

Research Article

The Role of Color Doppler US and triphasic MDCT with spleno-portography in differentiating benign from malignant portal vein thrombosis

Adel M.S. Mohsen, Nasr M.M. Osman, Ahmed S.I. Issa

Department of Radiology, Faculty of Medicine, Minia University.

Abstract

Objective: The aim of the work is to differentiate bland benign from malignant portal vein thrombosis. **Subjects and methods:** This study was carried out on 50 patients. All patients were subjected to color Doppler examination, MDCT and MRI diffusion. **Results:** Patients of this study were divided into 2 groups based on clinical suspicion, and images confirmation. Benign group include 17 patients and malignant group 33 patients. 24 patients (72.7%) of the malignant group have intra thrombus pulsatile flow by color Doppler. MDCT showed only one case of the first group showed focal lesion, all the second group patients had HFLs, 17 patients (51.5%) of the malignant group have PV neovascularization, 28 patients (84.8%) were found to have direct tumor invasion to the PV and 21 patients (63.6%) were found to have thrombus enhancement. The PV diameter with found to be of average 16.1 mm +/- 5.1mm in the benign cases, in comparison to 23.3+/- 8.1 in malignant group. 17 cases of the benign cases examined by DWI, 13 cases (76.5%) were found to have no focal lesions, 4 cases (23.5%) were found to have HFLs, by ADC mapping, 3 of them are confirmed to be malignant. **Conclusion:** Color Doppler study of patient with PVT should be an essential step in evaluation of patients with cirrhosis and HCC. The specificity of intra-thrombi pulsatile flow for the diagnosis of malignant PVT approaches 100%. MDCT showed sensitivity higher than color Doppler. DWI, MRI accurately detected small hepatic focal lesion.

Keywords: Color Doppler, triphasic, examination, hepatic focal lesion

Introduction

Portal vein thrombosis (PVT) is usually a complication of preexisting cirrhosis, abdominal malignancy (e.g., pancreatic or hepatocellular carcinoma), or abdominal inflammation (e.g., appendicitis, diverticulitis, pancreatitis)⁽¹⁾. PVT can cause or exacerbate portal hypertension; variceal bleeding or hypersplenism may then develop acutely or several years later. It also complicates portosystemic shunt surgery or liver transplantation⁽²⁾

Portal vein thrombosis (PVT) is increasingly recognized in cirrhotic patients, especially in those candidates for transplantation. Restoring both portal and arterial blood flow to the allograft is a necessary condition for liver transplantation to be successful. Restoration of the portal flow through the allograft rapidly reverses portal hypertension, which is a major source of

complications in cirrhosis⁽³⁾. The prevalence of PVT in cirrhotic patients at evaluation varies from 5% to 26%⁽⁴⁾.

Patients with cirrhosis and hepatocellular carcinoma (HCC) may have either malignant or benign (fibrin clot) portal vein thrombosis. Benign portal vein thrombosis was diagnosed by the application of the following criteria; lack of vascularization of the thrombus on contrast-enhanced ultrasound and on computed tomography or magnetic resonance imaging, absence of mass-forming features of the thrombus, absence of disruption of the walls of veins, and, if uncertainty persisted, biopsy of the thrombus for histological examination. Patients who did not fulfill the criteria for benign thrombosis were not placed on the transplantation list. Malignant portal vein thrombosis (PVT) is a well-recognized complication of hepatocellular carcinoma

(HCC); this is attributable to the frequency with which HCC invades the portal venous system. This complication occurs in approximately 35% of patients with HCC. In most cases of HCC, local or regional treatments are contraindicated and systemic chemotherapy is administered.⁽⁵⁾

Differentiating between benign and malignant thrombi in portal veins is difficult without histological examination of the thrombus.^(5,6) Benign PVT may also occur in patients with HCC, especially in those with early-stage HCC, who appear to be good candidates for LT. In such cases, if neoplastic involvement in the PVT cannot be excluded, a potentially lifesaving procedure will not be offered, and consequently, the prognosis will be poor. In this context, differentiation between benign and malignant thrombi may be achieved by ultrasound (US)-guided fine-needle biopsy of the lesion however, this procedure is invasive^(7,8).

Color Doppler imaging is often the initial technique of choice for the noninvasive assessment of abnormalities of the portal veins. In recent years, the possibility of using color Doppler sonography, contrast-enhanced color Doppler sonography, computed tomography (CT) and magnetic resonance imaging (MRI) to determine the benign or malignant nature of PVT has been reported^(9,10). The advent of contrast-enhanced ultrasound (CEUS) using second-generation US contrast agents has led to greater accuracy in the detection and characterization of PVT complicating HCC than that achieved by CT^(11,12,13,14,15). There are several advantages in evaluating PVT.

First, CT completely eliminates the superimposition of images of structures outside the area of interest. Second, because of the inherent high-contrast resolution of CT, differences between tissues that differ in physical density by less than 1% can be distinguished⁽¹⁶⁾. Phase contrast MRI sequences can also be used to evaluate the portal vein and phase contrast cine MR angiography can show the direction of

portal venous flow and the presence of portal vein thrombus. Magnetic resonance (DWI) evaluation of the portal venous system is accurate in demonstrating thrombosis and the collateral circulation. MRI is helpful if the hepatic parenchymal detail is required (in hepatic malignancies) and unlike CT scan, MRI can also quantitate portal and hepatic vessel flow, which is required in the planning of interventions such as shunt surgery, trans jugular intrahepatic Porto -systemic shunt (TIPS), or liver transplantation⁽¹⁷⁾.

