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Dan Alexandru Arhire et al., THAD or fat sparing? ARGV determining a pseudolesion at the border of the IInd/IIIrd liver segments

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ABSTRACT

The aberrant right gastric vein (ARGV) is a rare anatomical variation. It can be responsible for unexplained hyperdensities in the hepatic parenchyma on CT scans, also known as third inflow effects.

We present two cases sharing similar vascular pattern and slightly different imagistic findings on ultrasound and computer-tomography performed studies.

Both ultrasonographies showed a nodular-geographic hypoechoic area within a hyperechogenic fatty liver. Further CT evaluation showed in both cases a hyperattenuating homogenous area clearly visible on all four phases at the border of the IInd/IIIrd hepatic segment, that enhance especially on the portal venous phase, with no slow-fill, wash-out, central scar or rim-like features. The areas were considered to be focal fat sparing areas in diffuse fatty liver or a perfusion disorder due to the presence of an aberrant right gastric vein.

The aim of this paper is to discuss the embryological aspects which are the groundwork for this vascular anomaly and to correlate the findings with imagistic aspects.

These two ARGV produced pseudolesions, understood as focal fat sparing areas within diffuse fatty livers. These pseudolesions mimic liver tumours, therefore it is important to look for such an aberrant vessel in order to rule out other diagnoses.

Key words: liver hyperdensity, third inflow, vitelline veins, umbilical veins

INTRODUCTION

The aberrant right gastric vein (ARGV) can produce a pseudolesion at the posterior aspects of liver segments II/III. It can be explained either as a transient hepatic attenuation difference (THAD) or as a focal fat sparing area in a diffuse fatty liver.

Several studies reported various frequency values of THAD in groups without demonstrable hepato-biliary pathology, ranging between 1.78% [19], 3.2 % [13], 9.3% [25] and 13.1 % [4][CITATION Col02 \l 1048]. A THAD frequency of 10.9% was also reported in neonates [22]. THAD frequency rises substantially when various hepato-biliary pathologies are associated: 67.9% [19], 70.9% [13].

Focal fat sparing areas in diffuse fatty liver are well documented in literature [24] [21] [15]. They are attributed to third inflow sources, mainly the parabiliary veins, paraumbilical veins and aberrant right gastric veins. The liver parenchyma adjacent to the gallbladder fossa is the most common site for fat sparing. Fat sparing at the posterior aspect of segments II/III of the liver is not a common location and when it shows up it is probably determined by an aberrant gastric vein.

From the radiological point of view, differential diagnosis between haemangioma, adenoma, focal nodular hyperplasia, tumours (primary or secondary) or storage diseases may be taken into account.

The aim of this paper is to discuss the embryological aspects which are the groundwork for this vascular anomaly and to correlate the findings with clinical and imagistic aspects.

MATHERIALS AND METHODS

Two patients underwent a computer-tomography (CT) scan. Pre-contrast, arterial, portal venous and delayed phases were performed using a multi-detector 16-channel CT scanner (Aquilion 16, Toshiba Medical Systems) with a slice thickness of 1.0 mm. Using an automatic power injector, 110 mL of contrast agent (iohexol OMNIPAQUE 350 mgI/ml) was administered at a rate of 2.5 mL/s. By automatic bolus tracking, the arterial phase scan was performed 25 s after the attenuation of the pulmonary trunk (CASE 1) or the subdiaphragmatic aorta (CASE 2) reached a triggering threshold of 100 HU. The portal venous and delayed phase images were obtained 70 seconds and 4 minutes after injection of the contrast agent. Aberrant gastric veins are usually slender, with small diameters, best viewed with the help of selective angiography. In order to improve their detection on standard CT scans we used thinner sections, multiplanar reconstructions (with manipulated axes) and maximum intensity projection (MIP) images.

Both patients underwent also an ultrasound scan, but in different ways. For the first case an initial CT scan was performed, followed by an US. For the second case, the US was initially performed, followed by the CT scan.

RESULTS

Both ultrasonographies showed a nodular-geographic hypoechoic area within a hyperechogenic fatty liver. Doppler US could not assess an ARGV, but did manage to exclude suspicious vascularization of the pseudolesion as well as no portal vessel into the respective area (Fig. 1, Fig. 2).

