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Budd Chairi Syndrome- A Case Report and Review of Literature

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Keywords: Budd Chiari syndrome (BCS), Hepatic Encephalopathy, Alcoholic Liver Disease, Fatty Liver, Acute Gastroenteritis.

ABSTRACT

"Budd Chiari syndrome (BCS)" is an uncommon condition induced by thrombotic or nonthrombotic obstruction to hepatic venous outflow. This disease can be potentially life-threatening but prognosis is more favorable in patient with Inferior Vena Ceva webs but is extremely poor in malignant and haematological cases. A cooperative collaboration of hepatologist and cardiologist can make the situation easy for diagnosis, even in the treatment of some cases. The literature available on management of Budd Chairi Syndrome is scarce. In this Case report, the author present a case of 49 years old male patient who was admitted in Gastroenterology department of a tertiary care hospital with chief complaints of: Nausea, Vomiting, Pain in abdomen, Blood in the stool, Fever, Abdominal distension, Shortness of breath, Pedal oedema and from the final diagnosis the patient was diagnosed with Budd Chairi Syndrome, Hepatic Encephalopathy, Alcoholic Liver Disease & Fatty Liver Associated with Acute Gastroenteritis. The patient presented with acute deterioration in liver function and diffuse pain in abdomen and ascites. The liver enzymes stabilized on conservative management. There was feature of encephalopathy and bleeding tendency during hospital stay. However, ascites increased and became more tense and painful with prominent dilated veins over chest and abdomen. The Case report has been presented providing the detailed information of the Case in SOAP format. The author reviews the literature and discusses treatment options available for the management.

INTRODUCTION:

Budd-Chiari syndrome (BCS) was originally described as a rare vascular disorder that encompasses an array of symptoms due to obstruction of hepatic blood outflow at the level of the hepatic veins or hepatic portion of the inferior vena cava (IVC)[1]. The symptoms resulting from this type of occlusion of the hepatic outflow, "classical BCS", was first described by Budd[2,3] in 1845 and later by Hans Chiari in 1899. With the advancement of diagnostic and therapeutic techniques, providers have expanded upon these initial characterizations[4]. Historically, identifying the precise location of the obstruction was challenging, leading to the propagation of simplified descriptions. The precise location of the obstruction(s) is however clinically and prognostically significant. As Valla[5] proposed, the clinical manifestations of BCS (the selective group of symptoms that characterize the syndrome) can be explained by the location of the obstruction: Within the hepatic veins *vs* within the IVC at the level of the hepatic ostia. Over time, in order to incorporate novel and more detailed findings associated with BCS, the lexicon has evolved discordantly. The lexicon now includes a myriad of ambiguous terms or eponyms: Budd's disease, Chiari's disease, Chiari's syndrome, Rokitansky's disease, von Rokitansky disease, Hepatic vein outflow tract obstruction, membranous obstruction of the IVC, obliterative hepatocavopathy, Hepatic vena cava disease, Budd-Chiari syndrome with occlusion of hepatic vein, or hepatic vein thrombosis[6,8]. These eponyms have been used at some point during the course of further discovery; this disarray of terms, some of which are unclear and nonspecific, reflects not only the heterogeneous presentation of BCS, but also the possibility of distinct entities within this syndrome.

The currently accepted definition of primary BCS is hepatic outflow obstruction regardless of the cause or level of obstruction[6, 9]. The obstruction can range from the small hepatic veins to the orifice of the IVC into the right atrium. Sinusoidal obstruction syndrome is excluded from this definition[6, 9]. Secondary BCS is defined as a hepatic venous outflow obstruction due to compression or invasion by extravascular lesions, including benign or malignant diseases such as abscesses, hepatocellular carcinomas, and renal cell carcinomas, or secondary to cardiac or pericardial diseases[6, 9].

