



ICONOGRAPHIC REVIEW / *Gastrointestinal imaging*

CT imaging of peritoneal carcinomatosis and its mimics



A.D. Diop^a, M. Fontarensky^a, P.-F. Montoriol^a,
D. Da Ines^{b,*}

^a Clermont-Ferrand University Hospital, Estaing University Hospital, Department of Radiology and Medical Imaging, 1, place Lucie-Aubrac, 63003 Clermont-Ferrand cedex 1, France

^b Clermont University, University of Auvergne, ISIT, CNRS, UMR6284, BP 10448, 63000 Clermont-Ferrand, France

KEYWORDS

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Scalloping

Abstract Invasive peritoneal disease includes more than just peritoneal carcinomatosis. Although this is the most common aetiology, especially when a primary is found, other conditions may be responsible for peritoneal invasion. A rigorous analysis of CT features taken together with the clinical and biological context usually allows the main differential diagnoses, which entail different types of management, to be drawn out. Pseudomyxoma peritonei, peritoneal lymphomatosis, tuberculosis, peritoneal mesothelioma, diffuse peritoneal leiomyomatosis, and benign splenosis are the main differential diagnoses.

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Peritoneal carcinomatosis is the intraperitoneal dissemination of any tumour that does not originate from the peritoneum itself. It is the most common diffuse peritoneal disease. For a long time, it has been considered to be the terminal stage of malignant disease, with a very poor prognosis if untreated. Since the 1990s and the development of treatment combining cytoreduction surgery and hyperthermic intraperitoneal chemotherapy (HIPEC), the management of this disease has been totally turned on its head [1]. These techniques are aggressive and they are associated with high morbidity, so patient selection is crucial for optimum efficacy.

However, to improve the treatment of a lesion improvements in diagnosis are needed as well. In this sense, currently, the role of the radiologist consists not only of diagnosing peritoneal carcinomatosis at an early stage, but also of choosing the candidates who are likely to benefit from this extremely arduous surgery, and finally to exclude instances of

* Corresponding author.

E-mail address: ddaines@chu-clermontferrand.fr (D. Da Ines).

diffuse peritoneal disease that mimic carcinomatosis [2], diseases that for the most part require only medical treatment, in order to avoid any unnecessary surgical interventions. In current practice, lymphomatosis, tuberculosis, mesothelioma, and pseudomyxoma peritonei are the most common differential diagnoses to be considered when diffuse peritoneal involvement is seen.

Thus, the purpose of this article is to use a pictorial review of CT imaging as a reminder of the key signs in diffuse peritoneal involvement, to specify how and where to look for peritoneal carcinomatosis, and finally to describe the main differential diagnoses.

Routes of dissemination in peritoneal carcinomatosis

An understanding of the routes by which peritoneal carcinomatosis is disseminated and the dynamics of peritoneal circulation is an indispensable foundation for gaining a better understanding of the key signs of peritoneal carcinomatosis.

There are four dissemination routes [3]:

- the haematogenous route is the main route for primary tumours with a high grade of malignancy. They are able to invade the vascular walls, then disseminate and implant in the peritoneum because the tumour secretes a factor that increases capillary permeability: Vascular Permeability Factor (VPF). [4,5];
- contiguous spread consists of local or regional carcinomatosis that originates from a large tumour and crosses the serous membrane to invade neighbouring organs;
- lymphatic route: there are two main routes for lymphatic dissemination:
 - the lymphatic system of the greater omentum,
 - predominantly, the right side of the subphrenic lymphatic system, a site for true tumour cell entrapment, which drains into the anterior mediastinal lymphatic chain, then the right lymphatic duct, and the subclavian vein. When the subphrenic lymphatic system becomes obstructed, ascites result because peritoneal fluid is prevented from being reabsorbed;
- peritoneal surface spread:
 - by redistribution secondary to gravity, the carcinomatosis implants in the superior part of the sigmoid mesocolon, the inferior part of the mesentery, the ileocecal junction, the pouch of Douglas, and the right paracolic gutter,
 - using peristaltic motion, the carcinomatosis follows the peritoneal circulation and implants along the paracolic gutter, passing back up into the undersurface of the diaphragm, becoming implanted in Morison's pouch, the omental bursa, and along the left paracolic gutter.

Key signs of peritoneal carcinomatosis

Peritoneal carcinomatosis is the most common tumour of the peritoneum. Based on a pictorial review of CT imaging, we will describe its key signs as well as the main differential diagnoses encountered in current practice.

Ascites

Non-specific, free or loculated (Fig. 1) and present in 70% of cases [3], there are two main mechanisms causing ascites:

- the main cause is the subphrenic lymphatic vessels becoming obstructed by carcinomatosis, meaning they are unable to carry out their usual function of draining peritoneal fluid;
- this is associated with excess production of peritoneal fluid, resulting from an increase in capillary permeability, which is caused by the tumour cells secreting vascular permeability factor, with protein and albumin accumulating in the abdominal cavity. [6,7]

Greater omentum involvement

The greater omentum or omental apron, also known as the epiploon, is a peritoneal structure formed by the attachment of the two mesenteries of the visceral peritoneum. It begins at the posterior end of the omental bursa. It hangs like an apron from the greater curvature of the stomach and the proximal part of the duodenum, so that it covers the majority of the abdominal organs, in particular, the colon and the loops of the small intestine.

When the greater omentum is affected, this begins by invasion of omental fat, sometimes accompanied by small nodules within the fat (Fig. 2).

