

# Role of Triphasic CT in Evaluation of Causes and Hemodynamics of Transient Hepatic Attenuation Difference

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**Abstract:**

**Background:** THAD is important sign of underlying liver disorder.

**Purpose:** To evaluate role of triphasic CT in detecting causes and hemodynamics of transient hepatic attenuation difference that is important sign of an underlying liver disorder. **Patients and**

**methods:** We retrospectively evaluated 40 patients with THAD, their age ranges were from 25-75 years old. The study was done over a period of 24 months (Jan 2018–Dec 2019), for each patient triphasic CT liver examination was performed, during the arterial, portal and delayed phases. We assessed attenuation of THAD area in different phases, morphology, relation to focal lesions and etiopathogenesis of THAD. **Results:** 27/40 patients with THAD presented with hyperdensity in the arterial phase and become isodense to the normal liver parenchyma in other phases, 3/40 patients with THAD presented with hyperdensity in the arterial phase, persist in the porto-venous phase and become isodense in the delayed phase. Different morphology of THAD was detected as follow; sectorial pattern (32), polymorphous pattern (5), diffuse (2)

and multi-segmental THADs (1). 28/40 patients with THAD are related to focal lesion and 12/40 patients with THAD unrelated to focal lesion. We detected etiopathogenesis of THAD as follow; 35/40 patients with THAD was due to portal hypoperfusion, 4/40 was due to primary increase arterial blood inflow and 1/40 was cryptogenic. **Conclusion:** THADs are benign entities associated to focal lesions or other diseases of the liver, radiologists should be familiar with it to avoid false-positive diagnosis of pseudo lesions and not to overestimate the extent of the disease.

**Key words:** THAD, APS, Sectorial



**Abbreviations:**

THAD: transient hepatic attenuation difference

CT: computed tomography

HAP: hepatic arterial phase

PVP: Porto venous phase

IV: intravenous

RFA: radiofrequency ablation

APS: arterioportal shunt

HCCs: hepatocellular carcinomas

TACE:transarterial chemoembolization

HV:hepatic vein

PV:portal vein

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**Introduction**

The liver has a unique dual blood supply one coming from the hepatic artery (25%) and the second from the portal vein (75%). The arterial and portal venous supplies to the liver are not independent; communications exist between the two systems including trans sinusoidal, transvasal and transplexal routes. [1]

THAD reflects a hemodynamic change in the normal dual blood supply of the liver in area of surrounding lesion. The nature of THAD is perfusion differences due to redistribution of arterial blood flow among segments, sub-segments and lobes of the liver. [2]

THAD is a hepatic perfusion disorder and appears as areas of hepatic parenchymal enhancement visible on the hepatic arterial phase of contrast-enhanced imaging and returns to normal or nearly normal on the portal venous phase image. [3]

Among the reasons of THADs is portal vein inflow obstruction, arterio-portal shunting, and steal phenomenon by hypervascular tumors, inflammatory process, and aberrant blood supply due to anatomic variants, hepatic vein outflow obstruction and any other unknown reasons. [4]

They can be classified according to morphology into (lobar multi-segmental, sectorial, polymorphous, and diffuse), etiopathogenesis (arterialization can be secondary or primary) and association with focal lesions (benign or malignant). [5]

Although most cases of hepatic perfusion disorders are asymptomatic, it is important to recognize them to avoid false positive diagnoses (hypervascular tumors) or overestimation of the size of liver tumors, such as HCCs. [6]

**Aim of work:**

The aim of this retrospective study is to evaluate role of triphasic CT in detecting

causes and hemodynamics of transient hepatic attenuation difference that is important sign of an underlying liver disorder.

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## **Patients and methods:**

### **Patients:**

This retrospective study involved 40 cases; 31 males and 9 females. This study was done over a period of 24 months (Jan 2018–Dec 2019). The study was conducted in the radio-diagnosis department of Benha university hospitals as well as private radio-diagnosis centers after approval of ethical committee.

### **Inclusion criteria:**

- Any case underwent triphasic CT demonstrate THAD with or without cirrhotic liver.

### **Exclusion criteria:**

- People undergo only non-contrast CT.
- Contraindication to CT: pregnant women.
- Contraindications to contrast media: Patients allergic to contrast media; Patient with impaired renal function,
- Pediatric age group < 18 years old.

### **Technique of examination:**

### **Patient preparation:**

The patients were routinely fasting for 4-6 hours before the examination.

