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Transient hepatic attenuation difference (THAD) in biliary duct disease

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Abstract

Background: THADs are associated with a wide spectrum of hepato-biliary pathologies. The aim of this paper is to find out the role of THADs in the imaging assessment of biliary diseases. We performed a retrospective study to establish the frequency of arterial phenomena in patients with specific biliary diseases.
Methods: Out of 1833 patients who underwent upper abdomen biphasic CT (2003–2007), we selected those with the following diagnoses: biliary duct dilation due to extrahepatic obstruction (20 patients-group A), intrahepatic-hilar cholangiocellular-carcinoma (19 patients-group B), and cholangitis (14 patients-group C). THAD presence/pattern was assessed for each group. Patients without any demonstrable clinical/imaging signs of liver/biliary pathology (1124) were the control group.
Results: THADs were observed in 36/53 (67.9%) and 20/1124 (1.78%) in study and control groups respectively, with significant association between each diagnostic group and THAD patterns ($P < 0.0001$). Eleven out of 20 (55%) group A patients showed peribiliary-THAD around dilated biliary tracts; 15/19 (79%) group B patients demonstrated portal involvement and sectorial-THAD; 10/14 (71%) group C patients revealed polymorphous-THAD all along inflamed biliary duct.
Conclusions: However, THADs are complex phenomena, their evaluation can be an additional diagnostic tool in patients with a biliary pathology. Diffuse peribiliary, sectorial, and polymorphous-THADs show a good rate of correlation ($P < 0.0001$) with biliary duct ectasia, portal infiltration, and cholangitis, respectively.

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Key words: Biliary pathology—Hepatic arterialization—Hepatic perfusion abnormalities—Liver arterial phenomena—Transient hepatic attenuation difference

Transient hepatic attenuation difference(s) (THAD) are areas of healthy liver parenchyma which appear hyperdense only or mainly during the hepatic artery phase of spiral computed tomography (CT) [1, 2]. THADs derive from an increase in arterial blood supply, usually as a compensatory reaction to a decrease in portal flow. Nowadays, THADs are frequently observed as a consequence of the widespread use of dynamic imaging studies [3, 4]. These phenomena have been reported in literature prevalently associated with hepatic pathologies. Few studies have pointed out the relationships between arterial phenomena and biliary pathology [5–7]. Biliary duct diseases have a wide clinical spectrum with frequent challenging diagnoses. Imaging techniques have an important role in the assessment of site and cause of biliary obstruction, evaluation of biliary dilation degree, and detection of nodular lesion(s). To find out the role of THADs in the imaging of biliary diseases, we designed a retrospective study in order to establish the frequency of THADs in patients with specific biliary diseases (dilation, cholangiocarcinoma, and inflammation) and to identify couplings with associated patterns. The understanding of these phenomena could guide the radiologist toward a more accurate diagnosis.

Materials and methods

Patient selection

Images of 1833 patients, who underwent biphasic CT examinations of the upper abdomen at our Radiology Department between October 2003 and June 2007, were

revised on digital archives. All examinations were performed because of clinical indications according to standard procedure and all patients gave their written consent after being informed about possible risks of X-rays and contrast medium injection. Ethical committee approval and patient consent for this retrospective study were not required, as patient privacy was maintained and patient care was not impacted.

Inclusion criteria are as follows.

Group A—Extrahepatic biliary obstruction without signs of inflammation. The main bile duct was considered dilated when ≥7 mm in patients younger than 60 years, ≥9 mm in older patients, and ≥10 mm in patients with previous cholecystectomy, and a cause was detected [8]. The intrahepatic bile ducts were considered dilated when visible.

Group B—Cholangiocarcinoma with or without biliary duct dilation, with or without portal vein involvement (thrombosis, stenosis, or encasement). Diagnosis was always confirmed by pathological examination using a specimen obtained at surgery or during interventional treatment. Hilar tumors were classified according to Bismuth classification by Endoscopic Retrograde Cholangio-Pancreatography (ERCP) or MR cholangiography [9].

Group C—Cholangitis without biliary vessels dilation. Diagnosis was made according to the following clinical findings: symptoms and signs of infection (fever, leukocytosis), pain in the right upper abdominal quadrant, jaundice, serum bilirubin ≥2.5 mg/dL, high serum alkaline phosphatase, and resolution after an antimicrobial course [10]. If available, bile and/or blood cultures were considered as well. These patients underwent a CT examination within one week from the onset of symptoms. To obtain a clear correlation between THADs and biliary pathologic background, patients affected by mixed pathology (e.g., intra/extrahepatic biliary dilation and cholangitis) were not included in the study group. Age, gender, and diagnosis were recorded for every patient. Diagnoses were verified by consulting the patient, the referring clinicians, and/or the clinical folder. Patients without any demonstrable clinical signs, laboratory findings, or imaging involvement of the liver or biliary tree were considered the control group.

