Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.325392.3610 Volume 31, Issue 1, January. 2025
Manuscript ID:ZUMJ-2410-3610
DOI : 10.21608/zumj.2024.325392.3610
ORIGINAL ARTICLE

The Role of Shear Wave Elastography in Differentiation Between Tumoral and Bland Portal Vein Thrombosis

Mohamad Abowarda [1], Tamir Hassan [1], Nada Samir Abdelhalim Moustafa[1]*, Essam E Tharwat[1]
[1]Radiodiagnosis Department, Faculty of medicine, Zagazig University.

***Corresponding author:**
Nada Samir Abdelhalim Moustafa

Email:
Noooodyn93@gmail.com

Submit Date: 01-10-2024
Revise Date: 16-10-2024
Accept Date: 22-10-2024

ABSTRACT:

Background: Liver cirrhosis (LC) is considered the most prevalent cause of portal vein thrombosis (PVT) due to the defective synthetic function of the liver and the stagnation of blood flow. The present work aims to assess the impact of shear wave elastography (SWE) in distinguishing between tumoral and bland PVT by using a triphasic CT study of the liver as the gold standard.

Methods: This prospective cross-sectional study was conducted on 24 cases in the Radio-diagnosis Department, Faculty of Medicine, Zagazig University. All patients with suspected Portal vein thrombosis underwent an entire history and clinical assessment. All cases were conducted to complete history taking, clinical examination, and laboratory investigations, including Hemoglobin, platelet count, Liver function Test (Albumin, Total bilirubin, ALT, and AST), and Kidney function Test (Blood Urea and S. Creatinine), in addition to radiological assessment including triphasic CT examination of PV with 2 D portography and Shear wave elastography.

Results: There was significant variance between malignant and benign lesions detected by Triphasic CT with regard to age, age group, sex, site of thrombosis, SWE, and diagnosis (P=0.02, 0.008, 0.04, 0.04, <0.001, and 0.01, respectively). On conducting ROC curve analysis for discriminating between benign and malignant lesions by shear wave elastography, at cut-off point 13, it shows sensitivity (84.2%), specificity (100%), and AUC (0.921). There was a significant difference between the site of portal vein thrombosis and shear wave elastography, as the median of SWE was higher at the main trunk (P=0.05).

Conclusion: SWE can distinguish between benign and malignant PV thrombus without the need for contrast, radiation, or invasive techniques.

Keywords: Shear Wave ; Elastography ; Tumoral; Thrombosis

INTRODUCTION

The portal venous system transports blood from the intestine (including the lower portion of the rectum), pancreas, gallbladder, and spleen to the liver. It is made up of the splenic and superior mesenteric veins (SMV), which form the portal vein and flow directly into the liver. The portal vein (PV) accounts for approximately 75% of the hepatic blood flow [1].

PV thrombosis (PVT) can be described as whole or partial blockage of blood circulation in the PV caused by thrombus formation in the vein's core. It may arise in connection with liver cirrhosis or liver cancer or without any accompanying liver illness [2].

PVT can be classified as tumoral or bland, depending on the extension of the tumor into the veins. Characterizing PVT as tumoral versus bland is crucial to accurately detecting tumor stage and appropriate treatment for hepatic tumors, especially hepatocellular carcinoma (HCC). Tumoral PVT affects 6.5-44% of HCC cases, whereas bland PVT affects 4.5%- 26% of individuals with chronic liver disorders and 42% of cases with HCC. Bland PVT can be addressed with anticoagulant and thrombolytic medication [3].

Variables that make up the Virchow triad (blood stasis, impaired endothelial function, and hypercoagulability), especially liver cirrhosis and tumors, particularly HCC, may cause PVT [4].

MRI, contrast-enhanced computed tomography (CECT), and Doppler ultrasonography (DUS) imaging, in conjunction with clinical and laboratory results, are foundational for distinguishing between tumoral and bland PVT and deciding the therapeutic regimen [5].

Tri-phasic CT of PVT can reveal a filling deficiency that partially or completely obstructs the vessel lumen and rim augmentation of the vein wall that extends into the splenic and SMVs. Despite the accuracy of tri-phasic CT scans in identifying PVT, contact with ionizing radiation and injected contrast material hypersensitivity remains a crucial issue [6].