In later forms, omental fat is replaced by a solid mass that separates the colon or the small intestine from the anterior abdominal wall, giving the classic appearance of an omental "cake" (Fig. 3).

Invasion of the mesentery

The mesentery is a long double layer of peritoneal tissue that suspends the jejunum and the ileum from the posterior wall of the abdominal cavity.

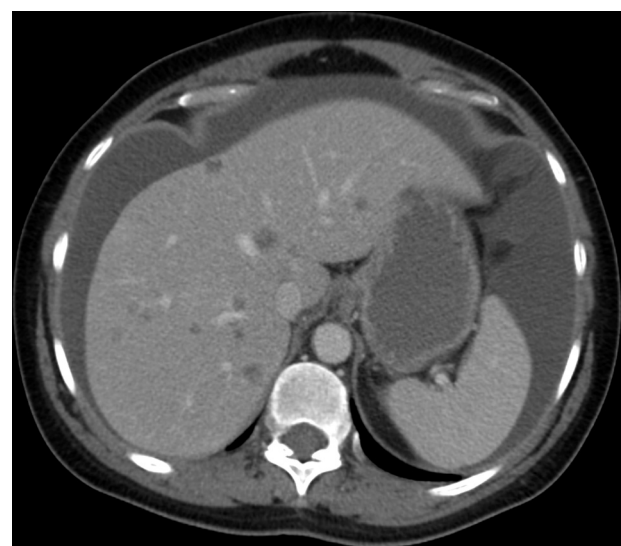


Figure 1. A 42-year old female with a cutaneous melanoma with hepatic metastases and peritoneal carcinomatosis. Free perihepatic and peri-splenic ascites with secondary hepatic lesions.

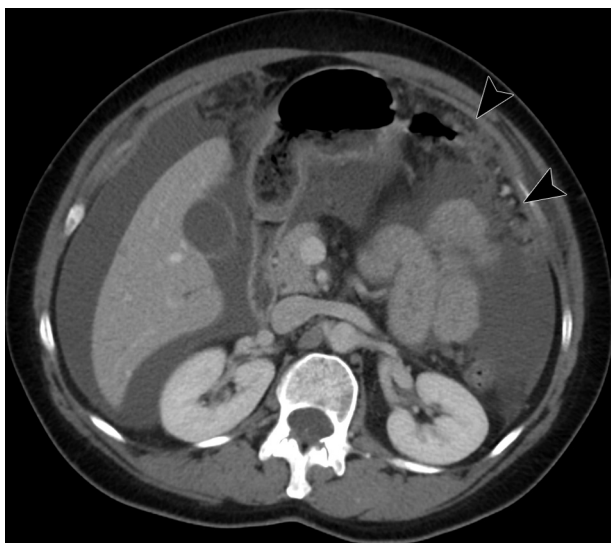


Figure 2. Same patient as Fig. 1: nodular invasion of the greater omentum (arrow heads).

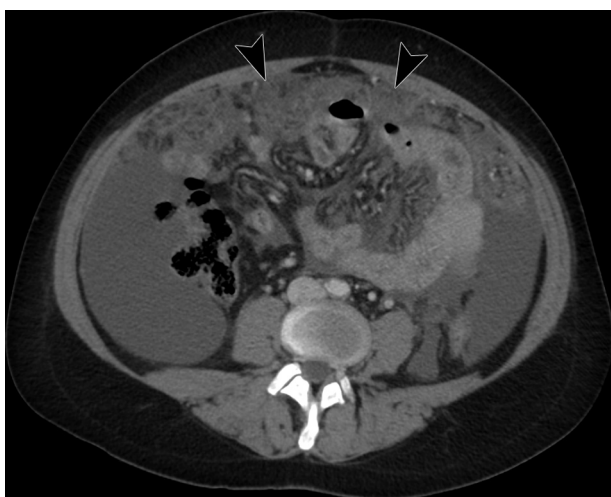


Figure 3. Mass of the anterior greater omentum or omental "cake" (arrow heads).

Invasion of the mesentery can manifest as anomalous fixation of the small intestine, thickening of the stomach walls, increased mesenteric fat density, and the presence of a stellate mesenteric mass or one or more mesenteric nodules that are more or less confluent.

Tumour implants in the peritoneal serous membrane

The peritoneum is the serous membrane of the abdominal and pelvic cavities. It is made up of two parts: the parietal peritoneum, which covers the internal walls, and the visceral peritoneum, which partially or totally covers the organs.

On CT, invasion of the peritoneal serous membrane is seen in the form of nodular or diffuse thickening of these layers, which enhances after contrast material administration. These can be micronodules or true tumour masses.

One of the indirect signs of serous membrane invasion is the adhesion of the small intestine or a segment of bowel becoming fixed to the wall, which blocks the free circulation of ascitic fluid.

It is important to look for tumour implants, as described above, in the paracolic gutters (Fig. 4), the pouch of Douglas (Fig. 5), the sigmoid mesocolon, and the ileocecal junction, but also in the anterior parietal peritoneum (Fig. 6). Involvement of the visceral peritoneum is clearly visible peri-hepatically at the round ligament (Fig. 7) and in the subphrenic space where it can mimic hepatic metastases, causing scalloping to the surface of the liver (Fig. 8).

Diffuse peritoneal invasion may also remain unseen on CT, especially when it consists of a scattered micronodules covering the peritoneum, and these would then only be diagnosed during surgery.

This exhaustive study of signs will produce a score on Sugarbaker's peritoneal cancer index (PCI) [1], taking into account lesion size and location in the peritoneum, and this must be recorded in the notes, preferably accompanied by a diagram (Fig. 9). The peritoneal cancer index has a prognostic purpose, as it allows the cancer's potential for resection to be assessed as well as enabling the response to treatment to be monitored.