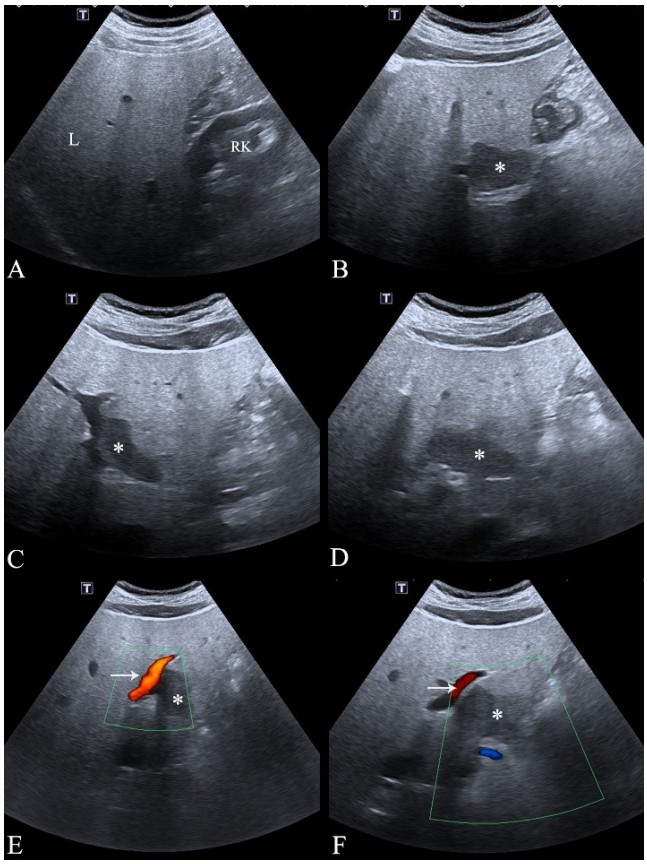

Figure 1. CASE 1. Ultrasound (US) liver images performed after the initial CT scan. A – Hyperechoic liver parenchyma (L) compared to hypoechoic right renal cortex (RK) and decreased posterior attenuation of the liver, both suggesting diffuse hepatic steatosis. B, C, D – different imaging planes showing a hypoechoic fat sparing area *(*)* located within the IIIrd segment. E, F – Doppler images showing no vascularization of the (pseudo)lesion *(*)* and no other vascular communication with the left branch of the portal vein *(arrows)*.

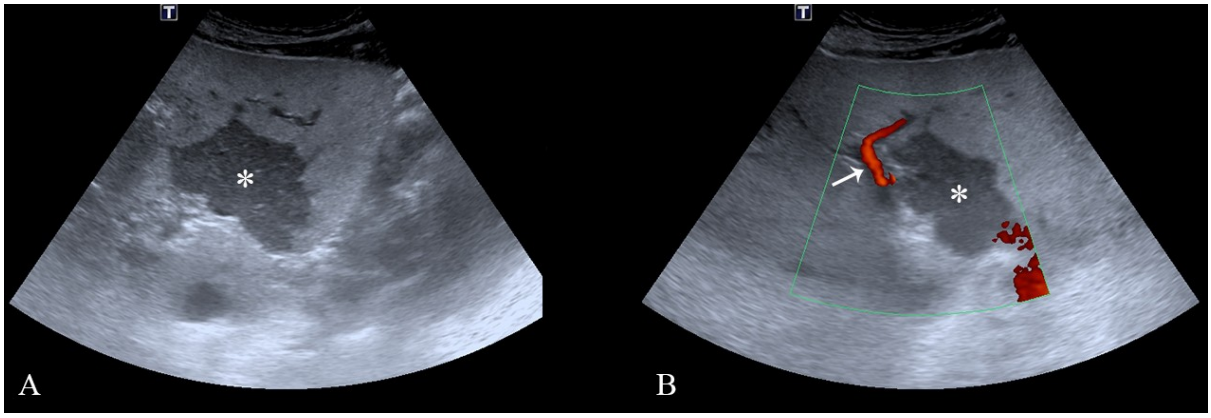

Figure 2. CASE 2. The initial ultrasound images required further CT evaluation. A - hypoechoic fat sparing area *(*)* located within the IInd/IIIrd segments. B – Doppler images showing no vascularization of the (pseudo)lesion *(*)* and no other vessel derived from the left branch of the portal vein *(arrow)*.

CASE 1

A 59 years old male patient addressed for further evaluation of a pulmonary nodule found on chest X-Ray. We performed a HRCT thoracic scan and on the abdominal images caught, we observed on all four phases a hyperdense area at the border of the IInd/IIIrd hepatic segment (Fig. 3, Fig. 4). The area showed increased attenuation on the portal venous phase and was considered to be a focal fat sparing area in diffuse fatty liver or a perfusion disorder due to the presence of an aberrant right gastric vein (Fig. 5).

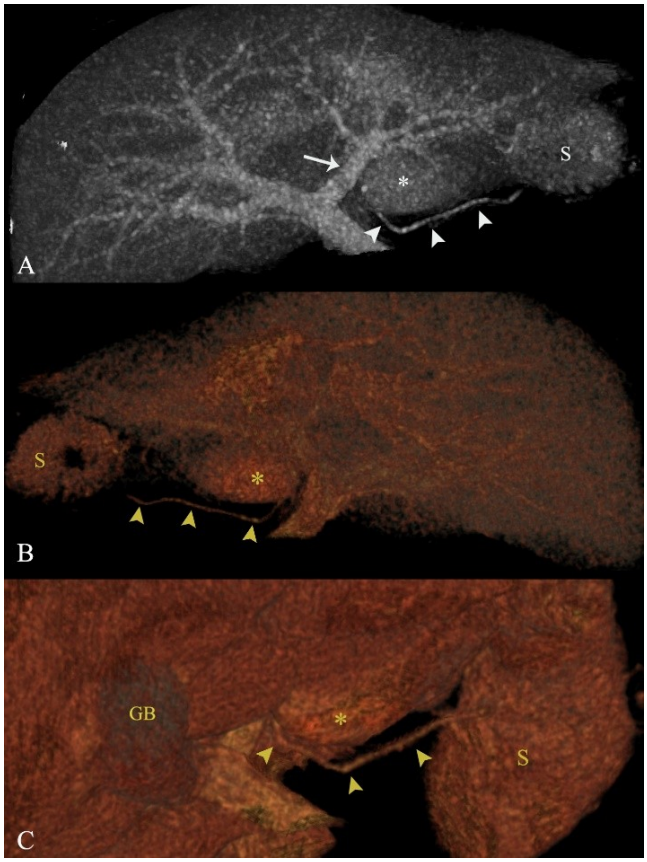

Figure 3. CASE 1. Portal venous phase 3D MIP reconstructed images of the liver. The aberrant right gastric vein (ARGV - *arrowheads*) drains from the lesser curvature of the stomach *(S)* into the IIIrd liver segment, enhancing a lesion at this level *(*)*. The left branch of the portal vein *(arrow)* does not have a distribution into this territory. A – right anterior view. B – left posterior view. C – right inferior view. *(Large portions of the stomach were cut out from the image in order to better highlight the ARGV course; GB – gallbladder).*