Study and control groups

Out of 1833 patients, 53 (79 CT examinations), 30 men and 23 women, age 42–89 years, mean 66 (13.7 SD) years, met the inclusion criteria and were the study group. The latter, demonstrated extrahepatic biliary obstruction in 20 (group A), cholangiocarcinoma in 19 (group B), and cholangitis in 14 (group C). Ten patients were excluded because they were affected by mixed pathologies such as biliary duct dilation and clinical signs of inflammation, 3 out of these with mass and portal involvement as well. Those without any demonstrable clinical signs, laboratory findings, or imaging involvement of the liver or biliary tree (1124 patients) were the control group, 526 men and 598 women, age 22–88 years, mean 62 (14.3 SD) years. The remaining 646 patients were excluded because they were affected by hepatic pathologies (diffuse hepatopathies, 177 patients; moderate to severe steatosis (≥30%) diagnosed by visual grading criterion, 20 patients [11]; neoplastic lesions, 155 patients; benign lesions, 294 patients).

Imaging acquisition

CT examinations were performed using a single-row scanner (Somatom Plus, Siemens, Erlangen, Germany). Spiral scans were obtained cephalocaudally with section thickness of 5/8 mm and pitch 1.5/1 (reconstruction 4.5/7 mm), matrix 512 × 512, 170 or 220 mAs and 120 kV. The entire liver parenchyma was scanned within a single breath hold of about 20 s. Three acquisitions were obtained: one before (unenhanced scan) and two after intravenous injection of a bolus of 1.5 mL/kg body weight of non-ionic iodinated contrast material (Ultravist 370, Schering, Berlin, Germany) at a rate of 3 mL/s. Contrast medium was administered by an automatic injector (Envision CT, MedRad, Pavia, Italy). Hepatic artery and portal vein phase CT scans started 30 and 75 s, respectively, after the beginning of contrast material injection.

Image evaluation

All the images of the study group were reviewed and reassessed by two radiologists, both experienced in body imaging for more than 15 years and unaware of the diagnosis. Disagreements were resolved by consensus. The presence and pattern of any arterial phenomenon were identified. THADs were classified (Table 1) as previously published [12]. Four different types of THADs were considered:

(1) Sectorial: arterialization areas, secondary to portal hypoperfusion and then following the portal dichotomy, appear with a triangular shape and at least one "straight border" sign (i.e., a clear separation line from the normally attenuating parenchyma) [1, 2, 12].
(2) Lobar: arterialization areas involving all or almost all the segments of a hepatic lobe, usually caused by a primary increase in arterial in-flow due to a large benign lesion (sump effect or siphoning phenomenon), following the arterial distribution.
(3) Polymorphous: arterialization areas that usually do not follow the portal dichotomy and show various shapes, without a straight border sign, in relation to

Table 1. Classification of transient hepatic attenuation differences

Type	Focal lesion	Morphology/pattern	Pathogenenesis
Sectorial	Yes	Wedge or fan-shaped	Secondary
	No	Wedge-shaped	
Lobar	Yes	Lobar multi-segmental not sectorial	Primary
Polymorphous	No	Various shape and size, marginal or central, without a clear straight border sign	Secondary
Diffuse	No	Patchy Central-peripheral Peribiliary	Secondary

Secondary: arterial hyperperfusion secondary to portal hypoperfusion; Primary: primary arterial hyperperfusion

Table 2. Demographic characteristics, pathology, and related transient hepatic attenuation differences of the 3 patient groups

Group	Gender (M/F)	Age mean (SD)	Pathology n (portal involvement)	Peribiliary THAD	Sectorial THAD	Polymorphous THAD	No THAD
A	(9/11)	64 (13)	Dilation 20 (0)	9	–	Scattered 2	9
B	(12/7)	67 (15)	Mass 4 (3)	–	2 Central-peripheral 1	–	1
			Dilation 7 (5)	1	5	–	1
			Mass + dilation 8 (8)	–	6	–	2
C	(7/7)	66 (15)	Cholangitis 14 (0)	–	–	Scattered 5 confluent 5	4

Group A: biliary tree dilation without clinical sign of inflammation. Mirizzi's syndrome (2), ampullary carcinoma (2), choledocal stones (8), pancreatic head carcinoma (8)
Group B: cholangiocarcinoma (classified according to Bismuth). Intrahepatic (7), Klatskin IIIB (4), Klatskin IV (8)
Group C: cholangitis without biliary duct dilation. After interventional procedure (4), in bile-digestive shunt (4), in gallstones (2), in duodenal diverticulum (1), unknown cause (3)
Central peripheral pattern should be considered generalized form of sectorial pattern

the cause, such as aberrant blood supply, inflammation, extrinsic compression, or percutaneous treatment outcome.

(4) Diffuse: arterialization areas involving the entire hepatic parenchyma (or at least large areas of it). They show three different patterns on the basis of the portal obstruction site: patchy (post-sinusoidal blockade with trans-sinusoidal system opening), central peripheral (pre-intra-sinusoidal obstacle with peribiliary plexus opening), or peribiliary (peribiliary plexus blockade).

