

BUDD CHIARI SYNDROME- A CASE REPORT

*P Salome Satya Vani¹, Manichandana G², C.vani³

^{1,2,3} Department of Pharmacy Practice, V-Pharm.D2,
Pullareddy Institute of Pharmacy

ABSTRACT

BUDD CHIARI SYNDROME results from obstruction to the venous outflow of liver from the hepatic veins till the inferior vena cava. Clinical presentation of Budd Chiari Syndrome is variable. Most of the patients present with abdominal pain, hepatomegaly and ascites. We report case of 44yrs female admitted in hospital for the evaluation of the Budd Chiari Syndrome. The importance of correct diagnosis by the physician and sub sequent management is reviewed.

KEY WORDS: Budd Chiari Syndrome, myeloproliferative disorders, thrombotic, non-thrombotic

ORIGINAL RESEARCH ARTICLE

ISSN : 2456-1045 (Online)

(ICV-MDS/Impact Value): 72.30

(GIF) Impact Factor: 5.188

Publishing Copyright @ International Journal Foundation

Journal Code: ARJMD/MDS/V-33.0/I-1/C-8/JAN-2019

Category : MEDICAL SCIENCE

Volume : 33.0/Chapter- VIII/ Issue -1(JANUARY-2019)

Journal Website: www.journalresearchijf.com

Paper Received: 29.01.2019

Paper Accepted: 05.02.2019

Date of Publication: 15-02-2019

Page: 33-34

Name of the Corresponding author :

P Salome Satya Vani*

Assistant Professor, Department of Pharmacy Practice, V-Pharm.D2, Pullareddy Institute of Pharmacy, TS,INDIA

CITATION OF THE ARTICLE



Vani PSS, Manichandana G, Vani C (2019) Budd Chiari Syndrome- A Case Report; *Advance Research Journal of Multidisciplinary Discoveries*; 33(8)pp. 33-34

I. INTRODUCTION

Budd-Chiari syndrome (BCS) was actually described as an obstruction of hepatic venous outflow or hepatic portion of the inferior vena cava (IVC) [1]. The symptoms resulting from this type of occlusion of the hepatic outflow, "classical BCS", were first described by Budd [2, 3] in 1845 and later by Hans Chiari in 1899. It has been described to occur in 1 in 100,000 of the population [4, 5]. This syndrome usually occurs when a clot narrows or blocks the hepatic veins, which carry blood out of the liver. Because blood flow out of the liver is impeded, blood backs up in the liver, causing it to enlarge and the spleen may also enlarge. This causes blood pressure in the portal vein (which carries blood to the liver from the intestines) to increase. This increased pressure, called portal hypertension, can result in dilated, twisted (varicose) veins in the esophagus (esophageal varices). Portal hypertension, plus the damaged liver, leads to fluid accumulating in the abdomen (called ascites). The kidneys contribute to ascites by causing salt and water to be retained. The clot may extend and also block the inferior vena cava (the large vein that carries blood from the lower parts of the body, including the liver, to the heart). Varicose veins in the abdomen near the skin's surface may develop and become visible. Finally, severe scarring of the liver (cirrhosis) occurs [6]. Symptoms may vary from a completely asymptomatic condition to fulminant liver failure [7]. The diagnosis of Budd-Chiari syndrome should be clinically diagnosed in patients who present with any one of the following findings. They are fulminant liver failure with abrupt onset of ascites and hepatomegaly, massive ascites with relatively preserved liver function, unexplained chronic liver disease, or liver disease and an associated thrombotic disorder [8]. Budd-Chiari syndrome may be classified into three types depending on the type of existing venous occlusion. Type I is limited to the inferior vena cava. In type II BCS, lesions are within the hepatic veins. If lesions are short-segment occlusions (< 4 cm), type II a Budd-Chiari syndrome is the diagnosis. Budd-Chiari syndrome type III is the mixed type with involvement of the IVC and hepatic veins. Imaging studies play an important role in confirming the diagnosis

of Budd-Chiari syndrome by showing the venous abnormalities. Probably the most useful imaging methods include conventional and Doppler ultrasound, CT, MRI, and catheter venography [9]. The management of Budd-Chiari syndrome can be divided into three main categories: medical, surgical, and endovascular [10-13].

II. CASE REPORT

A female patient of age 44 yrs was admitted in hospital with the complaints of severe abdomen pain and loss of appetite generalized weakness since 15 days. Past history was Known Case of post MHV stenting 8yrs back. Her laboratory investigations include Hemoglobin :11gm%; WBC: 1700 thousands/cumm; platelets :1.5 lakhs; Na:137 mmol/lit Ca:108mg/dl; K : 3.6 mmol/lit; Total Bilirubin :0.7 mg/dl; Direct Bilirubin :0.6 mg/dl; Ultra Sonograph of abdomen : Intra hepatic IVC narrowing; based on the subjective (chief complaints) and objective(laboratory data) evidences confirmed diagnosis was BUDD CHIARI SYNDROME.

