Portal venous pressure in non-cirrhotic bilharzial patients undergoing elective splenectomy, can it affect mortality? A prospective study

M. Tourky[1], A. Youssef[2], M. Salman[3], T. Abouelregal[2], M. Tag El-Din[4], A. Moustafa[5], A. Taha[5], A. El-Mikkawy[6], A. Saadawy[7], A. Salman[2]

(1) Department of Surgery, Great Western Hospitals NHS Foundation Trust, Swindon, United Kingdom; (2) Internal Medicine Department, Faculty of Medicine, Cairo University, Egypt; (3) General Surgery Department, Faculty of Medicine, Cairo University, Egypt; (4) General Surgery Department, Faculty of Medicine, Al-Azhar university, Cairo, Egypt; (5) Department of Endemic Medicine and Hepatology, Faculty of medicine, Cairo University, Egypt; (6) Theodor Bilharz Research Institute, Hepatology and Gastroenterology Department, Cairo, Egypt; (7) Radiology Department, Faculty of medicine-Ain Shams University, Cairo, Egypt.

Abstract

Background and study aims: **To evaluate the impact of intra-operatively measured portal vein pressure (PVP) on mortality in non-cirrhotic bilharzial patients undergoing splenectomy.**

Methods: **The present study is a prospective study that was conducted in Egypt from April 2014 to April 2018. Adult patients with non-cirrhotic bilharziasis who were scheduled to undergo splenectomy were included. Studied cases were divided into a survival cohort and a non-survival cohort. The main objective was the correlation between the incidence of mortality and intraoperative PVP.**

Results: **The present work comprised 130 cases with a mean age of 51.8 ± 6.4 years old. The in-hospital mortality rate was 22.3%, with sepsis as a major cause of death (37.9%). In term of the association between preoperative variables and mortality, survivors had statistically significant lower portal vein diameter (13.6 ± 1.8 versus 15.2 ± 1.8mm; p<0.001) and higher portal vein velocity (14.2 ± 1.8 versus 10.4 ± 2.3 cm/sec; p<0.001) than non-survivors. The survived patients had significantly lower PVP (13.9 ± 1.1 versus 17.7 ± 2.7; p <0.001). A cut-off value of ≥14.5 mmHg, the PVP yielded a sensitivity of 86.2% and a specificity of 69% for the prediction of mortality. The association analysis showed a statistically significant association between mortality and postoperative liver function parameters.**

Conclusions: **High intraoperative PVP is linked to early postoperative death in non-cirrhotic cases undergoing splenectomy. Our study showed that PVP > 14.5mmHg was an independent predictor of death and showed good diagnostic performance for the detection of early postoperative mortality.** (Acta gastroenterol. belg., **2021,** 84**, 549-556**).

Keywords: bilharziasis, portal venous pressure, splenectomy, postoperative mortality.

Introduction

Schistosomiasis (bilharziasis) is the third most common parasitic infestation worldwide, with a prevalence of 200 million individuals globally (1). The common complications of chronic schistosoma infection include polyp development, hepatosplenomegaly, and portal hypertension (PH) (2). Hepatic bilharziasis is depicted by deposition of bilharzial eggs in the hepatic tissue that leads to a host cell immune response and results in granuloma formation and neoangiogenesis (3). Schistosoma eggs reach the hepatic circulation through the portal blood; get extravasated to portal tracts where they initiate an inflammatory reaction, inducing periportal fibrosis, which causes perisinusoidal block and subsequently portal hypertension (2,4). Therefore, PH due to schistosoma infection is usually intrahepatic (5).

PH itself can have several serious complications as esophageal varices, ascites, and hepatic encephalopathy (6). Some studies classified PH as a contraindication for some surgical procedures as hepatic resection in patients with cirrhosis (7-9). Other studies argued that PH should not be considered a contra-indication for hepatic resection for hepatocellular carcinoma (10,11). Another population-based study by Nguyen and colleagues showed that cases with end-stage liver disease have a higher incidence of in-hospital mortality following colorectal surgery, especially in the existence of elevated portal pressure. (12). Therefore, portal venous pressure (PVP) appears to influence the outcomes of surgery in hepatic patients.