Aim of the work

The aim of the work is to assess the role of color Doppler, MDCT and MRI diffusion in differentiate benign from malignant portal vein thrombosis.

Patients and methods

This study was carried out on 50 patients presented to the Radiology department EL Minia University hospital from Tropical and Internal medicine departments from March 2014 to February 2015.

All patients in this study were subjected to the following:

Clinical examination: including general and abdominal examination.

Laboratory investigations including: Alpha-fetoprotein and hepatitis markers.

Radiological examination including:

Real time ultrasound was performed using GE (logic P5 or logic 5) ultrasound machines with 3.5 MHz transducer for examination of the liver pattern, diseased or not presence or absence of focal lesion.

Color Doppler ultrasound study of the portal vein:

color Doppler examination were performed on GE Logic P5 (GE Healthcare, Milwaukee, WI) Ultrasound machine using 3.5 MH Matched imaging and Doppler frequency transducer. The protocol consisted of obtaining grey scale images of the portal vein thrombus and color Duplex. Portal vein thrombi in the left portal vein were evaluated using an anterior sub-xiphoid approach; portal vein thrombi in the right or main portal vein were

evaluated between the right and mid-clavicular line and the right posterior axillary line using an inter-costal or subcostal approach. All portal vein thrombi were first evaluated by color Doppler sonography to locate flow within the thrombi and then by Duplex Doppler sonography to characterize any detected flow. The flow was categorized by spectral configuration as pulsatile or continuous. Pulsatile flow was called subjectively when a waveform had a marked difference between peak systolic and end diastolic velocities. Waveforms with mild sinusoidal variation in velocity such as that seen commonly in the portal vein interpreted as continuous obvious.

Triphasic CT examination of the liver with spleno-portography

Triphasic CT abdomen and pelvis was performed using MDCT scanner 16 detectors CT scanner (GE Healthcare, Waukesha, WI).

Technique:

One hour before scanning, patients received oral contrast medium (for GIT opacification.) Via a power injector, 150ml of Omnipaque 300 mg I/ml (iohexol 300 mg I/ml; Nycomed, Princeton, NJ) was injected at a rate of 4 mL/sec. Arterial phase scanning was performed during a single breath-hold using 35-sec scanning delays. The entire liver was scanned in a cephalic-to-caudal direction using a Detector collimation of 5 mm with a table speed per rotation of 15 mm/0.8 sec, pitch of 3 in the scanner's HQ mode and image thickness 5mm. After a brief period of quiet breathing, portal venous phase imaging of the entire abdomen was performed in a cephalic-to-caudal direction using scanning delay of 60 sec and an image thickness of 5 mm. Spleno-portography is done by coronal reconstruction in portal venous phase.

MDCT finding include; Liver parenchyma, presence or absence of focal lesions and pattern of enhancement, portal vein thrombus and its extension.

Assessment of portal vein thrombus: Filling defect in portal venous phase, rim enhancement, flow through dilated vasa vasorum, increased flow around periphery of clot and calcifications in chronic thrombus.

Criteria of malignant PVT includes: Intra thrombus neo-vascularity, arterial enhancement in arterial phase, thread and streak sign (multiple enhancing intraluminal and smaller vessels that can be seen at arterial phase) and direct invasion of hepatic tumor to the PV. Diameter of the thrombus: ≥ 23 mm likely to be malignant thrombus, near the tumor with no obvious extension (contiguous PVT), likely to be malignant, far from the tumor (remote PVT), likely to be benign.

MRI diffusion:

DW-MRI was performed using 1.5-T MRI scanner (Philips Achiva) equipped with phased-array torso surface coil. The examination included axial SPAIR as well as DW sequences as different b-values (0, 500 & 1000s/mm²), followed by computer-generated ADC mapping of the liver. The use of diffusion weighted single-shot echo-planar (DW SS-EP) sequences with a low b-value is important for the detection of hepatic focal lesions, especially for small-sized lesions. The acquisition of "black blood" images with DW SS-EP sequences makes it possible to easily differentiate vessels from focal small-sized lesions in the liver. In this study MRI diffusion was done to confirm the diagnosis of malignant PVT.

Results

This study included 50 patients with PV thrombosis were divided into 2 groups based on clinical suspicion, confirmed by images (ultrasound and color Doppler examination, MDCT evaluation and MRI-Diffusion.

Demographic data: The patient age ranged from 45-60 years old. Mean age in the first group 51.7+/- 9.3, and second group 52.8+/- 5.5 with no specific value relating the age to

the pathology. Out of 17 patients included in the first group, 9 were males (52.9%) and 8 females (47.1%) of the 33 patients in the second group patients, 20 patients were males (60.6%) and 13 females (39.4%).

Two cases in this study were had healthy hepatic parenchyma (non-cirrhotic) and the remaining cases were cirrhotic.

Grey scale and Doppler:

examination showed that 100% of the second group found to have hepatic focal lesions. The thrombus involvement was found to be variable with no significant value. Twenty four patients (72.7 %) of the suspected malignant patients were found to have intra thrombus pulsatile flow, with no intra thrombus vascularity detected in benign group.

15 cases (45.5%) of the malignant group were found to have solitary hepatic focal lesion, 18 cases (54.5%) found to have multiple hepatic focal lesions, most of the hepatic focal lesions found at the right hepatic lobe (22 cases, 66.7%) and the hepatic focal lesions were found to have heterogeneous echopattern.

MDCT finding

All patients underwent further confirmation using contrast enhanced multi-slice triphasic (MDCT), one case of the first group was found to have solitary focal lesion, on the other hand all the second group patients HFLs, varying from solitary (39.4%), Diffuse infiltrative (12.1%) or multi-centeric (48.5%), with the CT showing significant P-value in differentiation.