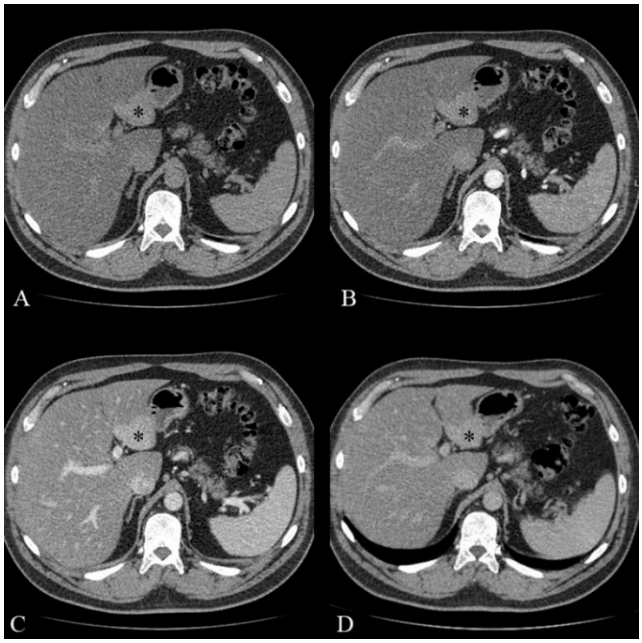

Figure 4. CASE 1. Axial liver images. A – pre-contrast scan (native); B – arterial phase; C – portal venous phase; D – delayed phase. The pseudolesion at the IIIrd segment level *(*)* can be easily seen on all phases.

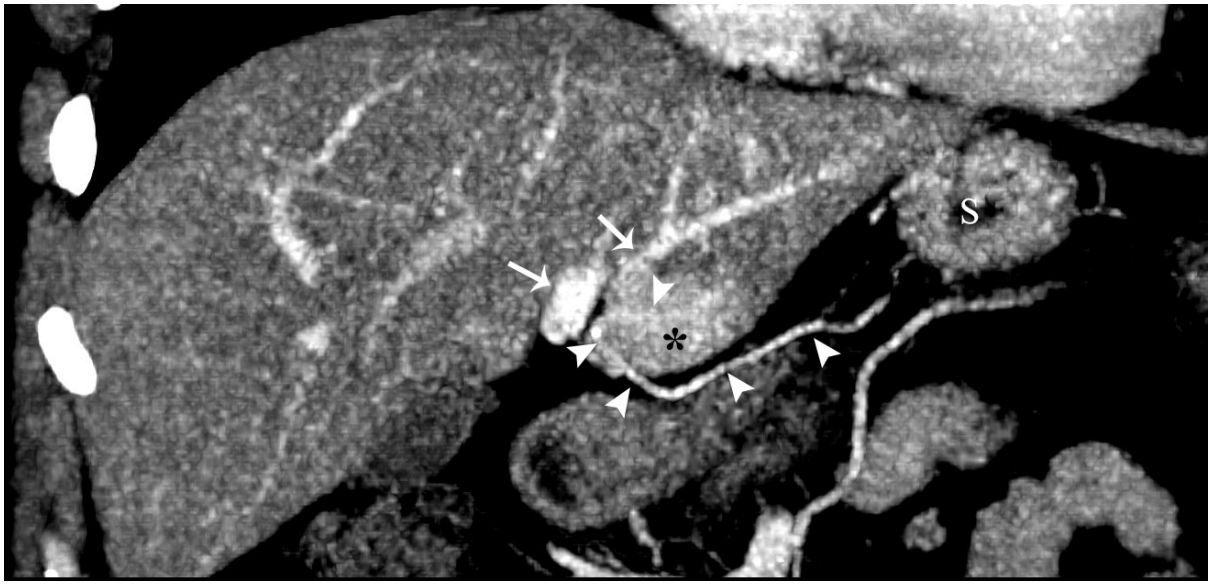

Figure 5. CASE 1. Portal venous phase coronal oblique MIP reconstructed image of the liver. There is an aberrant right gastric vein *(arrowheads)* draining from the lesser curvature of the stomach into a hyperdense pseudolesion *(*)* at the border of segments II/III. Neither a corresponding communication with the left branch of the portal vein *(arrows)* draining into this territory nor an anastomosis of ARGV with the portal system can be seen**.**

This aberrant right gastric vein collects small tributary veins from the lesser curvature of stomach, running almost straight along it for about 6 centimetres. It encompasses the inferior border of the liver, just below the third segment. At the hilum it sits anterior to the

portal vein, bending afterwards slightly posterior just to sit medially and parallel to the root of the left portal branch. From this position, inside the venous ligament's fissure, it approaches the third liver segment (from lateral to medial), branching out into small parenchymal vessels. As far as the CT scan details can show, the portal vein has no vessel distribution in the area where the ARGV drains. After the separation of the left branch of the portal vein, the only visible branches are heading towards the second and fourth liver segments.

CASE 2

On the other hand, for the second case, a 63 years old male, a routine ultrasonography of the abdomen was performed initially. The findings were similar to the ones described above: a fatty hepatic parenchyma and a hypoechoic geographic area within the same liver segments II/III (Fig. 2). For a better characterization of the lesion, a CT scan was highly recommended.

