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ORIGINAL ARTICLE

Prevalence of Portal Vein Thrombosis in Budd-Chiari Syndrome Patients

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ABSTRACT

Background and Aim: Thrombosis of the portal vein or obstruction of the hepatic venous outflow tract is the cause of portal vein thrombosis (PVT) and Budd-Chiari syndrome (BCS). Patients with PVT may experience a shorter survival rate compared to those without. The present study aimed to determine the portal vein thrombosis occurrence in Budd-Chiari syndrome patients.

Patients and Methods: This cross-sectional study was conducted on 46 Budd-Chiari syndrome patients in Gastroenterology department of DHQ Teaching Hospital and Mufti Mahmood Memorial Hospital, Dera Ismail Khan from November 2021 to October 2022. Written informed consent was taken from each individual. Doppler ultrasonography (colored-pulsed) was used to confirm the PVT and BCS cases. Clinical data, laboratory findings, and radiological data were recorded. SPSS version 26 was used for data analysis.

Results: The overall mean age of the BCS patients was 28.34±4.6 years. The prevalence of portal vein thrombosis in BCS patients was 12.8% (n=6). Of the total cases, the incidence of chronic and acute BCS presentation was 42 (91.3%) and 4 (8.7%) respectively. The most prevalent symptoms of BCS were abdominal pain and abdominal enlargement in 38 (82.6%) and 41 (89.1%) respectively. Hepatomegaly and ascites were most prevalent clinical signs found in 36 (78.3%) and 37 (80.4%) respectively. The incidence of esophageal varices, gastric extension, and fundal varices were 32 (69.6%), 4 (8.7%), and 2 (4.3%) respectively. About 21 (45.7%) cases had Portal hypertensive gastropathy (PHG).

Conclusion: A higher incidence of PVT was observed in the present study than previously reported. Sociodemographic data and underlying etiology of BCS were not significantly different between patients with and without PVT. Those with PVT were more likely to experience hepatic tenderness, increased white blood cells, and increased direct bilirubin. PVT and other patients had similar Doppler ultrasound findings.

Keywords: Portal vein thrombosis, Prevalence, Budd-Chiari syndrome

INTRODUCTION

Budd-Chiari syndrome (BCS) is a potentially fatal illness caused by a hepatic venous outflow blockage anywhere to the right atrium from the hepatic venules [1]. BCS can be characterized as primary or secondary [2]. Preceding research has suggested that primary BCS is a complex illness of multiple prothrombotic diseases. BCS patients from various topographical locations have diverse illness etiologies. In 74% of patients, acquired pro-coagulative condition or at least one genetic disorder exists [3, 4]. The traditional trinity of stomach discomfort, ascites, and hepatomegaly is thought to be non-specific [5]. Sub-acute, acute, or chronic are different classes of BCS depending on the length of the liver illness, with the chronic type being the most frequent [6]. Radiological imaging is crucial in the assessment of suspected instances of BCS. Doppler ultrasonography, magnetic resonance imaging (MRI), computed tomography (CT), and hepatic venography are all useful imaging modalities [7].

The portal vein thrombosis (PVT) occurrence ranges from four cases per million to 0.7 cases per 100,000 person-per annum, while the Budd-Chiari syndrome (BCS) ranges from 0.5 to 1 case per million per annum [8, 9]. Additionally, the PVT incidence was found 1% based on suspected thrombosis clinically diagnosed on abdominal CT [10]. As a result, the current prevalence of PVT is significantly higher than earlier reported, and that PVT diagnoses have progressively grown over time, notably with the growing utilization of imaging tests in diverse patient groups. A study conducted on the Danish population reported that mortality risk in splanchnic vein thrombosis increased to 20.6% and had a significant impact on PVT-associated mortality compared to BCS [11]. More research is needed to determine the association of these findings with occurrence of PVT with various comorbidities. Therefore, the present study intended to determine the prevalence of portal vein thrombosis in Budd-Chiari syndrome patients.

METHODOLOGY

This cross-sectional study was conducted on 46 Budd-Chiari syndrome patients in Gastroenterology department of DHQ Teaching Hospital and Mufti Mahmood Memorial Hospital Dera Ismail Khan from November 2021 to October 2022. Written informed consent was taken from each individual. Doppler ultrasonography (colored-pulsed) was used to confirm the PVT and BCS cases. Clinical data, laboratory findings, and radiological data were recorded. For each condition, the baseline characteristics and prevalence of concurrent diseases, as well as relevant risk factors, are reported individually. Incorporating obesity, diabetes mellitus, hypertension, peripheral arterial disease, neurological or pulmonary disease, and a history of cancer into the consideration of concomitant diagnoses. Statistical results were presented as a frequencies, percentages, and means with standard deviations and interquartile ranges. A gender-specific incidence rate was calculated based on a consideration of PVT and BCS separately. We regarded the disease listed in the primary discharge diagnosis when PVT and BCS were diagnosed concurrently during the same hospitalization.

RESULTS

The overall mean age of the BCS patients was 28.34±4.6 years. The prevalence of portal vein thrombosis in BCS patients was 12.8% (n=6). Of the total cases, the incidence of chronic and acute BCS presentation was 42 (91.3%) and 4 (8.7%) respectively. The most prevalent symptoms of BCS were abdominal pain and abdominal enlargement in 38 (82.6%) and 41 (89.1%) respectively. Hepatomegaly and ascites were most prevalent clinical signs found in 36 (78.3%) and 37 (80.4%) respectively. The incidence of esophageal varices, gastric extension, and fundal varices were 32 (69.6%), 4 (8.7%), and 2 (4.3%) respectively. About 21 (45.7%) cases had Portal hypertensive gastropathy (PHG). Table-I represent the BCS sociodemographic data (n=46) and the frequency of blocked hepatic veins, inferior vena cava, and portal veins were collected. Figure-1 depicts the prevalence of acute and chronic BCS. Clinical symptoms and signs are shown in Table-II.