In 1998, Okuda et al[4] proposed that primary hepatic venous thrombosis (classical BCS) and thrombosis of the IVC at the level of the IVC were two separate syndromes. Recent studies continue to suggest a clear division within the definition of "primary BCS" based on the location of the obstructive lesion[4,10]. Obstruction of the hepatic veins or "classical BCS"

appears to be more common in Western patient populations and usually has a known etiology[11, 12], acute onset of symptoms, and a greater severity of symptoms requiring a different therapeutic approach than obstruction of the IVC at the level of the hepatic veins[13, 14]. In comparison with "classical BCS", hepatic vena cava (HVC)-BCS appears to be more common in East Asian patient population and is more often idiopathic or due to membranous obstruction. HVC-BCS more commonly presents with a chronic onset of less severe symptoms, thus requiring a different therapeutic approach than "classical BCS". The location, size, and chronicity are clinically important as it dictates the patient's symptoms and directs the therapeutic approach for patient management[10].

ETIOLOGY OF BUDD CHAIRI SYNDROME:

CAUSES	DISEASE
Thrombosis	Pregnancy Pills Myeloproliferative disorder Paroxysmal nocturnal Hemoglobinuria Polycythemia rubra vera
Non Thrombotic	Compression or invasion of IVC Membranous obstruction/IVC diaphragm
Systemic	Behcet syndrome Inflammatory Bowel Disease

PATHOGENESIS OF BUDD CHAIRI SYNDROME:

Blockage of two or more major hepatic veins increases the sinusoidal pressure and reduces sinusoidal blood flow. Obstruction of a single hepatic vein is generally not evident; two veins must be blocked for clinical disease[16]. The result of these hemodynamic changes is sinusoidal dilation and filtration of interstitial fluid. Filtrated interstitial fluid passes through the liver capsule when it exceeds the capacity of lymphatic drainage. Thus, liver congestion, right upper quadrant pain and ascites occur.

Portal pressure increases and perfusion of the liver *via* portal vein is decreased. The combined effect of these changes in hepatic circulation on liver parenchyma is hypoxic damage of hepatocytes. Non-inflammatory centrilobular cell necrosis is found in nearly 70% of cases. Reperfusion injury may contribute to hepatocyte damage. Hepatocyte necrosis coordinates

with release of free oxygen radicals and inflammation. Massive hepatocellular damage with a fulminant course is rare. Usually, portal hypertension and ascites are seen in chronic form. Both the acute and chronic forms result in severe centrilobular congestion and hepatocellular necrosis and atrophy. Within a few weeks after obstruction, fibrosis develops predominantly in the centrilobular are Within a few months, nodular regeneration may be seen predominantly in the periportal area. Progressive fibrosis, nodular regenerative hyperplasia and cirrhosis develop during the course of disease. Interventional portosystemic shunts or development of portal venous collateral system may improve liver functions and delay the cirrhotic process[15,19].

The caudate lobe, which has direct venous drainage into the IVC, often undergoes compensatory hypertrophy. Caudate lobe hypertrophy is found in half of the cases and causes IVC stenosis. Obstruction of the portal vein is present in 10%-20% of cases and may be related to stagnant blood flow and underlying thrombophilic disorder[17,18].

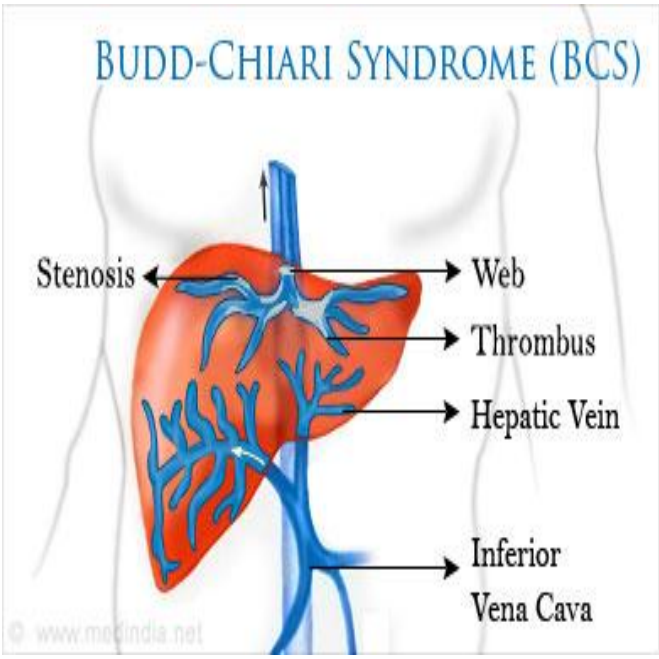

Figure 1: Budd Chairi Syndrome

REVIEW OF LITERATURE:

S.NO	AUTHOR	TITLE	CONCLUSION
1.	Aure´liePlessier, Dominique-Charles, Valla, et al..	Budd-Chiari Syndrome	A treatment strategy is recommended where anticoagulation is given first, followed by angioplasty when appropriate, then TIPS in patients not responding to previous measure, and finally liver transplantation. This strategy has achieved 5-year survival rates close to 90%.
2.	A.V. Kyriakidis , Vezyrgiannis , M. Pyrgioti	Budd-Chiari syndrome	Several works have enabled the comprehension of Budd-Chiari syndrome as well as the diagnosis and treatment. This clinical review attempted to present the Budd-Chiari syndrome overall and more particularly the history, etiology, pathophysiology, diagnosis and the understanding of the latest developments in conservative and surgical management as well as prognosis.
3.	• Praveen K Ro, BS Anand, Sarah D Komanapalli,	Budd-Chiari Syndrome	The prognosis is poor in patients with Budd-Chiari syndrome who remain

			untreated, with death resulting from progressive liver failure in 3 months to 3 years from the time of the diagnosis. Following portosystemic shunting, however, the 5-year survival rate for patients with the BC syndrome is 38-87%. The actuarial 5-year survival rate following liver transplantation is 70%.
4.	Naomi Shin, Young H Kim, Hao Xu, Hai-Bin Shi, Qing-Qiao Zhang, Jean Paul Colon Pons, Ducksoo Kim, Yi Xu, Fei-Yun Wu, Samuel Han, Byung-Boong Lee, and Lin-Sun Li	Redefining Budd-Chiari syndrome: A systematic review	Systematic review of recent data suggests that classical BCS and HVC-BCS may be two clinically different disorders that involve the disruption of hepatic venous outflow.
5.	PieterMartens and Frederik Nevens	Budd-Chiari syndrome	For the Budd-Chiari patient presenting with fulminant hepatic failure, first-line treatment with OLT(orthotopic liver transplantation) seems reasonable. It is clear that for these reasons patients with BCS should be treated in a center able to perform OLT.

6.	Hector Ferral, George Behrens, and Jorge Lopera	Budd-Chiari Syndrome.	BCS requires accurate, prompt diagnosis, and aggressive therapy. Treatment will vary depending on the clinical presentation, cause, and anatomic location of the problem. Patients with BCS are probably best treated in tertiary care centers where liver transplantation is available
7.	John D. Horton , Francisco L. San Miguel and Jorge A. Ortiz	Budd Chiari syndrome: illustrated review of current management	Multidisciplinary approach to diagnosis and treatment is advantageous in patients with BCS. Medical therapy consists of treatment of underlying disease, anticoagulation and symptom control. Emerging technologies have offered new minimally invasive treatment modalities such as percutaneous catheter-directed thrombolysis, angioplasty, stenting and TIPS.
8.	Dustin E Loomes, MA Albert Chang, , Douglas Webber, Charles H Scudamore ,and Eric M Yoshida	Acute Budd-Chiari syndrome	A 23-year-old woman presented to hospital with a two-week history of abdominal pain, nausea and increasing abdominal pain. Chronic Budd-Chiari

			syndrome is managed with anticoagulation and treatment of the complications of liver failure. Rarely, liver transplantation may be necessary.
9.	Musa Aydinli and Yusuf Bayraktar	Budd-Chiari syndrome: Etiology, pathogenesis and diagnosis	Budd-Chiari syndrome is a congestive hepatopathy caused by blockage of hepatic veins. The liver biopsy may be helpful for differential diagnosis. The prognosis of the chronic form is acceptable compared to other chronic liver diseases.
10.	Cura M, Haskal Z, Lopera J; Boozari B, Bahr MJ, Kubicka S, et al	Budd-Chiari Syndrome	Budd-Chiari syndrome (BCS) occurs when there is obstruction of the hepatic veins. Liver transplantation may be appropriate if there is decompensated liver cirrhosis.Reconstructing hepatic venous outflow post-transplant is sometimes a problem but successful venoplasty using autologous vein grafts has been reported.
11.	Vatsala Misra, Kachnar Verma, Dharmendra Kumar Singh,and Sri	The Budd-Chiari Syndrome in a Child: A Case	This case report highlights a rare case of BCS in a child. A high index of clinical

