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Value of Diffusion-Weighted Imaging in Differentiating Benign from Malignant Portal Vein Thrombosis: A Cross-Sectional Prospective Study

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Abstract

Background: It is still difficult to make noninvasive distinction between tumor invasion and bland clot in portal vein thrombosis [PVT]. The histopathologic examination is the gold standard to assess PVT. However, open laparotomy or percutaneous biopsy have been supplanted by imaging diagnostics in clinical practice to characterize PVT.

Aim of the work: By measuring apparent diffusion coefficient [ADC] values, this study aimed to validate diffusion weighted magnetic resonance imaging [MRI] for distinguishing benign from malignant PVT.

Patients and methods: Diffusion weighted sequences and dynamic liver MRI were performed on 159 adult patients with imaging-confirmed PVT. To determine ADC values and signal intensity ratios, regions of interest were positioned in each thrombus and within the adjacent spinal cord.

Results: Malignant PVT [n=129] occurred in older patients than benign PVT [n = 30] [mean age 62.1 ± 7.3 vs 50 ± 13.3 years; $p < 0.001$]. Portal hypertension, lower limb swelling, and CHILD score C were prevalent in malignant than benign PVT [72.9%, 45.7%, 61.2% vs 33.3%, 16.7% and 0.0% respectively]. Mean thrombus ADC was significantly lower in malignant than benign PVTs [$1.2 \pm 0.14 \times 10^{-3}$ mm²/s vs $1.4 \pm 0.05 \times 10^{-3}$ mm²/s respectively]. ROC analysis for ADC yielded an area under the curve [AUC] of 0.677 [$p = 0.02$] with a cutoff $\leq 1.2 \times 10^{-3}$ mm²/s, achieving 73.2% sensitivity and 56.7% specificity. The ADC ratio [PVT/cord] was also lower in malignant cases [1.6 ± 0.3 vs 1.9 ± 0.36; $p < 0.001$] but demonstrated poor discriminatory performance [AUC = 0.60; $p = 0.453$].

Conclusion: When combined with mean ADC values, DW MRI is a valuable noninvasive imaging method that is highly effective at characterizing tissue. In addition, it can be used to distinguish between benign and malignant PVT.

Keywords: Portal Vein Thrombosis; Diffusion-Weighted Imaging; Apparent Diffusion Coefficient; Benign; Malignant.

INTRODUCTION

Portal vein thrombosis [PVT] is a pathological disorder that can involve the splenic and superior mesenteric veins and is characterised by occlusion of the portal vein lumen and its intrahepatic branches. PVT typically occurs in silence and is frequently diagnosed by chance in the screening of chronic hepatopathy, whether or not portal hypertension is linked to it [1].

PVT may be caused by a number of diseases, such as neoplastic diseases, cirrhosis, infection, myeloproliferative disorders, intra-abdominal inflammatory diseases, and hypercoagulable states [2]. The total incidence of PVT varies between 0.05% and 1% in several autopsy assessments [3].

For those with neoplastic diseases, the presence of malignant PVT is crucial during staging tumours, selecting the best treatment, and assessing prognosis [4].

The identification and description of PVT in individuals with HCC depend heavily on imaging. Using morphological and dynamic contrast enhancement features, conventional imaging can readily identify PVT but may not always distinguish between benign and malignant PVT [5].

Up until recently, the detection of contrast enhancement and luminal expansion on CT or MRI was the main technique of imaging separation between benign and malignant PVT [6]. Due to a distinct variation in apparent diffusion coefficient [ADC], diffusion-weighted MRI [DWI] might distinguish between benign and malignant portal vein thrombi [6].

Variations in intracellular and extracellular water mobility are detected using diffusion-weighted MRI [DW-MRI] [7, 8]. DW signals from high cell density tumours are higher than those from inflammatory processes [7].

Tissue-specific characteristics, such as the apparent diffusion coefficient [ADC, [mm²/s]], can be computed for quantification. When tumours show a low ADC on initial imaging, DW-MRI could be a useful method for assessing response [9].

This study aimed to validate the value of DWI for differentiating benign from malignant PVT.

PATIENTS AND METHODS

This prospective study was carried out on 159 patients [119 males and 40 females] with portal vein thrombosis [PVT], either benign or malignant. The patients' ages ranged from 27 to 76 years, with a mean age of 60 ± 10 years. The study protocol was approved by the Ethics Committee of the National Liver Institute, Menoufia University, and informed consent was obtained from all patients before the procedure.

Our study was performed from May 2024 to April 2025 at the Diagnostic and Interventional Radiology Department at the National Liver Institute, Menoufia University [Approval code:00674/2025].

We included adult patients [age >18 years], patients with a known history of liver cirrhosis or hepatocellular carcinoma [HCC], and patients with proven PVT on dynamic MRI liver, either in the main portal vein branch or segmental branches. However, we excluded from the study: pediatric patients [0–18 years], patients who refused or were contraindicated to receive contrast material, patients with contraindications to MRI such as the presence of impact magnetizable devices, non-MRI-compatible pacemakers, or claustrophobia, patients diagnosed with malignancies other than HCC [such as gallbladder cancer, intrahepatic cholangiocarcinoma, gastric cancer, or unknown primary tumors], patients with thrombosis limited to the superior mesenteric vein without portal vein involvement, patients with recanalized chronic PVT associated with collateral formation, and patients with partial rather than complete PVT.

All patients were subjected to thorough history taking and clinical examination. Imaging evaluation of the liver was conducted, including the available previous imaging studies, such as abdominal ultrasound with portal vein color Doppler or triphasic CT liver.

MRI Technique: An abdominal coil and a 1.5-T GE closed MRI were used to perform a dynamic MRI liver. The coil extended from the nipples to the iliac crest while the patients were in the spine position. The following sequences were part of the standard dynamic MRI liver: Following the acquisition of supine localiser images, T2-weighted axial and coronal and series Diffusion-weighted imaging [DWI], T2 fat-saturated, T1-weighted axial and sequence: T1 in-phase and out-of-phase, T1 fat-saturated: Axial and sequential: single-shot diffusion-weighted fat-saturated.