Splenectomy is a common surgical approach in patients with hepatosplenomegaly and/or portal hypertension due to Schistosoma mansoni infection. Studies have shown that splenectomy in those patients improves the blood flow in the portal circulation and the synthetic capacity of hepatocytes (13,14). Moreover, it reduces the congestion in resulting esophageal varices and reduces upper gastrointestinal bleeding incidence (14). As mentioned earlier, abdominal operations in patients with abnormal PVP may carry some risk associated with controversy in the published literature.

However, – to our knowledge – no previous study assessed the correlation between PVP and mortality after splenectomy in bilharzial patients without hepatic cirrhosis. Therefore, we conducted this work to study the impact of intraoperatively measured PVP on mortality in non-cirrhotic bilharzial patients undergoing splenectomy and determine the PVP level that may increase the risk of mortality post-splenectomy.

Materials and Methods

The current work protocol was registered by the local ethics committee (number 0032/May 2016). We

Correspondence to : Ahmed Abdallah Salman, MD, Internal Medicine Department, Faculty of Medicine, Cairo University, Egypt. Phone: 00201000468664.
E-mail : awea844@gmail.com

Submission date : 07/05/2020
Acceptance date : 15/03/2021

confirm that all study's procedures were in concordance with ethical guidelines of the 1975 declaration of Helsinki and applicable local regulatory laws. Written informed consents were obtained from all cases prior to study participation. The surgery was performed after a full explanation of the possible benefits and risks of the operation. We adopted the recommendations of STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) guidelines during this prospective study's performance (15).

Study Design and Patients

The current study is a prospective study that was conducted from April 2014 to April 2018. Adults patients (aged 18-65 years old) with non-cirrhotic bilharziasis who were scheduled to undergo splenectomy were included. The diagnosis of bilharziasis was confirmed by demonstration of Schistosoma mansoni eggs in stool examinations or by rectal biopsy. We excluded patients with liver cirrhosis (based on preoperative ultrasound-guided liver biopsy), preoperative portal or mesenteric vein thrombosis, and patients with acute surgical emergency necessitating splenectomy.

Data collection

All cases were submitted to complete history and clinical examination before surgery. In addition, preoperative evaluation included complete blood count (CBC), coagulation profile, hepatic profile, kidney function tests, and bone marrow aspiration. A multi-disciplinary team (MDT) was established, including surgeons, internists, and hematologists, to confirm the need for splenectomy. Besides, cardiac evaluation was done to exclude cardiopulmonary complications associated with schistosoma infection or other non-related comorbidities.

Preoperative Doppler Ultrasound

Doppler ultrasound was done to all patients included in the study employing digital ultrasound machine provided with convex 5 MHz and linear 12 MHz probes (Gateway; Diasonics, Milpitas, CA, USA). An experienced sonographer performed all Doppler ultrasound assessments. The evaluation was done with the cases in the supine position following fasting for > 4 hours, and the patients were asked to breathe slowly during the examination. Maximum splenic length "from the upper pole to the lower pole" was measured three times, and readings were averaged. Portal vein (PV) anatomy was determined by following the splenic vein to the right until its connection with the superior mesenteric vein. The caliber of PV was assessed by a greyscale US. The velocity of blood flow in the PV was obtained from the Doppler tracings. The acceptable velocity of the bloodstream in the PV was 15-20 cm/s. Elevated portal pressure was associated with an increment in blood flow and congestion yet with a decline in blood velocity in the PV.

The velocity obtained was the maximum velocity (Vmax). The mean V was measured by the machine as follows: V peak registered multiplied by a correction factor of 0.57 acquired from an experimental analysis on a circulation mode. Three measurements were written, and the average was considered. Normal portal vein diameter (PVD) is less than 10 mm, with a more than 20-30% increase with food and respiration. In portal hypertension, the PV is dilated (>13 mm), with absent or less than 20% variation with respiration. Standard blood flow in the PV is toward the liver and is of low velocity, mildly affected by respiration.