	Prakash Misra	Report and Review of the Literature	suspicion, along with a radiological aid and a histopathological correlation, can lead to an early diagnosis and an appropriate management in such cases.
12.	Ouhadi L, Creemers E, Honoré P, Delwaide J, Marchetta S, Defraigne JO.	Budd-Chiari syndrome: a case report and review of the literature.	The history of a 20-year-old woman admitted for thrombosis of the hepatic veins and of the inferior vena cava (IVC) with extension of the thrombus into the right atrium. Finally, Hepatic transplantation should be considered in case of treatment ineffectiveness, of fulminant hepatic failure, or of an evolution towards cirrhosis.
13.	Daniela T Carvalho, Fernando T Oikawa, Nilce M Matsuda Paulo RB Évora and Alice T Yamada	Budd-Chiari syndrome in a 25-year-old woman with Behçet's disease: a case report and review of the literature	Severe vascular complications of Budd-Chiari syndrome in patients with Behçet's disease are much more common in young adult male patients; we present a rare case of Budd-Chiari syndrome in a young Afro-Brazilian woman with Behçet's disease.
14.	Alok Khanna, Vijay	Acute Hepatitis	The patient presented with

	Lakshmi Sood , Dalal, Pankaj Abrol, Shalini Agarwal,	Like Presentation of Budd Chiari Syndrome in a Child	progressively increasing ascites and pain abdomen but without any bleeding tendency, or encephalopathy. The initial diagnosis of acute hepatitis was proved wrong by contrast-enhanced CT scan of abdomen which showed atretic hepatic veins. Atretic hepatic veins causing Budd Chiari Syndrome (BCS) is a rarity in literature.
15.	Michael F. Sorrell	Budd-Chiari Syndrome: Case Report	A 23-year-old white woman presented to doctor with a 6-week history of progressive abdominal swelling. She received furosemide to treat fluid retention. Two weeks later, she returned to her physician with increasing abdominal discomfort and a 5-kg weight gain. She was referred for specialty care
16.	A. S. Katkar, Anderson H. Kuo,[1] S. Calle, K. Gangadhar, and K. Chintapalli	Budd-Chiari Syndrome Caused by TIPS Malposition: A Case Report	A case of Budd-Chiari syndrome that occurred in a transplanted cirrhotic liver from malpositioned proximal portion of the TIPS in IVC causing occlusion of the ostia of hepatic veins which was

			subsequently diagnosed on contrast-enhanced CT. Ten days later, a follow-up CT of the abdomen and ultrasound was performed showing interval development of a fluid collection at the site of the previously documented liver hypoattenuation, consistent with hepatic abscess formation, was later drained via percutaneous approach using ultrasound guidance.
17.	Md. Mukhlesur Rahman, KMHS Sirajul Haque, Md. Mahmudur Rahman Siddiqui, Tanjima Parvin, Md. Khurshed Ahmed	'Budd Chiari Syndrome' - A Case Report	Budd Chiari syndrome is a rare and confusing disease of the hepatic circulation. Sometimes it is misdiagnosed as a case of only chronic liver disease but a good venography can remove all kind of confusion like this patient.
18.	Khaled Ali Jadallah,Enas Walid Sarsak,Yara Mohammad Khazaleh, Rawan Mohammad, Khair Barakat	Budd-Chiari syndrome associated with coeliac disease: case report and literature review	In conclusion, this case report highlights the intriguing association between BCS and CD, and sheds some light on the putative pathogenic mechanism. An underlying pro-thrombotic condition was detected in less than