Despite the perfusion defect that may be found in patients with PVT, the MDCT was found to have a significant P-value in detection and differentiation of HFLs in correlation with the ultrasound. Only one case of the first group was having HFL, which attains globular enhancement at the arterial phase and following the venous pattern at the delayed phases (matching CT criteria of benign hemangioma).

17 patients (51.5%) of the malignant cases were found to have PV neovascularization in CT in comparison to benign group at which this finding is not detected with very high significance P-value. 28 patients (84.8%) were found to have direct tumor invasion to the PV in CT in relation to no patients in the benign group with very high significance P-value. 21 patients (63.6%) were found to have thrombus enhancement at the arterial phase with washout at the delayed and porto venous phases, in CT in relation to no patients in the benign group with very high significance P-value. The PV diameter with found to be of average 16.1 mm +/-5.1 mm in the benign cases, in comparison to 23.3+/- 8.1 in the malignant group with very high significance relating the PV diameter to nature of the thrombus.

In this study we found that the MDCT assessment has a significant P-value in diagnosis of malignant PVT, with a sensitivity of 91.7%, 100% specificity and 94% accuracy.

AFP correlation:

Correlation of color Doppler and CT findings to AFP levels, with cut off point for differentiating benign from malignant cases, 500 mg/dl. The first group patient only 14 patients were found to have low levels (less than 500 mg/dl), on the other hand out of the second group 36 cases were found to have high AFP level (more than 500 mg/dl), So patients in the first group need further evaluated by MRI, DWI for detection of any missed or small undetermined HFLs.

Out of the 17 benign group patients, 3 cases have further deterioration and extension of the portal thrombus in the follow up with very high levels of AFP, so MRI, DWI for more confirmation, examined those cases:

MRI DWI findings: Of the benign cases examined by DWI, 13 cases (76.5%) were found to have no focal lesions, 4 cases (23.5%) were found to have HFLs and ADC mapping confirms 3 cases confirmed to be malignant.

Table 1: Age and sex distribution of patients included in this study:

Doppler	Group I (suspected benign) N=17	Group II (suspected malignant) N=33	P value
Thrombus involvement:			
Main branch	17 (100%)	27 (81.8%)	0.061
RT branch	3 (17.6%)	17 (51.5%)	0.021*
LT branch	1 (5.9%)	4 (21.1%)	0.486
Thrombus flow pattern:			
Pulsatile.	0 (0%)	24 (72.7%)	
Non-pulsatile.	17 (100%)	9 (27.3%)	< 0.001*

Table 2: Extent of PV involvement by the ultrasound and color Doppler findings:

Characters of the thrombus	Group I (Suspected benign) N=17	Group II (suspected malignant) N=33	P value
Neovascularization	0 (0%)	17 (51.5%)	0.001*
Direct invasion	0 (0%)	28 (84.8%)	< 0.001*
Arterial enhancement	0 (0%)	21 (63.6%)	< 0.001*
Portal vein:			
Not Dilated.	5 (29.4%)	2 (6.1%)	0.024*
Dilated.	12 (70.6%)	31 (93.9%)	
PV diameter:			
Mean ± SD	16.1 ± 5.1	23.3 ± 8.1	< 0.001*

Table 3: Radiological characters of portal vein thrombus and diameter by MDCT

Diffusion MRI for benign cases:	Group I (suspected benign) N=17
HFL:	
No.	13 (76.5%)
Yes.	4 (23.5%)
ADC value:	
<1:	3 (75%)
>1:	1 (25%)

Table 4: characterization of suspicious cases by MRI, DWI.

Demographic data	Group I (suspected benign) N=17	Group II (suspected malignant) N=33	P value
Age: Mean ± SD	51.7 ± 9.3	52.8 ± 5.5	0.647
Sex:			
Male.	9 (52.9%)	20 (60.6%)	0.603
Female.	8 (47.1%)	13 (39.4%)	

