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Budd Chiari Syndrome: A Rare Case Report

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Abstract: ***Budd Chiari syndrome (BCS) is a rare disease which occurs one in million. It is caused by occlusion of hepatic veins that drains the liver. A 19-year-old female patient admitted with complaint of abdominal distension for 4 months. From CT scan its confirmed as BCS and treated with anticoagulants, thrombolytic agents and diuretics.***

Keywords: Budd Chiari syndrome, abdominal distension, CT scan

1. Introduction

BCS occurs in 1/100000 of the general population worldwide. Patient may present with acute signs and symptoms related of abdominal pain, ascites and hepatomegaly or more chronic symptoms related to long-standing portal hypertension.[1]BCS is classified as primary when the obstruction to hepatic venous outflow is related to primary venous problem, such as thrombosis, stenosis, or webs, and as secondary when it is related to extrinsic compression, such as that caused by abscess, tumor, cyst, or hyperplastic nodules. The presentation and cause of BCS may vary depending on the geographic area of presentation. Interestingly, obstruction of the IVC with or without involvement of the hepatic veins is predominant in Asia, and pure hepatic vein obstruction predominates in western countries. Medical management of BCS is focused on the treatment of the underlying cause: control of portal hypertension, ascites control, systemic or catheter-directed thrombolysis and anticoagulation. Medical management is usually insufficient and most patients will require either surgical or endovascular intervention. Most patients who undergo endovascular or endovascular management will need long-term anticoagulation because most have an underlying prothrombotic disorder. [2]

2. Case Report

A 19-year-old female patient admitted with chief complaints of abdominal distension for 4 months, as there was no significant past history and family history. In general physical examination she was found to have edema. Patient was apparently normal 4 months back then she noticed to have distension of abdomen progressive in nature, after 1 month it gotdecreased. Then again noticed distension of abdomen for 3 months progressed to current stage. No H/O fever, loss of appetite, melena, hematochezia and loss of weight. No H/O PTB in the past and PTB in family. On admission her BP was 120/100 mm/hg, PR- 150 bpm and SPO2 of 98 percentage at room air. Prognosis is explained to parents. On 3rd day of admission she complained of fever which was subsided after treatment. She was infused with albumin 100 ml over 2-3 hours on 9th day of admission and had an ascetic tapping on the same day and removed 2.5 liters. Provisional diagnosis made was CLD and also suspected to have abdominal TB. For confirmation of diagnosis she underwent colonoscopy, autoimmune test, 2D Echo, RFT, LFT, CBC and abdominal CT. USG report gave Liver parenchymal disease with caudate lobe hypertrophy and CT scan showed the evidence of hepatomegaly with heterogeneous enhancement of liver parenchyma and non -s visualized hepatic vein due to BUDD CHIARI SYNDROME. She was treated with Inj. Pan 40 mg OD, Tab. B- complex OD, Inj. Lasix 40 mg IV OD for 3 days and increased to 60 mg TID for remaining days, Tab. Aldactone 50 mg OD for 2 days and increased to 100 mg later,Tab. Iron + folic acid BD, Syp. Lactulose 15 ml TID, Inj. Ondansetron 4 mg iv BD, Inj. Paracetamol 1g BD, Inj. Vitamin K 10 mg OD for 2 days, Inj. Ceftriaxone 1 gm iv for 7 days, Inj. Vitamin B 12 OD, Inj.Optineuron in 100 ml NS for 4 days and Inj. Enoxaparin. On discharge she was given with T. Pan 40 mg, T. Lasix 60 mg BD, T. Aldactone 100 mg OD, Tab. Iron + folic acid BD and Tab. Vitamin B 12 OD.

3. Discussion

BCS is uncommon disease which occurs one in million. Clinically presented with ascites, portal hypertension, and hepatomegaly, swelling of legs, ankles and itching. One of the complications associated with BCS is liver failure which is present in this patient. There are different clinical variants of BCS such as acute, chronic and fulminant forms. From the physical examination it is evident that the patient is suffering from chronic BCS.Management includes anticoagulants, thrombolytic agents and diuretics, but in thiscase there is vitamin K which helps in coagulation and continued for 2 days, but after confirmation of BCS it's been stopped. Patient received Tab. Furosemide, Tab. Aldactone and Inj. Enoxaparin which is according to the standard treatment. Inj. Ceftriaxone is given as prophylactic, Tab. Folic acid and iron is given as supplement to treat low blood levels. Remaining drugs are given for symptomatic treatment.

4. Conclusion

This case report highlights a rare case. Currently the patient receives medical management alone, further follow up should be done to know the improvement and need of surgical care.

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