery has been associated with a 25% reduction of post-operative organ dysfunction **[7]**. PIH usually occurs immediately after induction of general anaesthesia, and it is not related to the type of surgical intervention, its invasiveness, duration of operation, or the amount of intraoperative blood loss **[8]**. Based on the timing of this complication, one should suspect an interaction between the inducting anaesthetic agents and patients' cardiac performance status, which may be responsible for PIH **[1, 9]**. Although many clinical risk factors

have been established for PIH, including old age, diabetes mellitus, high American Society of Anesthesiologists class, propofol administration, and high fentanyl doses **[10, 11]**. Nevertheless, studies linking PIH, and preoperative cardiac performance status are lacking in the current literature. Nowadays, transthoracic echocardiography has been widely used for the preoperative assessment of cardiac status, especially in patients with cardiac disease. It could ass the risk of cardiovascular events that might affect patient hemodynamics during general anaesthesia **[12, 13]**. Preoperative echocardiography was associated with a significant decline in post-operative heart failure, which ensures the value of this modality in the preoperative workup **[14]**. It is crucial for all physicians, including anesthesiologists, to seek independent echocardiographic parameters that may be significantly linked to PIH. This would allow us to understand the exact mechanism of PIH and to create a preventive strategy for this complication based on the preoperative echocardiographic data. Thus, we conducted the current investigation to estimate the incidence and predictors (clinical and echocardiographic) of PIH.

METHODS

This retrospective research was conducted at Mansoura University Hospitals after obtaining approval from the Institutional Review Board (IRB) of our medical school. The study was designed for adult patients who underwent elective surgical procedures, whatever their type, under general anaesthesia in the same hospitals during the period between January 2018 and December 2020. All these patients had undergone transthoracic echocardiography within six months prior to the surgical procedure. Contrarily, we excluded patients requiring emergency surgery, requiring spinal anaesthesia, missed medical data, having echocardiography older than six months before surgery, or having unstable ischemic heart disease. The data of consecutive 150 patients who met the previous criteria were collected and reviewed. They were divided into two groups based on the incidence of PIH: PIH and No-PIH groups. PIH was defined as a decrease in mean arterial pressure (MAP) ≤50 mmHg, at least a one-time point, after administration of the inducting anaesthetic agent and before surgery **[1]**. Before admission, all patients received the standard preoperative assessment, including detailed history taking, thorough clinical examination, routine preoperative investigations, and radiological assessment. During history taking, we focused on the presence of systemic comorbidities (diabetes, hypertension, ischemic heart disease, atrial fibrillation) and commenced medications with their dose and regimen.The echocardiographic assessment was performed by an experienced cardiologist with the aid of a 2D echocardiography machine (Phillips, Netherlands). Quantification of cardiac chambers, including left ventricular (LV) diameters, was done according to the published guidelines **[15]**. At the end-diastole, the thickness of the posterior ventricular wall along with the interventricular septum was measured. In addition, LV ejection fraction (EF) was estimated according to the Teichholz method in patients with no regional wall motion abnormality (RWMA). RWMA was established a cardiac segment, or more was classified as akinetic, hypokinetic, or dyskinetic after calculating the LV wall motion score **[16]**. If RWMA was detected, the biplane disc summation method was applied to estimate both end-systolic and diastolic volumes from the apical view, while LVEF was estimated using the following formula: "100 × (end-diastolic volume - end-systolic volume)/end-diastolic volume". The mitral inflow velocities were assessed via Doppler imaging, and they were used to evaluate the conventional diastolic parameters. The peak early (E) was estimated from the pattern of mitral inflow velocity, whereas peak A wave was expressed as the peak velocity in late diastole from the mitral inflow velocity pattern. The time interval between the peak E wave to the baseline zero velocity was taken as deceleration time. After measuring the peak early diastolic velocity (e') of the annulus, the ratio of peak early diastolic transmitral flow velocity to annular velocity (E/e') was calculated. In subjects with atrial fibrillation, E/e' was estimated using the average value of three heartbeats to decrease errors during measurement **[17]**. The diagnosis of valvular disease (stenosis or regurgitation) and grading of its severity was made according to the most recent guidelines **[18]**. The systolic pressure of the pulmonary artery was calculated by adding the pressure gradient of tricuspid regurgitation to the right atrial pressure (estimated from inferior vena cava diameter) **[19]**. On the operation day, patients were transferred to the operative theatre, where basic hemodynamic monitoring was established. Anaesthesia was induced by propofol (1 to 2 mg/kg) or midazolam (20 - 100 μg/kg), followed by morphine or fentanyl, while it was maintained via either isoflurane, sevoflurane, or propofol infusion. An epidural catheter was inserted before induction in some patients when required, and 2 to 3 ml of lidocaine 1% were administered as the initial dose.

