Primary Budd-Chiari Syndrome: Diagnostic Significance of Intrahepatic Collaterals on Color Doppler Sonography

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Abstract: Purpose: is to analyze the frequency of intrahepatic collaterals in primary Budd-Chiari Syndrome (BCS) on color Doppler ultrasound and asses its diagnostic significance compared to other sono-morphological signs. **Patients and methods**: Doppler findings in 23 diagnosed primary BCS patients (examined between 2005-2010) retrospectively reviewed. Diagnosed by clinical/laboratory data as primary BCS. CT confirmed Diagnosis (n=20).MRI confirmed remaining 3 pregnant patients. Doppler evaluation for hepatic veins, intrahepatic collaterals, IVC patency, portal & hepatic veins. **Results**: Using Doppler, intrahepatic collaterals seen in 21 of 23 (91.3%). Subcapsular collaterals alone in 7 (30.4%), Portovenous intrahepatic collaterals alone in 2 (8.69%), both combined in 12 patients (52.17%). Absence of hepatic veins/artery abnormal flow confirmed Veno-venous nature excluding arterio-portal and arterio-venous collaterals. Intrahepatic collaterals detected on CT in 17/20 (85%). Difference of venovenous collaterals detectability in primary BCS (without sub-typing) by US (91.3%) compared to CT (85%) was nonspecific as P. value was $P>0.05$. **Conclusion:** The non-invasive US / CDS has high detection rate of diagnostic signs in primary BCS. Highest significance of intrahepatic collaterals (particularly subcapsullar veno-venous) on CDS is significantly unique. This importance is more significant as CDS/US is the initial modality primary BCS.
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Keywords: Budd-Chiari, Doppler ultrasound, liver, intrahepatic collaterals.

Abbreviations: BCS = Budd Chiari Syndrome, CT = Computed Tomography, US = Ultrasound, TIPSS = Trans-jugular Porto Systemic Shunt, MRI = Magnetic Resonance Imaging, CDS = Color Doppler sonography

1. Introduction

Budd Chiari Syndrome (BCS) represents a spectrum of disease states resulting from hepatic venous outflow occlusion. Obstruction can occur at any level from hepatic venous to the right atrium of the heart.[1] Hepatic biopsy and venography are invasive procedures that gave good results in diagnosing BCS.[2] Hepatic biopsy results can be non specific in addition to sampling errors. [2&3] Venography cannot show collaterals when hepatic venous catheterization is unsuccessful. [3]

Radiological imaging plays an important role in the evaluation of patients suspected to have BCS. Recently, the detection rate of BCS has increased, because of progress in diagnostic imaging. [4] In fact under current consensus recommendations, radiological imaging is sufficient to make a diagnosis of BCS. [2] Awareness of its imaging findings is important for early diagnosis and appropriate treatment.[5]

With the improvement of ultrasound (US) resolution, US have become the first choice for non-invasive diagnosis of BCS. In most circumstances, US can provide the position, extent, degree and characteristics of Hepatic veins (HV) or inferior vena cava (IVC) abnormality, which provide important information for clinical therapy of BCS. [4] Color Doppler ultrasound (CDS) is a non-invasive procedure that has the advantage over Computed tomography (CT) and Magnetic resonance imaging (MRI) of hemodynamic vascular assessment and direct assessment of flow in examined vessels. Real time gray-scale sonography combined with color flow and duplex Doppler is considered the initial imaging technique in Budd-Chiari Syndrome [6, 7, 8] with a sensitivity and specificity of nearly 85%.[9, 10] Comparing modalities in BCS, sonography was found superior to CT in delineating IVC membranes and showing the relationship between the obstructed segment of IVC and the hepatic venous anatomy as well as intrahepatic venous collaterals and the direction of flow, especially with Doppler US. [11] Color and spectral Doppler ultrasonography allows demonstration of both the presence and direction of hepatic venous flow pattern even when they are not clearly visible on gray-scale examination. [12]