Post-Contrast Imaging: At the arterial phase [20–30 seconds], portal venous phase [60–70 seconds], equilibrium phase [3-5 minutes], and hepatobiliary delayed phase [10–30 minutes] with and without fat sat, post-contrast sequences included T1 2D or 3D gradient-echo sequences. When assessing lesions with an intrinsically high T1 signal, subtracted pictures are helpful.

Dynamic Contrast-Enhanced Imaging Protocol: Three consecutive post-contrast series, including early arterial, late arterial, and portal phase imaging, were included in a dynamic series. Each phase imaging began at 34-second intervals [20 seconds for image acquisition with breath-holding and 14 seconds for re-breathing]. Timing was done using the previously mentioned technique to ascertain the time delay for early arterial phase imaging. A dose of 0.1 mL/kg of gadolinium ethoxybenzyl-diethylenetriamine-pentaacetic acid [Gd-EOB-DTPA] was administered via the cubital vein at a rate of 1.0 mL/s. The patient conducted a breath-hold for arterial phase scanning once the contrast agent had reached the lower thoracic aorta. Scans of the portal venous phase, transitional phase, and hepatobiliary phase were performed 70 seconds, 2–5 minutes, and 15 minutes following the delay. Every scan was forwarded to the PACS.

Imaging Evaluation and Quantitative Analysis: On DWI, the thrombus intensity was classified as either hypointense or hyperintense in regard to the liver [b=800 s/mm²]. The ADC on the ADC map was measured for quantitative analysis by means of signal processing in Nuclear Magnetic Resonance software at the OsiriX and Interspace Philips workstation [Syngo.via]. Each observer used the same slice and regions to draw oval regions of interest [ROIs] in the spinal cord and thrombus that were as large as possible [≥10 mm²]. Three separate measurements of the spinal cord and thrombus signal intensity [SI] values on ADC were made, and the mean values of these measurements were noted. Finally, using the following formula, the signal intensity ratios [SIR] on ADC [SIRADC] were determined: SI thrombus/SI spinal cord. Avoiding visible artefacts including respiratory motion artefacts, magnetic susceptibility artefacts, and cerebrospinal fluid pulsation artefacts was a priority.

To assess the reproducibility of ADC measurements, two independent radiologists with experience in abdominal imaging separately evaluated the ADC values of the thrombus and spinal cord. Each observer placed regions of interest [ROIs] on the same pre-identified slices, blinded to the other's measurements and the clinical diagnosis. For intraobserver variability, one radiologist repeated the ADC measurements two weeks later under the same conditions, also blinded to the initial results. The mean values from each set of measurements were used to calculate the intraclass correlation coefficient [ICC] for both interobserver and intraobserver agreement. An ICC value above 0.75 was considered indicative of good agreement.

Standard of reference: Our study depended on the presence or absence of enhancement, T2 signal intensity, and diffusion as reference for diagnosis of benign or malignant PVT.

Statistical analysis: SPSS software version 26 [IBM Inc., Chicago, IL, USA] was used for statistical analysis. The unpaired t-test was used to compare groups, and quantitative parametric data were displayed as means and standard deviations. Frequencies [%] were used to show the qualitative data, and the chi-square test or Fisher's exact test, as appropriate, were used to compare groups. The Mann-Whitney U test was used to compare groups, and non-parametric quantitative data were displayed as medians and interquartile ranges [IQR]. A p-value below 0.05 was used to determine statistical significance. ROC curve analysis was used to evaluate each test's overall diagnostic performance.

RESULTS

This study consisted of 159 patients [119 males and 40 females] with portal vein thrombosis [PVT], either benign or malignant. The patients' ages ranged from 27 to 76 years, with a mean age of 60 ± 10 years.

Regarding the site of PVT, the right branch was involved in 25.2% of cases, with all occurring in malignant PVT [31.0%] and none in benign PVT. Thrombosis in the left branch was observed in 25.2% of cases, with a higher proportion in benign PVT [50.0%] compared to malignant PVT [19.4%]. Main portal vein involvement was seen in 21.4% of cases, with 16.6% in benign PVT and 22.4% in malignant PVT. Extensive thrombosis involving the main, right, and left branches was present in 28.3% of cases, affecting 33.3% of benign PVT cases and 27.1% of malignant PVT cases. The mean portal vein size was significantly larger in malignant PVT [19.3 ± 5.04 mm] compared to benign PVT [13 ± 1.2 mm, $p < 0.001$] **[Table 1].** Arterial enhancement was observed in 81.1% of cases, exclusively in malignant PVT [100.0%], while all benign PVT cases [100.0%] were non-enhanced [$p < 0.001$]. Thrombus signal intensity on T2-weighted imaging showed hyperintensity in 81.1% of cases, exclusively in malignant PVT [100.0%], whereas all benign PVT cases [100.0%] were hypointense [$p < 0.001$] **[Table 2].** Restricted diffusion was observed in 129 [81.1%] cases, all of which were malignant [p<0.001]. The mean ADC in the portal vein thrombosis was significantly higher in benign PVT [$1.4 \pm 0.05 \times 10^{-3}$ mm²/sec] compared to malignant PVT [$1.2 \pm 0.14 \times 10^{-3}$mm²/sec] [p<0.001]. The ADC ratio of the portal vein thrombosis to the cord was significantly higher in benign PVT [1.9 ± 0.36] than in malignant PVT [1.6 ± 0.3] [p<0.001] **[Table 3].**