In group B patients, detectable focal lesions, biliary ectasia and portal involvement (thrombosis, stenosis, or encasement) were noted. Stenosis was defined as restricted portal lumen, encasement as lumen vanishing into the mass and/or within dilated biliary duct during vein phase acquisition [13].

Statistical analysis

All data were analyzed with the Stats Direct statistical software, version 2.5.8 (Stats Direct Ltd. Bonville Chase Altrincham CHESHIRE WA14 4QA UK). Continuous data are presented as mean (SD); discrete variables (categorical data) are given as percentages. Discrete variables were compared by Chi-Square test (Yates-corrected for 2×2 contingency tables), two-tailed Fisher's Exact Test, or Fisher–Freeman–Halton Exact Test, where appropriate. A P-value <0.05 was considered statistically significant.

Results

A statistically significant difference in the incidence ($P < 0.0001$) of the arterial phenomena was observed between patients of the study (67.9%) and the control group (1.78%). Demographic features, diagnosis of patients, and THAD characteristics are shown in Table 2.

Group A—Dilation

Out of 20 patients 11 (55%) had a THAD, which appeared in 9 cases with a diffuse peribiliary linear railway-like pattern (Fig. 1), while 2 patients showed a polymorphous, more localized pattern, with globular enhancement (Table 1). Four patients, all with choledocal stones (2 peribiliary and 2 polymorphous patterns THAD), underwent a further CT examination from 15 days up to 3 months after stone removal, showing the disappearance of the arterial phenomena (Fig. 1).

Group B—Cholangiocarcinoma

Out of the 19 patients 15 (78.9%) showed at least one THAD. Four patients had a focal hepatic mass (21%), 7 biliary dilation (37%), and 8 both a mass and biliary ectasia (42%). The mean mass dimension was 2.1 (1.1 SD) cm, ranging from 0.8 to 4.5 cm. Sectorial pattern was found in 13 patients, all with portal branch stenosis or encasement (Table 2) (Fig. 2). One patient with complete hilum involvement and portal trunk thrombosis showed a diffuse central peripheral pattern; one other