We observed a diffuse hyperdense area at the IInd/IIIrd liver segments, accentuated mostly on the venous phase and minimally on the pre-contrast, arterial and delayed phases (Fig. 6). This pseudolesion was considered again to be a focal fat sparing area in diffuse fatty liver or a hepatic vascular disorder caused by an aberrant right gastric vein running along the lesser curvature of the stomach and draining into these segments (Fig. 7).

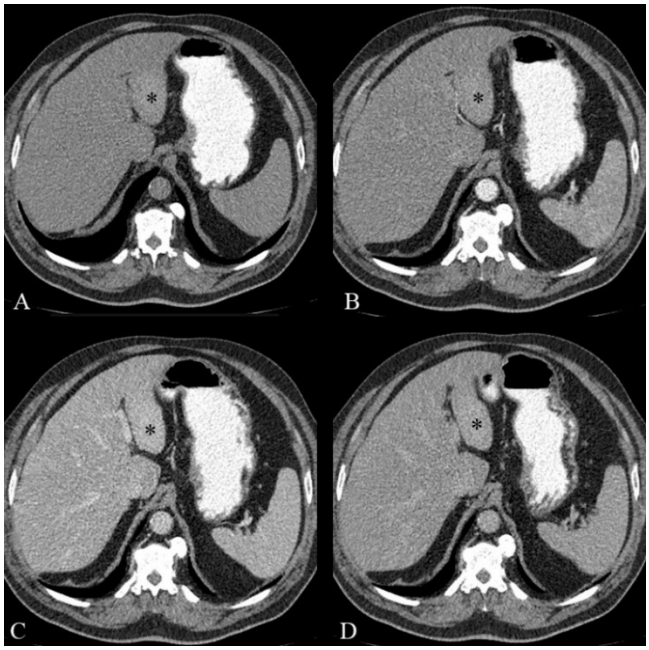

Figure 6. CASE 2. Axial liver images. A – pre-contrast scan (native); B – arterial phase; C – portal venous phase; D – delayed phase. The pseudolesion at the IInd/IIIrd segment level *(*)* is very little obvious on all phases, it can be better assessed on the portal venous phase, otherwise it could easily pass unnoticed.

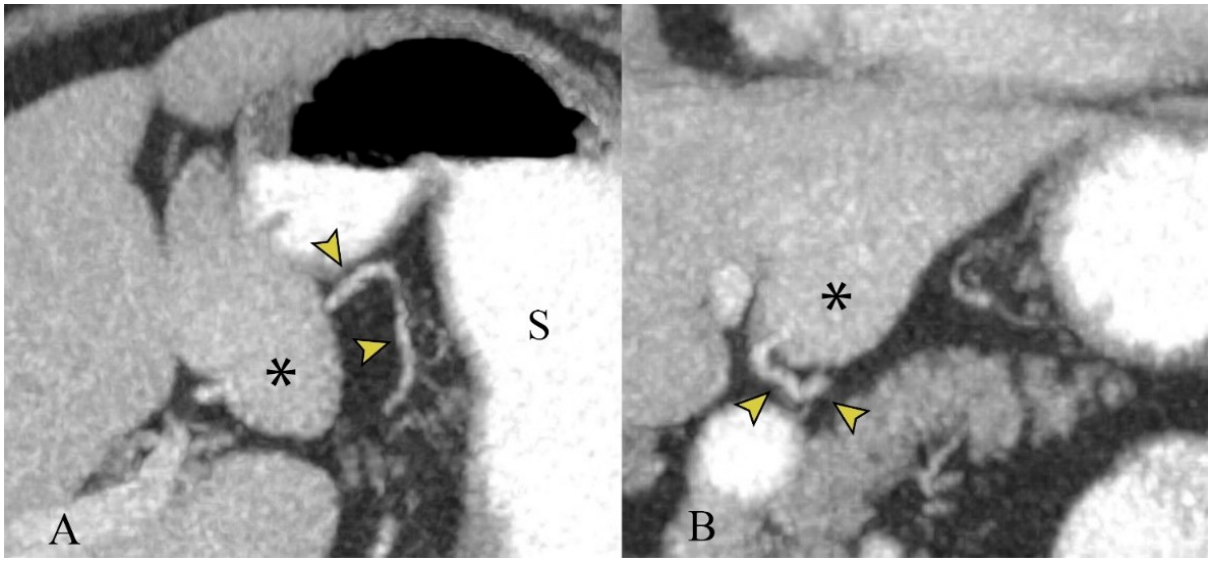

Figure 7. CASE 2. Portal venous phase axial (A) and coronal (B) MIP reconstructed image of the liver. The aberrant right gastric vein *(arrowheads)* drains from the lesser curvature of the stomach into a hyperdense pseudolesion *(*)* at the border of segments II/III.

The pathway of this aberrant right gastric vein resembles the one found on the first case scenario. After collecting tiny vessels from the lesser curvature of the stomach, it continues along it for about 6 centimetres in a slightly antero-posterior way. Afterwards, it goes along the inferior border of the IIIrd liver segment towards the hilum, where it sits antero-medial to the left branch of the portal vein. From this position it enters immediately into the IIIrd liver segment.

Besides the above described aberrant right gastric vein, in this case, there is another vascular variation, which is an accessory hepatic artery derived from the left gastric artery. After an ascent of 5 centimetres towards the eso-gastric junction, the left gastric artery divides into two small calibre vessels: a short one for the lesser curvature of the stomach and a long one (Fig. 8). The latter enters into the fissure of the venous ligament, following a postero-anterior trajectory between the IIIrd and IVth segments. It abuts the hyperattenuating pseudolesion, supplying a more antero-superior area of the IInd segment.