Preoperative preparation included upper GI endoscopy to assess the presence of varices and manage them as needed. All patients received pneumococcal and Haemophilus influenza vaccine one week before surgery. In addition, all patients were given preoperative IV cefazolin 2 gm one hour before skin incision

Operative technique

Splenectomy was done under general anesthesia in supine posture through a midline opening. A total splenectomy (with the removal of splenunculus) was performed through an open surgical technique. Devascularization or left gastric vein ligation was not performed routinely. Prophylactic antibiotics (amoxicillin/clavulanic acid and amikacin) were given initially and maintained after the operation as needed. If necessary, after the separation of the gastrocolic ligament, the splenic artery was doubly ligated in continuity at the pancreas' upper border prior to splenic mobilization. Platelets, plasma, and blood transfusion were given at this time if needed. Splenectomy was performed by the separation of its vascular attachments. Drains were placed selectively.

Intra-operative PVP

PVP was measured intra-operatively, before splenectomy, employing an 18 -gauge catheter put into one of the large jejunal, ileal mesenteric tributaries, or the main inferior mesenteric vein. The other side was joined through an extension-arterial line to a pressure transducer. If possible, both the main inferior mesenteric vein and one large mesenteric tributary pressure were assessed, and the mean value was obtained. The normal range for directly measured PVP values was considered to be 5 to 10 mm Hg. A figure of eight suture was performed if continuous oozing from the puncture site after proper pressure.

Intraoperative (IO) data comprised surgical time, IO central venous pressure (CVP), mean blood pressure (MBP), amount of IO blood loss, transfusion amount (number of transfused blood, plasma, and platelets units), intraoperative mean blood glucose, input, and output

during operation (IO average urine flow rate, crystalloid administration, net fluid balance during surgery), in addition to IO PVP.

Studied cases were categorized into a survival cohort and a non-survival cohort.

Postoperatively, patients were managed in an intensive care unit or regular ward, decided on a case by case basis with close clinical follow-up. Laboratory investigations included liver and renal functions, complete blood count, and bleeding profile. Oral feeding was initiated on postoperative day one and escalated, according to toleration. Drains were taken out if the output was not bloodstained and less than 500 ml/day.

Study outcomes

The primary outcome in the current work was the correlation between the incidence of mortality on one side and different pre, intra, and postoperative variables, including intraoperative PVP on the other side. The secondary outcome is to define the cut-off value of PVP that may increase postoperative mortality

Statistical Analysis

Data registry, processing, and statistical analysis were done through SPSS version 22.0. Frequency tables with percentages were employed for categorical variables, and descriptive statistics (mean and standard deviation) were used for numerical variables. The normality of the data was examined via the Shapiro-Wilk Test. Tests of significance (Chi-square, student's t-test, or Mann Whitney's test) were used according to the normality of the data. Multivariate logistic regression was obtained to define the predictors of in-hospital mortality. The recessive operative characteristics (ROC) curve was carried-out to assess the diagnostic performed of PVP for the detection of mortality. A P-values of less than 0.05 was considered statistically significant.

Results

The present study included 130 adult, non-cirrhotic, bilharzial cases for whom splenectomy was performed. The mean age of the included cases was 51.8 ± 6.4 years old, and most patients were males (86.2%). The most common cause of splenectomy was painfully enlarged spleen (26.9%), followed by pancytopenia (23.8%). The rest of the causes included bicyotopenia (10%), isolated hemolytic anemia (15.4%), isolated neutropenia (7.7%), and isolated thrombocytopenia (16.2%) (Table 1).

The average preoperative total bilirubin level was 1.3 ± 0.2 mg/dL, and the average serum albumin was 3.7 ± 0.31g/dL. In addition, the mean PVD and portal vein velocity (PVV) was 13.9 ± 1.9 mm and 13.3 ± 2.4 cm/sec, respectively. At the same time, the mean splenic size was 18.5 ± 2.1cm. Table 1 demonstrates the preoperative data of the enrolled cases.