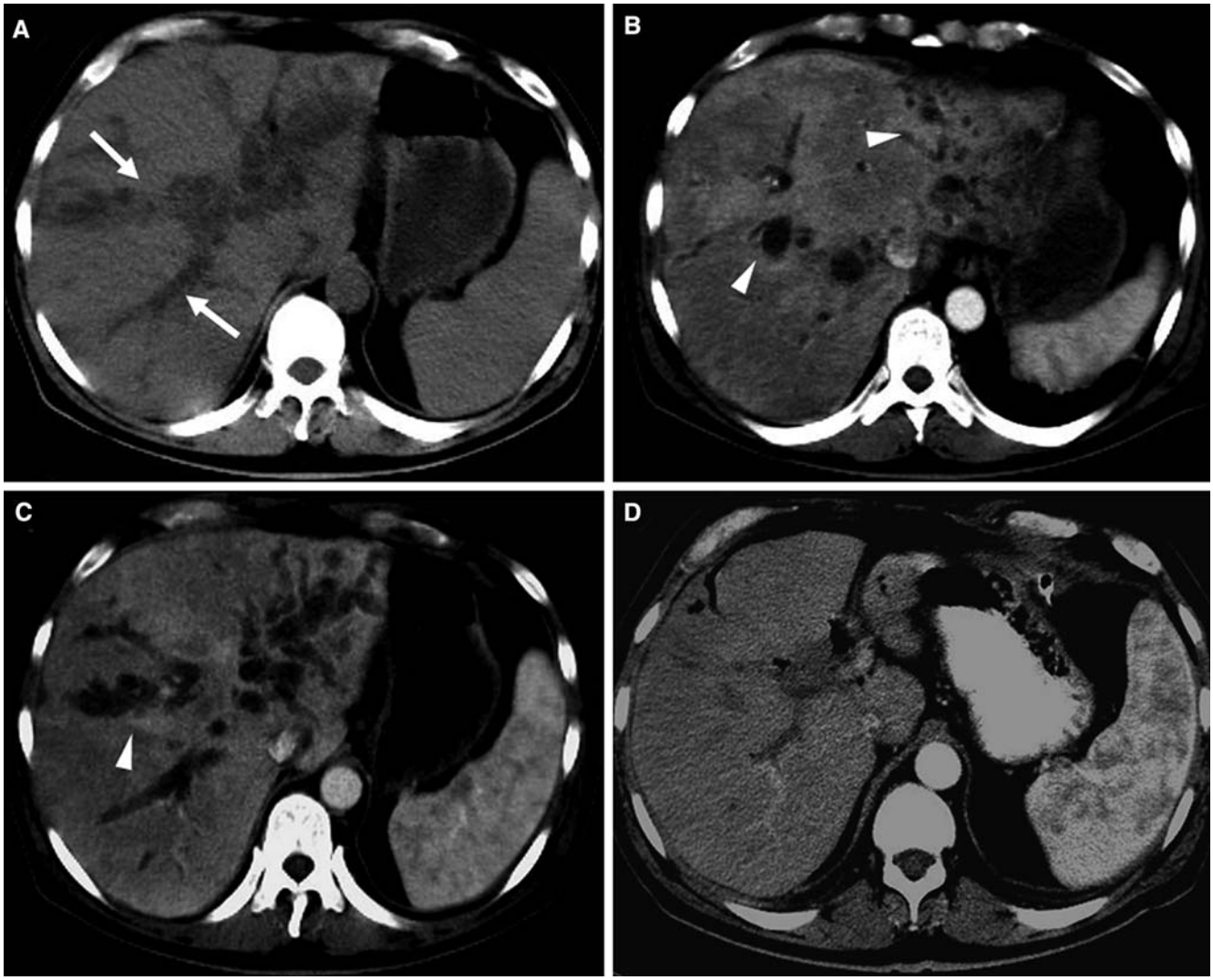

Fig. 1. A 59-year-old man with choledochal stones. (**A**) Unenhanced CT scan shows intrahepatic biliary tree dilation (*arrows*). (**B**), (**C**) CT artery phase scans reveal THAD around the biliary ducts (*arrowheads*). (**D**) CT 2 months after the removal of stones shows no arterial phenomena.

patient had dilated biliary vessels with peribiliary pattern (Table 1). Six patients had more than one CT examination. Two patients (one with a mass and one with biliary tree dilation) at a previous CT, performed 3 and 6 months before, showed only the arterial phenomenon without biliary duct dilation and no detectable mass (Fig. 3). Four patients with a mass and biliary ectasia, who never underwent open surgery (two received a biliary stent), showed a decrease in attenuation and in area of THAD (Fig. 2) at a further CT performed 2–12 months later.

Group C—Cholangitis

Of the 14 patients 10 (71.4%) had a THAD, which appeared as various dimensioned spots, roughly globular and irregularly distributed along biliary vessels (polymorphous pattern), scattered in 5 cases (Fig. 4), and confluent in the other 5 (Fig. 5). No clinical differences were observed between these patients and the other group C ones. Four patients, after clinical resolution of cholangitis, underwent further CT examinations from 15 days up to 3 months, which demonstrated THAD vanishing (Fig. 5).

Statistical analysis demonstrated a significant relationship between groups and THAD patterns ($P < 0.0001$). Post hoc analysis indicated the significant difference between each group vs. another group (A vs. B + C; B vs. A + C; C vs. A + B; $P < 0.0001$).

Discussion

Pathophysiological studies have demonstrated communication between the two hepatic inflows (70% portal vein, 30% hepatic artery) mainly through the sinusoid system and peribiliary plexus. These shunts attempt to