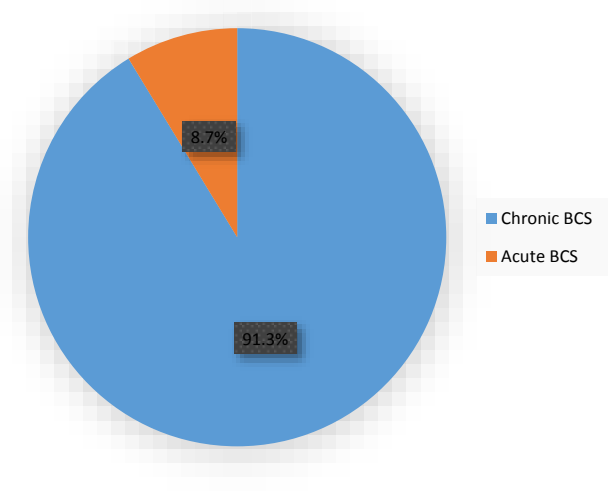

Figure-1: prevalence of acute and chronic BCS

Table-1: BCS sociodemographic data and frequency of blocked hepatic veins, inferior vena cava, and portal veins (n=40)

Doppler USD	PVT Patent N=40	P-value
Hepatomegaly N (%) Positive	36 (90)	0.05
Splenomegaly N (%) Positive	23 (57.8)	0.79
RHV Thrombosis N (%) Positive	35 (87.5)	0.41
MHV Thrombosis N (%) Positive	37 (92.5)	0.89
LHV Thrombosis N (%)	36 (90)	0.91
Ascites N (%) Positive	37 (92.5)	0.89
Esophageal varices N (%) Positive	28 (70)	0.22
Gastric extensions N (%) Positive	4 (10)	0.02
Fundal varices N (%) +ve	3 (7.5)	0.89
PHG N (%) +ve	21 (45.7)	0.02

Table-2: Prevalent Clinical symptoms and signs

Clinical symptoms and signs	Frequency N	Percentage %
Abdominal pain	38	82.6
Abdominal enlargement	41	89.1
Hepatomegaly	36	78.3
Ascites	37	80.4

DISCUSSION

The present study mainly focused on the portal vein thrombosis occurred in Budd-Chiari syndrome patients and found that a higher prevalence of PVT (12.8%) in BCS patients as compared to earlier reported prevalence 10.9% [12]. The sociodemographic characteristics and underlying etiology of BCS did not differ substantially between patients with and without PVT. PVT patients were more likely to have liver discomfort, a rise in white blood cells, and an increase in direct bilirubin. Doppler ultrasonography results were comparable in PVT and other patients.

Denninger et al. [13] found a mixed etiology for BCS in 25% patients. Mohanty et al, [14] reported that approximately 26% BCS were caused by the FVLM as a most prevalent risk factor. About 18.3% patients had MTHFR (Methylenetetrahydrofolate reductase) gene mutation. Another study reported an increased prevalence 45.1% of MTHFR found in BCS [15]. The prevalence of Behcet's disease (BD) 8.7% of the participants in the present investigation, similar to Turkey, which reported a 9% prevalence in BCS [16].

The present study found that PVT prevalence was 12.8% (6/46) among BCS patients composed of 4 males and 2 females. PVT was predicted to occur 14% of the time in a multicenter European study of BCS patients [17]. In the current study, the causes of PVT-BCS combination, unifactorial etiology, conclusive etiology, and multivariate cases were not found. However, previous studies conducted in Europe reported that unifactorial and multifactorial etiology was found in 18% and 58% respectively [18, 19].

Our instances were all symptomatic. PVT patients had a higher rate of liver soreness. In contrast, the most common presenting symptom in isolated PVT was variceal hemorrhage [20]. More severe manifestation of BCS could be the reason for major varices occur, drives medical attention [21]. Direct bilirubin levels were observed to be significantly higher in PVT patients. The cholestatic jaundice occurrence in PVT patients might be caused by portal hypertensive biliopathy development [22].

The severity of the condition is determined by amount of blockage (occluded arteries, total or imperfect occlusion) and the duration of symptoms [23]. Another study compared the single HVO with two and three occluded HVs and discovered that abdominal discomfort was statistically significant in 75%, 61%, and 86% respectively [24]. Recent results from several institutions indicate that primary BCS should be viewed as a complex illness, since multiple prothrombotic conditions may contribute to its development [25].

The present study found that BCS is most frequent in patients in their third decade of life, and it is more common in women. PVT is expected to occur in 12.8% of BCS patients. PVT in BCS patients has no effect on underlying etiology, clinical presentation, and venous thrombosis pattern in BCS.

CONCLUSION

A higher incidence of PVT was observed in the present study than previously reported. The BCS underlying etiologies and sociodemographic data were not significantly different between patients with and without PVT. Those with PVT were more likely to experience hepatic tenderness, increased white blood cells, and increased direct bilirubin. PVT and other patients had similar Doppler ultrasound findings.

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