Shear Wave Elastography (SWE) is an innovative diagnostic technique that assesses the ability of soft tissue to resist force-induced deformation due to its internal stiffness. Abnormal tissues are often less elastic than the normal tissue around them. SWE is regarded as an advanced elastography approach; it relies on evaluating the propagation of SW and allows for the quantitative evaluation of tissue stiffness based on the estimation of SW velocity [7].

The present work aims to evaluate the impact of SWE in distinguishing between tumoral and bland PVT using the Triphasic CT study of the liver as the gold standard.

METHODS

Patients: This prospective cross-sectional study was conducted on 24 cases in the Radio-diagnosis Department, Faculty of Medicine, Zagazig University. Informed consent has been obtained from all individuals involved in this investigation. This study was approved by Zagazig University Ethical Committee regulations. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research. We obtained the approval from the Institutional Review Board (IRB# 10586)

Cases with the following characteristics were included: patients with portal vein thrombosis diagnosed by U/S and Doppler U/S.

Cases with the following characteristics were excluded: patients with tense ascites or with hepatic encephalopathy, Morbid obese patients, non-operative patients, those contraindicated to the I.V. contrast agents, and patients with increased serum creatinine levels.

Sample size: Assuming all cases meet the criteria for inclusion and exclusion, they were conducted in the study. Throughout the study term (6 months), a thorough sample of 24 cases was collected at a rate of four per month.

Methods: All cases were subjected to complete history taking, clinical assessment, and laboratory investigations, including Hemoglobin, platelet count, liver function Test (Albumin, Total bilirubin, ALT, AST), and Kidney Function Test (Blood Urea and S. Creatinine), in addition to radiological assessment.

SWE of PV:

2D SWE was performed in all patients using an Ultrasound machine (CANON APLIO 500 system, TOSHIBA Medical Systems, Japan).

PVT was assessed using a C1-5-RS (1.75-4.95 MHz) convex probe through the intercostal or subcostal channel in a supine position with the associated arm maximally abducted to enlarge the intercostal space for easier inspection. Seven cases were conducted to 2D SWE in the left or right lateral positions to improve exposure to the liver and PV. Cases were instructed to hold their breath for 5 sec. Image acquisition was started, resulting in two or three colorful pictures.

Region Of Interest (ROI) was placed. Then, valid measurements were obtained in terms of Young modulus within each measurement region, which was automatically documented by the system in a worksheet. The system determined the median value of the accurate readings in kps. ROI was presented as a rectangular area measured 5*15 mm. The measurements were taken from the thrombosed portal vein's main trunk and left and right branches. The value of an average of 5-8 was considered a valid measurement.

Triphasic CT examination of PV:

Triphasic CT of the abdomen and pelvis was done in all instances using the MSCT PHILIPS 128.

Via a dual head pump injector, 150 mL of Omnipaque 300 mg I/ml (iohexol 300mgI/ml; Nycomed, Princeton, NJ) was injected IV at the anticubital vein with a rate of 4mL/sec followed by 100 ml of saline for flushing. Double arterial phase scanning, consisting of early and late arterial phase image acquisition, was performed during a single breath-hold using 20 and 35-second scanning delays from the initiation of contrast material injection. These timing delays were explicitly chosen so that both phases could be performed within a single breath-hold that ranged from 30-50 sec (which could be performed in all our patients). We elected to perform these two phases during a single breath-hold to minimize the effect of variation on lesion conspicuity and detection. The entire liver was scanned in a cephalad to caudad direction using detector collimation of 5 mm with a table speed per

rotation of 15mm/0.8 sec, a pitch 3 in the scanners HQ mode, and an image thickness of 5 mm. After a brief period of quiet breathing, portal venous phase imaging of the entire abdomen was performed in a cephalad to caudad direction during a breath-hold, using a scanning delay of 60 sec, detector collimation of 5 mm, table speed of 15 mm/0.8 sec, pitch of 3(HQ mode), and an image thickness of 5 mm. Using these parameters, the combined scanning duration for both arterial phase acquisitions was 20-24 sec, and for the portal venous phase,12-15 sec. A delayed phase scan was obtained (2-5 min after injection).