Differential diagnoses in peritoneal carcinomatosis

Pseudomyxoma peritonei

Pseudomyxoma peritonei, sometimes incorrectly referred to as gelatinous peritoneal disease, is characterised by the presence of a large amount of mucin in the abdomen.

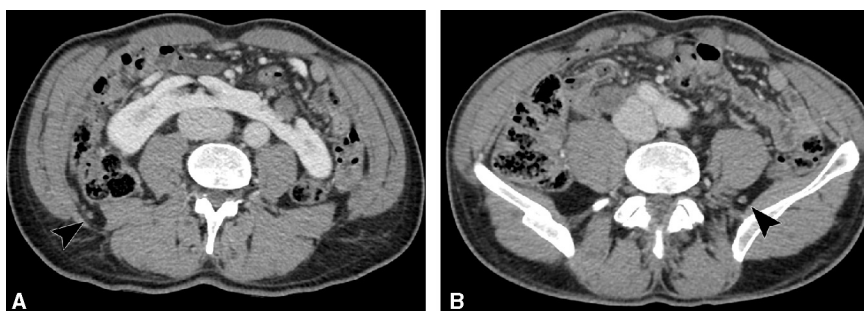


Figure 4. Male with cutaneous melanoma with micronodular peritoneal implants. A. Right paracolic gutter behind the ascending colon (Arrow head). B. Inferolateral rectovesical pouch in the left iliac fossa (Arrow head).

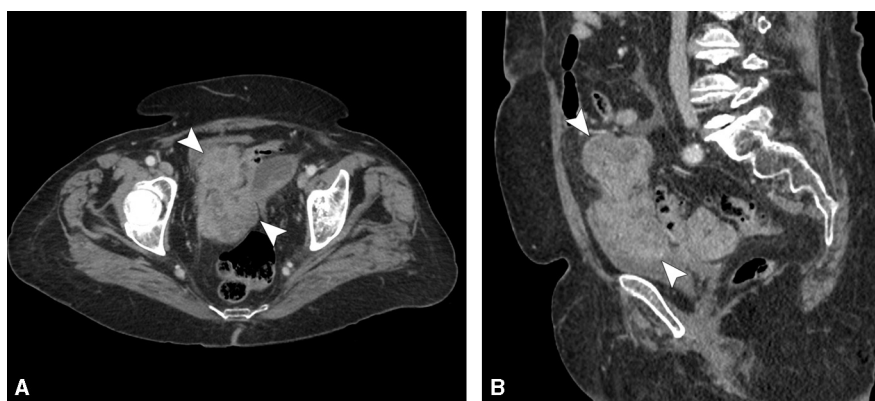


Figure 5. Female followed for carcinoma of the left ovary with peritoneal carcinomatosis. Axial view (A) and sagittal reconstruction (B) showing tumour thickening of the pelvic peritoneum (white arrow heads) and pouch of Douglas.

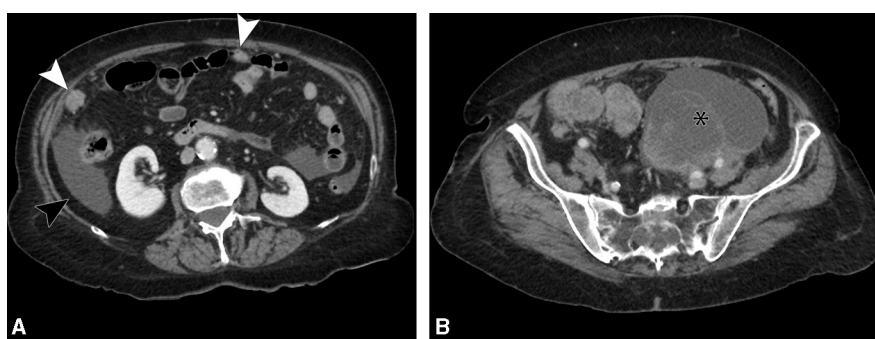


Figure 6. Same patient as Fig. 5. A. Macronodular metastases of the anterior parietal peritoneum (white arrow heads) with ascites (black arrow head). B. Cystic mass of the left ovary (asterisk).

Fundamentally, it is the presence of neoplastic mucin-secreting cells in the peritoneum [8].

Currently, and in the opinions of numerous authors, the primary tumour is thought to originate from the appendix [9,10]. In females, any concomitant involvement of the ovary is thought to be linked instead to a metastatic process that spreads mucinous tumour cells secondary to a ruptured

appendix. However, the theory of an ovarian origin has not been entirely excluded.

The CT signs of pseudomyxoma peritonei are not specific, combining peritoneal effusion, peritoneal nodules, and invasion of the greater omentum. Although these features are very similar to those seen in peritoneal carcinomatosis, there are, however, a number of signs that point to pseudomyxoma peritonei [3,9]:

- the extent to which there is scalloping (Fig. 10), which indicates extrinsic compression of the liver by gelatinous masses;
- loculation of intraperitoneal effusion (Fig. 11);
- calcifications, which are particularly suspicious when they are curvilinear;
- lesions predominating in the greater omentum and the diaphragmatic peritoneum, while the serous membrane of the digestive system is rarely involved;
- Visualisation of a fluid or soft-tissue mass on the appendix.