In BCS hepatic blood must find collateral pathways to exit the liver. [3] Two forms of intrahepatic

collaterals may develop: Those that communicate with systemic veins via the subcapsullar vessels and those that shunt blood from the occluded to non-occluded segments of hepatic veins. Usually collaterals develop between the right and left hepatic veins because the opening of the left hepatic vein is anatomically proximal to the IVC either at or above the obstructed IVC. [13] Intrahepatic venous collateral were considered relatively rare but have been reported in the literature with increasing frequency and are not as rare as once believed.[14]

Demonstration of intrahepatic veno-venous collaterals is a specific sign of BCS (in the proper clinical setting and supported by other imaging findings, and can include subcaspular veins, vessels shunting between hepatic veins, large veno-venous collaterals draining into the IVC, and the classical spider collaterals may also occur. [15] Intrahepatic and subcapsular venous collaterals are sensitive sonographic findings of BCS present in up to 80% of cases. [16] Synchronous presence of "alterations of flow in hepatic and/or caval veins" as well as "caudate lobe hypertrophy" provided the highest predictive value to identify BCS reaching a specificity of 100%. The absence of these signs exclude BCS, even if a combination of other ultrasound signs were present.[17]

A lot of care must be taken to observe the entire length of the detected collaterals by CDS combined with spectral analysis to assume the cause of collaterals. [14] Shunts between hepatic arteries and hepatic veins are rare but may occur in cavernous lymphangiomatosis or in Rendu-Osler-Weber syndrome. [(8)] More commonly arterio-systemic shunts occur in the setting of hepatocellular carcinoma with venous invasion. They may also develop after liver biopsy or penetrating trauma. CDS may reveal dilated tortuous hepatic arteries and aliasing at the junction with the draining hepatic vein. Although arterio-portal shunts are most typical vascular communications associated with hepatocellular carcinoma, identification of hepatic artery-to-hepatic vein shunt should also prompt a search for an underlying neoplasm.[16] In tumor patients, venous collaterals occurred around the tumor as tumor thrombus. This observation helps in assuming the obstructive site, and the presence of a tumor should be considered when localized venous collaterals are visible on CDS. [14] The hepatic veins may also show abnormal color-flow patterns and flow tracing s typically show arterializations.[16] Similar vessels (collaterals) are also reported with diaphragmatic hernia, Rendu-Osler-Weber syndrome and congestive heart failure. [4]

The addition of analysis of Doppler waveform proved to be very helpful in differentiating venous collaterals with venous obstruction from those without. Phasic flow in the venous collaterals in patients without veno-occlusive disease, instead of flat flow usually observed in the venous collaterals due to veno-occlusive disease. [14]

In BCS, CDS may reveal intrahepatic veno-venous collaterals that provide alternative pathways with obstruction. Gray-scale sonography in BCS may show low level echogenic material within normally sonolucent lumen of hepatic vein. However, if the hepatic veins appear patent on gray scale and CDS, spectral tracings should be obtained. [16]

Isolated abnormalities in the hepatic waveform morphology such as absence of phasic oscillation in flat flow are of limited value in the diagnosis unless they are associated intrahepatic collateral vessels because a similar Doppler pattern can be observed in patients with fatty liver, active chronic hepatitis or liver cirrhosis. [9]

The aim of this study is to analyze the frequency of intrahepatic collaterals in primary Budd-Chiari Syndrome (BCS) on color Doppler ultrasound and asses its diagnostic significance compared to other sono-morphological signs.

