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REVIEW ARTICLE

"Periportal neoplasms"—a CT perspective: review article

ANURADHA SINGH, MD, DNB, S H CHANDRASHEKHARA, MD, DNB, NAYHA HANDA MD, DNB, VINIT BALIYAN, MD and PAWAN KUMAR, MBBS

Department of Radiodiagnosis, All India Institute of Medical Sciences, Ansari Nagar, New Delhi

Address correspondence to: Dr SH Chandrashekhara
E-mail: drchandruaiims@yahoo.com

ABSTRACT

The periportal space is a potential space surrounding the portal vein and its intrahepatic branches. A variety of neoplasms can involve the periportal region, whether primary or secondary, owing to contiguous spread from surrounding hepatic parenchyma or from adjacent organs. CT plays an important role in not only diagnosing these lesions but also determining the extent of the disease. Most of the malignancies leading to the periportal spread manifest as periportal hypodensity either distinctly or in contiguity with the primary tumour. Even in known malignancies, periportal hypodensity commonly results from non-neoplastic causes like periportal oedema; hence, a knowledge of the imaging findings to ascertain its presence as well as to conclude the definite neoplastic spread is prudent. Periportal spread of neoplasm may suggest locally aggressive or disseminated disease (in extrahepatic malignancies), which may change management accordingly.

INTRODUCTION

The periportal region is a potential space surrounding the portal vein and its intrahepatic branches. Its contents include the hepatic artery, biliary radicle, nerves and lymphatics. A variety of neoplasms can involve the periportal region, whether primary (*via* haematogenous, lymphatics and biliary route) or secondary, owing to contiguous spread from surrounding hepatic parenchyma or adjacent organs. Differential diagnosis of periportal neoplasms is limited, which may be suggested on imaging; hence, radiologists need to be familiar with their findings. Nonetheless, diagnostic confirmation is a multidisciplinary approach, additionally requiring clinical and pathological correlation.

ANATOMY

In the process of becoming intrahepatic, the portal vein pierces the hepatic capsule (Glisson's capsule or fibrosa perivascularis). As a result, the intrahepatic portal vein and its branches carry with themselves a sleeve of loose connective tissue, resulting in the formation of a "periportal space". The contents of this space include neurovascular structures (hepatic artery, lymphatics and nerves) and biliary radicles, which embrace the portal vein.

On contrast-enhanced CT (CECT) scan, periportal lesions are most conspicuous in the portovenous or equilibrium phases, corresponding to the increased conspicuity of their anatomical landmark—the "portal vein". Owing to its

contents and strategic location, seedling of periportal space can occur from a variety of routes like haematogenous, lymphatic, neural, peritoneal or direct spread from contiguous neoplasms. Amongst these, the commonest cause of periportal pathologies is lesions affecting the portal vein.

For the purpose of simplicity, we have classified periportal neoplasms (Table 1) on the basis of mode of spread into primary (neurovascular or biliary origin) or secondary (infiltration from hepatic neoplasm or adjacent organ tumours).^{1–3}

Primary periportal neoplasm

Cholangiocarcinoma

Cholangiocarcinoma (CCA) is one of the commonest malignancies of the biliary tract, ranking next only to carcinoma gall bladder in incidence. The average age of presentation is 50 years, with a slight female preponderance. Of the three morphological subtypes of CCA—intrahepatic mass forming, intraductal growing and periductal infiltrating, the latter spreads exclusively in the periportal space, with only occasional infiltration by other subtypes. The biliary radicles are supplied by the peribiliary vascular plexus, lymphatics and nerve plexus, which serve as a conduit to the periportal spread.

On CECT, periductal CCA lacks a distinct mass and appears as infiltrating asymmetrical hypodense soft-tissue

Table 1. Classification of periportal neoplasms

Primary	Secondary
Biliary tumours	Hepatic neoplasm
Cholangiocarcinoma	Hepatocellular carcinoma
	Hepatoblastoma
Haematogeneous	Gall bladder
Chloroma	Gall bladder carcinoma
Lymphatics	Peritoneal spread
Lymphoma (including post-transplant lymphoproliferative disorders)	Peritoneal carcinomatosis
Langerhans cell histiocytosis	Pseudomyxoma peritonei
	Peritoneal lymphomatosis
	Peritoneal sarcomatosis
Neural	Contiguous spread from adjacent organs; <i>e.g.</i> carcinoma stomach, duodenum and colon
Nerve sheath tumour—neurofibroma or schwannoma	
Portal vein	Metastasis
Leiomyosarcoma	
Miscellaneous	
Mesenchymal tumour, atypical cystadenocarcinoma, inflammatory pseudotumour and Kaposi's sarcoma	