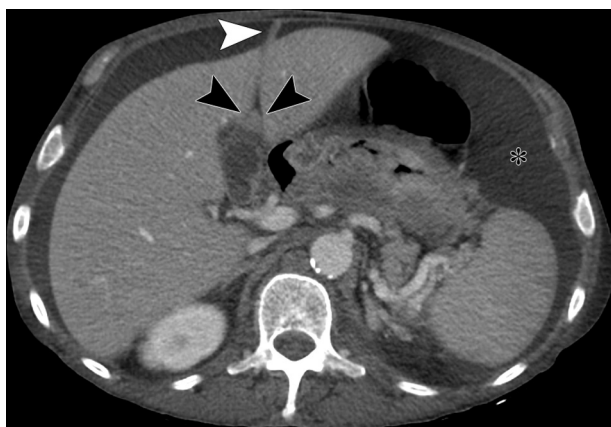


Figure 7. Peritoneal carcinomatosis nodule reaching the visceral peritoneum around the liver at the round ligament (black arrow heads). Note also the thickening of the falciform ligament (white arrow head) in contrast to the ascites (asterisk).

Correctly diagnosing pseudomyxoma peritonei is important, because these patients have prolonged survival [11] if an aggressive surgical approach is taken:

- surgical reduction of the tumour or “debulking” aims to remove the maximum amount of mucinous masses and tumours by dissection; this is in general restricted to a right hemicolectomy, partial resection of the greater omentum, and, for women, a hysterectomy with bilateral salpingo-oophorectomy. This “debulking” must be as



Figure 8. A and B. Axial view and sagittal reconstruction showing a tumour implant in the peri-hepatic visceral peritoneum, mimicking hepatic metastasis (arrow heads).

- thorough as possible, as this is an essential precondition for achieving longer-term survival;
- cytoreduction surgery combined with HIPEC with or without immediate postoperative intraperitoneal chemotherapy is recommended in the majority of centres, specialising in the therapeutic management of PMP [1,12].

Malignant peritoneal mesothelioma

Mesothelioma is a rare primary tumour of the connective tissue that can originate in the serous membranes of the

pleura, peritoneum, or pericardium. Peritoneal involvement is reported in 25% of cases [3,13].

Peritoneal mesothelioma, as with other forms of mesothelioma, is encouraged by asbestos exposure, which is found in one in every two cases.

There are different types of peritoneal mesothelioma that can be divided into four groups: malignant mesothelioma, cystic mesothelioma, adenomatoid tumour, and well-differentiated papillary mesothelioma [14].

Macroscopic features are similar to those seen in peritoneal carcinomatosis, including ascites, diffuse and/or nodular

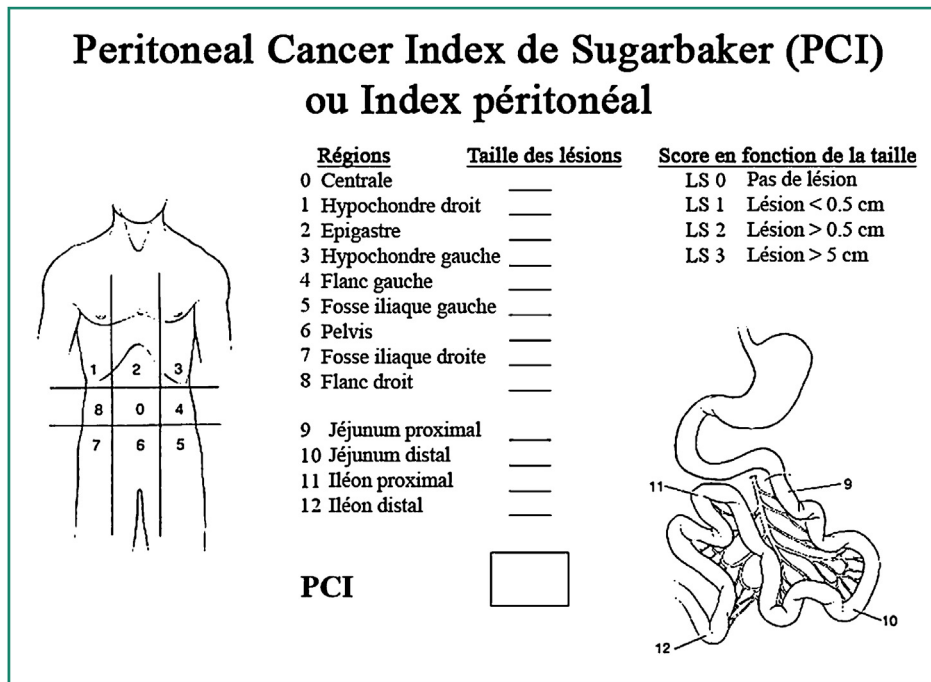


Figure 9. Sugarbaker’s Peritoneal Cancer Index (PCI). The abdominal cavity is divided into 13 regions (from 0 to 12). A score of 0–3 is assigned to each of these regions depending on the sizes of the nodules found there (0: no lesion; 1: lesion ≤ 0.5 cm; 2: lesions ≤ 5 cm; 3: lesions > 5 cm). The sum of these scores produces the PCI, ranging from 1 to 39.



Figure 10. Pseudomyxoma peritonei originating from the appendix. The intraperitoneal collections of mucin produce notches on the hepatic and splenic parenchyma (scalloping).

thickening of the peritoneal serous membrane, invasion of the greater omentum, sometimes with the formation of omental cakes, and mesenteric masses (Fig. 12). As with peritoneal carcinomatosis caused by ovarian cancer or a mucous-secreting gastric cancer, calcification of tumour masses may also be found. While a confirmed diagnosis is not generally established based on pathological assessment, there are, however, a certain number of features from clinical examination and other investigations that point to mesothelioma:

- occupational exposure to asbestos;
- the presence of pleural abnormalities, such as calcified plaques, that are suggestive of exposure to asbestos [3];
- the absence of a detectable primary tumour or secondary lesion of the liver or lymph nodes.