Abbreviation

PVT; portal vein thrombosis

MDCT; multi-detector computed tomography

LT; liver transplantation

LC; liver cirrhosis

HCC; hepatocellular carcinoma

HFL; hepatic focal lesion, HFLs; hepatic focal lesions

Figure 1

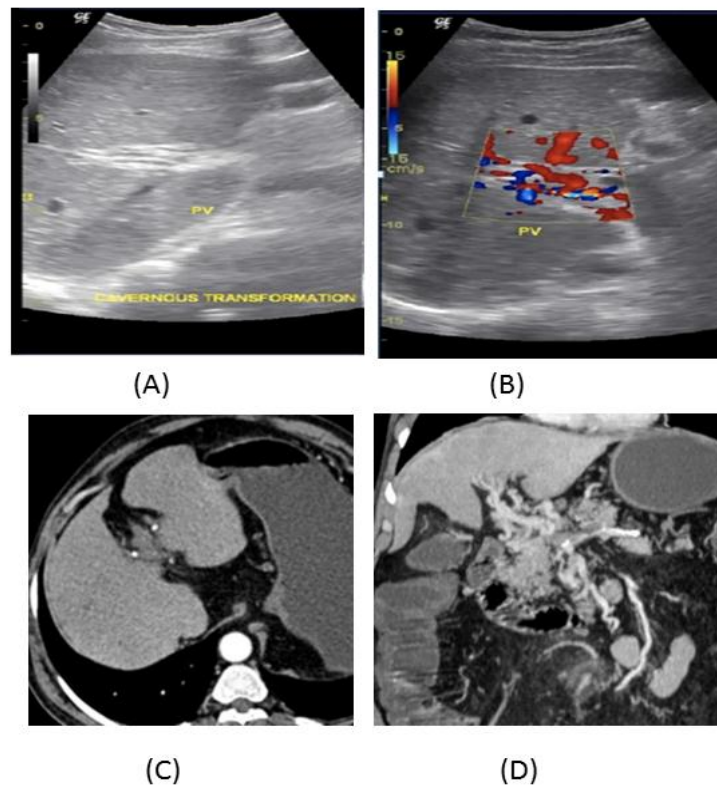


Figure 1: Male patient 38y old presented by attack of hematemesis. (A), B-mode and (B) color Doppler of the portal vein shows dilated vascular channels (cavernous transformation).(C) Axial arterial phase & (D) coronal portal venous-phase shows no enhancing of the portal vein thrombus with cavernous transformation

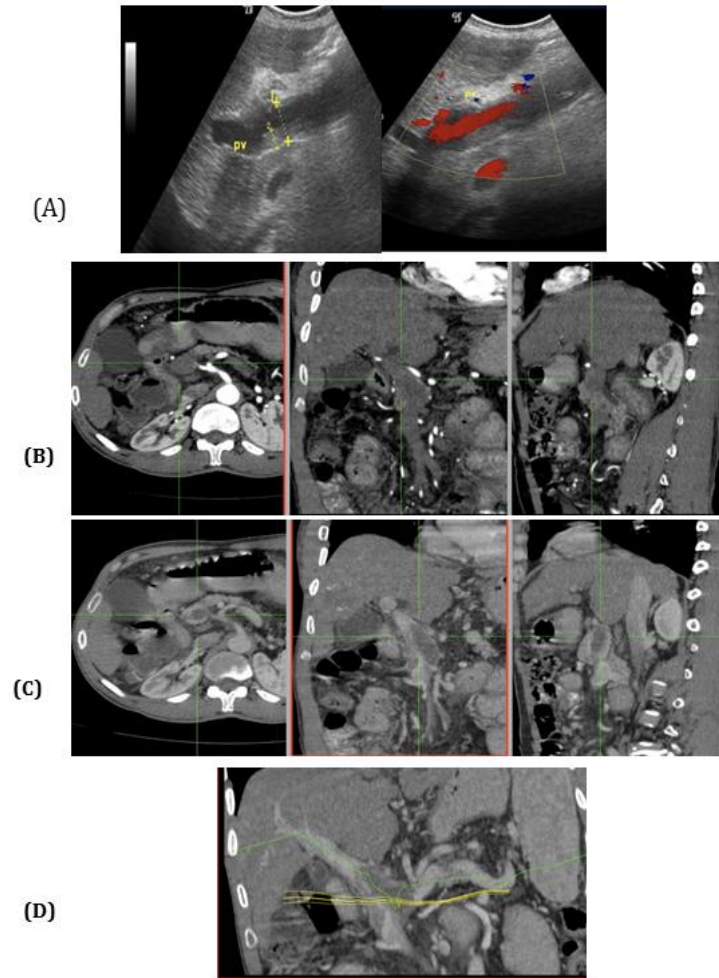


Figure 2: Female patient 65y presented by attack of hematemesis. A B-mode & color Doppler of the portal vein shows dilated main portal vein with posterior mural thrombus adherent to the PV wall, with no anterior eccentric flow detected on application of color Doppler, no intra-thrombus vascularity. (B) Axial, coronal and sagittal reformatted images in the arterial phase shows mural thrombus within the main PV, with no color detected on the arterial phase, no enhanced hepatic focal lesions. (C) Axial, coronal, sagittal images in the porto-venous phase show mural thrombus within the main PV. (Filling defect) (D) Splenoportography shows filling defect within the main PV.

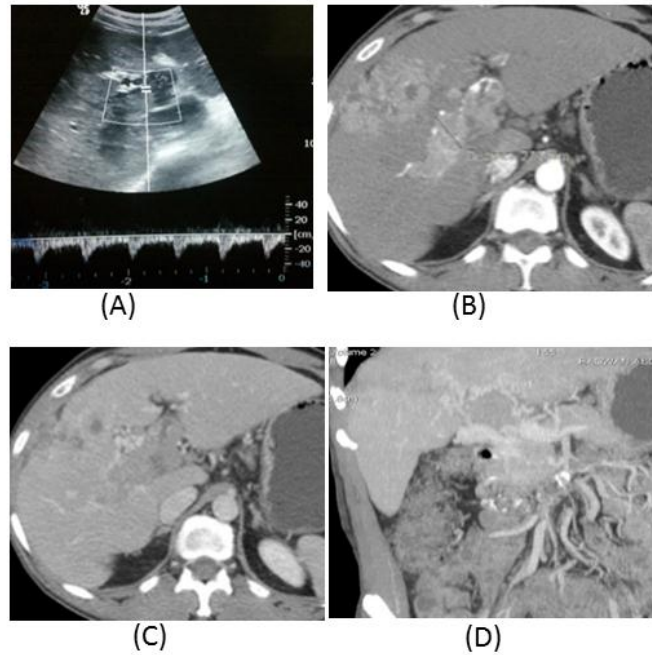


Figure 3: Male patient 55 y presented by right hypochondrial with attacks of hematemesis and hepatic encephalopathy. Image A: color Doppler assessment of the portal vein showing thrombosed right main portal vein branch with mono-phasic flow detected within the thrombus. Image B: axial CT image, arterial phase, showing heterogeneously enhanced hepatic focal lesion at the right hepatic lobe with enhanced right main portal thrombus. Image C: Axial Porto-venous phase showing washout of contrast from the right hepatic lobe hepatic focal lesion, associated with thrombus within the right main portal branch, which appears dilated. Image D: spleno-portography showing thrombosed right main portal branch.