For ADC in portal vein thrombosis, the area under the curve [AUC] was 0.677 [p = 0.02], with a cutoff value of ≤1.2 mm²/sec×10^{-3}, achieving a sensitivity of 73.2% and specificity of 56.7%. The positive predictive value [PPV] was 79.2%, while the negative predictive value [NPV] was 62.3% [Figure 1A]. For the ADC ratio [PVT/cord], the AUC was 0.60 [p = 0.453], with a cutoff value of ≤0.8. The sensitivity was 88%, specificity was 62%, PPV was 81.3%, and NPV was 73.2%. However, the low AUC suggests that the ADC ratio had limited diagnostic utility in distinguishing malignant from benign PVT **[Figure 2A].** The association between arterial enhancement and ADC values was significant. The mean ADC in PVT was lower in the enhanced group [1.22 ± 0.14] compared to the non-enhanced group [1.3 ± 0.16, p < 0.001]. Similarly, the ADC ratio in PVT/cord was lower in the enhanced group [1.6 ± 0.3] than in the non-enhanced group [1.8 ± 0.4, p = 0.01] **[Table 4].**

Case 1: A 64-year-old male patient was incidentally found to have a hepatic focal lesion during follow-up after completing DAA therapy for hepatitis C. His lab results showed a markedly elevated alpha-fetoprotein [AFP] level of over 1100 ng/mL. Imaging revealed a malignant main PVT. On axial T2-weighted images, the main portal vein appeared distended with a hyperintense intraluminal thrombus. Arterial-phase post-contrast scans demonstrated thrombus enhancement similar to HCC. DWI and the corresponding ADC map indicated restricted diffusion within the thrombus, with the ADC value measuring 1.13×10^{-3} mm²/sec—findings consistent with malignant PVT [Figure 2].

Case 2:

A 50-year-old male patient with a known diagnosis of HCC, undergoing follow-up after receiving immunotherapy. Radiological evaluation showed a malignant thrombus in the right portal vein. T2-weighted imaging revealed a distended right portal vein containing a hyperintense thrombus. Arterial-phase contrast images showed enhancement of the thrombus mimicking the vascular characteristics of HCC. DWI and ADC images demonstrated restricted diffusion, and the ADC value of the thrombus was recorded at 1.05×10^{-3} mm²/sec, supporting the diagnosis of malignant right PVT [Figure 3].

Case 3:

A 37-year-old female with a known history of Budd-Chiari syndrome presented with portal vein thrombosis. Her tumor marker levels were within normal range. Imaging findings were indicative of a benign thrombus in the left portal vein. On T2-weighted images, the affected portal vein appeared distended and contained a hypointense thrombus. Post-contrast arterial phase images showed no enhancement of the thrombus. DWI and ADC sequences confirmed the absence of diffusion restriction, and the measured ADC value was 1.64×10^{-3} mm²/sec, consistent with a benign thrombus [Figure 4].

Table [1]: Portal vein thrombosis site and size of the examined patients

		Total [n=159]	Benign PVT [n=30]	Malignant PVT [n=129]	P
PVT Site	**Right branch**	40 [25.2%]	0 [0.0%]	40 [31.0%]	<0.001*
	Left branch	40 [25.2%]	15 [50.0%]	25 [19.4%]	
	Main	34 [21.4%]	5 [16.6%]	29[22.4%]	
	Main, left, and right	45 [28.3%]	10 [33.3%]	35 [27.1%]	
Portal vein diameter		18.1± 5.2]	13± 1.2	19.3 ± 5.04	<0.001*

Data are presented as mean ± SD or frequency [%]. *: significant as P<0.05. PVT: Portal vein thrombosis

Table 2: Arterial enhancement and signal intensity on T2 of the examined patients

		Total [n=159]	Benign PVT [n=30]	Malignant PVT [n=129]	P
Arterial Enhancement	**Enhanced**	129 [81.1%]	0 [0.0%]	129 [100.0%]	<0.001*
	Non-Enhanced	30 [18.9%]	30 [100%]	0 [0.0%]	
Thrombus Signal Intensity on T2	**Hyperintense**	129 [81.1%]	0	129 [100%]	<0.001*
	Hypointense	30 [18.9%]	30 [100%]	0	

Data are presented as mean ± SD or frequency [%]. *: significant as P<0.05.

Table 3: Quantitative MRI Parameters of the examined patients

		Total [n=159]	Benign PVT [n=30]	Malignant PVT [n=129]	P
Diffusion	**Non-restricted**	30 [18.9%]	30 [100%]	0	<0.001*
	Restricted	129 [81.1%]	0	129 [100%]	
ADC in portal vein thrombosis		1.23 ± 0.15	1.4 ± 0.05	1.2 ± 0.14	<0.001*
ADC in Cord [mm²/sec][×10-3]		0.75 ± 0.12	0.75 ± 0.15	0.75 ± 0.12	0.17
ADC Ratio [PVT /cord]		1.7 ± 0.33	1.9 ± 0.36	1.6 ± 0.3	<0.001*

Data are presented as mean ± SD or frequency [%]. *: significant as P<0.05. PVT: Portal vein thrombosis. ADC: apparent diffusion coefficient.

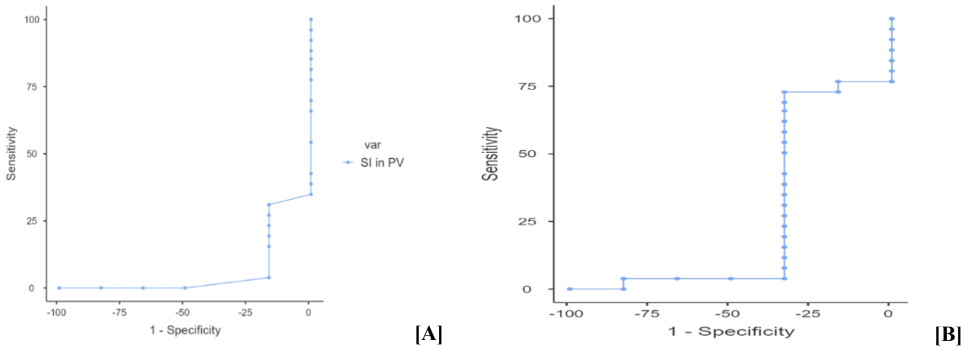

Figure [1]: [A] Roc curve of Portal vein ADC and [B] for ADC ratio [PV/cord] to discriminate Malignant from Benign

Table [4]: Imaging Features of the examined patients

	Total [n=159]	Enhanced	Non-Enhanced	P
ADC in Cord [mm²/sec] [×10^{-3}]	1.25 ± 0.06	1.22 ± 0.14	1.3 ± 0.16	<0.001*
ADC Ratio [PVT /cord]	1.7 ± 0.15	1.6 ± 0.3	1.8 ± 0.4	<0.01*

Data are presented as mean ± SD or frequency [%]. *: significant as P<0.05.