### **Triphasic CT protocol:**

The CT examinations were performed using different scanners; 16 slice Toshiba Aquilion at radio-diagnosis department of Benha university hospitals, 4 slice Toshiba Asteion and 8 slice Hitachi at private centers.

During the study period, our standard protocol for dynamic CT consisted of a total volume of 100–150 mL of nonionic IV contrast material (300–370 mg I/mL) administered by power injection at a rate of 3 mL/sec, with a scanning delay of 20–30 secs for the HAP and 60–70 secs for the PVP and 3-5 mins for parenchymal phase.

### **Analysis and Interpretation of the CT images:**

Images were sent to the workstation for further image processing and review attenuation pattern of THAD area, morphology, their relation to focal lesions and causes of it was analyzed.

### **Statistical analysis:**

Data management and statistical analysis were done using SPSS vs.25. (IBM, Armonk, New York, United states). Numerical data were summarized as means and standard deviations or medians and

ranges. Categorical data was summarized as numbers and percentages.

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## **Results**

The study group consisted of 40 patients of THADS and the results were analyzed as follows;

Thirty one were males (77 %) and 9 were females (23 %). Age of these patients ranged from 25 to 75 years. The mean and standard deviation value of age were  $58.35 \pm 11.74$ .

Twenty seven of forty (27/40) patients with THAD (92.5%) presented with hyperdensity in the arterial phase and become isodense to the normal liver parenchyma in other phases, 3/40 patients with THAD (7.5%) presented with hyperdensity in the arterial phase, persist in the porto-venous phase (with venous drainage blockage) and become isodense in the delayed phase.

Different morphology of THAD was detected as follow; 32 cases (80%) was sectorial pattern, 5 cases (12.5%) were polymorphous pattern, two cases (5%) were diffuse and one case (2.5%) of multi-segmental THADs.

Thirty two cases of sectorial pattern has different pattern and include 25 cases of wedge shaped area, 5 cases fan shaped area,

1 case had two areas of THADS presenting different morphology; fan and wedge shaped in two lobes and 1 case has two areas of THADS with similar morphology (wedge shaped) (**Fig.1**).

Twenty seven cases (84.4%) of sectorial THADs related to focal hepatic lesions (**Fig. 2**).

Five cases (15.6%) of sectorial THADs not related to tumor; 3 cases (60%) post RFA for HCCs, one case (20%) post TACE for HCC and 1 case (20%) was cryptogenic.

Those related to polymorphous pattern; 3 cases (60%) were due to acute cholecystitis (caused by primary increase arterial). Other two cases (40%) of polymorphus THADs were; one case (20%) post TACE for HCC complicated by infected cystic collection with compression of posterior branch right portal vein, other case (20%) was extrinsic compression.

Those related to diffuse THADs first case (50%) was acute Budd Chiari syndrome and other case (50%) was right sided heart failure.

There was one case (2.5%) of multi-segmental THADs (due to primary increase arterial blood flow with sump effect) cause by hypervascular focal lesion; metastasis.

Twenty eight patients (70%) of THAD their etiology was related to focal lesions, the other 12 cases (30%) their etiology unrelated to focal lesions. Those related to focal lesions; 21 cases (75%) were HCC, 3 cases (10.7%) were cholangiocarcinoma, 1 case (3.6%) was metastasis, 2 cases (7.1%) was hemangioma, 1 case (3.6%) was angiomyolipoma. Those their etiology unrelated to focal lesions; 3 cases (25%) was acute cholecystitis, 5 cases (41.7%) post managed HCCs (3 post RFA and 2 post TACE) 1 case (8.3%) was extrinsic compression (due to perihepatic collection), 1 case (8.3%) Budd Chiari syndrome, 1 case (8.3%) right sided heart failure and 1 case (8.3%) cryptogenic.

In our study we found 35 cases (87.5%) were due to portal hypoperfusion, 4 cases (10%) were primary increased arterial inflow and 1 case (2.5%) of unknown cause.

Causes of portal hypoperfusion are mentioned in Table 1 by their number and percentage of each cause.

Thirteen cases of portal obstruction (86.7%) were due to portal vein thrombosis in HCCs (**Fig. 3**). The other two cases (13.3%), the first one was left lobe cholangiocarcinoma with portal vein either compressed/infiltrated, the other case was post managed HCC with TACE complicated

with cystic collection that compressed the posterior branch of right portal vein.

Those related to hepatic vein occlusion; 1 case (50%) was acute Budd Chiari syndrome and other case (50%) was right sided heart failure.