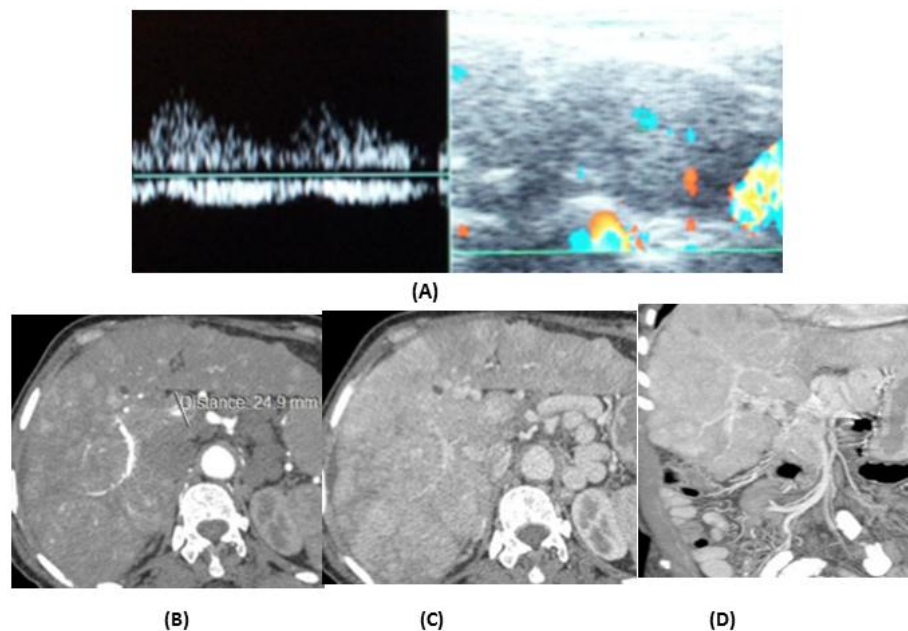


Figure 4: Male patient 51y old presented by right hypochondrial pain, jaundice and attacks of hematemesis. A) Color Doppler and spectral wave analysis of the main trunk showing neovascularity with arterial waveform pattern. Image B) arterial phase axial CT section showing thrombosis of the main trunk of portal vein(at porta hepatis) with thread and streak sign(arrowed) –neovascularity- that disappeared in portal phase(image C), diameter of the thrombus measures about 25mm, denoting malignant features. D) portography showing total thrombosis of the main trunk (arrowed).

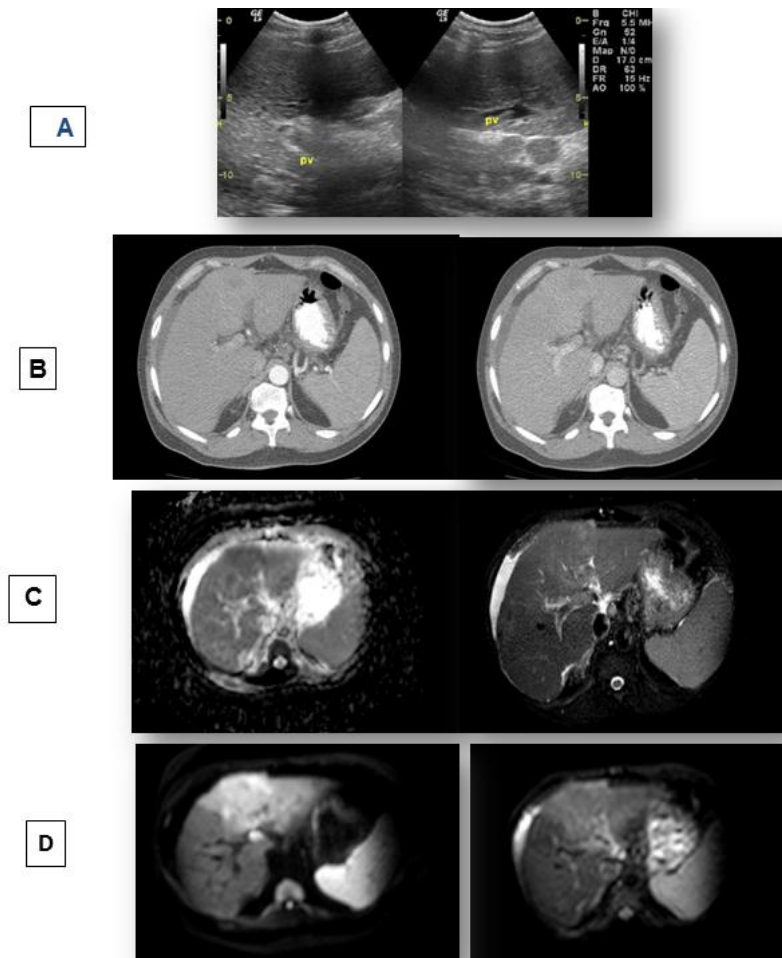


Figure 5: Male patient 60 y presented by jaundice and upper abdominal pain (A) Grey scale showed soft tissue thrombus that is seen at the main portal vein and extending to the left main portal vein. (B) MDCT examination at the arterial and Porto-venous phases shows no definite enhanced hepatic focal lesions with thrombosed left main portal vein at the Porto-venous phase. (C) MRI, T2WI showed PV thrombus with no definite focal lesion and (D) MRI DWI showed hepatic focal lesion (at the area of restricted diffusion) at the left hepatic lobe.