thickening, growing along an irregularly narrowed or dilated bile duct (Figure 1). It encases the biliary radicles and portal vein, causing their abrupt cut-off or significant luminal attenuation with peripheral dilatation, owing to its scirrhous nature. Similar to other periportal neoplasms, portovenous or equilibrium phase is best to characterize CCA, although the lesion may show enhancement in arterial phase. In CCA, periportal hypodensity not only indicates periductal spread but also portends a poor prognosis, making the lesion unresectable. Periductal infiltrating CCA needs to be differentiated from periportal lymphatic dissemination of extrahepatic malignancies. Periportal involvement in periductal CCA is usually focal or segmental and it results in dilatation of upstream biliary radicles. On the contrary,

in periportal lymphatic spread of extrahepatic malignancy, involvement is usually diffuse, and biliary radicles are usually not dilated.^{4–6}

Chloroma

Variously referred as “myeloid or granulocytic sarcoma”, chloroma denotes localized extramedullary proliferation of the primitive malignant myeloid cells. Of all the myeloid neoplasms, chloroma is more preponderant in acute (seen in 3–5% of cases) than chronic myeloid leukaemia or other myeloproliferative disorders. It is usually seen in the relapse or remission phase of the disease and portends a poor prognosis. Common sites of chloroma include the bones, lymph nodes, soft tissues,

Figure 1. Hilar cholangiocarcinoma: axial contrast-enhanced CT showing a heterogeneously hypodense infiltrating mass lesion at the hepatic hilum extending periportally [arrow in (a)] and causing isolation of primary and right secondary biliary [arrows in (b)] confluences (Bismuth–Collette–IIIa). Also, there is significant attenuation of the portal vein, especially its right branch. Owing to increasing jaundice, left-sided percutaneous transhepatic biliary drainage was performed with the drainage catheter *in situ* (asterisk).

Figure 2. A case of acute myeloid leukaemia with jaundice: contrast-enhanced CT image showing multiple variably sized well-circumscribed lesions in both lobes of the liver (arrows). Concomitant presence of periportal hypodensity is also noted, causing mild central intrahepatic biliary radicle dilatation.

skin and breast. It has a wide age of presentation but is commonly seen in children (<15 years) with no gender predilection.^{7,8}

Leukaemic involvement of the liver is extremely rare and usually occurs secondary to extensive marrow infiltration. Rare case reports in literature describe an entity called primary biliary chloroma, with isolated involvement of the peribiliary space in the absence of bone marrow involvement. However, recent times have seen a surge in the cases of hepatobiliary chloroma, as more patients of myeloid leukaemia are being treated by intensive chemotherapy and bone marrow transplant.^{7,9}

Myeloid cells can infiltrate either the hepatic sinusoids or periportal region. Corresponding to the pattern of spread, hepatic involvement may manifest as a nodular deposit (solitary or multiple) of varying sizes or the other rarer form, causing

peribiliary infiltration. On CT (Figure 2), there may be hepatomegaly, with the presence of either well-circumscribed hypodense heterogeneously enhancing lesions or periportal hypodensities paralleling the biliary radicles (peribiliary chloroma).

Being non-specific, both these patterns of chloroma are great imaging mimics. Discrete lesions resemble lymphoma from which it is difficult to differentiate; however, certain morphological features may help. Lymphoma deposits are relatively well margined, non-enhancing or mildly enhancing, and mass effect is considerably less for the extent of involvement. On the contrary, hepatic chloromas are usually infiltrating heterogeneously enhancing lesions with significant mass effect, resulting in biliary radicle dilatation. The presence of lesions at other sites may be a clue to lymphoma, especially in cases of secondary hepatic involvement. Peribiliary chloromas may remain undiagnosed until late, owing to their extremely rare occurrence as well as non-specific imaging findings, which are seen commonly in other conditions like cholangitis or CCA.⁹

Owing to its mass effect, hepatic chloroma can cause jaundice, the diagnosis of which may remain elusive until late, and radiologists are often the first ones to raise suspicion; nonetheless, prognosis remains poor. Early diagnosis of chloroma is prudent, as localized lesions in initial stages can be treated with curative intent by chemotherapy or focal ablative procedure like focused radiotherapy.^{8,10}