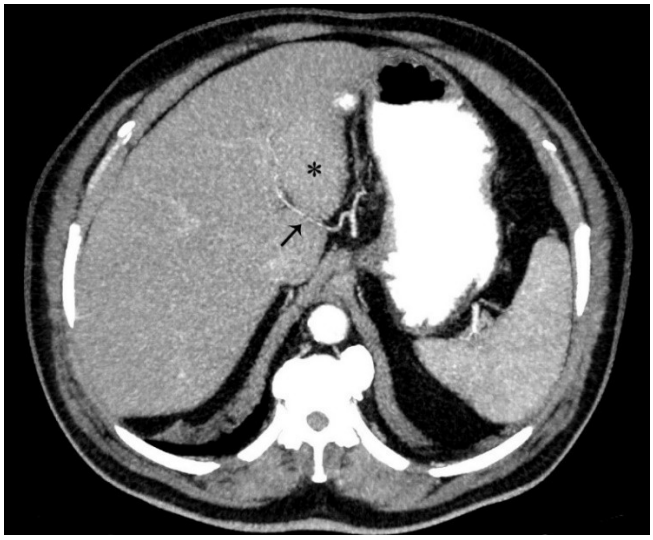

Figure 8. Arterial phase axial MIP reconstructed image shows the left gastric artery that divides, continuing with an accessory branch *(arrow)* that borders the hyperdense pseudolesion *(*)*.

Both two cases presented at the CT scan a diffuse homogenous hyperdense area with unclear edges at the border of IInd and IIIrd liver segments, encircled by diffuse hypodense steatosis hepatic parenchyma. We could not assess a visible portal branch in the territory of the pseudolesion, which proved to be supplied only by the aberrant vein (Fig. 9). A measurable density gap between our region of interest and the other liver segments proved to be evident constantly during the four phases. The calculated mean density of the pseudolesions was 58-64 HU on pre-contrast and arterial phases and 110-124 HU on portal venous phase, while the rest of the liver parenchyma had 22-30 HU and 40-53 HU on pre-contrast and arterial phases. On the portal venous phase most of the liver parenchyma reached the density of 58-62 HU and 91-95 HU respectively.

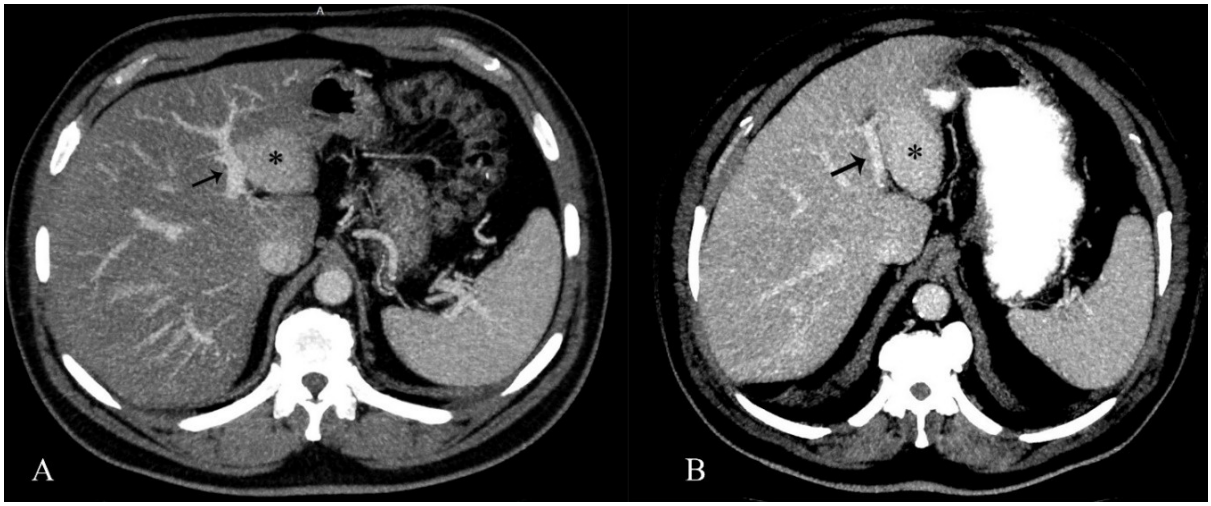

Figure 9. Portal venous phase axial MIP reconstructed images showing the pseudolesion at the border of segments II/III *(*)*, but no associated vessels deriving from the left branch of the portal vein *(arrows)*. A – CASE 1. B - CASE 2.

DISCUSSION

In order to better understand the peculiarities of our cases, a few ideas about the embryology background are highly necessary.

Embryology

The portal venous system develops between the fourth and the twelfth weeks of gestation from the anastomose of the vitelline veins (also known as omfalomesenteric veins) and umbilical veins. On the one hand, the vitelline veins arising from the yolk sac plexus pierce the transverse septum and drain initially into the sinus of the heart. The endodermal cells of the foregut which form the hepatic diverticulum will surround the two vitelline veins inside the transverse septum. At this point, the two veins will form a large anastomose which is the origin of hepatic sinusoids [1] [11] [27].

On the other hand, the vitelline veins form pre-hepatic anastomoses around the lower part of foregut. Two of them are situated ventral by duodenum (cranio-ventral and caudo-ventral) and one is situated dorsally [1] [11] [5]. The dorsal duodenal anastomosis, also called retro-duodenal anastomosis, is placed at the caudal edge of the dorsal pancreatic bud [11]. The caudo-ventral duodenal anastomosis is temporary formed distally by the dorsal anastomosis at the lower portion of the duodenum [11] [5]. The cranio-ventral duodenal anastomosis is located in subhepatic area, adjacent to the hepatic diverticulum. It lies above

the level of the dorsal duodenal anastomosis [5].