			50% of the patients, implying a possible thrombogenic role for CD. suggest that a diagnosis of CD should be pursued in the setting of BCS of undetermined cause. Similarly, CD patients with unexplained manifestations of acute or chronic liver injury should be assessed for BCS.
19.	Merz, Waltraut ,Rüland, Anna,Hippe, Valeria ,Poetzsch, Bernd, Meyer, Carsten, Pollok, Joerg M, Gembruch, Ulrich Trebicka, Jonel	Pregnancy in Budd-Chiari Syndrome: Case Report and Proposed Risk Score	The usefulness of these and additional criteria, however, needs to be established.With numbers of women with BCS seeking pregnancy expected to rise, the scoring system may help in preconception risk assessment and counseling; furthermore, it may support in the establishment of treatment algorithms of BCS in pregnancy.
20.	David Clain. M.B, James Freston , Louis Kreel. Sheila Sherlock.	Clinical diagnosis of the Budd-Chiari syndrome: A report of six cases	Five patients responded to a modern diuretic regimen and polycythaemia vera was successfully treated in three. Exploratory surgery is not advised in the Budd-Chiari syndrome.

			Special roentgenologic procedures are the only means of making an accurate diagnosis.

CASE REPORT:

CASE REPORT ON BUDD CHAIRI SYNDROME, HEPATIC ENCEPHALOPATHY, ALCOHOLIC LIVER DISEASE & FATTY LIVER ASSOCIATED WITH ACUTE GASTROENTERITIS

A 49 years old male patient was admitted in Gastroenterology department of a tertiary care hospital with chief complaints of:

- ✓ Nausea.
- ✓ Vomiting.
- ✓ Pain in abdomen.
- ✓ Blood in the stool.
- ✓ Fever.
- ✓ Abdominal distension
- ✓ Shortness of breath
- ✓ Pedal oedema
- ✓ Ascites

H_X OF PRESENT ILLNESS:

✓ Patient was asymptomatic 2days back, develop Nausea, Vomiting, Abdominal distention, Ascites and loose motions.

PM_X WITH ALLERGIC STATUS:

✓ K/C/O:- Alcoholic liver disease with Fatty Liver

FAMILY HISTORY:

- ✓ Mother-Arthritis
- ✓ Father- Hypertension, Type-2DM.

PHYSICAL EXAMINATION:

- ✓ Temp:-Normal
- ✓ RR:-22/mint
- ✓ PR:-82b/mint
- ✓ BP:-120/80mmHg

DISCUSSION

This is a rare case. The usual cause is obstruction of inferior vena cava (IVC), Atresia of hepatic veins. Two-third of cases of obstruction are due to membranes or webs in IVC and most of the remaining are due to thrombosis in the IVC or hepatic veins[11]. Lent(1899) his first pathological description of 'obliterative endophlebitis of hepatic veins'. The BCS refers to the clinical picture that occurs when there is obstruction to the hepatic venous outflow. The classic acute presentation is with the triad of ascites, hepatomegaly and abdominal pain[12].

BCS can present as fulminant, acute and chronic. Fulminant is characterized by acute deterioration in hepatic function and encephalopathy. Acute non-fulminant shows significant liver damage and lab abnormalities vary widely. About 25-30% of BCS present acutely[13].

This patient presented with acute deterioration in liver function and diffuse pain abdomen and ascites. The liver enzymes stabilized on conservative management. There was feature of encephalopathy and bleeding tendency during hospital stay. However, ascites increased and became more tense and painful with prominent dilated veins over chest and abdomen.

CASE REPORT OF BUDD CHAIRI SYNDROME(SOAP FORMAT)

SUBJECTIVE:

A 49yrs old male patient was admitted in the Gastroenterology department of a tertiary care hospital with chief complaints of Abdominal pain, Ascites, Nausea, Vomiting, Fever & blood in stool, Shortness of Breath(SOB), Pedal oedema.