Discussion

Portal vein thrombosis indicates thrombosis that develops in the lumen of the PV trunk and including its right and left intrahepatic branches⁽¹⁸⁾. A neoplastic thrombus of the portal vein is found in 6.5%-44% of patients with hepatocellular carcinoma (HCC). Presence of neoplastic thrombus serves as an important determinant of tumor staging, prognosis, and influences treatment selection. PVT increasingly diagnosed at the last years by the widespread use of ultrasound Doppler. PVT is increasingly recognized in cirrhotic patients, especially in candidates for transplantation. The prevalence of PVT in cirrhotic patients at

evaluation or at time of transplantation varies from 5%-26%^(19, 20, 21, 22).

In the current study liver cirrhosis was the most common cause of benign PVT; it was identified in 85.8% of patients with benign PVT included in this study (12 out of 14 patients). The other 2 patients were found to have other causes of hypercoagulability (one is diagnosed to have Behcet disease and the other have mylo-proliferative disorder).

The diagnosis of PVT has become easier than before because of the availability of real time sonography. However, the grey scale sonographic appearance of the thro-

mbus is not specific and cannot distinguish benign from malignant thrombi⁽²³⁾. The use of Doppler sonography in the diagnosis of malignant portal vein thrombosis has been evaluated by several studies as non-visualization of portal vein flow, pulsatile flow in the thrombus⁽²⁴⁾.

In this study, 24 patients from the malignant group (33 patients) had pulsatile flow within the thrombus; 9 patients had no detectable flow within the thrombus. On the other hand, no pulsatile blood flow could be detected in all benign thrombi, with a significant P value in differentiating benign from malignant thrombi. This matches a study done by Pozniak and Baus⁽²⁴⁾ who were the first to detect pulsatile arterial flow by color Doppler sonography within tumor related portal vein thrombi in patients with hepatocellular carcinoma. These authors had examined 5 patients with PVT; they found 60% having malignant PVT and 40% having the benign one. Pulsatile flow was detected in 66.6% of malignant thrombi, thus allowing the diagnosis of intravascular spread of the tumor. No detectable flow was found in 33.3% of malignant thrombi.

Tanaka et al.,⁽²⁵⁾ studied 18 patients with PVT related to HCC were studied, 9 of them had thread and streak sign by conventional angiography and are proved pathologically to be neoplastic, 8 of these 9 patients had pulsatile flow within the thrombi by color Doppler sonography and it was detected in none of the other 9 benign thrombi with sensitivity 89% and specificity was 100% and accuracy of 96%.

Dodd, et al.,⁽²⁶⁾ studied 47 patients with PVT, 26 of them were proved to be malignant by histopathology and CT findings and 21 proved to be benign. Pulsatility within the thrombus was detected in 16 patients of the malignant group and in 3 patients of the benign group, in those three patients; the flow in the portal vein thrombi was identical to the portal venous flow present in the non thrombosed segments of the portal vein. The pulsatile

flow detected in benign portal vein thrombi represented portal venous flow coursing through the inter slices of the thrombi and attributed to arterial portal venous shunting which is a common phenomenon in liver cirrhosis, the sensitivity was 62 % and the specificity was 95%.

Mohammed⁽²⁷⁾, studied 32 patients with PVT, 15 patients were proven to be malignant by FNAC. 13 patients of 15 showed color Doppler pulsatile flow, one patient (from 15) had continuous flow and the remaining one (from 15) showed no flow. The other 17 benign thrombi had no detectable pulsatile flow.

Rossi,⁽²⁸⁾ studied 83 patients with PVT, 81 patients were proved to be malignant- on imaging and pathological basis- and 2 to be benign. Pulsatility within the thrombus was detected in 51 patients of the malignant group and none of the 2 patients of the benign group, thus the sensitivity of color Doppler for detection of pulsatility in malignant thrombi was 63% with 100% specificity.

In the present study, pulsatile flow was the only sign detected during color Doppler study of neoplastic thrombi. It has been found in 72.7% of the malignant thrombi included in this study. Using the presence of intra thrombus pulsatile flow as a diagnostic criterion, we found that the sensitivity and specificity reaches 66.7% and 100% respectively, with an accuracy approaching 76%.

Consequently, if a pulsatile flow detected in portal vein thrombus and the character of the flow is clearly different from those of the hepatic artery and a patent segment of the portal vein, then the specificity of the intra thrombus pulsatile flow for the diagnosis of malignant portal vein thrombus approaches 100%.

Dynamic contrast enhanced CT is one of the best means of diagnosis of PV thrombus and evaluation of various causative diseases⁽²⁹⁾.

In the present study, 36 patients had malignant and 14 patients had benign PV thrombus. CT scans of patients with malignant PVT showed direct extension of hepatocellular carcinoma into the vein in 28 patients (84.8%). The mean diameters of malignant and benign portal vein thrombi were significantly different (23.3 mm versus 16.1 mm, respectively). CT scans of 21% (63.6%) of patients with malignant PVT and 0% (0/14) of patients with benign PVT showed enhancement in arterial phase. Neovascularity was seen on CT scans in 51.5% (17 /33) of patients with malignant PVT and in none of patients with benign PVT. 93.9% of patients with malignant PVT showed thrombus diameter ≥ 23 mm, while all the patients with benign thrombosis showed diameter < 23 mm.