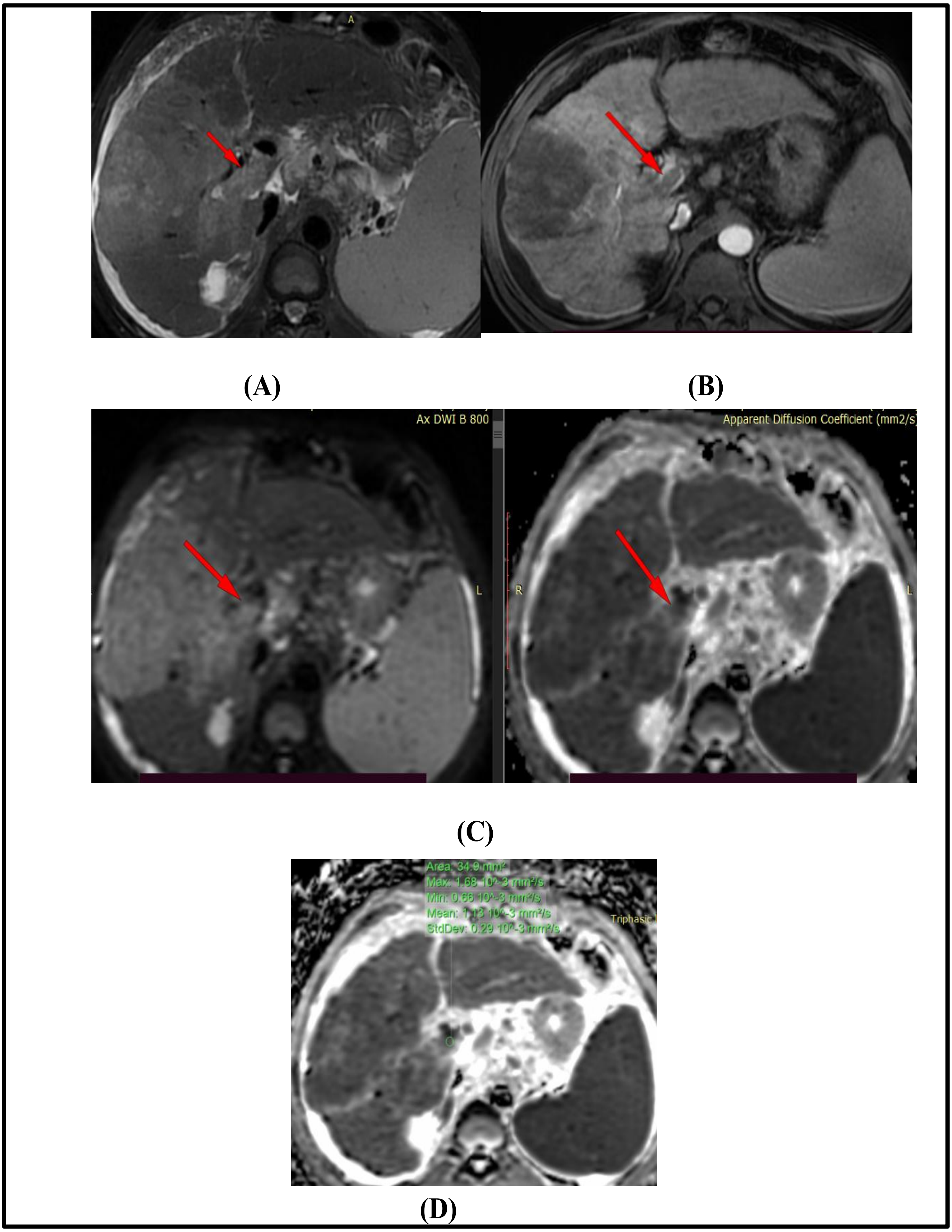

Figure (2): Malignant mains PVT: [A] axial T2 image shows distended main PV with intraluminal thrombosis showing hyperintense signal [red arrow].[B] axial post contrast arterial enhancement showing enhancement of the thrombus like HCC [red arrow]. [C] DWI&ADC map images of PVT showed diffusion restriction. [D] On ADC, the signal intensities within the thrombus were measured and the value of main PVT was 1.13 x10-3 mm2/sec].