The two umbilical veins pass through the transverse septum laterally by the hepatic diverticulum. Over time, the liver grows and eventually engulfs the umbilical veins [1].

Because of the coronary sinus left horn regression, the left omfalomesenteric vein regresses as well during the second month of gestation. Consequently, the right vitelline vein enlarges and drains to the heart all the blood of the vitelline system. This upper segment of the right vitelline vein connecting the liver and the heart, will form the cranial segment of inferior vena cava. After the third month of gestation, a small part of the right vitelline vein persists to form the portal vein and the superior mesenteric vein [1].

A selective involution occurs and the dorsal duodenal plexus persists in order to form the portal vein. The cranial-ventral duodenal plexus forms a segment of the left branch of the portal vein, meanwhile the caudal-ventral duodenal plexus involutes [11].

The umbilical veins suffers a similar selective involution which consist in the obliteration of the left one as well as the cranial segment of the right one. Consequently, the umbilical drainage passes through the hepatic sinusoids. Because the umbilical blood transports the oxygen from the placenta, a more rapid pathway is needed. In order to connect the left umbilical vein with the former inferior vena cava, an enlarged canal called *ductus venosus* appears. A hepatic segment of the left umbilical vein connects with cranio-ventral duodenal anastomosis and will persist in adult as the left branch of the portal vein. *Ductus venosus* and the rest of the left umbilical vein will suffer a fibrous transformation after birth. They persist in adults as venous ligament (Arantius' ligament) and the round ligament of the liver, respectively [1] [6] [10].

In conclusion, an aberrant right gastric vein is the consequence of an interrupted involution of the duodenal plexus in the early embryonic life stages, when the primitive foregut venous plexus anastomoses with *ductus venosus* [24] [6].

In our cases, a connection between cranio-ventral duodenal plexus and the venous duct persists and the aberrant vessel remains separated by the left branch of the portal vein, supplying thus, by itself, the corresponding lobe of the liver.

The prevalence and morphologic types

After the brief analisys of the embryologic development, we will discuss the prevalence and the documented types of such an aberrant right gastric veins.

Although rare in humans, sometimes a direct entry into the liver of a lesser curvature vein is observed. This vein is usually a right aberrant gastric vein, a left aberrant one being less frequent [20].

An aberrant right or left gastric vein draining into the hepatic parenchyma with associated atrophy of segment II was described by Choi et al. [3]. Although the drainage site in our cases was mostly the IIIrd liver segment, no atrophy was observed. Ünal et al. [23] found presence of hepatic artery variation in patients with aberrant gastric veins (AGV), while Caty et al. [2] described direct drainage of the right gastric vein into the liver accompanied by accessories right and hepatic arteries. Our first case had no hepatic artery variation, but second case's ARGV was accompanied by an aberrant left gastric artery (Fig. 8).

The prevalence of the aberrant right gastric vein in previous studies ranges from 1.5% to 49% [3]. An ARGV can drain into the portal vein, but also into the liver parenchyma without any connection with the portal system. Choi et al. [3] reported that such an independent right aberrant gastric vein commonly drains at the border between segments II and III. Our cases support these findings: both ARGVs drained at the posterior aspects of segments II/III.

On the contrary, Seong et al. [20] observed that hepatic segments II and III were uncommon drainage sites for the ARGV (only 4 out of 66 cases). He states also that the most common drainage site for the ARGV is the IVth liver segment and he reports a record prevalence of 49%. Matsui et al. [17] reports pseudolesions in the IVth liver segment caused by the direct drainage of ARGV, and an overall ARGV prevalence of 18%. The large difference between these two studies may be explained as follows: Seong et al. [20] performed selective arteriography of the left and right gastric arteries, while Matsui et al. [17] performed celiac or hepatic arteriography. Another difference is that the patients from Seong et al. [20] study had liver cirrhosis, altered hemodynamic and portal hypertension, which may affect angiographic visualization of aberrant gastric venous drainage. Another important remark is that aberrant gastric veins may be missed on CT or US scans because they are slender, while using angiography technique they are much more readily visualized.

The right gastric vein collects tributaries of the lesser curvature of the stomach, running along it and usually draining into the portal vein. Instead, an aberrant right gastric vein has various draining ways. After performing selective angiography, Seong et al. [20] classified the termination pattern of ARGV into four different types:

— type 1 — single channel smooth continuation of the peripheral portal vein;
— type 2 — collateral connection (via single/multi channels) of ARGV to the peripheral portal vein;
— type 3 — superficial hepatic parenchymal drainage without demonstrable portal branches;
— type 4 — connection of ARGV with the sectional/segmental portal vein via a network.

Seong et al. [20] found that type III termination pattern of ARGV is the most common one and suggest that it supplies only the capsular/subcapsular areas without penetrating deep into the liver parenchyma. The other way around, a relatively large type 1 or type 2 ARGV may sequester territory normally supplied by the portal vein, causing a pseudolesion on imaging studies. Because not all the termination patterns of ARGVs will cause pseudolesions, the true prevalence of ARGVs could be much higher.