In the study of Tublin⁽³⁰⁾, 47 patients had malignant and 11 patients had benign PVT. CT scans of patients with malignant PVT showed direct extension of hepatocellular carcinoma into the portal vein in 15 patients. In 29 patients with malignant PVT, CT scans showed PVT adjacent to tumor; CT scans showed tumor PVT remote from hepatocellular carcinoma in the remaining three patients with malignant PVT. The mean diameters of malignant and benign portal vein thrombi were significantly different (23.4 mm versus 16 mm). CT scans of 83% (39/47) of patients with malignant PVT and 18% (2/11) of patients with benign PVT showed generalized enhancement. Neovascularity was seen on CT scans in 82% (9/11) of patients with malignant PVT, in 31% (11/36) of patients with malignant PVT (but with conventional CT) and in no patient with benign PVT. Identification of a main PVT diameter greater than or equal to 23 mm resulted in a sensitivity and specificity for the CT characterization of malignant PVT of 86% and 100%, respectively.

In the study of Piscaglia⁽³¹⁾, the study was done on 33 patients who had HCC with portal vein thrombosis and came for liver transplantation, 9 patients of them were not placed on the transplantation list as 2 of them were diagnosed to have malignant

PVT simultaneously with HCC, while the other 7 were removed from transplantation list as they developed malignant PVT after diagnosis of HCC. The criteria that were considered by the authors and on their basis malignant PVT is diagnosed were Intra thrombus vascularity.

In the current study, the CT findings was found to have a sensitivity of (91.2%) and 100 % specificity, this near to Tublin et al.,⁽³⁰⁾ and it is different from that reported by Lee et al.,⁽³²⁾. Rossi et al.,⁽³⁰⁾ reported 67.6% sensitivity and 60% specificity of dynamic CT to detect malignant thrombi. The diameter of the portal vein thrombus (equal to or exceeding 23 mm) as a criterion for diagnosing malignant PVT showed 63.9% sensitivity, 57.1% specificity and 62% accuracy, this was near to that reported by Rossi et al.,⁽²⁸⁾ who showed 67.6% and 60% sensitivity, respectively.

Consequently, if a neo vascularity, thrombus enhancement in arterial phase, thrombus diameter ≥ 23 mm or direct invasion of the thrombus by tumor was detected (alone or combined) by CT, then the specificity for the diagnosis of malignant portal vein thrombus approaches 100%. MDCT and Doppler in comparison to MRI diffusion were found to have a sensitivity of 91.7% and (94%) accuracy.

A portal vein thrombus was defined as a filling defect in contrast enhanced images. According to the criteria described by Shah et al.,⁽³³⁾, a thrombus was considered neoplastic if either of the following criteria was met and bland if neither of the criteria was met: (a) expansion of the involved vessel (vessel diameter ≥ 1.8 cm for the main portal vein, ≥ 1.6 cm for the right portal vein, ≥ 1.8 cm for the left portal vein, and disproportionate enlargement when compared with non-affected same-order portal vein branches in the same lobe) or (b) clear evidence of enhancement on dynamic contrast enhanced MRI⁽³³⁾.

Summary and conclusion

Color Doppler study of the portal vein thrombus should be an essential step in the

evaluation of patients with HCC with PVT during planning for their management.

The specificity of intra-thrombi pulsatile flow for the diagnosis of malignant portal vein thrombosis approaches 100%. Multi-detector tri-phasic CT showed sensitivity higher than color Doppler in detecting malignant thrombi. MDCT have some falsies in detection of the nature of the thrombus and detection of small hepatic focal lesions, particularly in presence of perfusion defect that occurs in presence of PVT hence comes, the importance of DWI, MRI for detection of those small lesions.

References

1. Parvey H. R, Rava B and Sandier CM. Portal Vein Thrombosis: Imaging Findings. *AJR* 1994; 62:77. American Roentgen Ray Society.
2. Chawla Y, Duseja A and Dhiman R K: "the modern management of portal vein thrombosis". *Alimentary pharmacology & therapeutics*, 12 August, 2009.
3. Sarin SK, Sollano JD, Chawla YK: "Consensus on extra-hepatic portal vein obstruction". *Liver international*, 2006; 26: 512-9.
4. Ogren M, Bergqvist D, Björck M: "Portal vein thrombosis prevalence, patient characteristics and life time risk". *World journal of gastroenterology*, 2006; 12: 2115-9.
5. Fabio P, Alice G, Matteo R, Rita G, Alberta C, Emanuela G, Elisabetta S, Grazia I, Antonio D. Criteria for Diagnosing Benign Portal Vein Thrombosis in the Assessment of Patients with Cirrhosis and Hepatocellular Carcinoma for Liver Transplantation. *Liver transplantation* 2010;16:658-667.
6. Claire F, Dominique V, François D. Portal vein thrombosis, cirrhosis, and liver transplantation. *Journal of Hematology* 2012; 57: 203–212.
7. Bolondi L and the Bologna Liver Transplant Group. Toogood G: "Hepatobiliary system: liver in slandering S: "In Gray's anatomy, by Toogood G, 2008; 1213 – 1218". *El Sevier Churchill living stone*.
8. Gallego C, Velasco M, Marcuello P, Tejedor D, De Campo L, and Feriera A: "Congenital and acquired anomalies of portal venous system". *Radiographics*, 2002; 22:141-159.
9. Cosgrove D O, Arger P H, and Coleman B G: "Ultrasound anatomy of hepatic veins". *Journal of clinical ultrasound*, 1987; 15:231-235.
10. Von Herbay A, Frieling T, and Haussinger D: "Color Doppler sonographic evaluation of spontaneous portosystemic shunts and inversion of portal venous flow in patients with cirrhosis ". *Journal of clinical ultrasound*, 2000; 28: 332-339.
11. JT, Wu: "serum alfa-fetoprotein and its lactin reactivity in liver disease: a review. *Annual clinical lab science*, 1990; 20:98.
12. Jonathan M Schwartz and Robert L Carithers, Jr: "clinical features and diagnosis of primary hepatocellular carcinoma". Up to date, 2014; 1: 25.
13. Hidajat N, Stobbe H, Grieshaber V, Fleix R, and Schroder R-J: "Imaging and radiological intervention of portal vein thrombosis". *Acta radio*, 2005; 46: 336-343.
14. Mc-dicken W N, Hoskins PR: "physics: principles, practice and artifacts". In *Clinical Doppler ultrasound*. Churchill Livingstone, 2000;1-24.
15. O'Donohue J, Ng C, Catnacha S, Farrant P, and Williams R: "Diagnostic value of Doppler assessment of hepatic and portal vessels and ultrasound of the spleen in liver disease". *European journal of Gastroenterology & hepatology*, 2004; 16: 147-155.
16. Tamsel S, Demiropolat G, Killi R, Aydin U, Kilic M, Zeytunlu M, Parıldar M, Oran I, and Ucar H: "Vascular complications after liver transplantation, evaluation with Doppler ultrasound". *Abdominal imaging*, 2007; 32: 339-347.
17. Zirinsky K, Rubenstein WA, Cahill PT, Knowles RJ, Auh Yong Ho, Morrison H, Kazam E. MR Imaging of Portal Venous Thrombosis: Correlation with CT and Sonography *AJA*: February 1988;150.
18. Nonami T, Yokoyama I, Lwatsuki S, Starzl T. The incidence of portal vein thrombosis at liver transplantation. *Hepatology* 1992; 16:1195-1198.