OBJECTIVE:

Laboratory investigation revealed

- ✓ Hb- 10gm/dl,
- ✓ WBC-7800/mm3,
- ✓ Platelet count-370000/mm3,
- ✓ PCV-36%,
- ✓ Peripheral blood film was unremarkable.
- ✓ SGPT-56U/L(Normal <50U/L)
- ✓ SGOT 50U/L(Normal<45U/L)
- ✓ Serum bilirubin -2.3mg/dl,
- ✓ Prothrombin time was prolonged(patient 21 second, control 12 second)
- ✓ Serum albumin - 2.8gm/dl .
- ✓ Ascitic fluid was transudative and negative for malignant cell.
- ✓ APTT - 33 second (Normal 26-36second)
- ✓ D-dimers - 763 ng/ml (Normal <500ng/ml).
- ✓ Anti-phospholipid Ab(IgGand IgM) were negative.
- ✓ Chest Xray was normal and transthoracic echocardiography revealed thrombus in the right atrium and mild pericardial effusion but no evidence of constrictive pericarditis.

✓ Ultrasonogram of whole abdomen showed marked Hepatomegaly with enlargement of the caudate lobe of the liver and ascites.

✓ Doppler Ultrasonogram showed dilated inferior venacava with partial occlusion by thrombus, moderate portal hypertension, no evidence of intrahepatic venous obstruction. MRI of abdomen showed total occlusion in proximal inferior venacava with possible intraluminal thrombus in proximal Inferior Venacava (IVC), hepatomegaly, and ascites

✓ Upper GIT endoscopy showed grade 1 esophageal varises. Venography revealed right heart catheter could not be passed beyond the level of hepatic vein, complete obstruction in inferior venacava just 1 cm below the diaphragm. Superior venogram showed few millimeter of IVC. There was no indentation of caudate lobe.

✓ About 1 year back he was diagnosed as a case of cirrhosis of liver, but now finally after doing venography he is diagnosed as a case of "Budd Chiari Syndrome" due to membranous obstruction of the Inferior Venacava (IVC) with Cirrhosis of Liver.

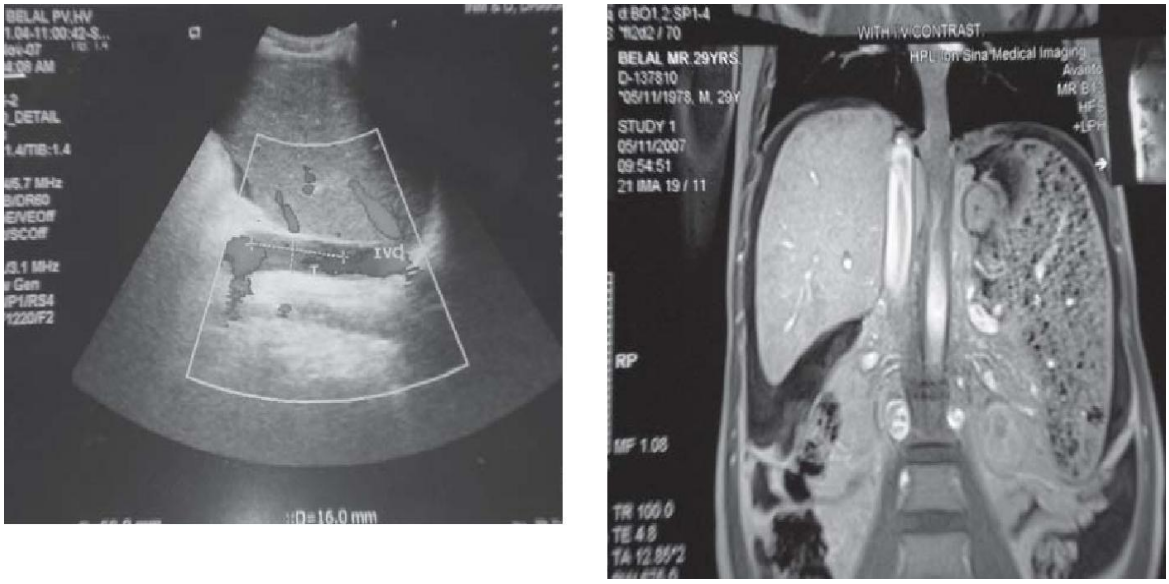

Figure 2: Dilated IVC, partial occlusion with Thrombus

Figure 3:IVC is total cut just after diaphragm

ASSESSMENT:

❖ A: From the above data the patient was diagnosed with Acute Gastroenteritis ,Hepatic Encephalopathy, Alcoholic Liver Disease, Fatty Liver & Budd chairi syndrome

I. Problem 1: Nausea &Vomiting.

II. Problem 2: Blood in the stool.

III. Problem 3: Fever.

IV. Problem 4: Burning sensation in Epigastric region.

V. Problem 5:Budd Chairi syndrome

VI. Problem 6:Alcoholic Liver Disease

VII. Problem 7:Fatty Liver

VIII. Problem 8:Hepatic encephalopathy

PROBLEM 1:-NAUSEA AND VOMITING:

✓ Anti-emetic therapy has been started i.e., Zofer, it should be continued.

✓ Add Zofer(Ondansetron)4mg IV TID.

✓ Management of the diet is a first priority for the treatment of Diarrhoea

✓ Rehydration & maintenance of water & electrolytes are the primary treatment measures until the diarrheal episode ends.

✓ Advise the patient to maintain Hygienic conditions.

✓ Advising patients to increase fluid intake

PROBLEM 2: BLOOD IN STOOL:

✓ This is due to Enterotoxin induced inflammatory Gastroenteritis.

✓ Sporolac 2Tablets and Inj. Metronidazole 100ml IV TID is suggested by the physician.

PROBLEM 3: FEVER

✓ Temperature is to be monitored q 4^{th} hour,.

✓ So, Ibuprofen & Paracetamol can be administered(if Fever persists).

PROBLEM 4: BURNING SENSATION IN THE EPIGASTRIC REGION:

✓ Syrup Sucralfate 10ml PO TID is administered and it is monitered carefully as it Adverse effects are many as it may cause Diarrhoea, Nausea, Constipation and Headache.

PROBLEM:5 BUDD CHAIRI SYNDROME:

✓ Formation of blood clot within the hepatic veins can lead to Budd chairi Syndrome.

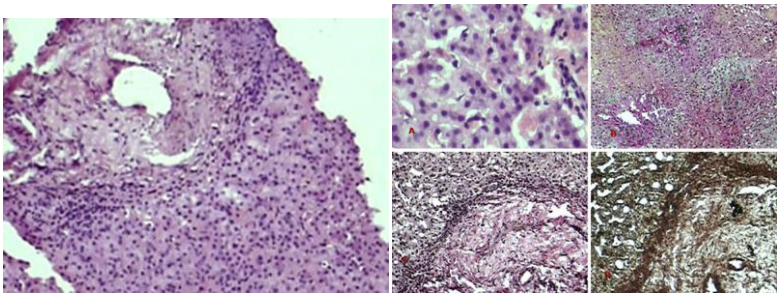

Figure 4: Photomicrograph showing histopathologic features of Budd Chairi Syndrome with centrizonal congestion, perivenular fibrosis.

Figure 5: Photomicrograph showing centrizonal congestion and mild degeneration.

TREATMENT OF BCS:-

✓ **Anticoagulant therapy** - If the blockage is caused by a clot, anticoagulant drugs such as warfarin can help in preventing the clots from recurring. The patient should be monitored regularly to maintain the anticoagulant effect within the acceptable range.

✓ **Percutaneous transluminal angioplasty** - can help widen the veins narrowed by clots. This procedure involves placing a stent to keep the vein open.

✓ **Liver transplant** - is another life-saving option especially for people with severe liver decompensation and failure.

✓ **Transjugular intrahepatic portal-systemic shunting (TIPS)** - can create an alternate route for blood flow thereby bypassing the liver. This reduces the pressure on the hepatic portal vein. This procedure is necessary if the patient presents with portal hypertension.