Sample size calculation Sample size was calculated using the equation published by Burmeister, E. and Aitken, L.M (2012) **[20]**

modification of Green's equation for regression sample size calculation (1991) **[21]** with Risk factors for post-induction hypotension in general anesthesia as the primary outcome. The null hypothesis was considered as absence of predictors of post-induction hypotension. K. Tarao et al. **[1]** reported 8 predictors of post-induction hypotension. According to by Burmeister, E. and Aitken, L.M equation, $N \geq 50 + 8p$ where p is the number of predictors. A sample size of 114 patients is needed to achieve 80% power (1-β or the probability of rejecting the null hypothesis when it is false) in the proposed study with a significance level (α or the probability of rejecting the null hypothesis when it is true) of 0.05. The result will be rounded up to 150 an ensure sufficient regression model power. **Statistical analysis** IBM's SPSS statistics (Statistical Package for the Social Sciences) for windows (version 25) will be used for statistical analysis of the collected data. Shapiro-Wilk test will be used to check the normality of the data distribution. Normally distributed continuous variables will be expressed as mean ± SD while categorical variables and the abnormally distributed continuous ones will be expressed as median and inter-quartile range or number and percentage (as appropriate). Student t test and Mann-Whitney will be used for normally and abnormally distributed continuous data respectively. Chi square test will be used for categorical data using the crosstabs' function. All tests will be conducted with 95% confidence interval. If needed, bivariate correlations will be assessed using Pearson's or Spearman's correlation coefficient depending on the nature of data. P (probability) value < 0.05 will be considered statistically significant.

Table (1): Patient demographic characteristics according to the presence of PIH in the current study.

		No PIH (n= 94)	PIH (n= 56)	95% CI/ Odds ratio	P
Age (years)		58.88 ± 11.419	61.64 ± 9.627	-6.4, 0.8	0.132
Gender	**Male**	61.7% (58)	44.6% (25)	**1.99**	**0.042**
	Female	38.3% (36)	55.4% (31)		
BMI (kg/m^2)		26.37 ± 5.617	27.47 ± 4.717	-2.9, 0.7	0.219

ACEI: Angiotensin-converting enzyme inhibitors; ARB: Angiotensin receptor blockers; BMI: Body mass index; CCB: Calcium channel blockers.

Table (2): Laboratory and echocardiographic variables according to the presence of PIH in the current study.

	No PIH (n= 94)	PIH (n= 56)	95% CI/ Odds ratio	P
Hemoglobin (gm/dl)	12.03 ± 1.182	11.19 ± 1.150	0.4, 1.2	**< 0.001**
End-diastolic left ventricular diameter (mm)	46.28 ± 6.724	48.46 ± 5.985	-4.3, 0.0	**0.047**
End-systolic left ventricular diameter (mm)	32.59 ± 5.642	35.09 ± 5.667	-4.4, -0.6	**0.010**
End-diastolic left ventricular posterior wall thickness (mm)	8.63 ± 1.444	9.04 ± 1.584	-0.9, 0.1	0.109
Interventricular septum thickness (mm)	9.18 ± 1.502	9.54 ± 1.640	-0.9, 0.2	0.178
Regional wall motion abnormality	13.8% (13)	19.6% (11)	1.52	0.348
Ejection fraction (%)	59.41 ± 6.100	56.18 ± 10.275	0.6, 5.9	**0.017**
E/A ratio	0.87 ± 0.194	0.76 ± 0.159	0.0, 0.2	**0.001**
Deceleration time (ms)	239.27 ± 56.707	271.32 ± 46.844	- 49.8, - 14.3	**< 0.001**
E/e'	9.50 ± 2.432	10.88 ± 2.244	-2.2, -0.6	**0.001**
Pulmonary artery systolic pressure (mmHg)	29.41 ± 5.391	28.93 ± 5.201	-1.3, 2.3	0.589
IVC diameter during inspiration (mm)	5.63 ± 1.653	5.02 ± 1.646	0.1, 1.2	**0.030**
IVC diameter during expiration (mm)	11.49 ± 3.555	10.38 ± 4.043	-0.1, 2.4	0.080
Moderate Aortic regurgitation	2.1% (2)	7.1% (4)	**3.54**	0.129
Moderate Aortic stenosis	3.2% (3)	3.6% (2)	1.12	0.900

	No PIH (n= 94)	PIH (n= 56)	95% CI/ Odds ratio	P
Moderate Mitral regurgitation	5.3% (5)	14.3% (8)	**2.97**	0.059
Moderate Tricuspid regurgitation	1.1% (1)	1.8% (1)	1.69	0.709

IVC: Inferior vena cava.