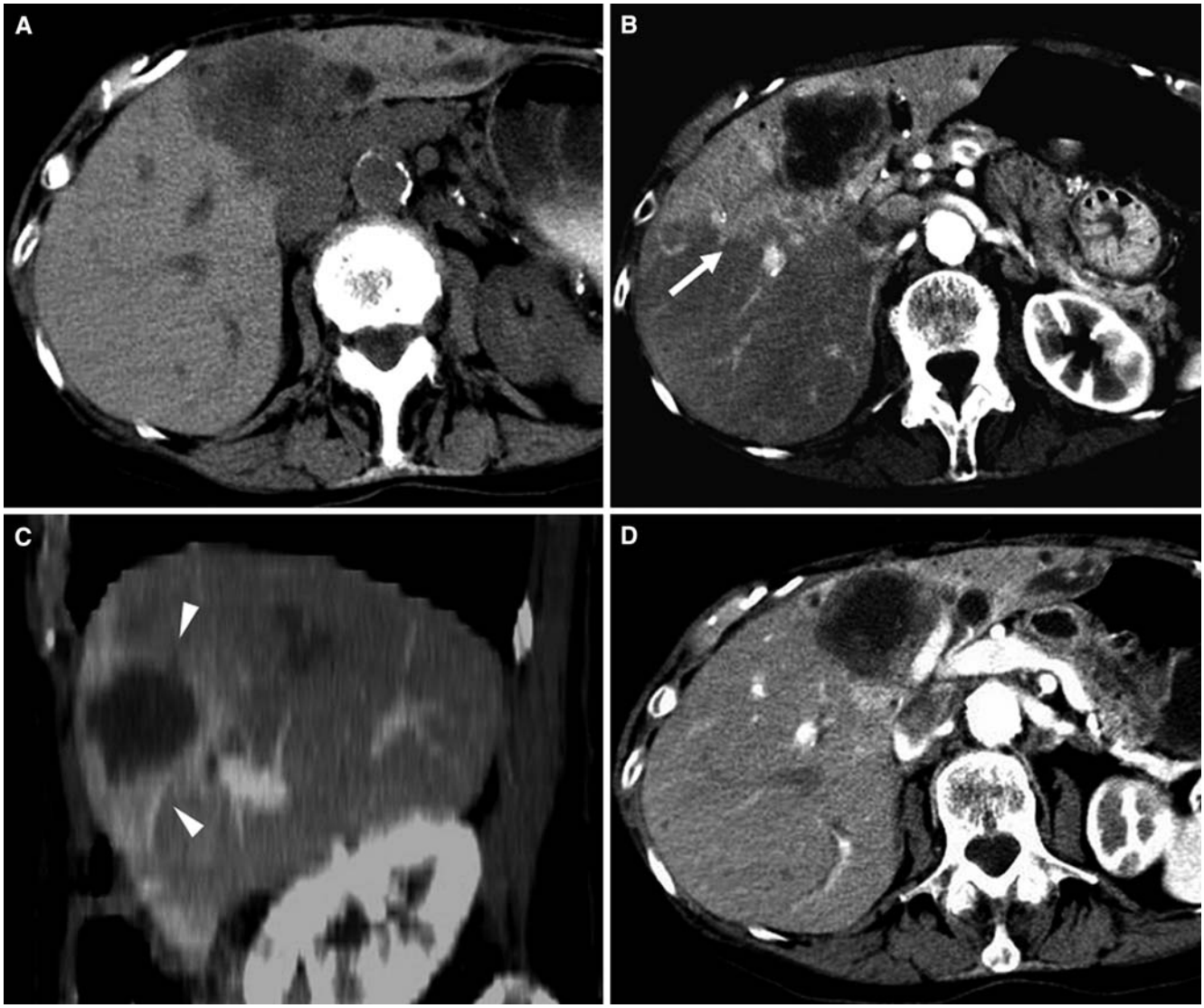

Fig. 2. An 80-year-old women with cholangiocellular carcinoma. (**A**) Unenhanced CT scan reveals a hypodense mass in the left hepatic lobe and biliary tree dilation. (**B**) On CT artery phase a sectorial hyperdense area (*arrow*) surrounding the mass is visible. (**C**) Sagittal plane reconstruction confirms the sectorial shaped THAD (*arrowheads*). (**D**) Two months later, the mass persisting, at artery phase CT, the THAD appears smaller and with a weaker visual intensity.

locally compensate for reduced portal inflow under the action of autonomic nervous system and humoral mediators (primarily adenosine and prostacycline) [2]. Thus, an important increase in arterial inflow can be demonstrated only in the parenchymal area involved by acute portal inflow reduction. The arterial reaction is proportional to the portal lessening; however, even a slight decrement in portal inflow determines an important arterial increment [1, 14]. The THAD is the CT appearance of the described events.

The main shunting system involved in our series is the peribiliary plexus, which surrounds the main bile ducts like a mesh, allowing the passage of a minor, non-quantifiable fraction, of the portal inflow into the sinusoid system, while the main part of portal blood passes directly from the triads to the sinusoids. The peribiliary plexus, lacking muscular walls, collapses when the bile duct dilates. So, functional impairment or failure of the plexus causes a decrease in blood inflow from the portal vein into the sinusoid system which induces a slight arterial compensation due to the only partial blockade of portal inflow [1, 15]. When biliary pressure decreases as a result of drainage, the bile duct becomes narrower with consequent decompression of the peribiliary plexus: portal flow is restored and the arterial phenomena vanish (Fig. 1). However, if stasis persists, the arterial phenomena slowly disappear as well, due to receptor saturation and collateral shunt consolidation [1, 2]. Both clinical experience and the literature indicate that the outcome in case of biliary stasis is atrophy of the involved parenchyma [1, 2, 15].

In our series, we found a greater prevalence of THADs (68%) in patients with biliary diseases than in