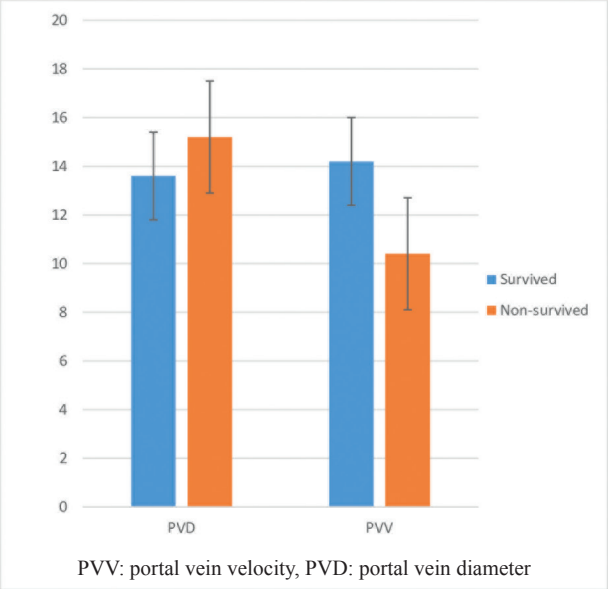
Table 1. — **The demographic and preoperative data of study groups**

	Patients (n = 130)
Male, No. (%)	112 (86.2%)
Age (years), Mean ± SD.	51.8 ± 6.4
BMI (kg/m²), Mean ± SD.	27 ± 3.1
HTN, No. (%)	29 (22.3%)
DM, No. (%)	44 (33.9%)
Associated HCV, No. (%)	82 (63.1%)
Indication of Splenectomy, No. (%)	
– Bicyotopenia	13 (10%)
– Isolated hemolytic anemia	20 (15.4%)
– Isolated neutropenia	10 (7.7%)
– Isolated thrombocytopenia	21 (16.2%)
– Painfully enlarged spleen	35 (26.9%)
– Pancytopenia	31 (23.8%)
Platelet counts x1000, Median (IQR).	100 (33.7-179)
INR, Mean ± SD.	1.2 ± 0.12
Hemoglobin (g/dL), Mean ± SD.	9.4 ± 1.9
TLC x1000 (cell/mm³), Median (IQR)	5.2 (3.1- 7.1)
Total bilirubin (mg/dL), Mean ± SD.	1.29 ±0.17
Serum albumin (g/dL), Mean ± SD.	3.7 ± 0.31
Serum AST (IU/dL), Median (IQR)	42 (34-45.3)
Serum ALT (IU/dL), Median (IQR)	42.5 (34-48)
Serum GGT (mg/dL), Median (IQR)	114 (87-187)
Portal vein diameter (mm), Mean ± SD.	13.9 ± 1.9
Portal vein velocity (cm/sec), Mean ± SD.	13.3 ± 2.4
Splenic size (cm), Mean ± SD.	18.5 ± 2.1

BMI: Body mass index; HTN: Hypertension; DM: Diabetes mellitus; HCV: Hepatitis C; TLC: Total leucocytic count; INR: International normalized ratio; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; GGT: Gamma-glutamyl transferase.

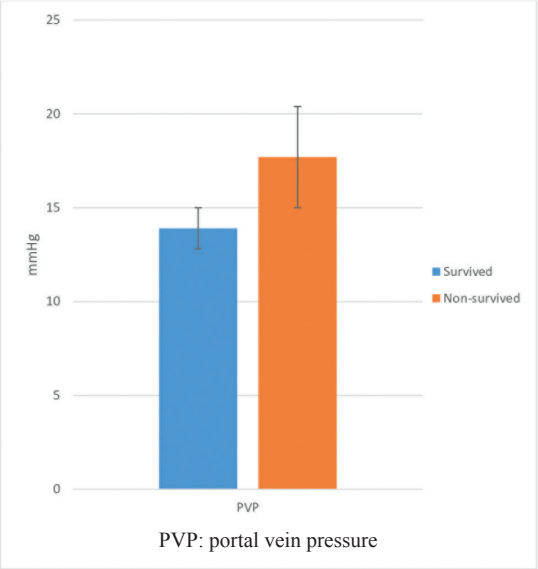
Sixty-three patients had esophageal vaices. Nine had gastric varices. Preoperative variceal bleeding was encountered in thirty patients. Seventeen patients underwent IO devascularization, according to surgeon clinical judgement. Postoperatively, the in-hospital mortality rate was 22.3%, with sepsis as a major cause of death (37.9%), followed by myocardial infarction (17.2%), pulmonary embolism (10.3%), and gastrointestinal bleeding due to variceal etiology (6.8%). In term of the association between preoperative variables and mortality, Survivors had statistically significant lower PVD (13.6 ± 1.8 versus 15.2 ± 1.8mm; $p<0.001$) and higher PVV (14.2 ± 1.8 versus 10.4 ± 2.3 cm/sec; $p<0.001$) than non-survivors (Figure 1a). In addition, survivors had significantly higher serum GGT compared to non-survivors ($p <0.001$). In contrast, there were no statistically considerable associations between other preoperative liver function test parameters, CBC findings, or serum creatinine with mortality ($p >0.05$; Table 2a).