2. Materials and Methods:

Twenty three patients with Budd-Chiari Syndrome (10 males and 13 females) were evaluated by US & CDS. Sonographic findings in 23 patients with BCS were retro respectively reviewed. Patients are examined, in addition to US, by CT (14 patients), MRI (3 patients) and Venography (5 patients). US were done using Philips-ATL HDI 5000 US machine, (Philips Ultrasound, Bothell, WA), 5MHz curvilinear probe. Examinations were done in Grayscale for liver size, lobar ratios, parenchymal echo texture, focal lesions & abnormal possible vascular channels. Power Doppler, Color Doppler and Pulsed Doppler are used for hepatic veins, IVC and portal vein. For each patient the following were evaluated using Doppler Imaging: The appearances of hepatic veins, the direction of flow (normal or reversed) and the morphology of the Doppler Spectra, the presence, distribution and nature of intrahepatic collaterals, the patency of IVC and portal system and the direction of flow in portal vein. Doppler analysis of the hepatic veins and IVC were considered normal when they exhibited a triphasic spectral pattern with two periods of forward flow (corresponding to the two phases of right atrial filling and one period of reversed flow (corresponding to right atrial systole). The portal spectrum was considered normal when it had steady typical wave form with only respiratory variations, and a velocity ranging from 0.15-0.30m/sec. Patients with thrombosis secondary to venous compression or tumor invasion were excluded from this study to obtain homogenous population of primary BCS.

Statistical method:

We used SPSS statistical package ver. 15.0 for data analysis. We calculated the frequencies (percentages) for different variables. Also, we used Fisher's Exact Test to compare between CT findings and US findings. We assumed that there is a statistically significant difference if $P<0.05$.

3. Results:

Intrahepatic collaterals were seen in 21 patients out of 23 (91.3%) (Table1) and (Fig.1). There were seen as subcapsular collaterals alone in 7 patients (30.4%), portocaval collaterals alone in 2 patients (8.69%) while they were combined in 12 patients (52.17%) (Table 2). They were confirmed as veno-venous collaterals with absence of hepatic artery abnormal appearance or abnormal (arterial) flow in hepatic veins to exclude the possibility of arterio-portal or arterio-venous collaterals. Detectability of collaterals (without sub-typing or flow characterization) by US was (91.3%) as compared to CT (85.7%) (Table 3). The difference was nonspecific as *P*. value was around 0.05.

Table (1) Color & Pulsed Doppler Findings (n=23)

P.V. Thrombosis	**(n=6/23) (26.09%)**
Reversed (hepatofugal)	**(n=2/23) (8.69%)**
IVC Thrombosis	**(n=7/23) (30.43%)**
IVC Segmental narrowing	**(n=4/23) (17.39%)**
Bi-directional Flow (in hepatic veins)	**(n=3/23) (13.04%)**
HVs attenuation	**(n=11/23) (47.83%)**
HVS Thrombosis	**(n=7/23) (30.43%)**
HVs Uniphasic flow	**(n=17/23) (73.91%)**
HVs Dilatation	**(n=1/23) (4.35%)**
Intrahepatic Collaterals	**(n=21/23) (91.3%)**

PV = Portal Vein IVC = Inferior Vena Cava HVs = Hepatic Veins

Table (2) Subtypes of Intrahepatic Collaterals

Intrahepatic Collateral Total (n=23)	*Subcaspular Alone*	*Porto caval Alone*	*All types together (in same patient)*
21 (91.3%)	**7 (30.4%)**	**2 (8.69%)**	**12 (52.17%)**

Table (3) Comparison between Enhanced (Post-IV Contrast) CT & Sonographic Findings:

	US (B-Mode & Doppler) (n=23)	CT (n=14)	P-Value
Caudate lobe hypertrophy	(n=20/23) (86.96%)	(n=12/14) (85.7%)	**0.6384 (N.S)**
Intrahepatic collaterals	(n=21/23) (91.30%)	(n=12/14) (85.7%)	**0.4905 (N.S)**
Heterogeneous parenchyma	**(n=18/23) (78.26%)**	**(n=14/14) (100%)**	**0.0772 (N.S)**