Regarding our cases, it is very difficult to determine solely on CT scans which type of termination they have. We could not demonstrate any anastomosis between the ARGV and the portal system or any portal branches draining into the pseudolesions; in line with these findings it could be a type 3 termination, which Seong et al. [20] found to be the most common one, but type 3 termination does not penetrate deep into the liver parenchyma, supplying only the subcapsular areas, which cannot be the case on our reviewed images. Taking into account that a pseudolesion is more prone to manifest with type 1 or type 2 termination patterns, there is a high possibility to be one of those two.

Third inflow effect

Because an aberrant right gastric vein is a third pathway of hepatic blood supply, explanations about some related hemodynamic consequences will be further addressed.

The blood supply of the liver is provided by two main sources: the portal vein (75%-80%) and the hepatic artery (20%-25%). The mixture of the deoxygenated blood (from the portal vein) and the oxygenated blood (from the hepatic artery) takes place at the hepatic lobule level, through the sinusoids, the blood being drained thereafter into the hepatic veins.

There are also frequently encountered some other supplying systemic veins, known under the name of third inflow tracts. The most common ones are the pericholecystic veins draining into segment V, the parabiliary veins draining into segment IV, the epigastric or paraumbillical veins draining into the anterior aspect of segment III/IV and also an aberrant

right/left gastric veins draining into the posterior edge of segment II/III [9] [26].

On different imaging techniques (US, CT, MRI) these third inflow veins can cause perfusion abnormalities and pseudolesions such as focal fatty infiltration or focal fatty sparing. Although a well-known issue in the literature, pseudolesions can still pose diagnostic challenges for unexperienced radiologists. Imaging the third inflow during the enhanced CT is dictated by the distance between the hepatic pseudolesion and the region from which the blood is drained. A pseudolesion explained by cholecystic veins enhances quickly, at the arterial phase. A pseudolesion produced by the parabiliary venous system enhances at the early portal phase, while the one caused by the seldom drainage of the epigastric-paraumbilical venous system enhances in late portal phase because the blood reaches the liver slightly later [26].

However, recognizing and following the unusual vessel through its course up to the pseudolesion may help establishing the diagnosis with greater confidence at intravenously enhanced CT scan [26].

THAD – transient hepatic attenuation difference

Perfusion abnormalities can occur in the liver not only because of the third inflow, but also within the normal dual supply. Via similar hemodynamic mechanisms (inflow mismatches), perfusion abnormalities are frequently encountered on CT scans as THADs, which are pseudolesions seen only on the arterial phase, that vanishes on the portal venous phase, when the liver parenchyma homogenously enchances. THADs are produced because of a transient change in the liver hemodynamics immediately after contrast injection, when a localized mismatch between arterial and portal venous blood supply occurs [7]. Because the hemodynamic mechanisms of both third inflow effect and THADs are strongly related, we think it is also important to discuss this latter topic, although our described cases do not show true “transient” pseudolesions, but rather “permanent” ones.

It is well known that there is a good balance of all the hepatic blood sources. If the portal inflow diminishes (e.g. thrombosis), the hepatic arterial inflow will increase in order to maintain the total blood flow constant. These regional perfusion variations can manifest on the arterial phase CT scans as unexpected areas of hyper- or hypodensity that are not linked with a mass lesion and they were termed transient hepatic attenuation differences (THADs). Any mechanism that increases arterial inflow or decreases portal inflow may result in a

THAD [7].

Other examples of THADs produced by decreased portal flow can be: venous thrombosis (due to hypercoagulable states or pylephlebitis), occlusion of hepatic venules (in the cirrhotic liver due to fibrosis) or even direct compression of portal vein by external processes.

As mentioned above, a THAD can manifest also in the absence of a decrease in portal flow. Hypervascular tumours can increase the arterial flow either by “stealing” or by “siphoning” arterial blood in relation with the adjacent parenchyma [7]. Drugs like gentamicin or sulphamethoxazol can modify the blood parameters and alter the liver architecture [8].

Another venous system, the aforementioned “third inflow”, may supply small regions of hepatic parenchyma and therefore produce a THAD. The most common such finding is usually seen near the gallbladder (segments IV and V) due to the parabiliar venous system drainage [7]. THAD produced by an aberrant gastric vein (AGV) is a much rarer entity, but could occur due to the delay between opacification of the aberrant vein and the main portal pathway.

Temporal separation of the contrast inflow between the portal vein and an aberrant vein can lead to a THAD, seen as a focal enhancement in the liver (usually during the arterial phase), because the blood travels at different speeds in the portal vein compared to third inflow routes [7]. But it is not mandatory for an AGV to produce a THAD. Firstly, it depends on the presence or absence of a communication with a branch of the left portal system and secondly on the calibres of both vessels involved. If there is an anastomosis between the ARGV and the portal system’s branch, the usually greater diameter of the latter may wash out the aberrant venous blood and no pseudolesion will be noticed. Conversely, the amount of ARGV blood may be enough to dilute the contrasted portal blood, resulting in a THAD. In the absence of an anastomosis between portal and third inflow systems, their corresponding hepatic regions may receive two different amounts of blood, producing thereby attenuation differences even on the pre-contrast CT scan as well as on the contrasted phases [12]. In the latter scenario, the “hepatic attenuation difference” is not anymore a “transient”, but rather a “permanent” mismatch and this can be well seen on the acquired images of our both cases.

Regarding the first case (Fig. 4), already on our pre-contrast scan we observed a main attenuation difference between IIIrd hepatic segment and the adjacent liver parenchyma,

equally maintained during the arterial and venous phases. As to the second case (Fig. 6) the pseudolesion would have probably passed unnoticed if an ultrasound had not been performed prior. There is a better contrast between hypo and hyperdensities regarding the first case, explained by the fact that here the liver is much more fatty than the one examined in the second case.