Lymphoma

Hepatic involvement in lymphoma can be either primary or secondary to the involvement at other sites, the latter being commonly seen in nearly half of the cases. The age of presentation is wide, ranging from childhood to eighth decade, males being affected twice as common as females. Owing to the paucity of lymphocytes, primary hepatic lymphoma (PHL) is an extremely rare entity and is diagnosed by the absence of extra-hepatic involvement for at least 6 months after the onset of hepatic disease.⁸

On imaging, there can be various patterns of involvement—diffusely infiltrating form resulting in hepatomegaly, discrete

Figure 3. Primary hepatic lymphoma: (a) axial sections of the liver showing conglomerate mass-like asymmetrical hypodensity encasing and attenuating the main portal vein and its left and right branches (arrows), causing volume redistribution of the liver with lobulated contour. (b) Follow-up scan 6 months post chemotherapy showing complete resolution of periportal masses; however, there is persistence of atrophy and contour deformity.

Table 2. Differentiating primary hepatic lymphoma (PHL) from hepatocellular carcinoma (HCC) and intrahepatic cholangiocarcinoma (IHCC)⁸

Feature	HCC	IHCC	PHL
Background liver parenchyma	Usually cirrhotic	Usually non-cirrhotic	Can be cirrhotic
Contrast enhancement	Arterial enhancement with washout in portovenous and delayed phases	Continuous rim enhancement in arterial phase with progressive centripetal fill in portovenous and delayed phases	Subtle or no enhancement in all the phases of dynamic scan
Vessel involvement	Vascular thrombosis (tumour in vein) is a feature	No vascular thrombosis	Vascular encasement without attenuation or thrombosis
Capsular retraction and dilatation of biliary radicles	Not a feature	Characteristic feature	May be present in periportal infiltrating form
Lymphadenopathy below the renal hilum	Usually absent	May be seen in disseminated disease	May be present
FDG-PET	Hypometabolic	Hypometabolic	Usually intensely FDG avid
Serum alpha-fetoprotein	Usually elevated (in approximately 70% of cases)	Not elevated	Not elevated

FDG, fludeoxyglucose; PET, positron emission tomography.

nodules or masses, combined nodular infiltrating and an extremely rare periportal infiltrating form (Figure 3). Diffusely infiltrating is the most common pattern in secondary hepatic lymphoma, whereas in the primary form, solitary nodules are more common (Table 4).

On CT, lymphoma is usually hypodense and shows no enhancement. Central necrosis, scar and/or calcification may be seen in the aggressive variety or post chemotherapy. As lymphomatous deposits are soft lesions lacking desmoplastic response, the mass effect is disproportionately less compared with their size. In peribiliary lymphoma, by virtue of its distribution, biliary dilatation is relatively commoner and occurs early in the course of disease.

Labelling hepatic deposits as lymphomatous involvement with the evidence of disease elsewhere as in secondary hepatic lymphoma is usually not a diagnostic concern. However, dilemma arises in cases of PHL, in which differentiating it from other focal hepatic neoplasms can be painstakingly difficult. PHL with nodular deposits needs to be differentiated from other common hepatic malignancies like hepatocellular carcinoma (HCC), mass-forming intrahepatic CCA and metastasis, mainly owing to different therapeutic approaches. MRI may remain inconclusive, as all the above-mentioned lesions can be of intermediate signal intensity on T_2 weighted images and may show restricted diffusion. Hence, differentiating amongst these conditions is a holistic approach requiring correlation of the imaging findings with clinical, laboratory profile and histopathological analysis (Table 2).^{8,11}

“Periportal halo” resulting from dilated obstructed lymphatics can be seen in any morphological form of lymphoma. It differs from periportal lymphoma in being a linear circumferential hypoattenuation along the portal triad as opposed to the latter, which presents as a circumscribed mass in peribiliary distribution.

Persistence of periportal halo may suggest refractoriness to chemotherapy or disease exacerbation.^{1,12,13}

Lymphoma as a part of the spectrum of post-transplant lymphoproliferative disorders (PTLD) seen in the transplant recipients are usually extranodal, the liver being the commonest solid visceral organ to be involved. Three imaging patterns in PTLD of the liver have been described—poorly enhancing circumscribed masses, diffusely infiltrating pattern and an uncommon periportal infiltrating lesion. The periportal infiltrating form encases the portal vein and may cause its attenuation or thrombosis; however, the incidence of venous involvement is significantly lower than in HCC. Post-transplant recipients are vulnerable to

Figure 4. A 4-year-old child with Langerhans cell histiocytosis and hyperbilirubinaemia. Contrast-enhanced CT image showing conglomerate ill-defined hypodensities (arrows) surrounding the portal vein and its bilobar intrahepatic branches.