**by Fisher's Exact Test **N.S. = Not Significant since P>0.05*

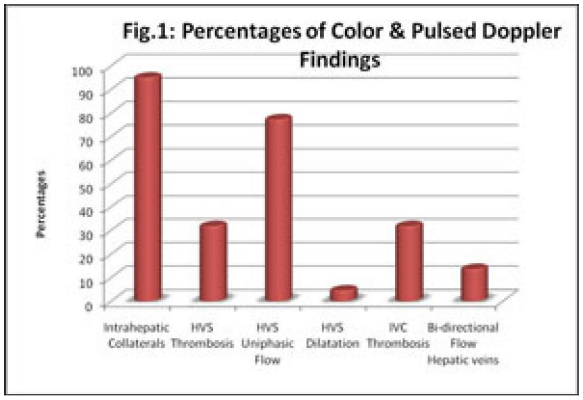

Fig.1: Percentage of Color and pulsed Doppler findings in primary BCS

4. Discussion:

As Budd–Chiari syndrome comprises a group of disorders characterized by hepatic outflow obstruction, it is evident that imaging techniques directly proving the obstruction are the basis for diagnosis. In practice BCS is regarded primary when no causes of secondary obstruction (tumor, abscess, cysts) are found. Recent advances in imaging modalities have enabled early diagnosis of asymptomatic cases and early surgical or interventional (TIPSS) treatment. In most cases, diagnosis of BCS can be made solely on the basis of imaging without the need for liver biopsy[18] Ultrasound (& CDS) now play a major role in diagnosis and follow-up of BCS. Availability and noninvasiveness allows it to play larger role as compared to angiography which was the "gold standard". [9] Doppler

US of the liver has the sensitivity and specificity of approximately 85-90% for the diagnosis of BCS. It is considered the initial imaging technique of choice in a patient suspected to have BCS. [6- 8, 26] Additional valuable benefit of sonography and CDS is easy to use in follow up and post-treatment repeated assement of those patients.

In the BCS, CDS can clearly assess the hepatic veins and IVC; detect their thrombotic occlusion (or stenosis). It can also clearly identify intrahepatic collaterals and characterize their veno-venous nature in contrast to these seen in other etiologies. In addition to CDS, gray-scale ultrasound can exclude clearly the presence of hepatic malignant neoplasm. This clarity for exclusion is magnified by the fact that, the hepatic neoplasm that can cause invasion of the hepatic venous are large enough or is an invasive one which is not easily missed on Gray-scale US. It is worth mentioning that CDS is superior to other modalities (cross sectional ones) in detection of the direction and pattern of flow in collateral, hepatic veins & IVC. This adds to the value of CDS in diagnosing BCS as CT and US (in our study) has approximate equal sensitivity in detection of intrahepatic collaterals (CT=85.7%, US & CDS =91.3%) in addition to other nonspecific findings like caudate lobe hypertrophy and coarse or heterogeneous parenchyma. (Table 3) In all these three criteria the difference between detecibility on CT or US was insignificant as *P* was > 0.05.

When diagnosing BCS (by US) there are different categories of signs. Specific signs such as evidence of hepatic vein involvement (e.g. non-visualization, fibrous cord, thrombosis and stenosis. [9] Non visualization (alone) of the hepatic vein does not clearly indicate its thrombosis, while prevalence or detection of thrombosis and stenosis is operator dependent and can be missed easily in early stage of thrombosis. In our study, IVC or H.V. thrombosis were clearly identified in 30.34% of cases (7 out of 23 patients). IVC web as an etiology is uncommon and in our study, it was seen in two cases (8.69%).