Statistical Analysis:

Data were analyzed by employing SPSS version 26. Qualitative data was provided as frequencies and relative percentages. The qualitative variables were compared using the chi-square test ($\chi 2$) and Fisher exact test, as specified. Quantitative data were presented as mean ± SD. The Independent Samples t-test was used to compare two independent groups of regularly distributed variables, whereas the Mann-Whitney U test was used for non-normally distributed data. ANOVA test and Kruskal- Wallis were utilized to compare quantitative variables of more than two groups. Spearman's correlation was employed to examine the relationship between two variables with non-parametric quantitative data. ROC curve analysis is utilized to examine the predictive value of continuous numerical variables. P-values <0.05 are considered significant.

RESULTS

This cross-sectional study included 24 patients with portal vein thrombosis. Their ages ranged from 43 to 72 years with a mean ± SD of 57.9 ± 9.28; most of the patients (79.2%) were males, and (20.8%) were females. SWE levels ranged from 4.8 to 33 with a median (IQR) of 18.7 (14.2), (66.7%) were malignant lesions, and (33.3%) were benign lesions. The laboratory data among the studied patients were presented in Table (1). Triphasic CT detected 19 (79.2%) malignant and 5 (20.8%) benign lesions. As regards the site of portal vein thrombosis; (79.2%) of the lesions were located at the main trunk, (8.3%) of the lesions were located at the right PV, and (12.5%) of the lesions were located at the left PV.

There was a substantial variance between benign and malignant lesions detected by Triphasic CT with regard to age, age group, sex, site of thrombosis, SWE, and diagnosis (P=0.02, 0.008, 0.04, 0.04, <0.001, and 0.01, respectively). (Table 2)

Cut off the value of 13, above which case is considered malignant, while below it is considered benign. On conducting ROC curve analysis for discriminating between benign and malignant lesions by shear wave elastography, at cut-off point 13, it shows sensitivity (84.2%), specificity (100%), and AUC (0.921). (Table 3)

Table (4) showed a substantial variance between the site of portal vein thrombosis and SWE, as the median SWE was higher at the main trunk (P=0.05).

Table 1: Demographic, clinical, laboratory, SWE, and diagnosis data among studied patient

Variables	All patients (n=24)
Age *(years)*	
Mean ± SD	57.9 ± 9.28
Range	(43 – 72)
Age groups *(N. %)*	
<50	6 (25%)
50 – 60	8 (33.3%)
>60	10 (41.7%)
Sex *(N. %)*	
Male	19 (79.2%)
Female	5 (20.8%)
Hb *(g/dL)*	
Median (IQR)	9.8 (1.075)
Min-Max	8.6-12.1
PLT *($10^3/mm^3$)*	
Median (IQR)	163 (55.25)
Min-Max	39-258

Table 1: Demographic, clinical, laboratory, SWE, and diagnosis data among studied patient	
Albumin *(g/dL)* Median (IQR) Min-Max	 3.1 (0.54) 2.7-3.7
Bilirubin *(mg/dL)* Median (IQR) Min-Max	 1.15 (0.15) 0.96-8.5
ALT *(U/L)* Median (IQR) Min-Max	 39 (10) 25-55
AST *(U/L)* Median (IQR) Min-Max	 46 (10) 34-108
Urea *(mg/dL)* Median (IQR) Min-Max	 39 (12.25) 16.5-65
Creatinine *(mg/dL)* Median (IQR) Min-Max	 1.02 (0.1) 0.9-1.2
SWE Median (IQR) Range	 18.7 (14.2) (4.8 – 33)
Diagnosis (N. %)by SWE Benign Malignant	 8 (33.3%) 16 (66.7%)
Diagnosis by Triphasic CT Benign Malignant	 5 (20.8%) 19 (79.2%)
Site of PVT Main trunk Right Left	 19 (79.2%) 2 (8.3%) 3 (12.5%)

Hb: Hemoglobin, IQR: Interquartile Range, PLT : Platelet Count, ALT : Alanine Aminotransferase, AST : Aspartate Aminotransferase, SWE: Shear Wave Elastography, PVT : Portal Vein Thrombosis

Table 2: Comparison between benign and malignant lesions as regard demographic, clinical, laboratory, SWE, and diagnosis data.