Those related to combined portal vein obstruction and occlusion of hepatic vein; two cases (66.7%) were left lobe cholangiocarcinoma. One case (33.3%) left lobe infiltrating HCC.

Those related to arterio-portal shunts were 9 cases (69.2%) associated with tumor and 4 cases (30.8%) not related to tumor. The tumoral APS in our study include: 6 cases (66.7%) of HCC, 2 cases (22.2%) of hemangioma and 1 case (11.1%) of fat containing lesion; angiomyolipoma. In our study there were 4 cases (30.8%) of nontumorous APS associated with THADs caused by post intervention procedure and include: 3 cases (75%) post radiofrequency ablation (for right hepatic lobe segment VIII HCC, right hepatic lobe segment VI HCC and left lobe segment III HCC in different patients) and 1 case (25%) post TACE (for right hepatic lobe HCC segment VII).

Those related to combined APS and PV obstruction was multicentric HCCs case presented with two different areas of

THADs; one at the right hepatic lobe segment VI caused by right portal vein thrombosis other at the left lobe segment III caused by APS.

Those related to extrinsic compression was by perihepatic collection with scalloping hepatic surface in pancreatic head cancer patient.

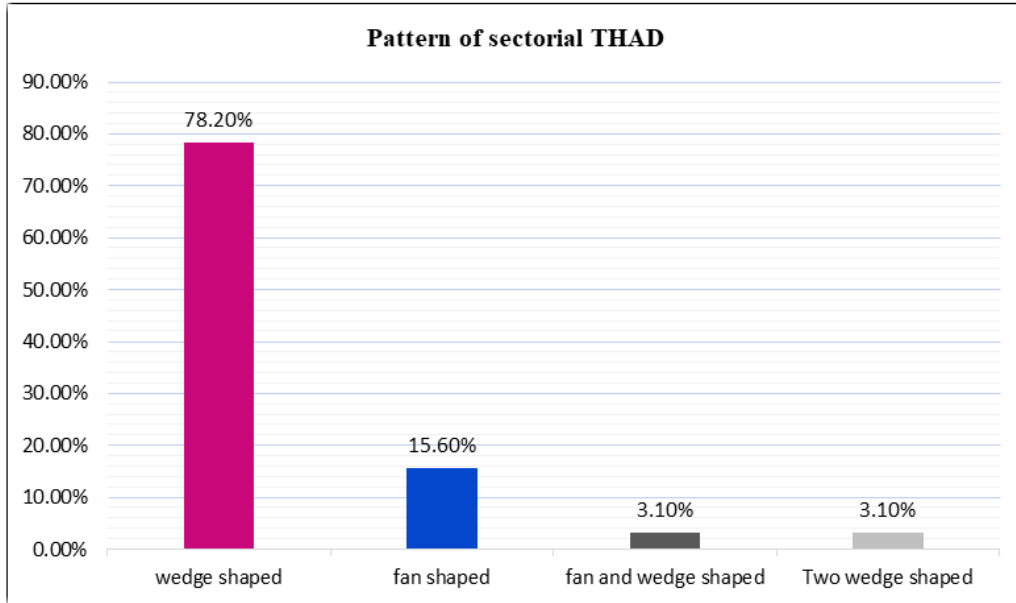
In our study there were 4 cases (10%) of primary increase in arterial blood flow, 3 cases (75%) of them were local inflammatory changes; acute cholecystitis. The other case (25%) was due to metastasis in case of obstructive jaundice.

There was one case (2.5%) of subcapsular small area of THAD with unexplained pathology.