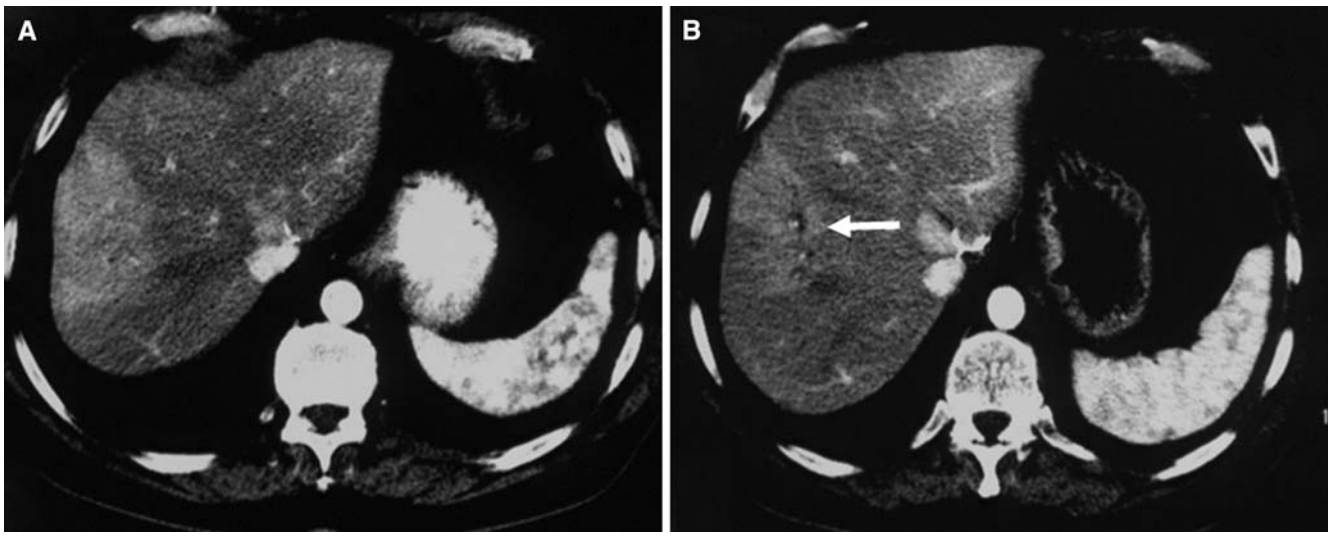

Fig. 3. A 63-year-old man with cholangiocellular carcinoma. (**A**) CT artery phase demonstrates only a sectorial hyperdense area without a clear explanation. (**B**) Three months later, at artery phase CT, a bile ducts dilation can be observed as well (*arrow*).