Variables	Benign (n=5)	Malignant (n=19)	P value
Age *(years)* Mean ± SD Range	 49.2 ± 8.58 (43 – 64)	 60.2 ± 8.19 (45 – 72)	 0.021
Age groups *(N. %)* <50 50 – 60 >60	 4 (80%) 0 (0%) 1 (20%)	 2 (10.5%) 8 (42.1%) 9 (47.4%)	 0.008[2]
Sex *(N. %)* Male Female	 2 (40%) 3 (60%)	 17 (89.5%) 2 (10.5%)	 0.04[2]
Hb *(g/dL)*	9.3 (0.2)	10 (1.5)	0.09[1]
PLT *($10^3/mm^3$)*	258 (115)	163 (65)	0.13[3]
Albumin *(g/dL)*	3.1 (0)	3.08 (0.65)	0.89[3]

Variables	Benign (n=5)	Malignant (n=19)	P value
Bilirubin *(mg/dL)*	1.1 (0.07)	1.2 (0.3)	0.06[3]
ALT *(U/L)*	38 (7)	40 (10.5)	0.53[1]
AST *(U/L)*	46 (2)	43 (10)	0.26[3]
Urea *(mg/dL)*	36 (4)	40 (19)	0.41[1]
Creatinine *(mg/dL)*	1 (0.1)	1.02 (0.1)	0.87[1]
Site of lesion Main trunk Right Left	 3 (60%) 2 (40%) 0 (0%)	 16 (84.2%) 0 (0%) 3 (15.8%)	 0.04[2]
SWE *Median (IQR)*	 6.3 (1.5)	 20.2 (5.15)	 <0.001[3]
Diagnosis by SWE Benign Malignant	 5 (100%) 0 (0%)	 3 (15.8%) 16 (84.2%)	 0.001[2]
*[1]*Student's T test,* [2]*Fisher's exact test,* [3]*Mann-Whitney U test, Significant: P ≤0.05*			

Hb: Hemoglobin, IQR: Interquartile Range, PLT : Platelet Count, ALT : Alanine Aminotransferase, AST : Aspartate Aminotransferase, SWE: Shear Wave Elastography

Table 3: ROC curve analysis of shear wave elastography in differentiating benign from malignant lesions

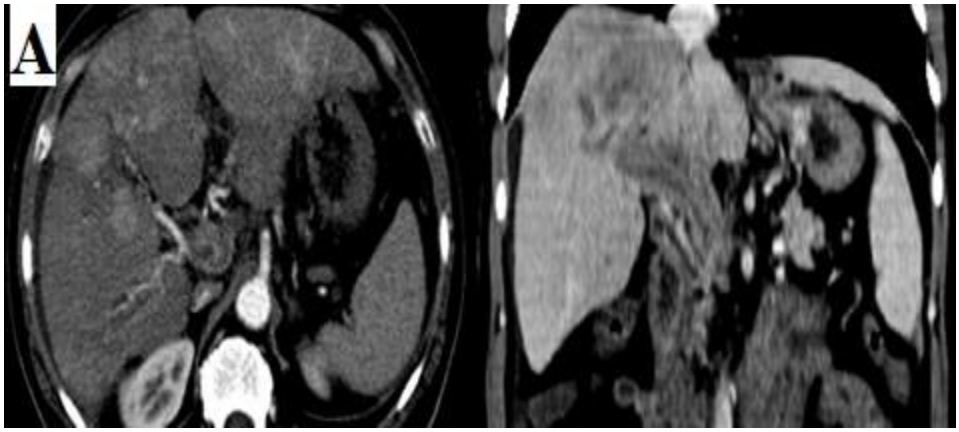
Cut-point	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	AUC
13	84.2%	100%	100%	62.5%	0.921

PPV: Positive predictive value, NPV: Negative predictive value, AUC: Area under curve

Table 4: Comparison between site of PVT as regard SWE

Variables *(N. %)*	Main trunk (n=19)	Right (n=2)	Left (n=3)	P value
SWE *Median (IQR)*	 20.2 (14.5)	 4.8 (0)	 17.5 (0)	 0.05[1]
*[1]*Kruscal-Wallis, Non-significant: P >0.05, Significant: P ≤0.05*				