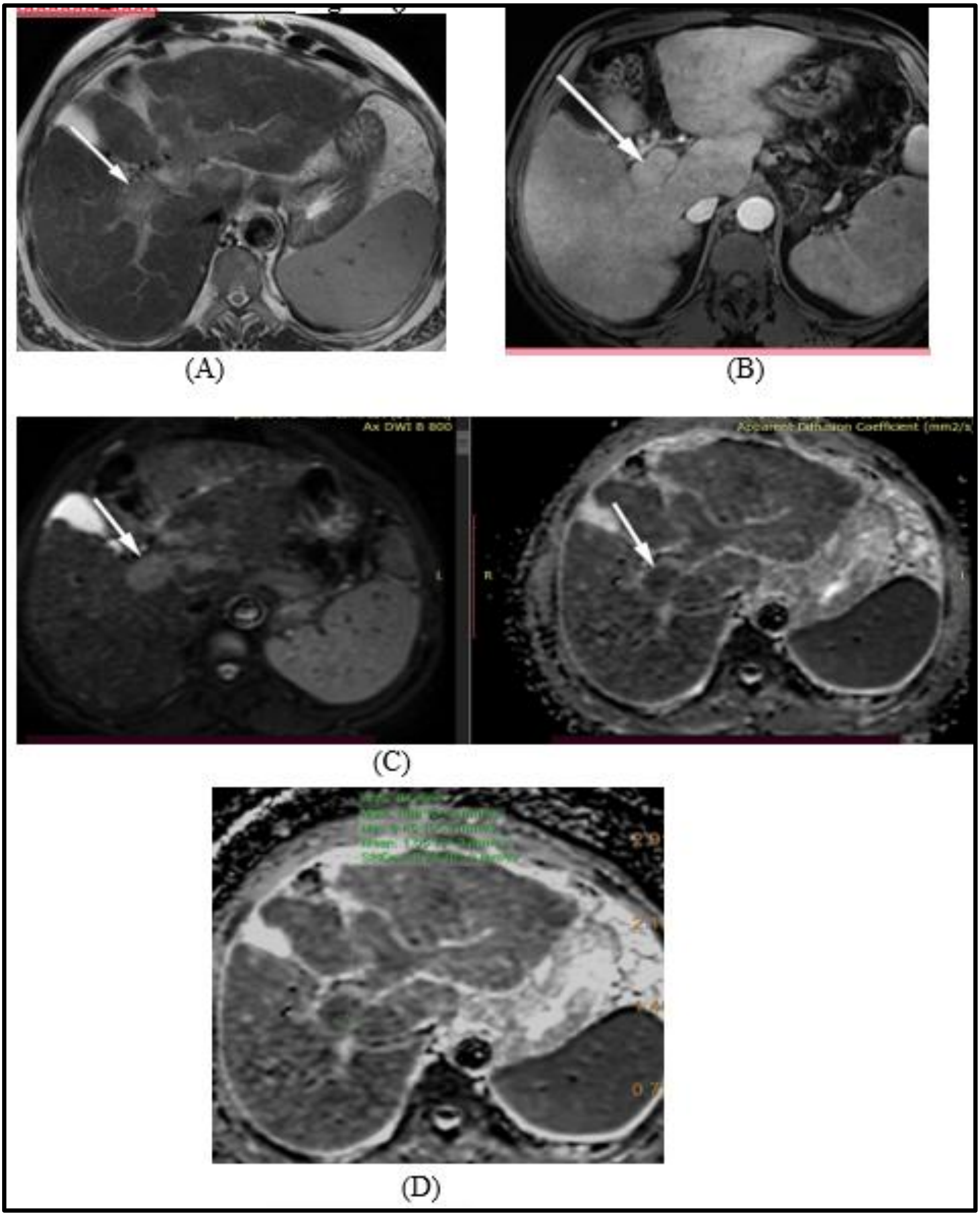

Figure (3): Malignant right PVT :[A] axial T2 image shows distended right PV with intraluminal thrombosis showing hyperintense signal [white arrow].[B] axial post contrast arterial enhancement showing enhancement of the thrombus similar to HCC [white arrow]. [C] DWI&ADC map images of PVT showed diffusion restriction. [D] On ADC, the signal intensities within the thrombus were measured and the value of main PVT was 1.05 x10^{-3} mm^2/sec