Figure 5. Intrahepatic periportal neurofibroma: a case of neurofibromatosis-1 showing non-enhancing lobulated infiltrating soft tissue in the retroperitoneum and bilateral paraspinal regions [arrows in (a) and (b)] encasing the vessels and infiltrating the hepatic hilum [arrow in (a)] along the hepatoduodenal ligament with further intrahepatic spread along the left branch of the portal vein [arrow in (b)].

opportunistic infections and other malignancies, which may metastasize to the liver. It is prudent to differentiate these lesions, as PTLN usually responds favourably by mere reduction in the immunosuppressant doses.¹

Langerhans cell histiocytosis

Langerhans cell histiocytosis (LCH) is the most common histiocytic proliferative disorder, and the majority of patients are younger than 2 years of age. Hepatobiliary involvement is usually a part of the disseminated disease, seen in approximately 40–60% of the cases. On the contrary, isolated hepatobiliary LCH is relatively rare, with an incidence of approximately 14–18%. Hepatobiliary involvement of LCH can be of two types—diffusely infiltrating resulting in hepatomegaly or periportal involvement, owing to the marked proclivity of histiocytes for biliary radicles.

The periportal form can be further categorized into four stages—proliferative, granulomatous, xanthomatous and fibrotic. Initial

proliferative and granulomatous stages are essentially a periportal triaditis, resulting in oedema on histopathology. On ultrasound, it manifests as hyperechoic nodularity and band in the periportal region with corresponding hypodensity on CT (Figure 4), showing occasional enhancement. In the xanthomatous stage, there is clustering of lipid laden nodules in the hepatic parenchyma in the peribiliary location. On ultrasound, it appears as a circumscribed hyperechoic nodule, which is hypodense on CECT. Ultimately, if allowed to progress, periportal fibrosis ensues, resulting in secondary biliary cirrhosis.^{14,15}

Neurofibroma

Neurofibroma, a benign nerve sheath tumour and a feature of neurofibromatosis-1 (NF-1), can be either peripheral or plexiform. Peripheral neurofibromas are usually supradiaphragmatic in location—the head, neck and thorax being the usual sites of involvement. Gastrointestinal tract involvement in NF is extremely rare and is usually restricted to the occasional presence

Figure 6. Periportal metastasis: (a) a case of jejunal gastrointestinal stromal tumour (GIST); coronal contrast-enhanced CT showing exophytic GIST (arrow) with hepatic metastasis (curved arrow). (b) Axial scan showing metastasis centring around the left and main portal vein branch (arrow). Another discrete lesion can be seen in the right lobe of the liver (curved arrow).

Figure 7. (a) A case of carcinoma colon with hepatic metastases epicentred in the periportal region (arrow) conspicuously seen in the left lobe of the liver. (b) A metastatic carcinoma breast with hepatic lesions predominant in the subcapsular and periportal distribution (arrows), leading to “pseudocirrhosis” appearance.

of leiomyoma or neurofibroma of the hollow viscera. Plexiform neurofibromas, which are pathognomonic lesions of NF-1, rarely occur below the diaphragm. Only a handful of case reports in literature describe its occurrence in the retroperitoneum and paraspinal region, which may contiguously infiltrate the hepatic hilum.

Intrahepatic periportal plexiform NF results from the involvement of the hepatic nerve plexus constituted by autonomic nerve branches of splanchnic and phrenic nerves. Further periportal spread is explained by the distribution of the hepatic nerve plexus along vessels and biliary radicles. Hepatic

involvement may be either diffuse or segmental in distribution. On CT, it appears as homogeneous low-attenuation non-enhancing/hypo-enhancing masses (approximately 15–20 HU) in the periportal distribution emanating from the hepatic hilum (Figure 5). Low attenuation is variably attributed to the presence of the myxoid matrix, entrapment of adipose tissue and cystic degeneration. Quite characteristic of these lesions is vascular and biliary radicle encasement, without causing luminal narrowing or thrombosis, hence preserving the native architecture. Contiguous extension of the lesion from the retroperitoneum and/or mesenteric root is another key distinguishing feature.^{1,3,16–19}

Figure 8. Hepatocellular carcinoma: arterial-phase image (a) showing large infiltrating heterogeneously arterially enhancing lesion in the right lobe and medial segments of the left lobe of the liver. Note the expanded right portal vein branch showing arterial enhancement suggestive of tumour thrombus [arrows in (a)]. Portovenous-phase images (b, c) showing periportal infiltration of the tumour along the main portal vein and its intrahepatic branches (arrows).