On day one of hospital admission patient is conscious/coherent, afebrile vitals are stable. Patient has Complaining abdominal pain. the treatment given the physician was inj. Tramadol 50mg in 100ml NS IV BD ,inj.pan 40mg IV OD, tablet Warfarin 5mg OD, Tablet dropin (Digestive Enzymes) 4mg IV BD. On the next day patient has complaining again abdominal pain, fever on and off. Then physician prescribed Tablet Dolo (paracetamol) 650mg (SOS). On third day on examination of abdomen soft and mild tenderness in right hypochondriac region, dilated vein over right hypochondriac region. Then the physician said continue the same treatment to the patient on that day and discharged. During discharge physician said come for review after 10 days. The discharge medications are Pylokit (tinidazole+ clarithromycin+lansaprazole) 500mg OD, T. colospa (mebeverine- used to treat abdominal cramps) 135mg BD, Warfarin 5mg OD.

III. DISCUSSION

This syndrome is less common in female than compared to males and the prevalence rate is 5.29 per million populations and the survival rate is 38-87%. In 2009-13 national wide South Korea patients are 424 patients and in France 4.04.BCS is associated with thrombotic or non- thrombotic obstruction and characterized by abdominal pain hepatomegaly and ascites. The pre disposing factors include Hemoglobin :11gm%; WBC: 1700 thousands/cumm; platelets :1.5 lakhs; Na:137 mmol/lit Ca:108mg/dl; K : 3.6 mmol/lit; Total Bilirubin :0.7 mg/dl; Direct Bilirubin :0.6 mg/dl; Ultra Sonograph of abdomen : Intra hepatic IVC narrowing. Hemoglobin :11gm%; WBC: 1700 thousands/cumm; platelets :1.5 lakhs; Na:137 mmol/lit Ca:108mg/d ;l K : 3.6 mmol/lit; Total Bilirubin :0.7 mg/dl; Direct Bilirubin :0.6 mg/dl; Ultra Sonograph of abdomen : Intra hepatic IVC narrowing. A diagnosis of Budd-Chiari syndrome should be considered in any patient who presents with acute or chronic liver disease, as the clinical manifestations are extremely diverse. The most

common cause in adults is thrombosis, which is secondary to an underlying myeloproliferative disease. And In children, the most common cause is a membranous obstruction of the inferior vena cava.

IV. CONCLUSION

Budd-Chiari syndrome is a rare case in females this syndrome can be fast and lead to death in couple of months and may be considered sudden onset of abdominal pain and hepatomegaly. After diagnosis antithrombotic medications must be given immediately.

V. REFERENCES

- [1] **Valla DC.** Vascular disorders of the liver. *Acta Gastroenterol Belg.* 2003;66:294-297.
- [2] **Budd G.** On diseases of the liver. Philadelphia: Lea and Blanchard; 1846.
- [3] **Budd G.** On diseases of the liver. Philadelphia: Blanchard and Lea; 1857.
- [4] **Aydinli M, Bayraktar Y.** Budd-Chiari syndrome: etiology, pathogenesis and diagnosis. *World J Gastroenterol* 2007; 13:2693-2696 .
- [5] **Valla DC.** Primary Budd-Chiari syndrome. *J Hepatol* 2009; 50:195-203 [Crossref] [Medline]
- [6] **Full review/revision October 2018 by Nicholas T. Orfanidis, MD**
- [7] **Senzolo M, Cholongitas EC, Patch D, Burroughs AK.** Update on the classification, assessment of prognosis and therapy of Budd-Chiari syndrome. *Nat Clin Pract Gastroenterol Hepatol* 2005; 2:182-190.
- [8] **Aydinli M, Bayraktar Y.** Budd-Chiari syndrome: etiology, pathogenesis and diagnosis. *World J Gastroenterol* 2007; 13:2693-2696.
- [9] **Patil p.** Deshmukh H, Popat B, Rathod K.Spectrum of imaging in Budd Chiari syndrome. *J Med imaging Radiat Oncol* 2012;56:75-83.
- [10] **Darwish Murad S, Plessier A, Hernandez-Guerra M, et al;** EN-Vie (European Network for Vascular Disorders of the Liver). Etiology, management, and outcome of the Budd-Chiari syndrome. *Ann Intern Med* 2009; 151:167-175.
- [11] **Inafuku H, Morishima Y, Nagano T, Arakaki K, Yamashiro S, Kuniyoshi Y.** A three-decade experience of radical open endvenectomy with pericardial patch graft for correction of Budd-Chiari syndrome. *J Vasc Surg* 2009; 50:590-593.
- [12] **Kazimi M, Karaca C, Ozsoy M, et al.** Live donor liver transplantation for Budd-Chiari syndrome: anastomosis of the right hepatic vein to the right atrium. *Liver Transpl* 2009; 15:1374-1377.
- [13] **Klein AS.** Management of Budd-Chiari syndrome. *Liver Transpl* 2006; 12(suppl 2):S23-S28.