Table (3): Anesthetic agents used for induction and maintenance of anaesthesia.

		No PIH (n= 94)	PIH (n= 56)	Odds ratio	P
Induction agent	**Propofol**	87.2% (82)	87.5% (49)	0.98	0.962
	Midazolam	12.8% (12)	12.5% (7)		
Opioid	**Fentanyl**	91.5% (86)	94.6% (53)	0.61	0.474
	Morphine	8.5% (8)	5.4% (3)		
Epidural analgesia		16.0% (15)	30.4% (17)	2.3	**0.037**
Maintenance agent	**Isoflurane**	56.4% (53)	67.9% (38)	-	0.374
	Sevoflurane	33.0% (31)	25.0% (14)		
	Propofol infusion	10.6% (10)	7.1% (4)		

Table (4): Univariate regression analysis for predictors of PIH in the current study.

	R^2	Exp (B)	95% CI of Exp (B)	p
Female gender	3.7%	1.99	1.02, 3.91	**0.043**
Heart failure	4.2%	4.33	1.07, 17.51	**0.040**
ARB	-	-	-	0.105
β blocker	-	-	-	0.065
CCB	-	-	-	0.100
Hemoglobin (gm/dl)	14.8%	0.54	0.39, 0.74	**< 0.001**
End-diastolic left ventricular diameter (mm)	3.6%	1.06	1, 1.11	**0.049**
End-systolic left ventricular diameter (mm)	6%	1.08	1.02, 1.15	**0.012**
Regional wall motion abnormality	-	-	-	0.350
Ejection fraction (%)	16.3%	0.95	0.91, 0.99	**0.025**
E/A ratio	10.3%	0.04	0.01, 0.27	**0.001**
Deceleration time (ms)	10.8%	1.01	1.01, 1.02	**0.001**
E/e’	10.1%	1.28	1.1, 1.49	**0.001**
IVC diameter during inspiration (mm)	4.3%	0.79	0.64, 0.98	**0.032**
Moderate Aortic regurgitation	-	-	-	0.152
Moderate Mitral regurgitation	-	-	-	0.069
Epidural analgesia	3.8%	2.3	1.04, 5.08	**0.040**

ARB: Angiotensin receptor blockers; CCB: Calcium channel blockers; IVC: Inferior vena cava.

Table (4): Multivariate regression analysis for predictors of PIH in the current study.

	R^2	Exp (B)	95% CI of Exp (B)	p
Hemoglobin (gm/dl)	33.5%	0.46	0.32, 0.67	**< 0.001**
E/A ratio		0.01	0.00, 0.12	**< 0.001**
IVC diameter during inspiration (mm)		0.77	0.61, 0.98	**0.033**
Epidural analgesia		2.7	1.08, 6.76	**0.033**

IVC: Inferior vena cava.

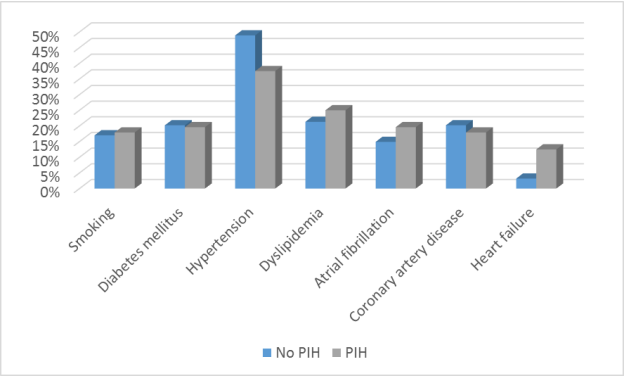

Figure (1) Medical history and special habits according to the presence of PIH in the current study

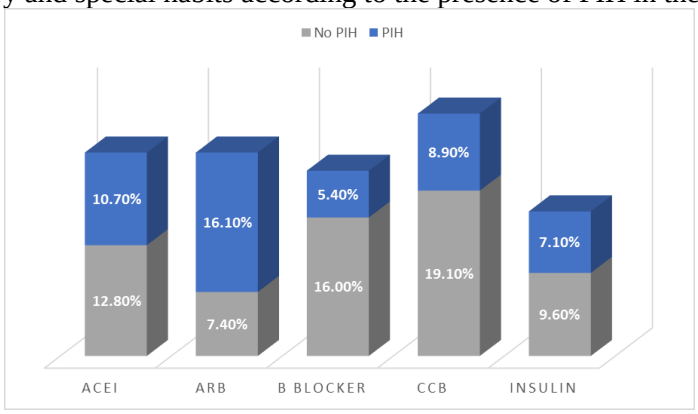

Figure (2) Medical treatment according to the presence of PIH in the current study