Although changes in hepatic waveform morphology such as Bi-directional Flow (73.91% in our cases) and Uniphasic flow (13.04% in our cases) (Fig.1 and Table 1) are important findings for CDS in BCS, they are of limited value in the diagnosis unless they are associated with intrahepatic collaterals vessels(Fig. 2) because a similar Doppler pattern can be observed in patients with fatty liver, active chronic hepatitis or liver disease.[16] A distinctive feature in primary BCS is the association with intrahepatic or subcapsular hepatic venous collaterals. This collateral circulation is the most sensitive feature for the diagnosis, being found in over 80% of cases. [2, 20, 21] Intrahepatic collaterals are of two types: (1) Intrahepatic veno-venous collaterals: the newly formed vessels can be classified into spider web collaterals (Fig. 4), large veno-venous collaterals draining to the IVC and subcapsular veins (Fig. 3) and hepatic vein to hepatic vein shunting, and (2) Intrahepatic portocaval collaterals: (Spontaneous direct portocaval shunts) they imitate in a natural way the available treatment option (Surgical Porto-Systemic shunts, TIPS). [13] Several patients of BCS can have more than one type of collaterals. [15] Aydinilli, *et. al* [22] demonstrated subcapsular and Intrahepatic collaterals in primary BCS in 80% of primary BCS patients. In our study, intrahepatic veno-venous collaterals were demonstrated by CDS in 21 out of 23 patients (91.3%) of primary BCS. (Table 1) This was the highest prevalence of diagnostic signs of primary BCS. Out of the sub types, subcapsular collateral alone were seen in 7 patients (30.4%), portocaval collaterals were seen alone in 2 patients (8.69%) and both are seen combined in 12 patients (52.17%). (Table2) In other words, subcapsular collaterals are more prevalence (19 out of 23 patients), where combination of both types of Intrahepatic were seen in 12 patients (overall presence of veno-venous collaterals was 21 out of 23 patients (91.3%). This highlights the diagnostic significance of intrahepatic veno-venous collateral in general and that of subcapsular collaterals in particular, in diagnosing primary BCS.

The second category is suggestive signs such as evidence of intrahepatic circulation and caudate vein dilatation. The third category is other findings shared with other conditions such as benign regenerative nodules, caudate lobe hypertrophy, nonhomogenous parenchymal structure, portal vein thrombosis, recanalized umbilical vein and ascites.[9] Nonvisualized or tortuous hepatic veins are common but nonspecific sonographic findings of BCS, while intrahepatic or subcapsular venous collaterals are sensitive sonographic findings. [18, 23- 25]

In our cases the overall presence of intrahepatic veno-venous collaterals in primary BCS on CDs was (91.3%) as compared to other diagnostic finding such as hepatic vein thrombosis, IVC, thrombosis (30.43% of each), bi-directional flow and bi-colored HVs (13.04%) (Fig.1).

Intrahepatic collaterals are not only seen in BCS, but they are also noted with hepatic tumors, Osler-Weber-Redu disease, veno-occlusive disease and chronic liver congestion.

Naganuma et al., [14] stated that the presence of tumor should be considered when localized venous collaterals are visible on color Doppler sonography. All the cases of secondary BCS are excluded from this study as in this situation has clear role in assessment of the etiology of IVC thrombosis.

In the entity of veno-occlusive disease[16] hepatic veins are usually patent because the obstruction occurs at the level of Centrilobular hepatic vessels or sinusoids, so the demonstration of normal non-occluded hepatic veins (and IVC) favors the diagnosis of veno occlusive disease & excludes Budd-Chiari.[26]

In patients with BCS, evaluation of the portal vein is essential because thrombosis precludes decompression of the liver via a Porto-systemic shunt with poor prognosis and reported in 10-20% of patients. [2, 9, 27] In our patients portal vein thrombosis was detected in 26.09% (n=6/23) (Table 1).

Additionally US/CDS can be used easily and repeatedly in post-treatment assessment and follow up.