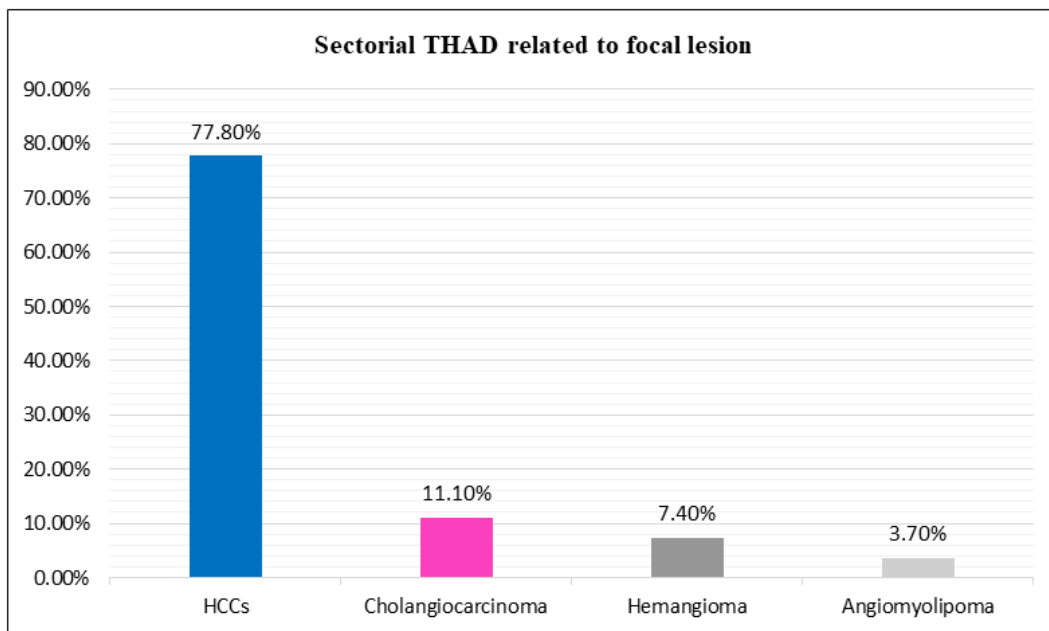
**Table (1): Causes of portal hypoperfusion of THAD in the studied group**

<b>Causes of Portal <u>hypoperfusion</u></b>	<b>N= 35</b>	<b>Percentage</b>
Portal vein obstruction	15	42.9 %
Hepatic vein occlusion	2	5.7 %
Combined cause (PV + HV obstruction)	3	8.6 %
Flow diversion; <u>arterio-portal shunt</u>	13	37.1 %
Extrinsic compression	1	2.9 %
Combined APS and PV obstruction	1	2.9 %

**Fig. (1):** Chart representing percentile for pattern of sectorial THAD in the studied group.

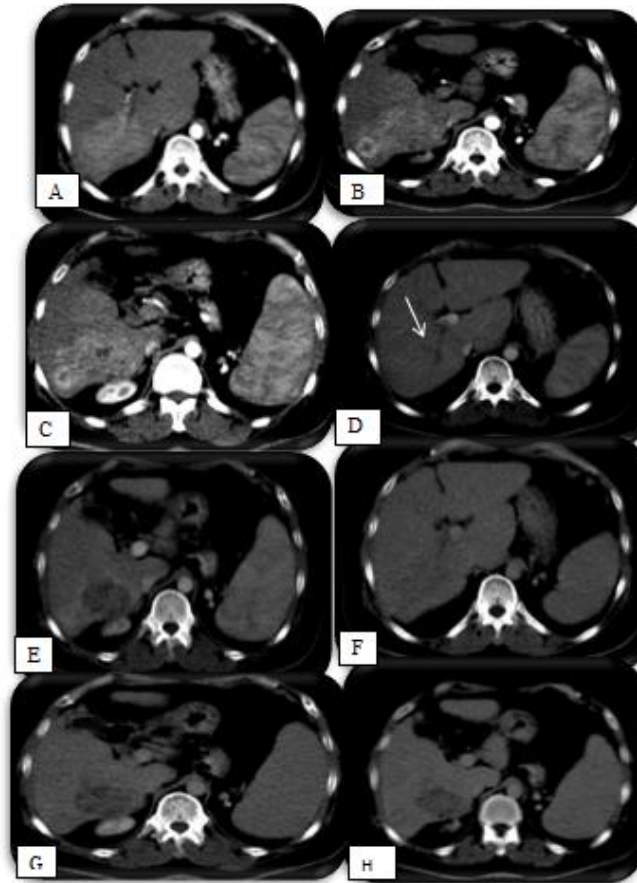


**Fig. (2):** Chart representing percentile for sectorial THAD related to focal hepatic lesions in the studied group.





**Fig. (3)**



Triphasic CT shows two right hepatic lobe segment VI focal lesions (HCCs) are noted, the nearby posterior branch of the right portal vein is infiltrated by the mass and extending to bifurcation of the right portal vein (D). The lesion is surrounded with sectorial area of THAD that appears hyperintense in the arterial phase (A-C) with isodense to parenchyma in the portovenous and delayed phases (D-H).

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## Discussion

With recent advances in diagnostic imaging techniques, hepatic incidentalomas is increasing in both the oncology and non-oncology patient population that in the past remained undiscovered. [7]

In our study, the etiology of 40 cases of THADS have different morphology, 32/40 cases (80%) was sectorial pattern with at

least one straight border which is higher than results obtained by [8] who mentioned that sectorial THADs represent (60.60%) in his study and also higher than [9] that detected 23% sectorial THADs in his study.