On the other hand, non-survivors had comparable averages for the transfused blood units ($p =0.38$), platelet units ($p =0.68$), and plasma units ($p =0.98$) compared to survivors. Similarly, there were no statistically considerable variations between survivors and non-survivors regarding other intraoperative characteristics, except PVP and CVP (Table 2b). The survived patients had significantly lower PVP (13.9 ± 1.1 versus 17.7 ±



PVV: portal vein velocity, PVD: portal vein diameter

Figure 1. — The distribution of PVD and PVV among survivors and non-survivors.



PVP: portal vein pressure

Figure 2. —The distribution of PVP among survivors and non-survivors

Table 2a. — **The association between preoperative characteristics and mortality**

	Survivors (n = 101)	**Non-survivors (No = 29)**	**P-value**
Male, No. (%)	95 (87.2%)	17 (81%)	0.32
Age (years), Mean ± SD.	51.9 ± 6.2	51.4 ± 7.1	0.69
BMI (kg/m²), Mean ± SD.	27.3 ± 2.9	26.1 ± 3.5	0.09
HTN, No. (%)	25 (22.9%)	4 (19%)	0.47
DM, No. (%)	35 (32.1%)	9 (42.9%)	0.54
Platelet counts x1000, Median (IQR).	122.3 ± 103.5	116.5 ±87.5	0.76
INR, Mean ± SD.	1.2 ±0.12	1.2 ±0.15	0.13
Hemoglobin (g/dL), Mean ± SD.	9.4 ±1.9	9.4 ± 2.2	0.87
TLC x1000 (cell/mm³), Median (IQR)	5.4 ± 2.3	5.8 ± 3.1	0.45
Total bilirubin (mg/dL), Mean ± SD.	1.3 ± 0.17	1.35 ± 0.19	0.155
Serum albumin (g/dL), Mean ± SD.	3.7 ± 0.31	3.6 ± 0.27	0.11
Serum AST (IU/dL), Median (IQR)	42 (32 – 47.5)	43 (49 – 50)	0.108
Serum ALT (IU/dL), Median (IQR)	42 (33 – 46)	41 (38.5 – 44)	0.477
Serum GGT (mg/dL), Median (IQR)	128 (88 – 194.5)	87 (70 – 110)	<0.001**
Serum creatinine (mg/dL), Mean ± SD.	1.11 ± 0.16	1.14 ± 0.17	0.551
Splenic size (cm), Mean ± SD.	18.4 ± 2.1	18.8 ±2.1	0.39
PVD, Mean ± SD.	13.6 ± 1.8	15.2 ± 1.8	<0.001**
PVV, Mean ± SD.	14.2 ± 1.8	10.4 ± 2.3	<0.001**
Biopsy results			
F0	12	4	0.943
F1	21	5	
F2	34	10	
F3	29	10	

*P value < 0.05 significant, ** P value < 0.001 highly significant.
BMI: Body mass index; HTN: Hypertension; DM: Diabetes mellitus; HCV: Hepatitis C; TLC: Total leucocytic count; INR: International normalized ratio; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; GGT: Gamma-glutamyl transferase, PVD: Portal vein diameter, PVV: Portal vein velocity

2.7 in non-survivors, respectively; p <0.001; Figure 1b) and intraoperative CVP (p <0.001) than non-survivors. At a cut-off value of ≥14.5 mmHg, the PVP yielded a sensitivity of 86.2% and a specificity of 69% for prediction of mortality (AUC =0.903, p <0.001; Figure 2).