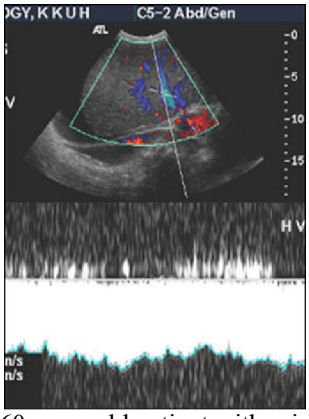

Fig. 2: Male 60 years old patient with primary BCS. Note mono-phasic flat flow in middle hepatic vein multiple intrahepatic collaterals.

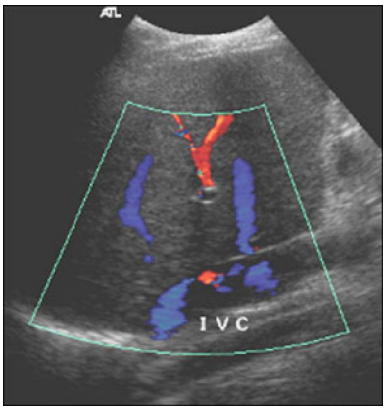

Fig.3: Color Doppler in 25 years old male patient with Primary BCS due with partially thrombosed IVC. Incomplete color filling of the IVC lumen with intrahepatic collateral vessels.

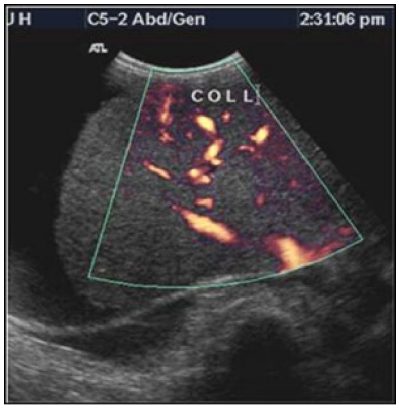

Fig 4: (A) CDS and Power Doppler (B) in 60 years old male with primary BCS: power Doppler showed multiple intrahepatic and subcapsular collaterals. Note also right sided pleural effusion and ascites (left image).

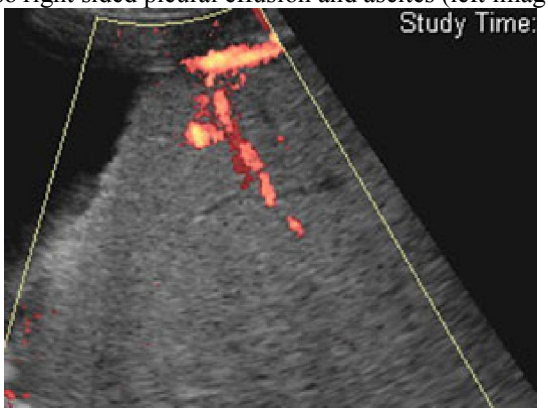

Fig 4: (B) same patient of Fig.4(A) with subcapsular intrahepatic collateral vessel.

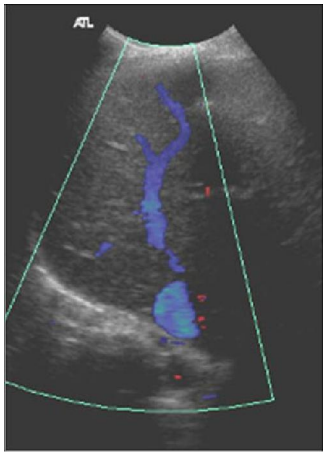

Fig.5: Color and power Doppler in SLE female patient with primary BCS: veno-venous "Hockey-stick" intrahepatic collateral.

Conclusion:

Doppler Ultrasound is a non-invasive modality that have high sensitivity rate in detection of diagnostic signs for primary BCS. US and CDS plays a major role in diagnosis of primary BCS by direct

visualization & characterization of veno-venous intrahepatic and subcapsular collaterals, which has significant role in its diagnosis as it has the highest rate of prevalence and hence more specific than other signs. Among the diagnostic signs presence of intrahepatic collaterals particularly subcapsular veno-venous collaterals had the highest rate and diagnostic significance on CDS.

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