IQR: Interquartile Range
SWE: Shear Wave Elastography



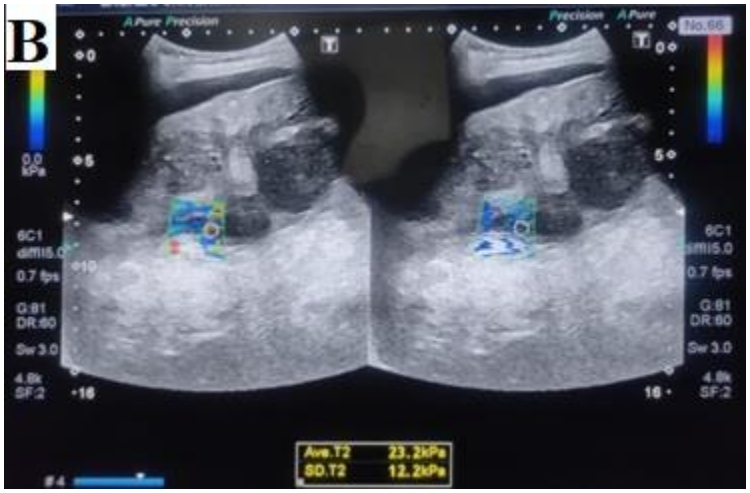


Fig. 1. Male patient aged 74 years old. (A) Triphasic CT reveals cirrhotic liver and hepatic focal lesions with main portal vein thrombosis. (B) SWE shows cirrhotic liver, ascites, hepatic focal lesions and main portal vein thrombosis with ROI measured elasticity of 23.2 kpa denoting tumoral PVT.