The association analysis revealed a statistically significant association between mortality and post-operative liver function parameters (Table 2c). Non-survivors had statistically significant higher postoperative serum total bilirubin (p =0.02), lower serum albumin (p =0.006), and serum gamma-glutamyl transferase

Table 2b. — **The association between intraoperative characteristics and mortality**

	Survivors (n = 101)	Non-survivors (No = 29)	P-value
Transfused blood units, Mean ± SD.	1.8 ± 1.4	2.1 ± 1.4	0.38
Platelet units, Mean ± SD.	11.7 ± 8.2	12.6 ± 9.3	0.68
Plasma units, Median (IQR).	1.9 ±0.7	1.9 ±0.9	0.98
Blood glucose (mg/dL), Mean ± SD.	120.9 ±21.8	120.4 ±18.6	0.91
Urine flow rate (mL/kg/h), Mean ± SD.	1.7 ±0.3	1.6 ± 0.3	0.68
Crystalloid administration (mL/kg), Mean ± SD.	44.1 ±7.5	44.1 ± 8.3	0.96
Net fluid balance during surgery, (mL/kg), Mean ± SD.	29.4 ±6.8	31.1 ±5.4	0.21
Blood loss (mL), Mean ± SD.	992.3 ± 361.2	1045.2 ± 503.5	0.65
MAP (mmHg), Mean ± SD.	75.5 ± 5.5	75.7 ± 4.8	0.87
Operative time (minutes), Mean ± SD.	176.8 ±35.1	180.2± 26.5	0.61
CVP, cm H2o, Mean ± SD.	9.6 ± 1.7	12.3 ± 1.3	<0.001**
PVP mmHg	13.9 ± 1.1	17.7 ± 2.7	<0.001**
Gradient (difference between portal pressure and CVP), mmHg	5.8 ± 1.2	3.9 ± 0.9	<0.001**

*P value < 0.05 significant, ** P value < 0.001 highly significant
PVP: Portal venin pressure, MAP: Mean arterial pressure; CVP: Central venous pressure

Table 2c. — **The association between postoperative characteristics and mortality**

	Survivors (n = 101)	Non-survivors (No = 29)	P-value
Hospital Stay, Mean ± SD.	7.5 ± 1.4	11.3 ± 1.3	<0.001**
Platelet counts x1000, Median (IQR).	161.1 ±78.2	138.5 ±72.5	0.85
INR, Mean ± SD.	1.1 ±0.13	1.5 ±0.16	0.13
Hemoglobin (g/dL), Mean ± SD.	9.8 ±1.4	9.9 ±1.4	0.74
TLC x1000 (cell/mm³), Median (IQR)	5.6 ±1.9	6.2 ± 2.8	0.33
Total bilirubin (mg/dL), Mean ± SD.	1.2 ± 0.14	1.3 ± 0.13	0.02*
Serum albumin (g/dL), Mean ± SD.	3.8 ± 0.26	3.6 ± 0.28	0.006*
Serum AST (IU/dL), Median (IQR)	41 (35-43)	42 (37-45.5)	0.11
Serum ALT (IU/dL), Median (IQR)	39 (35-43)	43 (40-45.5)	0.07
Serum GGT (mg/dL), Median (IQR)	123 (92.5-176)	99 (93-110.5)	0.02*
Serum creatinine (mg/dL), Mean ± SD.	1.12 ± 0.17	1.1 ± 0.17	0.54

*P value < 0.05 significant, ** P value < 0.001 highly significant
TLC: Total leucocytic count; INR: International normalized ratio; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; GGT: Gamma-glutamyl transferase

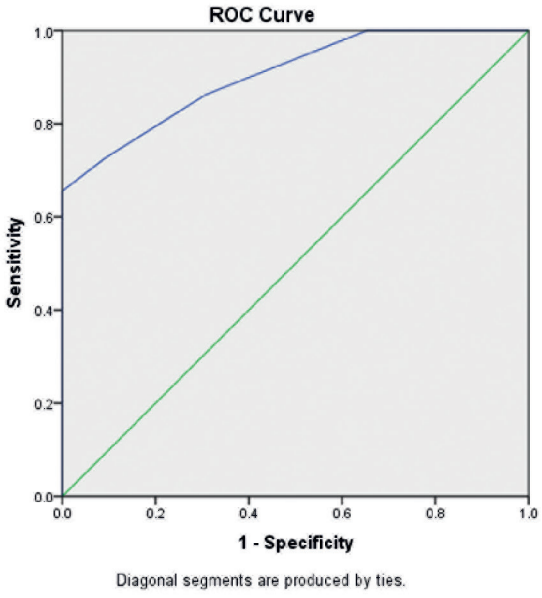


Figure 3. — ROC curve for the diagnostic accuracy of PVP for discrimination of mortality