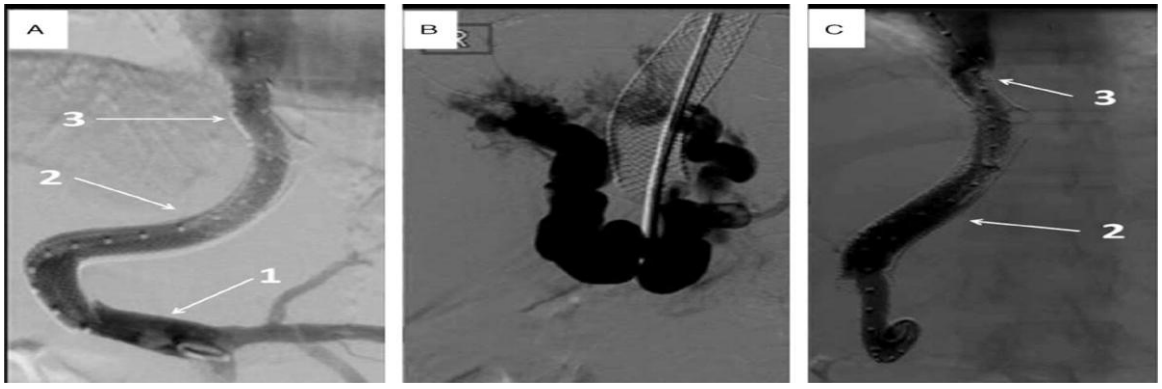

Figure:6(A) Invasive portography after left-sided Transjugular intrahepatic portal-systemic shunting (TIPS) placement demonstrating patent TIPS perfusion. (B, C) Invasive TIPS-control demonstrating cavernous transformed portal occlusion and restored hepatopetal flow after TIPS elongation: (1) main portal vein; (2) left portal vein; (3) left hepatic vein. TIPS = transjugular portosystemic shunt.

PROBLEM 6:-ALCOHOLIC LIVER DISEASE(ALD):

✓ Alcoholic Liver Disease(ALD) is a heterogenous disease & caused by alcoholism and can be divided into 3types:-

✓ Hepatic steatosis, Alcoholic hepatitis & Cirrhosis.

Pharmacists intervention:-

✓ All the patients with alcohol related liver disease should abstain from alcohol.(Stop drinking alcohol)

Preventing relapses:-

✓ Once the patient stopped drinking alcohol, they need further treatment to help ensure that they don't start drinking again.

✓ The first treatment usually offered is psychological therapy.

✓ If the psychological therapy alone is not effective, the patient may also need medications to help abstain from alcohol

✓ The medications include:

- ✓ Acamprosate
- ✓ Naltrexone
- ✓ Disulfiram

DIET AND NUTRITION:-

✓ Malnutrition is common in people with ARLD(Alcohol- Related Liver Disease), so it is important to take a balanced diet.

PROBLEM 7:-FATTY LIVER:

✓ Fatty liver disease consists of Alcoholic liver disease(ALD), and non-alcoholic liver disease(NAFLD).

✓ Symptoms:-Malaise, Fatigue, Snores, Disturbed sleep, Chronic pain disorders.

✓ Physical examination:-Abdominal obesity, Enlarged liver, RUQ(Right Upper Quadrant) tenderness on palpitations

Pharmacists Interventions:-

✓ FATTY LIVER may resolve with weight loss.

✓ Diet and exercise improve insulin sensitivity, increase oxidative capacity

✓ Modify underlying metabolic risk factors i.e., Diet and Exercise

PROBLEM 8:-HEPATIC ENCEPHALOPATHY

✓ For those with severe disease i.e.,hepatic encephalopathy and no contraindications to their use steroids should be considered

✓ Liver transplantation remains an option for selected patients with end stage liver disease due to alcohol

PLAN:

✓ Tab.Sporolac(Lactic acid bacillus)2tabs PO TID.

- ✓ Inj.Zofer(Ondansetron)4mg IV TID.
- ✓ Tab.Parasafe(Paracetamol)500mg PO TID.
- ✓ Inj.Metrogyl(Metronidazole)100ml IV TID.
- ✓ Syp.Sucral(Sucralfate)10ml PO TID.
- ✓ IVF 30RL(Ringer Lactate)@100ml/hr
- ✓ IVF 10NS(Normal saline)@100ml/hr
- ✓ Tab Lasix 40mg PO BD.
- ✓ Tab Fruselac(Spironolactone+furosemide) 20mg PO BD
- ✓ Tab.Bilefix(Ursodeoxycholic acid)300mg PO BD

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