Peritoneal lymphomatosis

Diffuse peritoneal involvement in lymphomatous disease is encountered above all in high grade lymphomas, lymphomas complicating AIDS, and Burkitt lymphomas [15].

In contrast to peritoneal carcinomatosis, peritoneal lymphomatosis can be cured with no surgical intervention. Once again, apart from ascitic fluid that is usually not loculated, invasion of the greater omentum and the

mesentery, and abnormal thickening of the peritoneal membrane (Figs. 13 and 14), there are other signs that allow this diagnosis to be proposed:

- frequent lymph node involvement, associating pre-aortic and retroperitoneal lymphadenopathy. It appears as confluent masses encasing the mesenteric vasculature, producing the “sandwich” sign (Fig. 15) [16]. These masses are bulky, soft, non-obstructing, homogeneous without significant necrosis, and they seem to be less vascularised than carcinomatosis;
- splenomegaly, although this is not always present;
- the presence of tumours in the gastrointestinal tract, especially the stomach and the terminal ileum.

Peritoneal tuberculosis

Tuberculosis is still endemic in developing countries. It is even seeing some resurgence in the developed world [17–19]. Peritoneal tuberculosis accounts for 1–3% of cases, making it the sixth most common extra-pulmonary site of tuberculosis [20,21]. The AIDS pandemic, increased immigration rates, and the ever-increasing use of immunosuppressant drugs are the main causative factors for this [19].

There are three forms of peritoneal tuberculosis [18,21,22]:

- the “wet” type, with abundant ascites and increased density (20–45 HU) due to a high concentration of protein and cells;
- the “fixed fibrotic” type, with peritoneal masses adhering to the adjacent structures of the digestive system and sometimes with loculated ascites;
- the “dry” or “plastic” form, which is less common and causes a fibrous reaction in the peritoneum.

There is often an overlap between the latter two types.

While peritoneal tuberculosis may be difficult to diagnose, there are nonetheless signs that will assist in guiding diagnosis [2,3,21,22] (Fig. 16):

- the presence of mesenteric macronodules;
- enhancement and regular thickening of the parietal peritoneum being identified (Fig. 16C–E);
- splenomegaly and calcifications of the spleen;
- associated involvement of the ileocecal wall;
- retroperitoneal and peri-pancreatic lymphadenopathy with a hypodense centre and ring-enhancement (Fig. 15A and B).

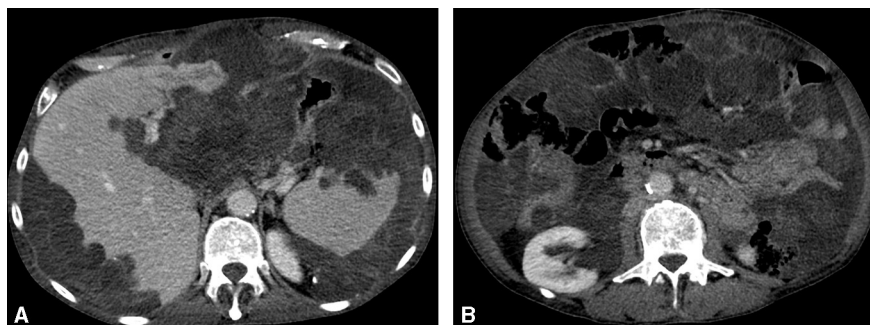


Figure 11. A and B. Multi-loculated gelatinous ascites with hepatic and splenic scalloping in a PMP of appendiceal origin.

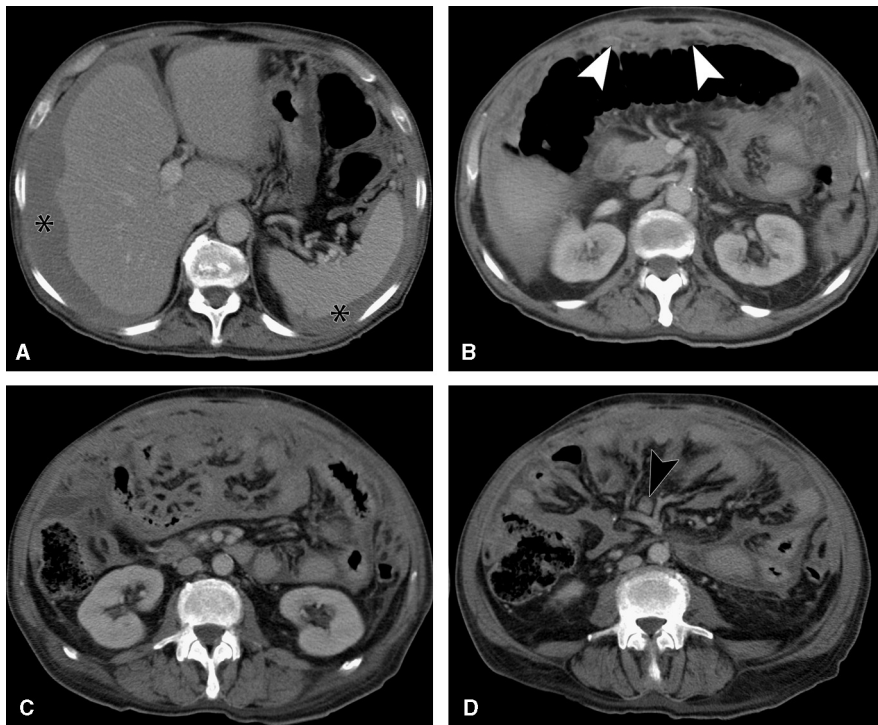


Figure 12. Male with diffuse malignant peritoneal mesothelioma. A. Highly abundant peri-hepatic and peri-splenic ascites (asterisk). B. Tumour mass anterior to the greater omentum (white arrow heads). C and D. Invasion of the mesentery and mesenteric lymphadenitis (black arrow head).