Table 3. — **The linear and multivariate logistic regression for predictors of Mortality**

	Odds ratio (95% CI)	P-value
PVP ≥14.5	1.21 (0.86–2.56)	0.24
Preoperative PVD	1.68 (0.75–3.73)	0.21
Preoperative PVD	0.63 (0.23–1.69)	0.35
CVP	0.53 (0.19–1.69)	0.34
Serum bilirubin	0.75 (0.66 – 1.06)	0.091

PVP: Portal venin pressure; PVD: portal vein diameter, CVP: Central venous pressure

(GGT) (p =0.02) than survivors. The hospital stay was significantly higher in non-survivors as well (11.3 ± 1.3 versus 7.5 ± 1.4 days, p <0.001).

The logistic regression analysis demonstrated that none of the perioperative variables were independent predictors of mortality (Table 3).

Discussion

In recent years, there has been increasing evidence suggesting that intraoperative PVP is a significant predictor of poor postoperative outcomes, especially mortality, in cases with end-stage liver disease undergoing hepatic surgery (16,17). Yet, the impact of intraoperative PVP on postoperative mortality in non-cirrhotic patients is still debatable. In the present prospective study, we found that higher intraoperative PVP was linked to in-hospital death among adult, non-cirrhotic, bilharzial patients submitted to splenectomy operation. The PVP exhibited excellent diagnostic performance with a sensitivity of 86.2% and a specificity of 69% for predicting in-hospital mortality. Besides, the logistic regression analysis showed that the intraoperative PVP was an independent predictor of death in cases undergoing splenectomy. Alongside these findings, we found significant associations between in-hospital mortality and preoperative PVD and PVV, further supporting the considerable role of high intraoperative PVP in postoperative mortality. On the other hand, in-hospital mortality in patients undergoing splenectomy was associated with intraoperative CVP and postoperative liver function parameters.

Portal hypertension is one of the major complications of end-stage liver disease and the main contributor to the development of many cirrhosis-related complications, including esophageal varices and ascites.(18) Significant elevation of PVP was previously linked with impaired liver functions and, hence, higher risk of coagulopathy and bleeding complications in cirrhotic patients.(19,20) These findings formed the rationale of recent studies that highlighted the potential role of elevated PVP as an objective predictor of death in cirrhotic cases submitted to surgery (16,17). However, PVP can be considered as a proxy for the general health condition of the patients, not only as an indicator of liver function; clinically-meaningful elevation in the PVP is associated with the hyperdynamic circulatory state which has deleterious effects on cardiac, pulmonary, renal, and brain functions (21). Therefore, we hypothesized that elevated PVP could have a negative impact on postoperative mortality, even among non-cirrhotic patients. The present study showed that higher intraoperative PVP was a significant predictor of in-hospital mortality among adult, non-cirrhotic, bilharzial patients scheduled to undergo splenectomy. At level $\geq$ 14.5mmHg, the PVP exhibited excellent diagnostic performance with a sensitivity of 86.2% and a specificity of 69% for predicting in-hospital mortality.

Although, it may be assumed that the higher PVP is caused by higher dissemination of schistosoma in the liver, and hence mortality and higher PVP are rather consequence of a more severe/spread disease; unfortunately, we did not have registered the data regarding parasitic load. However, the gradient CVP-PVP is lower, suggesting a lower intrahepatic vascular resistance. Increased CVP in non-survivors may be a potential surrogate of disease severity.

To the best of our knowledge, this is the first prospective work that assessed the predictive role of PVP in mortality among non-cirrhotic patients undergoing splenectomy. Allard and colleagues (22) demonstrated that post hepatectomy PVP significantly predicted the in-hospital mortality in patients who underwent major hepatic resection. In cirrhotic patients, it was found that high intraoperative PVP was associated with mortality following hepatic resection (23) or emergency surgery (17).