19. Nakayama Y, Imuta M, Funama Y, Kadota M, Utsunomiya D, Shiraishi S, Hayashida Y, and Yamashita Y: "CT portography by multidetector helical CT: Comparison of three rendering models". *Radiant med*, 2002; 20:273-279.
20. Amitrano L, Guarduscione MA, Branccio V, Margagione M, Manguso F, Lannaccone L, et al. Risk factors and clinical presentation of portal vein thrombosis in patients with liver cirrhosis. *J hepatol* 2004; 40:736-741.
21. Garcia-pagan JC, Valla DC: "portal vein thrombosis: apredictable milestone in cirrhosis". *Journal of hepatology*, 2009.
22. Fimognari FL, and Violi F: "Portal vein thrombosis in liver cirrhosis". *Internal emergency medicine*, 2008; 3: 213-218
23. Prof. Alexander Julianov: "Ultrasound imaging of liver tumors-current applications", liver tumors. s.l.: In Tech, 03, feb. 2012.
24. Pozniak MA, Baus KM: " Hepatofugal arterial signal in the main portal vein: An indicator of intravascular tumor spread". *Radiology*, 1991; 180:663-666.
25. Tanaka K, Numata K, Okazaki H, Nakamura S, Inoue S, and Takamura Y: "Diagnosis of Portal Vein Thrombosis in Patients with Hepatocellular Carcinoma: Efficacy of Color Doppler Sonography Compared with Angiography". *AJR*, 1993; 160:1279-1283.
26. Dodd GD, Memel DS, Baron RL, Eichner L, Santiguida LA: "Portal Vein Thrombosis in Patients with Cirrhosis: Does Sonographic Detection of intra thrombus Flow Allow Differentiation of Benign and Malignant Thrombus ". *AJR*, 1995; 165:573-577.
27. Mohammed WM A: " Role of color Doppler and FNAB in differentiating between benign and malignant portal vein thrombosis, Thesis for master degree". Alexandria, 2005.
28. Rossi S, Ghittoni G, Ravetta V, Viera FT, Rosa L, Serassi M, Scabini M, Vercelli A, Tinelli C, Bello BD, Burns PN, and Calliada F: "Contrast-enhanced ultrasonography and spiral computed tomography in the detection and characterization of portal vein thrombosis complicating hepatocellular carcinoma". *Eur Radiol*, 2008; 18: 1749-1756.
29. Tarantino L, Francica G, Sordelli I, Esposito F, Giorgio A, Sorrentino P, De Stefano G, Di Sarno A, Ferraioli G, and Sperlongano P: "Diagnosis of benign and malignant portal vein thrombosis in cirrhotic patients with hepatocellular carcinoma: color Doppler US, contrast-enhanced US, and fine-needle biopsy". *Abdom Imaging*, 2006; 31:537-544.
30. Tublin ME, Dodd GD, and Baron RL: "Benign and Malignant Portal Vein Thrombosis: Differentiation by CT Characteristics". *AJR*, 1997; 168:719-723.
31. Piscaglia F, Gianstefani A, Ravaioli M, Golfieri R, Cappelli A, Giampalma E, Sagrini E, Imbriaco G, Pinna AD, Bolondi L, and the Bologna Liver Transplant Group: Criteria for Diagnosing Benign Portal Vein Thrombosis in the Assessment of Patients with Cirrhosis and Hepatocellular Carcinoma for Liver Transplantation". *Liver transplantation*, 2010; 16:658-667.
32. Lee H, Park S, Yi B, yeon E, Kim JH, and Hong H: "portal vein thrombosis: CT features". *Abdominal imaging*, 2008; 33: 72-79.
33. Shah ZK, McKernan MG, Hahn PF, Sahani DV. Enhancing and expansile portal vein thrombosis: value in the diagnosis of hepatocellular carcinoma in patients with multiple hepatic lesions. *AJR Am J Roentgenol* 2007; 188:1320-1323. CrossRef, Medline