Compared to that seen in peritoneal carcinomatosis, peritoneal thickening is smoother and more regular in tuberculosis [2, 3]. Peritoneal calcifications, especially when seen in mesenteric nodules, are a classic finding, but they are not specific since they may be present in peritoneal metastases from ovarian cancer or a mucous-secreting gastric cancer, and in mesothelioma.

Splenositis implants

Implants of splenositis can mimic tumour implants in the peritoneum, but an assessment of the context looking above all at whether there is a history of splenectomy will allow the correct diagnosis to be made [23]. Often asymptomatic,

diagnosis will usually be made incidentally and should not lead to aggressive management.

The majority of splenositis implants are found after a trauma to the spleen that has undergone splenectomy. At the time of the trauma, fragments can become implanted anywhere in the abdominal cavity, mainly in the peritoneal cavity, but also subcutaneously [24] along the path of the incision, and in the chest if there is a rupture of the diaphragm, specifically in the mediastinum and pleural regions [25].

This is a separate entity from accessory spleen, as this is formed due to incorrect migration of nodules of primitive splenic tissue during embryogenesis.

On computed tomography without intravenous contrast material, splenositis implants are as dense as the hepatic

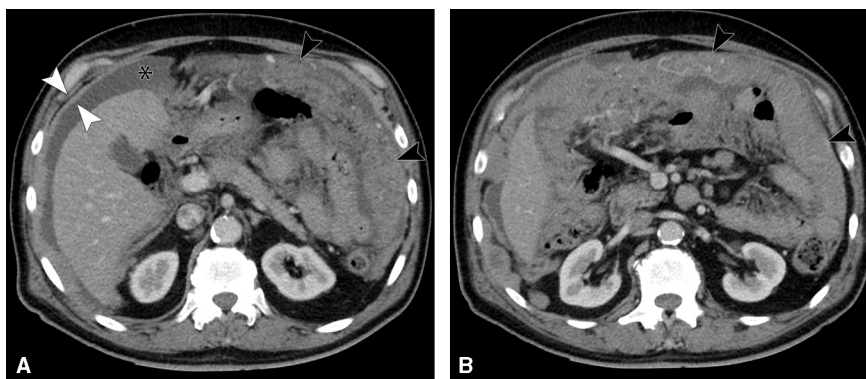


Figure 13. A 63-year old female with a large B-cell lymphoma. A and B. Peri-hepatic ascites (asterisk), associated with a significant tumour invasion of the greater omentum (black arrow heads) and parietal peritoneal thickening (white arrow heads).



Figure 14. Large calcified mesenteric mass (arrow heads) associated with an intraperitoneal effusion (asterisk) in a 69-year old male with follicular lymphoma.



Figure 15. Retroperitoneal lymphomatous mass (arrow heads) encasing the vasculature: "sandwich" sign associated with invasion of the mesentery in a 60-year old male with non-Hodgkin lymphoma.