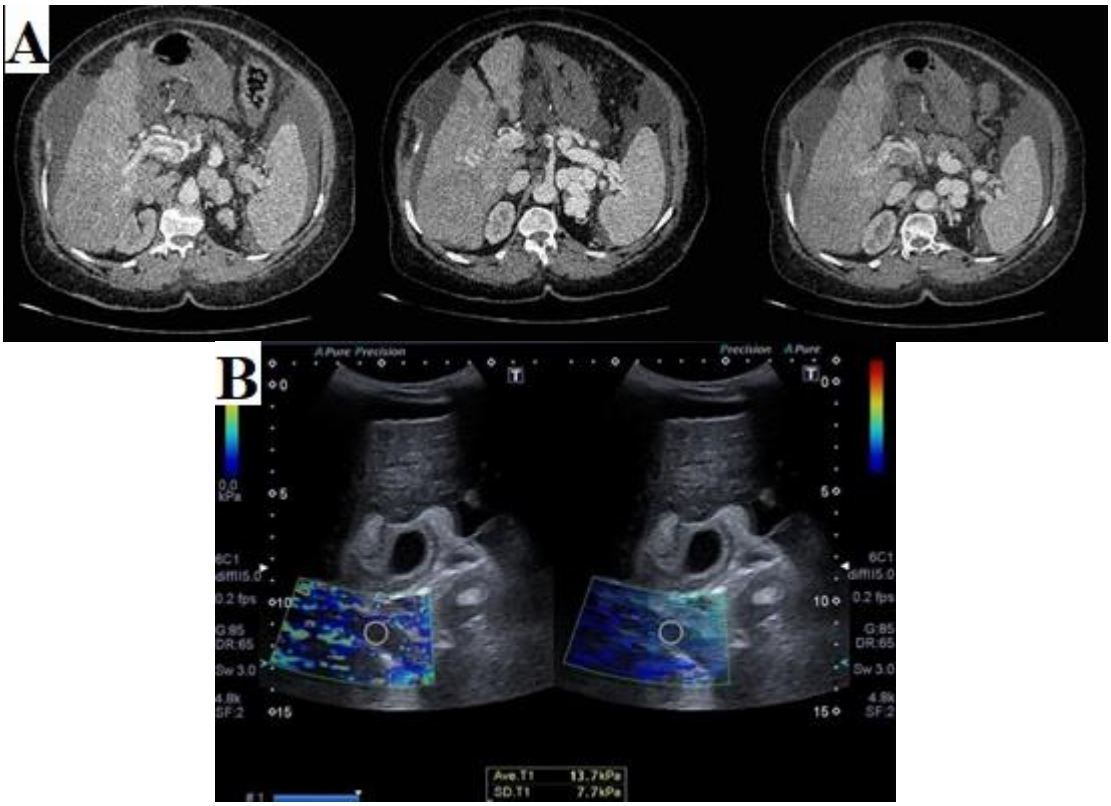


Fig. 2. Male patient aged 72 years old. (A) Triphasic CT reveals multiple focal lesions with main PVT. (B) SWE showed ascites, main portal vein thrombosis with ROI measured elasticity of 13.7 kpa denoting tumoral PVT.

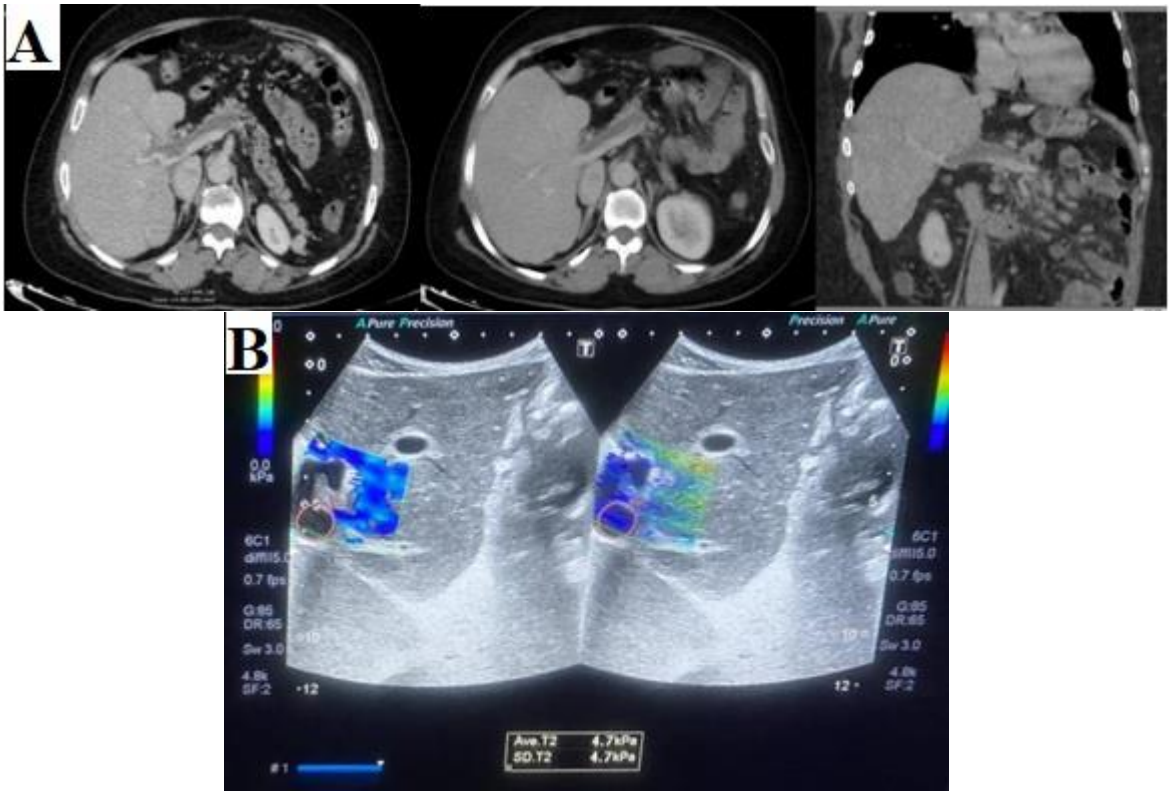


Fig 3. Female patient 44 years old. (A) Triphasic CT reveals splenectomy and healthy liver with long segment main stem portal vein bland thrombosis. (B) SWE shows main portal vein thrombosis with ROI measured elasticity of 4.7 kpa denoting bland PVT.

DISCUSSION

This study aimed to assess the accuracy of SWE in distinguishing between bland and malignant PVT.

A quantitative evaluation of PVT stiffness by SWE was done and compared with the final diagnosis obtained by triphasic CT.