Therefore, we theorize that the increased attenuation of the posterior edges of segments II/III may be due to the early venous drainage from the aberrant right gastric vein compared with the portal vein drainage, but also to the lack of a portal vein branch into this territory. Thus, the venous blood from these two sources does not mix at all. Because the calibre of the ARGV is greater than a portal branch would normally be in such a small territory, hypervascularity can also be responsible for the difference of attenuation.

In conclusion, a THAD-like hemodynamic mechanism may also explain our case findings, but perhaps not solely *per se*. There may be also a THAD related metabolic substrate; previous studies have shown that a hyperdense pseudolesion at the posterior aspect of segments II/III correlates with a focal fat sparing area due to third inflow effect [23] [17].

Thereby, non-portal venous drainage (i.e. third inflow) not only may produce a THAD, but it may also spare a small hepatic region of diffuse fatty infiltration. One reason of such a focal fat sparing could be that the liver does not receive the same nutrient-rich venous drainage via third inflow pathways (e.g. an AGV) as it normally happens with the porto-mesenteric venous route [7]. On our pre-contrast scans (native) we found diffuse hepatic steatosis with a mean density value of 22-30 HU (CASE 1) and 40-46 HU (CASE 2), but, in the region supplied by the aberrant right gastric vein, the measured mean density value was not less than 60 HU. These findings are highly suggestive of a fat sparing area and correspond to the ones already reported elsewhere in the published literature [24] [20] [3].

The stomach is not a site for absorbing rich nutrients as the small bowel is par excellence. Thereby, an aberrant right gastric vein that directly links the stomach with the liver, eluding the mix with mesenteric lipid-rich blood coming from the small bowel, may preserve a “fat free” hepatic area.

It is speculated that this process may be hormone modulated [24]. The role of insuline of esterification of fatty acids into triglycerides stimulates their accumulation in hepatocytes. When a third inflow source drains the stomach region (as an AGV does), where the levels of dietary fat and insulin are low, focal sparing is observed. Vice versa, fatty infiltration is noted

in the hepatic area where high-insulin blood level coming from the pancreaticoduodenal vein is drained [21].

Whether it is the vascular hypothesis (i.e. third inflow effect or THAD-like) that stands up as an explanation, whether it is the metabolic one (i.e. fatty changes, hepatotoxicity) or even both of them, the most important feature for radiologists and clinicians is that such a finding has no harming potential and should not be confused with true focal masses, although a differential diagnosis with them should always be kept in mind. That is why, further on, we will briefly discuss some important differences between liver masses.

Differential diagnosis

Judging solely by the ultrasound scan, our findings would normally pose differential diagnosis problem between an atypical haemangioma, a “fat free” area or a tumour. That is the reason why a CT-scan is mandatory in such cases. For a better comparison between different hepatic lesions, let us remember once again our main CT findings: two hyperattenuating homogenous areas clearly visible on all four phases, that enhance especially on the portal venous phase, with no slow-fill, wash-out, central scar or rim-like features.

Haemangioma typically manifests on non-contrast scans as hypoattenuating area with peripheral enhancement on the arterial phase and progressive centripetal fill-in on the portal venous phase.

Focal nodular hyperplasia with its homogeneous hypervascularity may manifest as a focal fat sparing area or as a THAD-like, but usually it presents a pathognomonic central scar with progressive enhancement. Hepatic adenoma becomes isodense on the portal venous phase.

Typically, liver metastases are hypoattenuating on pre-contrast CT scan, enhancing peripheral following contrast and showing rapid washout on portal venous/delayed phase. Associated hepatic steatosis may mask metastases, making them isodense or discrete hyperdense lesions. Neuroendocrine tumours, renal or thyroid carcinoma may produce hyperenhancing liver metastases. A delayed phase was performed only for the second case and showed the same attenuation difference seen on all the other phases; the density gap between segments II/III and the rest of the adjacent hepatic parenchyma pointed around the same 20 HU, ruling out with greater confidence a possible tumour.

Focal hemochromatosis entered also on our list of differential diagnoses, but its homogeneous increase liver density (75-130 HU) did not correspond to our lesions' mean measurements (~62 HU), the latter being more appropriate to normal hepatic parenchyma. Wilson disease sparing almost the entire liver except for the IInd/IIIrd segments, was a less probable diagnosis.

Knowing about the presence of an ARGV is not only important for making differential diagnosis between its related tumor mimicker (pseudolesion) and the aforementioned lesions, but also for other reasons. Firstly, it can cause unexpected haemorrhage during surgery, pre-operative planning and evaluation based on imaging techniques being thus strongly encouraged [16]. Secondly, the presence of an ARGV can serve as a collateral pathway of the portal system whenever portal vein obstruction or hypertension occurs. Lastly, gastric tumours can directly spread metastases to the liver through an ARGV, careful assessment of such anatomical variation being thus highly important in gastric cancer patients.

CONCLUSIONS

An aberrant right gastric vein is a rare anatomical variation that usually runs along the lesser curvature of the stomach, draining directly into the liver parenchyma. The ARGV may produce a pseudolesion due to hepatic blood inflow mismatch (third inflow and THAD related hemodynamic mechanisms), but also due to an associated and underlying metabolic cause: focal fat sparing area within diffuse hepatic fatty infiltration. These pseudolesions may mimic liver tumours, therefore it is important to look for such an aberrant vessel in order to rule out other diagnoses.

Institutional Review Board Statement

The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Ethics Committee of the Arhimed Radiology (no. 926/09.06.2023).

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