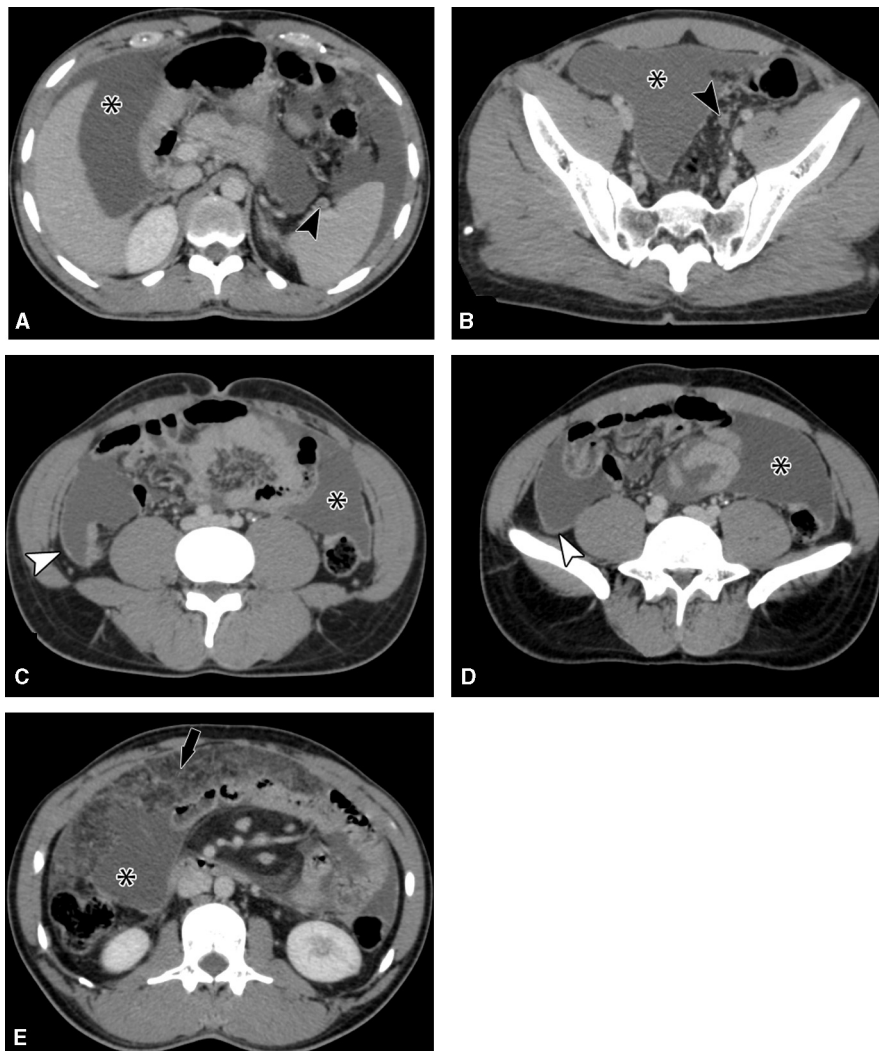


Figure 16. Peritoneal tuberculosis in a 45-year old Senegalese male. A and B. Presence of left iliac and splenic hilar lymphadenopathies with necrotic centre (black arrow heads). C and D. Enhancement and regular thickening of the parietal peritoneum, iliac fossae, and pelvis (white arrow heads) with free ascites (asterisk). E. Invasion of the greater omentum (black arrow) and lymph nodes of the mesenteric root.

parenchyma and well-circumscribed, while their enhancement pattern mimics that of healthy spleen parenchyma, and they are heterogeneous in the arterial phase becoming homogeneous during the portal phase (Fig. 17). There is not usually an associated effusion of ascitic fluid.

On sonography, they are hypo-echoic compared to the liver parenchyma, with well-delineated borders. MRI exploration will find nodules with low signal intensity on T1 and T2 sequences with the same enhancement pattern as seen on CT, and high signal intensity on diffusion-weighted sequences. A technetium-99m red blood cell scintigraphy will also allow the diagnosis of splenosis to be confirmed [23].

Diffuse peritoneal leiomyomatosis

Diffuse peritoneal leiomyomatosis (DPL) is a rare and benign disorder of unknown origin [24] that is characterised by the presence of myoma nodules in the peritoneum that have similar histologic features to those of uterine leiomyomas (smooth muscle fibres) [25].

The CT finding of disseminated peritoneal nodules can give rise to fears of peritoneal carcinomatosis [24].

It is most often found in black women of childbearing age, and in 70% of cases, it is associated with the use of oestrogen–progesterone contraception or pregnancy, which strongly suggests that this proliferation of smooth muscle cells is hormone-dependent [26]. Only one case of PDL has been reported in a male [27].

Although principally located in the pelvic peritoneum and greater omentum, they can also be found in the uterus, ovaries, and on the visceral side of the intestinal peritoneum. Nodules are less common in the superior areas of the peritoneum.

On computed tomography (Fig. 18), multiple diffuse peritoneal nodules are seen, associated with a pelvic soft-tissue mass with multiple lobules that displaces the pelvic organs. The tumour shows delayed enhancement. There is no associated lymphadenopathy or gastric wall thickening.

The absence of ascites and hepatic metastasis points diagnosis away from peritoneal carcinomatosis.

DPL generally has a good prognosis, with nodules regressing once oral contraceptives are discontinued. However, cases of recurrence at a later stage and malignant transformation have both been reported [28–31].