This study showed a substantial variance in stiffness between bland and malignant portal vein thrombi with mean ±SD 6.3 ±1.5 kPa for bland PVT and 20.2 ±5.15 kPa for malignant PVT with substantial variance (p-value < 0.001).

SWE was used because it is a recent method for assessing organ stiffness and is usually incorporated in US machines. It was found that using SWE on the portal vein thrombus has an excellent predictive value in evaluating the stiffness of the thrombus and the differentiation between bland and malignant thrombi. The cut-off value for SWE was 13, at which sensitivity was 84.2%, specificity was 100%, PPV was 100%, NPV was 62.5%, and accuracy was 92%.

Regarding the demographic data of this study, the majority of patients were male (79.2% of total studied cases), and their ages ranged from 43 to 72, with a mean of 57.9 ± 9.28. A retrospective multi-center study done between 1995 and 2004 by Rajani et al. [8] showed that the prevalence and incidence rates of PVT were 0.7 per 100,000 persons each year.

Alhaddad et al. [9], who were studying the prevalence and clinical characteristics of PVT in hepatitis C virus-related cirrhotic cases in an Egyptian cohort, also observed male predominance (71.8%).

In our study, the majority of PVT cases (62.5%) were having liver cirrhosis. On the other hand, Amitrano et al. [10] proposed that their examination of risk factors and the clinical signs of PVT in cases with liver cirrhosis revealed that it occurs primarily in cirrhotic individuals with severe liver damage. They stated that among 701 cirrhotic patients, PVT was found in 79 patients (11.2%).

Regarding laboratory investigations, most of our laboratory results showed non-significant relations when differentiating benign from malignant PVT, such as serum albumin, bilirubin, serum creatinine, and alanine aminotransferase (ALT). Nery et al. [11] assumed similar non-significant associations of serum creatinine, serum albumin, and bilirubin with PVT.

Aspartate transglutaminase (AST) or serum glutamic oxaloacetic transaminase (SGOT) was not significantly higher in malignant cases than benign ones (median =46mg/dl and 43mg/dl), respectively. Correlated results found by Ponziani et al. [12] adopted that laboratory investigations in PVT will be normal unless another hepatic disorder is concurrence.

In contrast to J. Carlos García-Pagán et al. [13], who documented the common mild increase in transaminases in cases of PVT, they mentioned that these results are most likely attributable to altered hepatic perfusion, notwithstanding the arterial compensatory vasodilation.

In our study, triphasic CT showed that main trunk PVT accounted for 79.2%, while right and left PVT accounted for 20.8% of all studied cases. Amitrano et al., in their study, revealed that thrombosis involved in the main trunk portal vein in 85% of patients, right and left PVT (8-9%), while isolated mesenteric or splenic thrombus in 6.1%.[10]

Regarding SWE, our study corresponds to Aboelezz et al. [14] since they stated that SWE could be a valuable tool to differentiate between tumoral and benign tissue within the portal vein. Our study reveals that Tumoral PVT has higher SWE values than Benign PVT (Sig 0.001), while They revealed (Sig 0.012). However, SWE showed 3 cases as benign PVT, while triphasic CT diagnosed these as malignant PVT. These misdiagnosed cases were due to the patient was not fully cooperative, the morbidly obese patient, and the lesion location beyond the penetration limits of SWE.

The current study determined a cut-off value of 13 to distinguish between benign and malignant thrombus. Closer to this study, Aboelezz et al. [14] selected 15.5 as the cut-off point for distinguishing between benign and malignant hepatic PVT, with a sensitivity of 93, specificity of 90, and accuracy of 92.5.

However, SWE showed 3 cases as beginning PVT, while triphasic CT diagnosed these as malignant PVT. These misdiagnosed cases were due to the patient not being fully cooperative, the patient being morbidly obese, and the lesion location being beyond the penetration limits of SWE.

Numerous constraints are associated with employing SWE: The first reason for failure was the lesion's position outside the SWE penetration limitations (>8 cm) and closeness to the vasculature. Other limitations were ascribed to cases' disability to breath holding long enough to achieve a steady SWE image acquisition, particularly in cases with lung cancers and significant ascites. The diagnosis was not blinded before the acquisition of elastography pictures, which may have introduced bias.