In our study 25 cases (78.2%) were wedge shaped sectorial THADs which is higher than result obtained by [9] that revealed 10%

wedge shaped sectorial THADs in his study. Also we detected 5 cases (15.6%) fan shaped sectorial THADs which is lower than result obtained by [9] that mentioned 87% fan shaped sectorial THADs in his study. Also we detected 1 case (3.1%) had two areas of THADs presenting different morphology; fan and wedge shaped and 1 case (3.1%) has two areas of THADs with similar morphology (wedge shaped).

In our study 27 cases (84.4%) of sectorial THADs related to focal hepatic lesions; 21 (77.8%) HCC, 2 (7.4%) hemangioma, 3 (11.1%) cholangiocarcinoma, and 1 (3.7%) fat containing lesion) which is lower than result obtained by [9] that showed 90% of sectorial THADs related to focal lesions; 52% metastasis, 15% HCC, 3.5% cholangiocarcinoma, 15% hemangioma, 11% abscess, 3.5% focal nodular hyperplasia.

5 cases (15.6%) of sectorial THADs not related to tumor ( 1 case (20%) was cryptogenic, 3 cases (60%) post RFA for HCCs and one case (20%) post TACE for HCC) which is higher than result obtained by [9] that revealed 10% of sectorial THADs unrelated to focal lesions with different atiology from our study (67% arteriportal shunt and 33% traumatic).

In our study there were 5 cases (12.5%) of THADs of polymorphous pattern which is lower than results obtained by [8] that revealed 14.14% of THADs of polymorphous pattern (12.5%) in his study.

In our study 3 cases (60%) of polymorphous THADs were due to acute cholecystitis (caused by primary increase arterial) which is lower than result obtained by [10] that revealed 48% appearance of THADs related to acute cholecystitis curvilinear or nodular attenuation (polymorphous) adjacent to the gallbladder fossa in his study.

Other two cases (40%) of polymorphous THADs in our study were; one case (20%) post TACE for HCC complicated by infected cystic collection with compression of posterior branch right portal vein, other case (20%) was due to extrinsic compression by perihepatic collection.

In our study there were two cases (5%) of diffuse THAD which is lower than result obtained by [8] that mentioned 10.10% of THADs of diffuse pattern in his study. First case (50%) was acute Budd Chiari syndrome and other case (50%) was right sided heart failure.

There was one case (2.5%) of multi-segmental THADs (due to primary increase arterial blood flow with sump effect) which

is lower than result obtained by [8] that revealed 26.7% of multi-segmental THADs due to primary increase arterial blood flow (sump effect). The cause in our study was hypervascular metastasis for a case of obstructive jaundice in contrast to [8] that mentioned that all lesions associated with sump effect were always benign

Transient hepatic attenuation differences may have a wide range of causes, with an arterialization phenomenon being primary increased arterial flow without portal hypoperfusion or secondary (increased arterial supply caused by decreased portal inflow or by mixing of arterial and portal blood due to shunting). [12]

In our study 28/40 cases of THADs (70%) their etiology related to focal lesions which is higher than results obtained by another study that showed that 14.3% of THADs were related to focal lesions [11]. This result revealed that 64% of THADs were related to focal lesions. This result is higher than what was revealed before [12]. We detected 21 cases (75%) HCC, 3 cases (10.7%) cholangiocarcinoma, 1cases (3.6%) metastasis, 2 cases (7.1%) hemangioma , 1 case (3.6%) angiomyolipoma while it was detected that 10% HCC, 4% metastasis, 44% hemangioma and 42% hepatic abscess. THADs were not visible in

cholangiocarcinoma in a study [11]. It was reported that 50% metastasis, 39% HCC, 7% abscess and 4% hemangioma. THADs were not visible in cholangiocarcinoma [12].