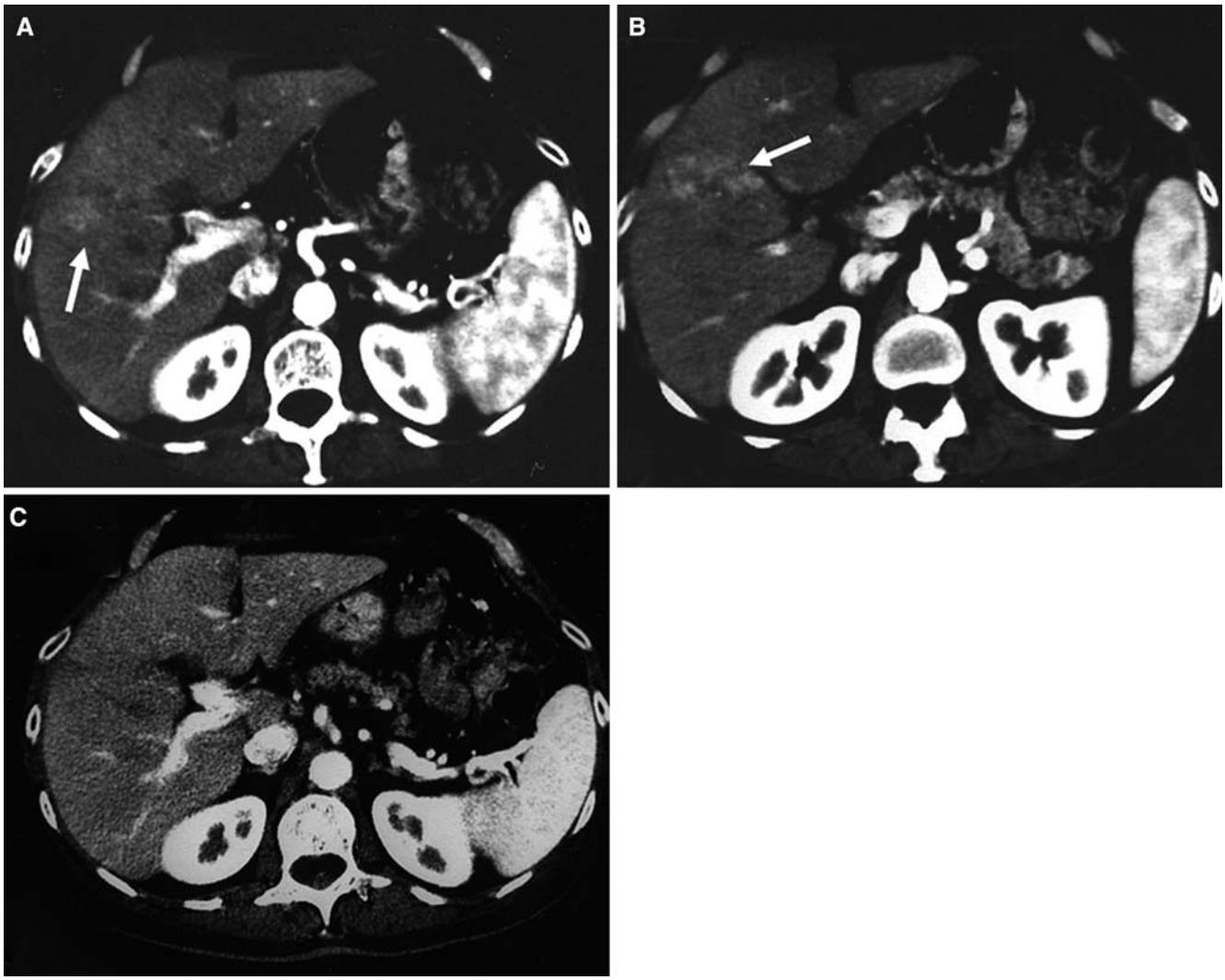

Fig. 4. A 64-year-old man with cholangitis. (**A**), (**B**) CT artery phase shows polymorphous THADs (*arrows*) without dilation of biliary vessels. (**C**) Three months later, after therapy, at artery phase CT, the arterial phenomena disappeared.

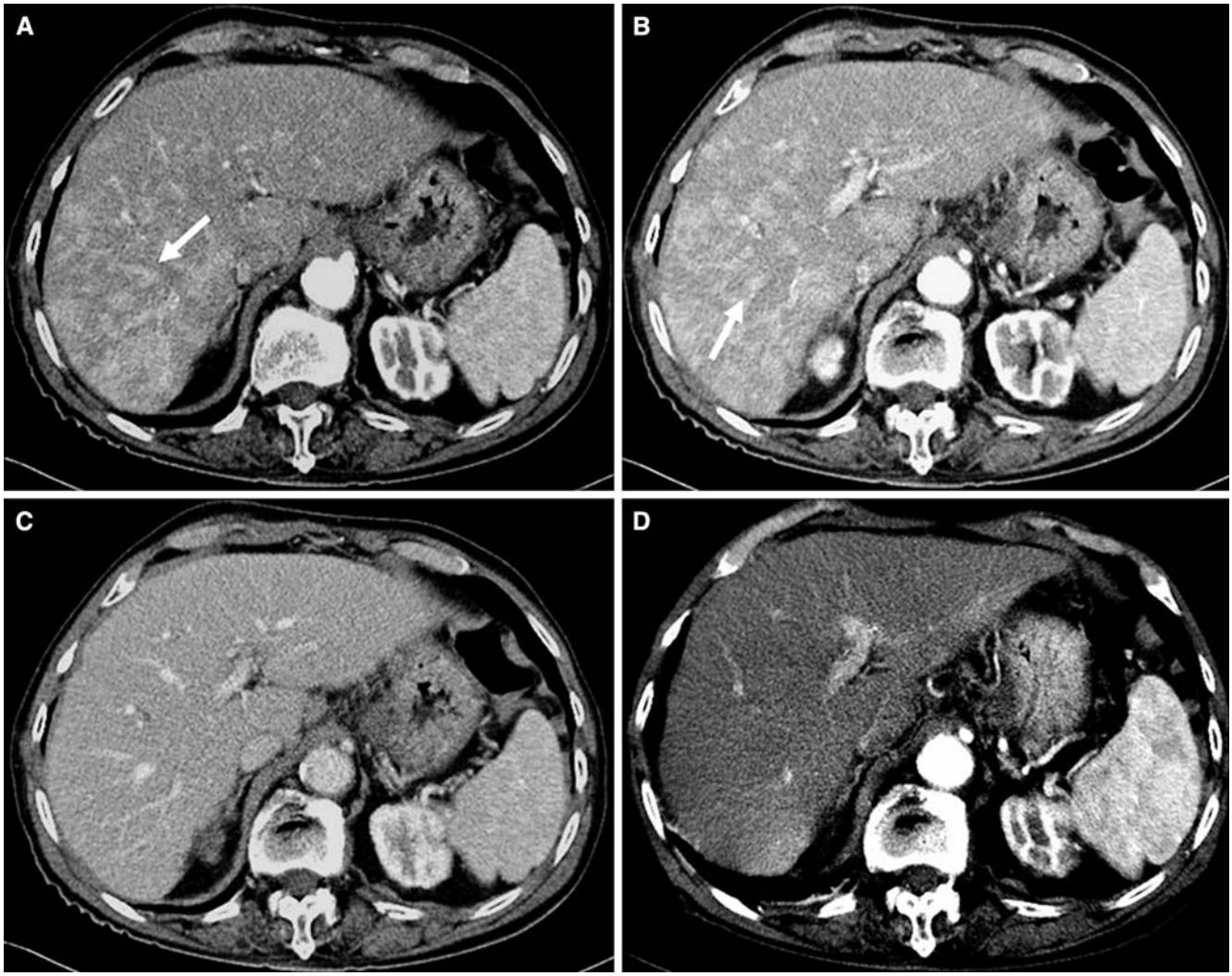

Fig. 5. An 84-year-old man with cholangitis. (**A**), (**B**) CT artery phase demonstrates multiple globular spots around non-dilated biliary tree (*arrows*). (**C**) On portal phase, arterial phenomena vanish. (**D**) Fifteen days later, after therapy, at artery phase CT, the arterial phenomena disappeared.

control subjects (1.8%). When an infiltrative pathology with portal involvement was considered (group B), the incidence reached 79%. These findings indicate that biliary tree diseases cause perfusional abnormalities of liver parenchyma in the early phase on dynamic CT. We also found a significant correlation between each group of alterations and a specific THAD pattern.