Conclusion

SWE is a fast, noninvasive indicator for PVT detection. Our study concluded that SWE can distinguish between benign and malignant PV thrombus without the need for contrast, radiation, or invasive techniques.

Conflict of interest: None.

Financial Disclosures: None.

REFERENCES:

1. Corness JAG, McHugh K, Roebuck DJ, Taylor AM. The portal vein in children: radiological review of congenital anomalies and acquired abnormalities. Pediatr Radiol. 2006;36:87–96, quiz 170–1.

2. Chawla Y, Duseja A, Dhiman RK. Review article: the modern management of portal vein thrombosis. Aliment Pharmacol Ther. 2009;30:881–94.

3. Benevento F, Pecorelli A, Stefanescu H, Sparchez Z, Vukotic R, Pettinari I, et al. Presence of Hepatocellular Carcinoma Does Not Affect Course and Response to Anticoagulation of Bland Portal Vein Thrombosis in Cirrhotic Patients. J Hepatocell Carcinoma. 2023;10:473–82.

4. Young K, Wong R. Evaluation and management of acute and chronic portal vein thrombosis in patients with cirrhosis. Clin Liver Dis (Hoboken). 2017;10:152–6.

5. Catalano OA, Choy G, Zhu A, Hahn PF, Sahani DV. Differentiation of malignant thrombus from bland thrombus of the portal vein in patients with hepatocellular carcinoma: application of diffusion-weighted MR imaging. Radiology. 2010;254:154–62.

6. Nakayama Y, Yamashita Y, Takahashi M. CT Portography by Multidetector Helical CT. In: Reiser MF, Takahashi M, Modic M, Bruening R, editors. Multislice CT [Internet]. Berlin, Heidelberg: Springer; 2001 [cited 2024 Sep 21]. p. 187–96.

7. Sigrist RMS, Liau J, Kaffas AE, Chammas MC, Willmann JK. Ultrasound Elastography: Review of Techniques and Clinical Applications. Theranostics. 2017;7:1303–29.

8. Rajani R, Björnsson E, Bergquist A, Danielsson A, Gustavsson A, Grip O, et al. The epidemiology and clinical features of portal vein thrombosis: a multicentre study. Aliment Pharmacol Ther. 2010;32:1154–62.

9. Alhaddad O, Elsabaawy M, Elshaaraawy O, Elhalawany M, Houseni MM, Abdelsameea E. Portal vein thrombosis in hepatitis C virus-related cirrhotic patients: Prevalence and clinical characteristics in an Egyptian cohort. Trop Doct. 2021;51:314–8.
10. Amitrano L, Guardascione MA, Brancaccio V, Margaglione M, Manguso F, Iannaccone L, et al. Risk factors and clinical presentation of portal vein thrombosis in patients with liver cirrhosis. J Hepatol. 2004;40:736–41.
11. Nery F, Chevret S, Condat B, de Raucourt E, Boudaoud L, Rautou P-E, et al. Causes and consequences of portal vein thrombosis in 1,243 patients with cirrhosis: results of a longitudinal study. Hepatology. 2015;61:660–7.
12. Ponziani FR, Zocco MA, Campanale C, Rinninella E, Tortora A, Maurizio LD, et al. Portal vein thrombosis: Insight into physiopathology, diagnosis, and treatment. World J Gastroenterol. 2010;16:143–55.
13. García-Pagán JC, Caca K, Bureau C, Laleman W, Appenrodt B, Luca A, et al. Early use of TIPS in patients with cirrhosis and variceal bleeding. N Engl J Med. 2010;362:2370–9.
14. Aboelezz Ahmad AF, Elsawy AA, Omar HM, Abofrekha MH, Gabr MT. The role of shear wave elastography in differentiation between benign and malignant portal vein thrombosis in hepatocellular carcinoma. Egypt J Radiol Nucl Med. 2022; 53: 88.

Citation

Abowarda, M., Hassan, T., Moustafa, N., Tharwat, E. The Role of Shear Wave Elastography in Differentiation Between Tumoral and Bland Portal Vein Thrombosis. ***Zagazig University Medical Journal*****, 2024; (373-381): -. doi: 10.21608/zumj.2024.325392.3610**