In our study 12/40 cases (30%) their etiology were unrelated to focal lesions which is lower than results obtained by before that reported that 36% of THADs were unrelated to focal lesions [12] and also lower than another study [13] that reported 33% of THADs with unrelated to focal lesions. Our causes were 3 cases (25%) acute cholecystitis, 5 cases (41.7%) post managed HCCs (3 post RFA and 2 post TACE) 1 case (8.3%) extrinsic compression (due to perihepatic collection), 1 case (8.3%) Budd Chiari syndrome, 1 case (8.3%) right sided heart failure and 1 case (8.3%) cryptogenic. A study reported 31% portal vein thrombosis, 6% hepatic vein thrombosis, 31% post managed lesion (arterio-portal fistula), 13% acute cholecystitis and 19% cryptogenic [12]. Another study reported 33% portal branch thrombosis, 11% trauma (post biopsy), 11% arterioportal shunt, 6% partial Budd Chiari syndrome, 22% inflammation from surrounding organs and 17% extrinsic compression by ribs, 32% diffuse Budd Chiari syndrome, 12% right sided heart failure, 16% portal trunk

thrombosis, 28% cirrhosis and 12% biliary obstruction [13].

In our study we found 35 cases (87.5%) of Portal hypoperfusion (secondary increased arterial supply), 4 cases (10%) primary increased arterial inflow and 1 case (2.5%) of unknown cause.

In our study portal hypoperfusion was the most common cause of THADs and similar to a previous study which showed that portal vein obstruction (due to thrombosis or compression by malignant mass) is the most common cause to portal hypoperfusion [12, 14].

In our study 15 cases (42.9%) of portal hypoperfusion was portal vein obstruction, 13 cases of them (86.7%) was due to portal vein thrombosis in HCCs. The other two cases (13.3%), the portal vein either compressed/infiltrated by lesions; the first one was associated with left lobe cholangiocarcinoma, the other case was post managed HCC with TACE complicated with cystic collection.

In our study there were two cases (5.7 %) of hepatic vein occlusion, 1 case (50%) was acute Budd Chiari syndrome which is higher than result those obtained before [15] in which it was mentioned that 30% of acute Budd Chiari were due to hepatic vein

occlusion. The other cases (50%) were right sided heart failure.

In our study there were three cases (8.6 %) of combined portal vein obstruction and occlusion of hepatic vein; two cases (66.7%) of left lobe cholangiocarcinoma One case (33.3%) left lobe infiltrating HCC

In our study there were 13 cases (37.1 %) of arterio-portal shunts. 9 cases of them (69.2%) associated with tumor and 4 cases (30.8%) not related to tumour.

The tumoural APS in our study include: 6 cases (66.7%) of HCC which is higher than results obtained before where it was reported that detection rate of APSs associated with THADs in of cases of HCC (4.2%) and (63%) respectively [16,17]. We detected 2 cases (22.2%) of hemangioma in our study which is almost near to another study done [16] and higher than results reported by a similar study [11]. We detected 1 case (11.1%) of fat containing lesion; angiomyolipoma.

In our study there were 4 cases (30.8%) of nontumorous APS associated with THADs caused by post intervention procedure which is lower than results obtained others [18]. We reported 3 cases (75%) post radiofrequency ablation (for right hepatic lobe segment VIII HCC, right hepatic lobe

segment VI HCC and left lobe segment III HCC in different patients) and 1 case (25%) post TACE (for right hepatic lobe HCC segment VII) which is lower than results obtained before [19].

In our study there was one case (2.9%) of extrinsic compression which is lower than results obtained by others [13] that mentioned the detection rate of THADs 17% due to extrinsic compression. In our study the cause was perihepatic collection with scalloping hepatic surface in pancreatic head cancer patient while in that previous study [13] the cause was compression by ribs.

In our study there was one case (2.9%) of combined APS and PV obstruction due to portal vein thrombosis in multicentric HCCs case with two different areas of THADs; one at the right hepatic lobe segment VI caused by right portal vein thrombosis other at the left lobe segment III caused by APS.

In our study there were 4 cases (10%) of primary increase in arterial blood flow, 3 cases (75%) of them acute cholecystitis which is higher than results obtained before [10] where it was reported that 71% of THADs were associated with acute cholecystitis. The other case (25%) was metastasis in case of obstructive jaundice.

There was one case (2.5%) of subcapsular small area of THAD with unexplained pathology. This was lower than the results obtained before [12] that mentioned the detection rate of cryptogenic THADs was 19%.

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## **Conclusion:**

We conclude that THADs are benign entities associated to focal lesions (benign or malignant) or other diseases of the liver, characterized by hepatic hemodynamic changes. It is very important for radiologists to be familiar with the various appearances of THADs in order to determine the etiopathogenesis of this lesion, avoid false-positive diagnosis of pseudo lesions and not to overestimate the extent of the disease. If there is no underlying cause should lead to clinical attention and regular follow-ups, if necessary to exclude hidden nodule.

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