Doppler evaluation of portal vein hemodynamics is a widely accepted, non-invasive, accurate method for detecting the presence and severity of portal hypertension. In cases with definite portal hypertension, Doppler studies showed a significant increase in PVD and sluggish portal blood flow (reduced PVV) (24,25). In the present study, we found that in-hospital mortality was linked to increased PVD and decreased PVV. These findings further support the significant role of high intraoperative PVP in postoperative mortality.

Notably, we found statistically significant associations between in-hospital mortality and postoperative liver function parameters; non-survivors had statistically substantial higher postoperative serum total bilirubin, lower serum albumin, and serum GGT than survivors. Previous works suggested that patients with early deterioration in the postoperative liver functions are at higher risk of mortality than patients with normal liver function. Balzan and colleagues (26) demonstrated that postoperative elevation in serum bilirubin was associated with mortality in patients who underwent elective liver resections.

It should be noted that the rate of mortality was quite high in this studied cohort compared to the published literature. This relatively high mortality rate may confirm, in an indirect way our results that elevated portal pressure may have a serious impact on mortality in this category of patients, so probably taking care of this point is of atmost importance. Also, the relatively high mean age of the studied patients and associated comorbidities such as diabetes and other perioperative parameters may play an additional role regarding this issue.

In all cases, there was a demanding indication to proceed to the operation. The patients with painful spleen had a very dull aching pain that most of them could not withstand it due to very poor life quality. The other patients had very frequent symptoms due to significantly decreased blood counts such as bleeding from different body orifices due to severe thrombocytopenia, recurrent infections attributed to leucopenia, and disabling anemic manifestations with marked fatigue and weakness that impaired their routine activities. Prior to surgery, all patients were informed about the possible benefits and risks of the operation.

Porto-systemic shunt was not performed in any of the studied patients. Our surgeons considered as an old fashion and time consuming technique, with noticeable rate of complications. The surgical team only performed

devascularization on a case by case basis such as those with moderate to large varices and/or previous variceal bleeding.

We acknowledge that the current work had some limitations. Patients were recruited using a non-probability, convenient sampling technique, which may increase selection bias. Also, further future studies on a larger number of cases should be done to affirm our findings. In this work, we did not assess if splenectomy had survival benefit or a favorable impact on long-term survival, especially given a relatively high mortality rate; we were only focusing on short-term outcomes. This point may be the topic of further work. Other important points, such as previous variceal bleeding, should be included in the analysis as they probably indicates more severe portal pressure; furthermore, fluctuation of the portal pressure over the day and effect of anesthesia and surgery should be taken into account in future work. Elaboration on the possible clinical implementation of PVP measurements, such as when the surgeon should consider to stop the procedure if the PVP was more elevated than a certain point; is an another interesting piont to be addressed in next studies. It should be also noted that pre-operative PVP measurements should be considered in these type of patients (for example by interventional radiologist).

In conclusion, high intraoperative PVP is linked to in-hospital death in non-cirrhotic patients undergoing splenectomy. Our study showed that PVP > 14.5mmHg was an independent predictor of death and showed good diagnostic performance to detect in-hospital mortality. Preoperative Doppler studies of portal vein hemodynamics can represent promising indicators for poor postoperative outcomes as well. However, further well-designed studies are still required to underline our findings.

Novelty of the study

It is well known that bilharziasis is a common parasitic infestation, especially in the developing world. It is a recognizable indication of splenectomy in these areas, as it may cause huge splenomegaly. To our knowledge- no previous study assessed the correlation between portal venous pressure and mortality after splenectomy in bilharzial patients without hepatic cirrhosis. Assessment of PVP in these patients might present as a new indicator or supplement to preexisting preoperative indicators. And so with higher PVP (especially > 14.5 mmHg as in our study), physicians could get ready more thoroughly, probably resulting in better overall outcomes after splenectomy. Therefore, the high lightening of PVP's role might be viewed as a novel, worthy aspect of the current work.

Conflict of interest

All authors affirm no financial or personal relationship with a third party whose interests could be positively or negatively affected by the article's content.

Funding souce

Authors confirm they did not receive any funding to do this work.

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