RESULTS

PIH was detected in 56 out of the included 150 subjects, which makes the incidence rate of this complication 37.33%. Based on that incidence, the participants were allocated into two groups: PIH (56 patients) and No-PIH (94 patients) groups. Starting with demographic characteristics, the included patients had mean ages of 58.88 and 61.64 years in the No-PIH and PIH groups, respectively, which was comparable between the two groups ($p = 0.132$). Regarding gender distribution, male patients represented 61.7% and 44.6% of the included subjects in the same two groups, with a higher prevalence of women in the PIH group ($p = 0.042$).

The included cases' BMI had mean values of 26.37 and 27.47 kg/m2 in the same two groups, respectively, with no statistically significant difference between them ($p = 0.219$). The prevalence of smoking and other systemic comorbidities (diabetes, hypertension, dyslipidemia, atrial fibrillation, did not show any statistical difference between the two groups. Nonetheless, heart failure patients showed a significant increase in the PIH group (12.5% vs 3.2% in the other group – $p = 0.027$). When it comes to the drug history, it showed no significant difference between the two groups, and that included ACEI, ARB, B blockers, CCB, and insulin. The previous data are shown in table 1. Hemoglobin levels showed a significant decline in the PIH group (11.19 vs 12.03 gm/dl in the other group – $p < 0.001$). The same group expressed significantly higher end-systolic and diastolic left ventricular diameters ($p = 0.01$ and 0.047, respectively). Nevertheless, the two groups expressed comparable other echocardiographic parameters, including end-diastolic posterior wall thickness, interventricular septum, and the presence of wall motion abnormality ($p > 0.05$). Ejection fraction ($p = 0.017$) and E/A ratio ($p = 0.001$) were significantly lower in the PIH group. In the same time, the same group showed significantly longer deceleration time ($p < 0.001$) and higher E/e' ratio ($p = 0.001$). Pulmonary artery systolic pressure showed comparable readings between the two groups ($p = 0.589$). Although inspiratory IVC diameter was significantly decreased in the PIH group (5.02 vs 5.63 mm in the other group – $p = 0.03$), the expiratory diameter of the same vessel did not show any significant difference between them ($p = 0.08$). Valvular pathologies including stenosis or regurgitation had comparable prevalence in the two groups ($p > 0.05$). Table 2 shows the previous data. As shown in Table 3, drugs used for induction and maintenance of anaesthesia, along with the type of opioid administered, showed no significant difference between the two groups. However, epidural analgesia showed a significant increase in association with PIH (30.4% vs 16% in the other group – $p = 0.037$). Univariate regression analysis showed that female gender, heart failure, low haemoglobin levels, high end-systolic and diastolic

ventricular diameter, low ejection fraction, low E/A ratio, prolonged deceleration time, increased E/e', decreased inspiratory IVC diameter, and epidural analgesia were significant risk factors for PIH (Table 4).