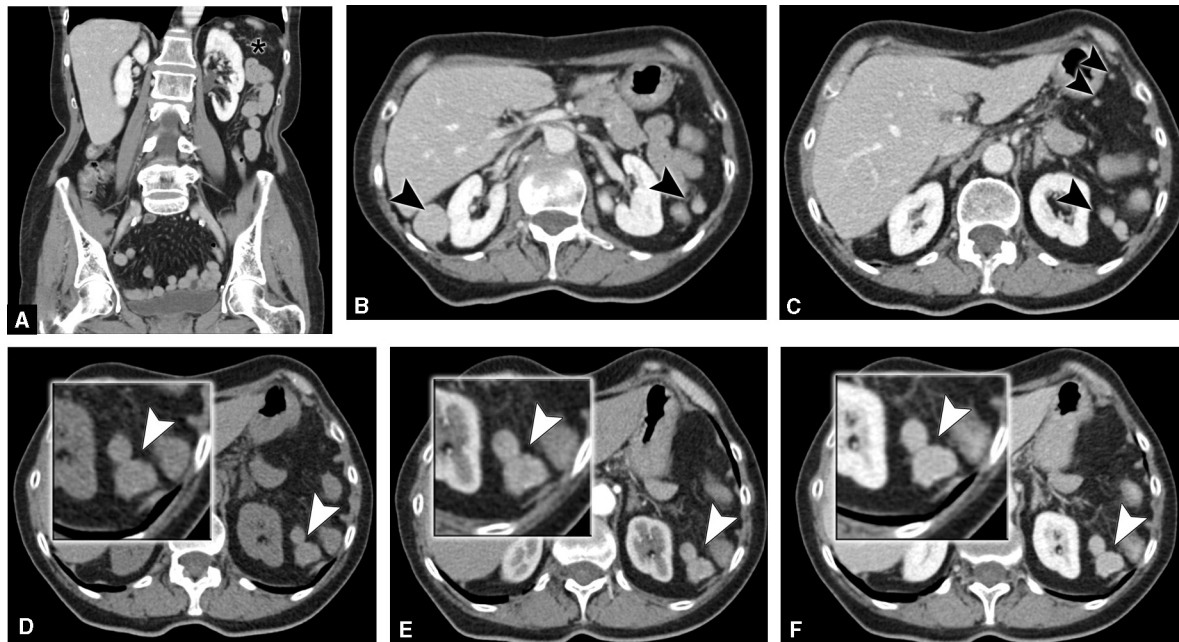


Figure 17. Incidental sonographic discovery of soft-tissue lesions that are splenosis implants in a 45-year old female with a history of splenectomy following a road accident. A. Note the history of splenectomy (asterisk). Presence of peritoneal tissue lesions (black arrow heads) in the splenectomy site (C) and in the right peri-renal space (B). An enhancement study (white arrow heads) found isodensity to the hepatic parenchyma before administration of intravenous contrast material (D), then a non-homogeneous appearance in the arterial phase (E), becoming homogeneous in the venous phase (F).

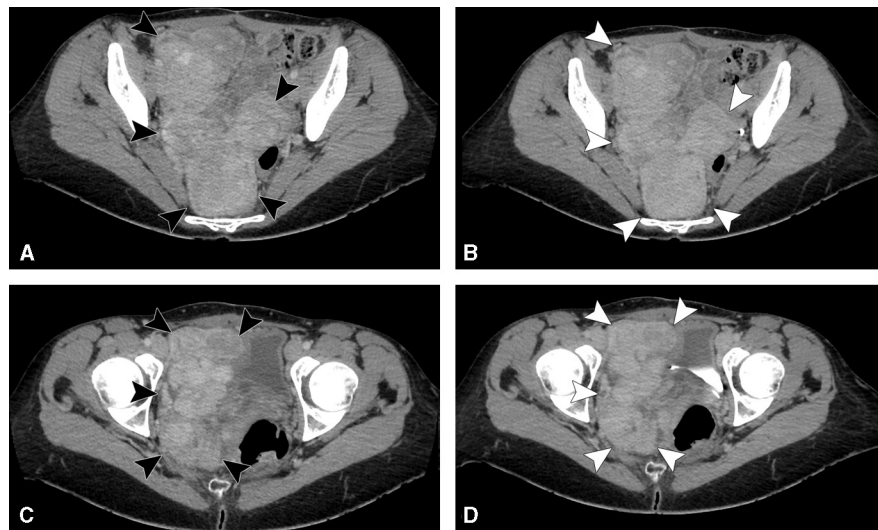


Figure 18. A 45-year old female was investigated for a feeling of pressure in the pelvis leading to the incidental discovery of diffuse peritoneal leiomyomatosis (DPL) confirmed histologically by diagnostic peritoneoscopy. A and C. Large pelvic soft-tissue mass displacing the adjacent organs, enhancing progressively and non-homogeneously in the venous phase (black arrow heads). B and D. It becomes homogeneous and enhances more markedly in the delayed phase at 5 min (white arrow heads).

Conclusion

Invasive peritoneal disease includes more than just peritoneal carcinomatosis (Table 1). There are quite a number of differential diagnoses that are easy to recognise or propose based on a rigorous assessment of computed tomography features and the clinical context. Although not an exhaustive

list, peritoneal lymphomatosis, especially Burkitt lymphoma and large cell lymphomas (concomitant involvement of retroperitoneal lymph nodes and a tumour), malignant peritoneal mesothelioma (asbestos exposure), pseudomyxoma peritonei (scalloping), and peritoneal tuberculosis (necrotic lymph nodes and calcifications) are the main disorders that mimic peritoneal carcinomatosis.

Table 1 Causes of diffuse peritoneal invasion and key computed tomography features.

	Ascites	Peritoneal features	Greater omentum features	Mesenteric features	Other associated signs
Carcinomatosis	Present in 70% of cases, free or loculated	Irregular thickening and enhancement Peritoneal nodules present	Invasion of the fat of the greater omentum with nodules in later forms, produces the renowned omental cake	Invasion of the fat of the mesentery with stellate masses and nodules	Presence of a primary tumour
Mesothelium	Ascites, diffuse and/or nodular thickening of the peritoneal serous membrane, invasion of the greater omentum, sometimes with the formation of omental "cakes", and mesenteric masses may be seen				Asbestos exposure Pleural plaques No detectable primary tumour
Pseudomyxoma peritonei	Multi-loculated, ascites compressing other structures to cause scalloping on the visceral organs	Predominates in the diaphragmatic peritoneum and greater omentum while the serous membrane of the digestive system is rarely invaded		Rare	Curvilinear calcifications Appendiceal mass
Tuberculosis	Wet type comprises ascites with increased density due to their high protein content	Regular thickening and enhancement of the peritoneal serous membrane	Invasion of the mesentery and serous membrane of the greater omentum with macronodules present		Lymphadenopathy with a necrotic centre, involvement of the ileocecal wall Calcifications in the spleen
Lymphoma	Present, and in most cases not loculated	Tumour invasion of the greater omentum or mesentery and abnormal thickening of the peritoneal serous membrane may be seen			Retroperitoneal lymphadenopathy with sandwich sign Splénomegaly Gastrointestinal tract involvement
Splenosis	Absent	Solitary or multiple nodules with enhancement pattern mimicking that of the splenic parenchyma		Rare	History of splenectomy
Diffuse peritoneal leiomyomatosis (DPL)	Absent	Diffuse soft-tissue nodules Pelvic soft-tissue mass	Common (multiple nodules)	Possible (nodules)	Context of hormonal change (pregnancy, oral contraception, hormone-secreting tumour etc.) No deterioration of patient's general health

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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