Figure 9. Hepatoblastoma: (a and b) axial contrast-enhanced CT scan of a 4-year-old patient showing heterogeneously enhancing mass in the right lobe of the liver infiltrating and extending along the right branch of the portal vein [arrow in (b) pointing to malignant thrombus in the right branch of the portal vein]. In addition, mass-like hypodensities can be seen along the left branch of the portal vein (curved arrow in a).

Schwannoma, the other nerve sheath tumour seen in NF-1, can occasionally involve the liver. Akin to intrahepatic neurofibroma, it is also extremely rare. Its most common site of involvement is the hepatic hilum (porta hepatis), owing to abundance of nerve fibres in the hepatoduodenal ligament.²⁰ As opposed to the unencapsulated intrahepatic neurofibroma, which spreads longitudinally along the biliary radicles, schwannomas are usually encapsulated heterogeneously enhancing cystic masses, and they may also show calcification.¹

Metastasis

Metastatic spread to the liver can occur through a variety of routes. Haematogenously disseminated metastases derive their vascular supply through the portal vein. Correspondingly, in the initial stages, such lesions seed along the periportal and subcapsular locations (Figures 6 and 7).

Malignant lymphadenopathy at the porta hepatis or lesser omentum can lead to periportal hypodensity by either impeding the lymphatic drainage resulting in periportal lymphoedema or there may be a retrograde dissemination of malignant cells from the hilum. Hence, periportal hypodensity should always be interpreted cautiously to rule out occult neoplasms in the porta hepatis or lesser sac.^{2,21–23}

Secondary periportal neoplasms

Hepatocellular carcinoma

HCC is the most common primary malignant hepatic tumour. It may show periportal spread either by invasion of the portal vein or through lymphatic dissemination. On CECT, it appears as an arterially enhancing mass lesion with washout in the portovenous and/or delayed phases, usually in the background of cirrhosis. Well-differentiated lesions may be circumscribed with the presence of a capsule; however, poorly differentiated forms are

Figure 10. Carcinoma of the gall bladder (Ca GB) with periportal spread. An operated case of Ca GB on palliative chemotherapy and percutaneous transhepatic biliary drainage: (a) axial contrast-enhanced CT demonstrates ill-defined hypodensity paralleling the course of the main portal vein (MPV) and its segmental branches (thin arrow). Note its spread along the anterior and posterior divisions of the right branch (asterisks). GB fossa is empty. Solid arrow points to external biliary drainage catheter. (b) Coronal images showing periportal spread along the MPV (thin arrow).

Figure 11. A carcinoma ovary with peritoneal carcinomatosis. (a) Axial contrast-enhanced CT section at the level of the liver showing conglomerate hypodense deposits (arrow) along the falciform ligament. (b) Cranial sections showing deposits (arrows) along the main and left branch of the portal vein.

usually infiltrating lesions (Figure 8). Venous invasion is a remarkable feature of HCC. Periportal spread, which may be diffuse or segmental in distribution, is indicated by the presence of hypodensity tracking along the invaded portal vein (tumour in vein) or biliary radicles.³

Hepatoblastoma

It is the most common primary malignant hepatic tumour in children of less than 5 years of age. On CT, it is usually a well-encapsulated heterogeneously enhancing mass lesion, with variable presence of calcification or cystic degeneration. In some cases, periportal hypodensity (Figure 9) is seen, which may be secondary to the involvement of periportal lymphatics.²⁴

Gall bladder carcinoma

Gall bladder carcinoma is the most common malignancy of the biliary tract. As the gall bladder lacks submucosa, there is early

rapid spread of malignancies to the adjacent organs and hepatic parenchyma. Hepatic involvement can be either due to direct infiltration or can be haematogeneous. Further, the disease may show periportal spread (Figure 10), which can be due to direct portal tract invasion, lymphatic dissemination or involvement of the hepatoduodenal ligament.^{25,26}

Peritoneal tumours

Glisson's capsule, which surrounds the liver, forms a sleeve of connective tissue around the intrahepatic portal vein referred to as Glisson's sheath. Glisson's sheath further continues into the subperitoneal spaces of gastrohepatic and hepatoduodenal ligaments, hence serving as a conduit to the periportal spread of peritoneal malignancies.