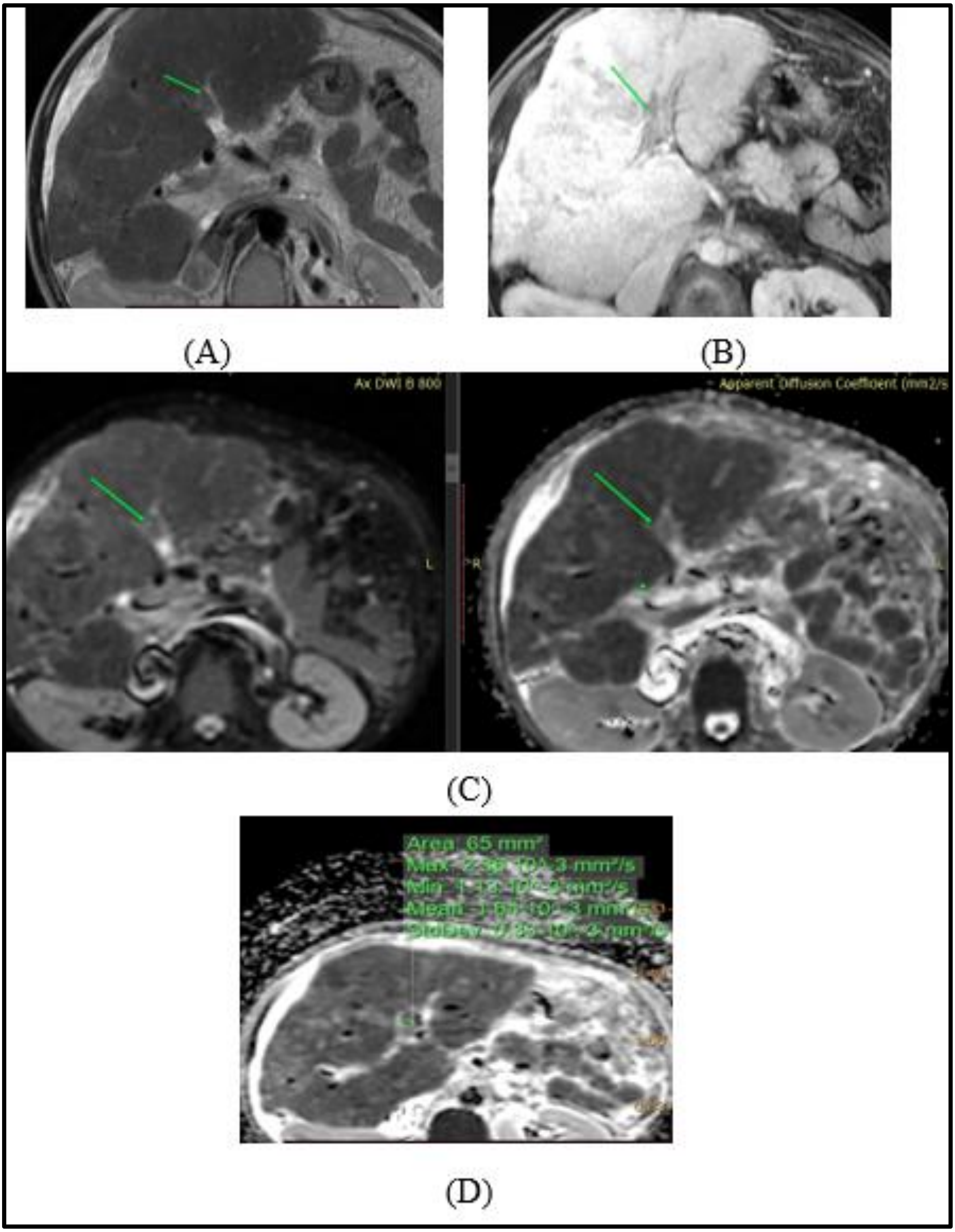

Figure (4): Benign left PVT: [A] axial T2 image shows distended left PV with intraluminal thrombosis showing hypointense signal [orange arrow]. [B] axial post contrast arterial enhancement showing no enhancement [orange arrow]. [C] DWI&ADC map images of PVT showed no diffusion restriction. [D] On ADC, the signal intensities within the thrombus were measured and the value of main PVT was 1.64 x10^{-3} mm^2/sec.

DISCUSSION

In individuals with HCC, tumor thrombus in the portal vein is a significant problem and a predictor of outcome [10]. The histopathologic examination is the gold standard for assessing PVT [11]. But in clinical practice, imaging diagnostics has taken the place of percutaneous biopsy or open laparotomy in order to characterize PVT [12]. Several imaging features of malignant PVT have been documented, and the Doppler US, contrast-enhanced US, contrast-enhanced CT, and contrast-enhanced MRI have been shown to be appropriate modalities for distinction [13]. Dynamic MRI is essential for identifying PVT and differentiating between benign [bland] and malignant thrombi, especially when paired with DWI. Because acute thrombi contain a lot of water, they frequently appear hyperintense on T2-weighted imaging. By emphasizing regions of restricted diffusion, which are suggestive of high cellularity or dense fibrin content, DWI improves detection even more. Because of their dense cellular structure, malignant thrombi usually show real limited diffusion, appearing as hypointense on ADC maps and hyperintense on DWI maps. On the other hand, benign thrombi may exhibit hyperintensity on DWI and ADC maps as a result of the T2 shine-through effect, where the high signal is caused by longer T2 relaxation durations rather than restricted diffusion [3].

Our study exclusively focused on acute portal vein thrombosis [PVT], excluding chronic thrombi characterized by recanalization, lysis, cavernous transformation, or collateral vessel formation. Acute thrombi were identified by imaging features such as increased vessel caliber and intraluminal hyperdensity or hyperintensity on CT, US, and MRI, without evidence of collateralization or cavernoma formation seen in chronic PVT [14-16].

Moreover, emerging MR–noncontract thrombus imaging techniques, such as 3D T1 Dixon and fast field echo sequences, effectively distinguish acute from chronic PVT based on methemoglobin progression and tissue signal changes [17], further justifying our acute-only cohort. By focusing on acute thrombi, our findings gain greater clinical relevance, as these lesions are more likely to respond to therapies and present reliable diffusion and enhancement profiles distinct from chronic stage thrombi.

In our study, among the 159 patients, 119 [74.8%] were male and 40 [25.2%] were female, with ages ranging from 27 to 76 years and a mean age of 60 ± 10 years. The median age was 60 years [IQR: 27–76]. Benign PVT was found in 30 patients, with ages ranging from 27 to 76 years, a mean age of 50 ± 13.3 years, and a median age of 55.5 years. Malignant PVT was observed in 129 patients, aged between 48 and 76 years, with a mean age of 62.1 ± 7.3 years and a median age of 62 years.

In our study, PHT was significantly more frequent in malignant PVT cases [72.9%] compared to benign PVT [33.3%] [$p < 0.001$]. Splenomegaly was the most common organomegaly [65.4%], and lower limb swelling was notably more common in malignant PVT [45.7%] than benign [16.7%] [$p = 0.004$]. CHILD Score A was exclusive to benign PVT, while Score C appeared only in malignant cases.

In our study, the right portal vein branch thrombosis was involved in 25.2% of cases, exclusively in malignant PVT [31.0%]. Left branch thrombosis was more common in benign PVT [50.0%] than in malignant cases [19.4%]. Main portal vein involvement was seen in 21.4% overall [16.6% benign, 22.4% malignant], while extensive thrombosis affecting the main, right, and left branches occurred in 28.3% of patients, slightly more frequent in benign PVT [33.3%] than malignant [27.1%].

In line with our findings, Osman and Samy [18] found that right branch involvement more common in malignant PVT [51.51% vs. 17.6%, $p = 0.01$] and reported that benign PVT more frequently involved the main portal vein [100% vs. 81.81%, $p = 0.03$]. Additionally, they observed that PVT diameter was <16 mm in benign and >18 mm in malignant cases, reinforcing vein size as a potential differentiator.

Notably, the mean portal vein diameter was significantly larger in malignant PVT [19.3 ± 5.04 mm] compared to benign PVT [13 ± 1.2 mm; $p < 0.001$]. These findings are consistent with **Mohakud *et al.*** [19], who reported larger lesion sizes in benign vs. malignant cases and noted cavitation in some lesions. **Koc** [20] similarly observed significantly larger portal vein diameters in malignant PVT [18.2 mm] versus benign [15.8 mm; $p < 0.05$].