DISCUSSION

This study was conducted to estimate the prevalence and risk factors of PIH in patients undergoing surgical procedures under general anaesthesia. This complication was encountered in 56 patients (out of 15) with an incidence rate of 37.33%. Tarao et al. reported that the same complication occurred in 63 patients out of the included 201 participants (incidence rate = 31.34%) **[1]**, which is near to our findings. Other authors reported a higher incidence, as the same complication was noticed in 121 out of 172 participants (70%) **[22]**. The difference in patient and disease criteria or the definition of PIH could attribute to the previous heterogenicity in incidence rates. We did not detect any significant association between age and the development of that complication. Yatabe et al. Confirmed our findings regarding age ($p = 0.71$) **[22]**. However, another study linked that complication with old patient age. The included patients had median values of 65 and 59 years in the PIH and No-PIH groups, respectively **[3]**. Our findings showed that the female gender was significantly associated with PIH (OR = 1.99 – $p = 0.04$). This is in agreement with Tarao et al., who confirmed our findings regarding the same gender **[1]**. However, Südfeld and his coworkers reported that the male gender is a significant risk factor for PIH **[3]**, and that contradicts the previous findings. In the current study, no significant association was detected between BMI and PIH. This was also confirmed by another study **[22]**. Contrarily, Südfeld et al. noted that PIH was more noticed in patients with lower BMI ($p < 0.01$). Patients with PIH had a median BMI of 24.7 kg/m2 compared to 25.5 kg/m2 in the other group **[3]**. In the current investigation, most of the systemic comorbidities, including smoking, diabetes, hypertension, and dyslipidemia, did not have a significant impact on PIH. Others also noted the previous findings, like atrial fibrillation, hypertension, and diabetes prevalence was comparable between the PIH and No-PIH groups, respectively **[22]**. Our study showed that neither of the reported medications had a significant impact on PIH development. Although some researchers agreed with our findings regarding calcium channel blockers (CCB) as they denied its relation with PIH **[23]**, others incriminated angiotensin receptor blockers (ARB) and angiotensin convertase enzyme inhibitor (ACEI) in the development of the same complication **[1, 24]**. Although Südfeld et al. reported no significant impact of ACEI, ARB and CCB on the development of the same complication, nevertheless, the same study found a significant association between B blocker intake and PIH ($p = 0.04$) **[3]**. In our study, the PIH group had a significantly lower inspiratory IVC diameter. We think that decreased IVC diameter may reflect a state of dehydration in the patient, which could attribute to the development of PIH. Previous studies have reported that IVC measurement by echocardiography may reflect the fluid status of the patients and decreased IVC diameters are correlated with low central venous pressure **[25-27]**. Our findings showed that the PIH group expressed higher end-systolic and diastolic left ventricular diameters compared to the other group. Other researchers have denied the previous findings, as they did not detect any significant difference between the PIH and No-PIH groups regarding both variables ($p > 0.05$) **[1]**. In the current study, we noted that the PIH group expressed higher values of E/e' ratio, along with higher values of E/A ratio. Another study also confirmed our perspective, as the former ratio had mean values of 11.2 and 9.6, while the latter had mean values of 0.75 and 0.93 in the PIH and No-PIH groups, respectively **[1]**. Our findings showed a significant increase in the deceleration time in the PIH group, and this was also confirmed by Tarao and his colleagues, who reported that the mean values of the same parameter were 260 and 229 msec in the PIH and No-PIH groups, respectively ($p = 0.02$) **[1]**. We did not detect any significant association between PIH and RWMA ($p > 0.05$). On the other hand, another study reported that the same variable was significantly linked to the occurrence of PIH (OR = 6.65) **[1]**. The authors hypothesized that RWMA might reflect an old myocardial ischemic or inflammatory event, which had affected circumferential wall contraction **[28]**. Our study showed that low EF was significantly associated with PIH. It is reasonable that patients with low EF are more prone to hypotensive episodes due to low stroke volume and decreased peripheral resistance associated with general anaesthesia. This was also evident in patients with heart failure, which was a strong predictor of PIH in our study. Our findings failed to detect any significant impact of valvular pathologies on the development of PIH. The absence of severe cases could explain the previous findings. Severe valvular pathology, like severe aortic regurgitation, increases the risk of PIH due to decreased stroke volume and decreased peripheral resistance with anaesthetic induction **[29]**. Our findings showed that propofol administration was not significantly

associated with PIH ($p > 0.05$). In a previous similar study, propofol administration, either during induction or maintenance, did not have a significant impact on the development of PIH **[1]**. Contrarily, other researchers reported that propofol administration is associated with hypotension because of its vasodilator and negative inotropic action **[30, 31]**. Others also noted that these cardiac side effects of propofol did not improve with the decline of its serum levels **[30]**. We did not notice any significant impact of the anaesthetic agents used on the development of PIH. Yatabe and his coworkers confirmed the previous findings **[22]** Our findings showed that epidural catheter insertion was associated with the development of PIH. We all agree that the installation of local anaesthetic agents into the epidural catheter is associated with sympathetic flow block, leading to vasodilation and functional hypovolemia **[32]**. In contrast to the previous findings, another study denied the previous findings, as the epidural catheter was installed in 5.2% and 3.4% of patients in the PIH and No-PIH groups, respectively, with no significant difference between them **[3]**. Our research has some limitations. The included patients were collected from a single medical centre, and the sample size was relatively small. More studies should be conducted to cover these drawbacks.

CONCLUSIONS

Based on the previous findings, female gender, heart failure, low haemoglobin levels, high end-systolic and diastolic ventricular diameter, low ejection fraction, low E/A ratio, prolonged deceleration time, increased E/e', decreased inspiratory IVC diameter, and epidural analgesia were significant risk factors for PIH. Patients having either of the above risk factors should be closely monitored during general anaesthesia for early identification and management of this complication.

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