Peritoneal neoplasms like peritoneal carcinomatosis, pseudomyxoma peritonei or peritoneal lymphomatosis usually stud the hepatic surface corresponding to the peritoneal fluid circulation. Involvement of the lesser omentum leads to further spread to the periportal region contiguously from the gastrohepatic and hepatoduodenal ligaments.

On CT, peritoneal implants appear as low-attenuation plaque-like or nodular masses involving the liver surface and may show periportal spread, predominantly at the porta hepatis and along the left branch of the portal vein (Figure 11).

This periportal spread needs to be differentiated from the hepatic parenchymal metastasis especially in ovarian malignancies, as the latter portends a poorer prognosis, making the lesion usually unresectable. Periportal spread is relatively ill defined and is not circumferentially harboured by hepatic parenchyma, as opposed to the intraparenchymal metastasis.

As periportal spread of all neoplastic peritoneal disease processes remain similar, these can be differentiated by the primary site elsewhere. However, scalloping of the liver surface is a distinguishing feature of pseudomyxoma peritonei (Figure 12).^{21,27,28}

Spread of malignancy from an adjacent organ along the perihepatic ligaments

Apart from peritoneal neoplasm, malignancies of adjacent organs like the stomach, duodenum, colon and pancreas or even from the anterior abdominal wall can contiguously infiltrate the perihepatic ligaments with further periportal spread (Figures 13 and 14). In the absence of distant metastasis, such cases are usually resectable, although surgery is more extensive.^{1,21,29}

Miscellaneous tumours

Kaposi's sarcoma is a low-grade vascular tumour and is the commonest intrahepatic malignancy in patients with acquired immune deficiency syndrome. CT examination shows hepatomegaly with the presence of ill-defined hypodense periportal nodules, which may show conglomeration. After contrast administration, these nodules may show enhancement with gradual filling in the delayed phases, making them indistinguishable from haemangioma. Similar lesions may be present in the spleen

Figure 12. Pseudomyxoma peritonei. (a) Axial contrast-enhanced CT showing lobulated hypodense lesions extending along the portal vein (arrow) and causing scalloping of the adjacent hepatic parenchyma. On coronal reformat (b), scalloping of the liver surface and periportal spread (arrows) is well demonstrated along with the presence of high-attenuation septated ascites (asterisk).

coupled with hyperenhancing lymphadenopathy, serving as a clincher to its diagnosis.³⁰

Non-neoplastic causes of periportal hypodensity (periportal oedema)

Periportal hypodensity is most commonly due to non-neoplastic aetiologies (periportal oedema) like fluid overload, trauma, hepatitis, liver transplantation etc. (Figure 15). It is imperative to differentiate these causes from neoplastic aetiologies, in which certain morphological features of periportal hypodensity may help. In periportal oedema due to non-neoplastic causes, hepatic involvement is diffuse and the periportal halo is thin and sharply marginated with regular or symmetrical contour. On the contrary, periportal hypodensity resulting from neoplasm is usually

focal or segmental in distribution and asymmetrically thickened with infiltrating margins.

CONCLUSION

A vast majority of neoplastic as well as non-neoplastic conditions can affect the periportal space, resulting in periportal hypodensity. By itself, periportal hypodensity is a non-specific finding. However, it may herald the presence of a sinister aetiology either within the liver or at other extrahepatic sites like the lesser omentum, peritoneum or adjacent organs. Arriving at a definite diagnosis is nonetheless a multidisciplinary approach, additionally requiring clinical and histopathological correlation. Radiologists need to be familiar with these imaging findings to suggest as well as curtail the differential diagnosis.

Figure 13. Gastric non-Hodgkin's lymphoma with periportal spread. (a) Axial contrast-enhanced CT scan showing an eccentric hypodense exophytic mass arising from the antropyloric region of the stomach without causing luminal narrowing (thin arrow). (b) The lesion has further infiltrated the hepatic hilum (solid arrow) with spread along the falciform ligament and left branch of the portal vein (thin arrow) and causing atrophy of the left lobe of the liver (asterisk).

Figure 14. A post-operative case of periampullary carcinoma showing confluent mass-like hypodensities (arrow) along the main portal vein and its intrahepatic branches, causing their attenuation. Discrete hepatic metastasis also present (curved arrow). Note the upstream dilatation of the main pancreatic duct (asterisk).

Figure 15. A case of polytrauma having hypotension at initial presentation. Subsequent fluid correction led to volume overload, manifesting as periportal oedema paralleling the portal vein and its branches (arrow).

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