In our study, elevated AFP levels were observed in 139 [87.4%] patients, with a significantly higher prevalence in malignant PVT [92.2%] compared to benign PVT [66.7%] [$p<0.001$]. Low AFP levels were found in 20 [12.6%] patients, with benign PVT cases having a higher proportion [33.3%] than malignant PVT cases [7.8%]. Similarly, **Huang *et al.*** [21] demonstrated that there were statistically significant differences in AFP level between benign and malignant PVTs [15 [40.5%] vs 87 [84.5%] respectively, $P<0.05$].

In a study by Shah and colleagues [22], the imaging features of “enhancing and expansile” portal vein thrombus were evaluated. The authors observed that malignant PVT [usually due to HCC invasion] often exhibited heterogeneous enhancement along with elevated AFP levels, serving as a valuable diagnostic indicator of malignancy.

In our study, arterial enhancement was observed in 81.1% of cases, exclusively in malignant PVT [100%], whereas all benign PVT cases [100%] showed no enhancement [$p < 0.001$]. Thrombus signal intensity on T2-weighted imaging showed hyperintensity in 81.1% of cases, found only in malignant PVT [100%], while all benign PVT cases [100%] were hypointense [$p < 0.001$]. Arterial enhancement on contrast studies generally indicates the presence of neovascularization—that is, the formation of new blood vessels. In the context of PVT in patients with underlying malignancy [such as HCC], such enhancement is often seen when tumor cells invade the thrombus, inducing a blood supply that supports rapid growth. This is in contrast to benign thrombi, which lack such vascularization [23].

In our study, restricted diffusion was noted in 129 [81.1%] cases, all of which were malignant [$p < 0.001$]. The mean ADC SI in PVT was significantly higher in benign PVT [mean $1.4 \pm 0.05 \times 10^{-3}$ mm²/sec and median 1.4[1.3-1.4]] compared to malignant PVT [mean $1.2 \pm 0.14 \times 10^{-3}$ mm²/sec, and median 1.2[0.8-1.4] $p < 0.001$]. In our study, for ADC in PVT, the area under the curve [AUC] was 0.677 [$p = 0.02$], with a cutoff value of ≤ 1.2 mm²/sec $\times 10^{-3}$, yielding a sensitivity of 73.2%, specificity of 56.7%, positive predictive value [PPV] of 79.2%, and negative predictive value [NPV] of 62.3%, indicating moderate diagnostic performance in differentiating malignant from benign PVT.

In line with our findings, **Mohakud *et al.*** [19] reported that the mean ADC in PVT was higher in benign PVT [$1.49 \pm 0.38 \times 10^{-3}$ mm²/sec] compared to malignant PVT [$1.11 \pm 0.20 \times 10^{-3}$ mm²/sec, $p < 0.001$]. They exhibited that ROC curve for 5-point rank scale on DWI to differentiate benign and malignant lesions showing an AUC as 0. 842 [95% confidence interval [CI], 0. 666–1.000]. The AUC for minimum ADC is 0.860 [95% CI, 0. 691–1.000].

In agreement with our results, **Huang *et al.*** [21] noted that the mean signal intensity ratio $[SIR]_{ADC}$ of benign and malignant PVTs were 0.72±0.32 and 0.62±0.17 respectively. There were significant differences between benign and malignant PVTs in DWI [t=–2.138; 95% CI: −0.179–0.007; P=0.034]. They demonstrated that for SIR_{ADC} in PVT, the AUC was [0.619; 95% CI: 0.500–0.737, p = <0.001], with a cutoff value of 0.791 mm²/sec, yielding a sensitivity of 45.9%, and specificity of 83.3%. Also, **Sonbel *et al.*** [4] reported that patients with malignant focal lesions had significantly lesser mean ADC when contrasted with cases with benign focal lesions [0.96 ± 0.17 vs 1.88 ± 0.60; P=0.000]. Among cases with malignant focal lesions, cases with malignant PVT had significantly lower mean ADC PVT [1.08 ± 0.16 vs 2.07 ± 0.13; P=0.000], as well as significantly lower ADC ratio [1.07 ± 0.07 vs 2.42 ± 0.50; P< 0.05] when compared to patients with benign PVT. They reported that ROC curve revealed that cut off value of ADC 1.42 or less had significant discriminative capability detect malignant PVT among the studied malignant FL cases with AUC 1.000, 100 percent sensitivity, 100 percent specificity, 100% PPV and 100% NPV. However, **Koc** [20] stated that the mean ADC values of benign thrombus were calculated as 1.03±0.27 x 10-3 mm²/sec for b400, and $1.01 \pm 0.23 \times 10^{-3}$ mm²/sec for b1000. The mean ADC values were calculated as 0.93±0.13 x 10-3 mm²/sec for b400 and $0.88 \pm 0.26 \times 10^{-3}$ mm²/ sec for b1000 for malignant thrombus. No statistically significant difference was found between the groups [p=0.778]. The use of normalized metrics [such as ADC ratios comparing the PVT with adjacent structures] in our investigation may have enhanced the ability to detect differences by compensating for inter-patient variability. Koi et al.'s approach, which focused solely on absolute ADC values, may not have accounted for such variability, leading to overlapping values between benign and malignant thrombi.

In our study, the ADC ratio of the PVT to the cord was also significantly higher in benign PVT [mean 1.9 ± 0.36 and median 2.1[4.4-2.25]] than in malignant PVT [mean 1.6 ± 0.3 and median 1.47[1-2.23] [$p < 0.001$]. ADC ratio [PVT/cord], the AUC was 0.60 [$p = 0.453$], with a cutoff value of ≤0.8. The sensitivity was 88%, specificity 62%, PPV 81.3%, and NPV 73.2%. However, the low AUC suggests limited diagnostic utility of the ADC ratio in distinguishing malignant from benign PVT. In line with our findings, **Mohakud *et al.*** [19] exhibited that the AUC for lesion to spinal cord ratio [LSR] on DWI to differentiate benign and malignant lesions is 0. 810 [95% CI, 0. 584–1. 000]. The AUC for lesion to spinal cord ADC ratio [LSAR] to differentiate benign and malignant lesions is 0. 774 [95% CI, 0. 520–1. 000].