We detected a peribiliary pattern in more than half of the patients (55%) with biliary duct dilation (group A) (Fig. 1). This is due to functional impairment of the peribiliary plexus, and when the biliary pressure decreases, the portal inflow is restored and the THAD disappears or lessens (as we observed in the four cases in which control examinations were available) (Fig. 1). This reversibility, and the non-quantifiable amount of portal inflow to the sinusoids passing through the peribiliary plexus, almost certainly with wide inter/intra-individual variability, could explain the inconstancy of THAD in the patients with dilated biliary duct.

The peribiliary plexus plays a main role when the pathological background is a biliary duct inflammation (group C), as well. We observed a polymorphous pattern in 72% of our patients (Fig. 5). The pattern and incidence we detected, agree with what previous investigators found (85%) [10]. However, they suggested that the arterial phenomenon was induced by inflammatory enlargement of the plexus, whereas we believe that THAD patterns derive from impairment of the peribiliary plexus attributable to inflammatory edema. The dissimilarity between THAD pattern observed in dilation and that detected in inflammation (without dilation) of biliary duct is, in our opinion, related to the spread of inflammatory mediators by contiguity with consequently more evident arterial pattern. The wide variability in extension and severity of cholangitis presentation can explain the polymorphous pattern and absence of any arterial phenomenon (4 patients in our series), as well. When inflammation is relieved after successful therapy,

THADs consequently disappear, as demonstrated in other 4 patients in our series (Fig. 5).

The most prevalent THAD pattern in patients with cholangiocarcinoma (group B) is represented by sectorial arterialization i.e., a roughly triangular shaped hyperattenuating area with the apex directed towards the hepatic hilum (Fig. 2). Noteworthy, while peribiliary arterialization is specific of biliary dilation and inflammation, sectorial THADs are not only due to cholangiocarcinoma. Actually, sectorial THADs are the most frequent arterialization phenomena associated with non-nodular and nodular (prevalently malignant masses) hepatic pathologies. As a general rule, arterial phenomena with sectorial morphology are secondary to portal hypoperfusion due to portal branch stenosis, encasement, thrombosis or by flow diversion caused by an arterioportal shunt.

Then, in case of cholangiocarcinoma, if a hepatic mass, with or without biliary duct dilation, is present, sectorial high attenuation THAD is determined by malignant involvement of the portal branches and peribiliary sites [1, 2, 12]. In fact in 8/11 patients of our series with mass and portal involvement we observed a sectorial pattern. However, in 5/7 patients with biliary duct dilation without any detectable mass, but with sign of portal encasement, we found a sectorial shaped THAD as well. We postulate that in these cases the arterial phenomenon is caused by a focal lesion, not detectable for dimensional or contrastographic reasons, but already inducing a portal inflow blockade. So, arterialization may suggest an underlying abnormality and precede the CT detection of the nodular lesion. This seems to be confirmed by the 2 cases with a mass and biliary tree dilation (Fig. 3), in which the first CT scan showed only the sectorial arterial phenomenon. In the only case in which the obstacle determined by cholangiocarcinoma is at the level of the portal axis with thrombosis of both main branches, the observed THAD pattern is a "central-peripheral" type, which can be considered as the generalized equivalent of the sectorial arterialization. We interpreted the unique cholangiocarcinoma presented with mass and dilation associated with a non-sectorial THAD as a non-infiltrative nodule and then as a neoplasm inducing only biliary duct dilation determining a peribiliary pattern. Similarly, we postulate that the lack of major portal infiltration could also clarify the absence of any THAD in 2 of our selected cases [1, 2]. In other 2 patients with portal involvement but without any perceptible THAD, we believe the absence of arterial phenomena a consequence of receptor saturation and collateral shunt consolidation with resulting parenchymal involution. This looks to be validated by the 4 cases of sectorial THADs vanishing or disappearing (Fig. 2), despite the permanence of the cause.

The patients affected by mixed pathology were not included in the study group in order to avoid confusing CT patterns. In fact, in these cases we detected overlapping appearance of THAD's patterns. When peribiliary plexus is impaired due to duct dilation and inflammation, the resultant CT pattern appears mainly as a diffuse arterial background (i.e., great confluent spots). If a mass with portal involvement is also present, the major portal obstruction determines sectorial THAD(s) as well.

In conclusion, although our series is limited, it indicates the existence of three main THAD's patterns which can represent additional diagnostic tools: diffuse linear and polymorphous peribiliary types, exclusively found in patients with biliary dilation and inflammation, respectively; sectorial high attenuation type, typically due to portal involvement and in our study related to cholangiocarcinoma.

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