In our study, a significant association between arterial enhancement and ADC values was observed. The mean ADC in PVT was lower in the enhanced group [1.22 ± 0.14] compared to the non-enhanced group [1.3 ± 0.16, $p < 0.001$]. Similarly, the ADC ratio [PVT/cord] was lower in the enhanced group [1.6 ± 0.3] than in the non-enhanced group [1.8 ± 0.4, p = 0.01]. Median values followed the same trend, indicating a significant correlation between arterial enhancement and ADC parameters.

The main limitation in our study were: This study was conducted at a single center, which may lead to different findings compared to multicenter studies. The cohort was restricted to patients with cirrhotic liver disease or HCC, which may not fully represent the broader spectrum of patients with PVT. The absence of additional imaging techniques, such as dynamic contrast-enhanced MRI, may have limited the comprehensive evaluation of vascular thrombotic features.

In conclusion, when combined with mean ADC values, DW MRI is a valuable noninvasive imaging method that is highly effective at characterizing tissue and can be used to distinguish between benign and malignant PVT. A cut off value of [$\leq 1.2 \times 10^{-3}$ mm²/sec] for mean ADC of PVT was reached and proven to be highly diagnostic of malignancy. A cut off value of [≤0.8] for mean ratio ADC PVT/cord was reached and proven to be limited diagnostic and less specific of malignancy. The arterial enhancement and the ADC value of PVT are strongly correlated. Additionally, there is a substantial correlation between arterial enhancement and the ADC ratio PVT/cord. Similar to HCC, malignant PVT exhibits arterial enhancement, a low ADC PVT value, and a low ADC ratio of PVT to cord. DWI is an important for distinguishing between benign and malignant PVT. This correlation between diffusion characteristics and enhancement patterns may significantly aid in noninvasive clinical decision-making, potentially reducing the need for biopsy or invasive procedures in differentiating malignant from benign PVT.

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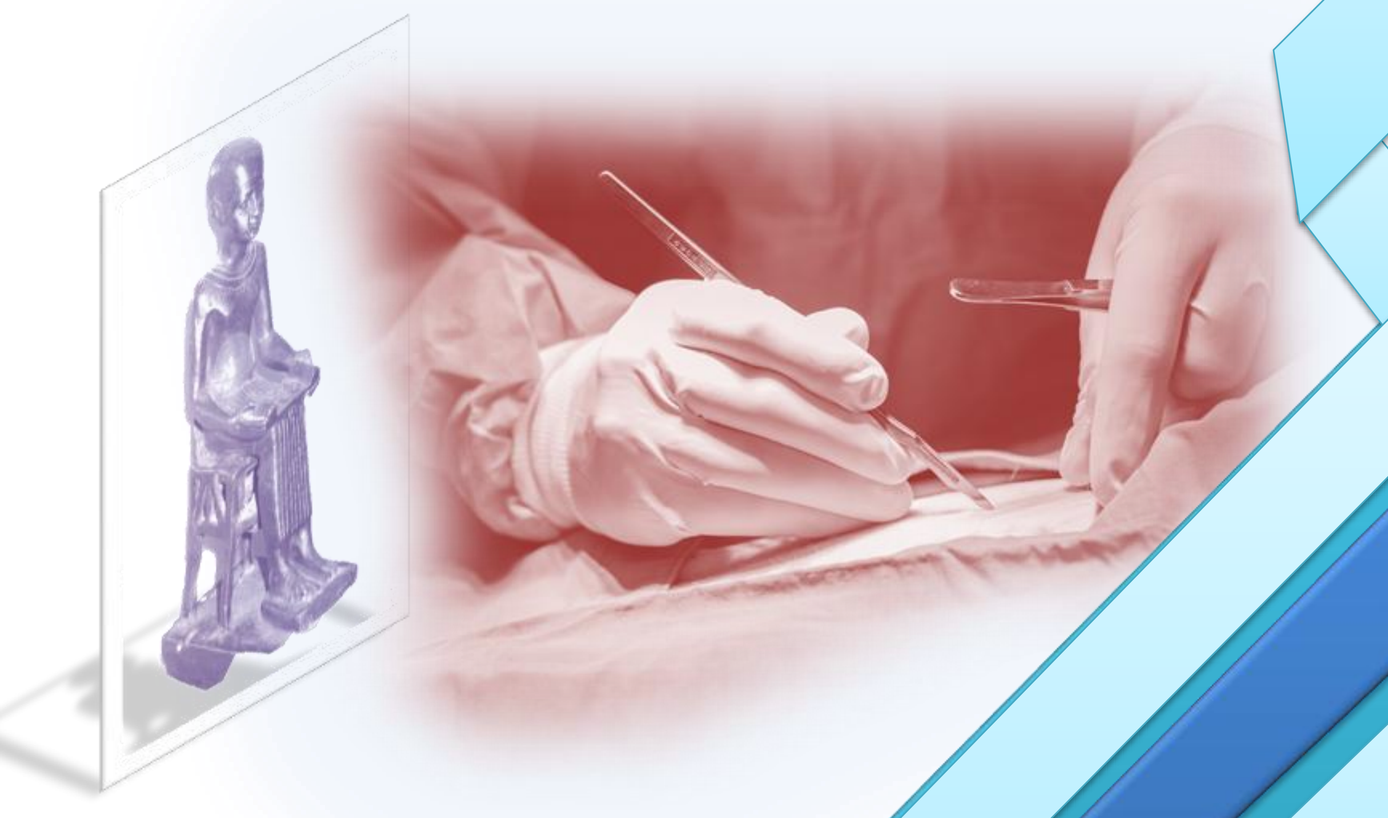

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