Unravelling the Mysteries of the Mesentery

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Abstract
The mesentery and its folds tether the small bowel loops to the posterior abdominal wall. It transmits nerves, vessels, and lymph ensconced in a fatty sponge layer wrapped in a thin glistening peritoneum, from and to the small bowel. Not only does this flexible dynamic fatty apron house various localized primary benign and malignant lesions, it is often involved in and gives an indication of generalized or systemic diseases in the body. An understanding of the anatomy, components, and function of the mesentery helps to classify mesenteric abnormalities. This further allows for characterizing radiological patterns and appearances specific to certain disease entities. Recent reviews of mesenteric anatomy have kindled new interest in its function and clinical applications, heralding the possibility of revision of its role in diseases of the abdomen.

Keywords
► mesentery
► diseases
► masses
► nonneoplastic

Introduction
New concepts in mesenteric anatomy have brought it to the limelight in recent years as a possible new organ.¹ Traditionally the mesentery is defined as a double layer of peritoneum that encloses an organ (bowel) and attaches it to the posterior abdominal wall. It is a supple lipomatous structure that allows and controls movement of the bowel. It transmits the vascular, lymphatic, and neural network that supplies the bowel. In its supporting role of the bowel, it gets intimately involved in the pathologies and diseases of bowel. However, it also retains its identity as an independent entity by virtue of its contents. This review aims to help gain an understanding of primary and secondary diseases of the mesentery both mass and nonmass abnormalities and the varied radiological patterns of presentation. It also attempts to categorize diseases of mesentery with a case-based approach to direct interpretation when presented with a mesenteric abnormality. It briefly touches upon historical and traditional concepts of the mesentery, as well as current and future concepts, to engage the reader’s interest in the possibility of new developments in the role of the mesentery in disease management.

Historical Outline
The earliest description of the “mesenterium” as a continuous membrane subtermining the bowel dates back to Aristotle.² Thereafter in the 18th century, Toldt and Rosa revisited the anatomy of the mesentery and described a fascial plane found between the mesothelium of the mesocolon and the retroperitoneum that contained lymphatics.³⁻⁵ However, the current and traditional approach to mesenteric anatomy has been derived from the lectures published and expounded by a Royal British Surgeon to Queen Victoria, Sir Frederick Treves,⁴ and the 19th century radiologist Wylie J. Dodds who propounded the presence of a primary retroperitoneum and a secondary/anterior retroperitoneum “bounded by folded fused leaves of mesentery that contained distinct pancreatocoduodenal and colonic subcompartments.”

Anatomy of the Mesentery
The abdomen and its organs are lined by parietal and visceral peritoneum. The reflections of the peritoneum around the solid and hollow organs of the abdomen connect them to...
one another and the posterior abdominal wall, holding them within the peritoneal cavity. The nerves to the small bowel from the splanchnic system have both sympathetic and parasympathetic components that course through the mesentery, but the definitive anatomy of these fibers in the mesentery is yet not clearly defined. Lymphatic vessels and nodes in the mesentery complete the mesenteric structure and contribute to its physiology and pathology.

Traditionally, a true mesentery is one that connects bowel to the posterior abdominal wall as follows:

- The small bowel mesentery proper containing the superior mesenteric artery (SMA), vein, and its branches.
- The transverse mesocolon containing the middle colic artery vein and its branches.
- The sigmoid mesocolon containing the sigmoid artery and vein and the superior rectal vessels.

The root of the small bowel mesentery is approximately 15-cm long, extending from the left of L2 vertebra at the duodenojejunal flexure to the ileocecal valve. It communicates with the transverse mesocolon at the formation of the gastrocolic trunk and with the hepatoduodenal ligament via the superior mesenteric vein (SMV) and portal vein. The transverse mesocolon divides the peritoneal cavity into supramesocolic and inframesocolic compartments, while the root of the small bowel mesentery divides the inframesocolic compartment into right and left infracolic spaces directing the flow of fluid within the abdomen.

**Imaging of the Mesentery**

**Conventional Radiography**

The mesentery being a predominantly fat-filled structure cannot be adequately imaged by conventional radiography. Calcifications and displacement of bowel loops due to masses may be seen on radiographs of abdomen and direct further imaging.

**Ultrasound**

Ultrasound (US) is the most accessible modality for the initial screening of the abdomen in both adults and children. Being freely available and radiation free, it is the investigation of choice in any patient presenting with an intra-abdominal mass.

**Barium Studies**

Mesenteric lesions, such as lymphoma, carcinoid, metastases, and tumors, may be seen as separation and or displacement of bowel loops in a barium meal follow-up through examination. However, there is currently no role for barium studies or enteroclysis in the evaluation of the mesentery.

**Computerized Tomography**

Multidetector computerized tomography (MDCT) with intravenous contrast-enhanced CT (CECT) with or without positive or negative oral contrast is possibly the frequently used and rational cross-sectional imaging modality for assessing and characterizing the mesentery and its diseases, with the added advantage of reconstruction in multiple planes. Volume and three-dimensional (3D) rendering enhance viewing aesthetics.

![Diagrammatic representation of the small bowel mesentery.](image-url)
Magnetic Resonance Imaging
Although magnetic resonance imaging (MRI) lends itself to high soft-tissue resolution, MRI is used less commonly in routine practice for mesenteric abnormalities, being used more often as a problem-solving tool, especially in the characterization of tumors. Longer time of acquisition, as well as respiratory and bowel motion artifacts, may limit its usefulness depending on the case scenario. MR enterography is currently an important imaging tool used in assessment and follow-up of inflammatory bowel disease in both pediatric and adult patients.

Mesenteric Diseases
Mesenteric diseases have been categorized variously by various authors. In this review, mesenteric pathologies have been differentiated under two sections: mass like lesions and nonmass lesions of mesentery. Mass and mass-like lesions have been discussed and categorized simply as cystic, solid, and fatty lesions. Commonly and uncommonly encountered systemic, mechanical, and vascular entities involving the mesentery have been discussed under nonmass lesions of the mesentery for completion (►Fig. 3).

Cystic Lesions of the Mesentery
Cystic mass and mass like lesions of the mesentery can be further discussed as follows (►Table 1):

#### Pseudomyxoma Peritonei and Mucinous Carcinomatosis
Low-grade pseudomyxoma peritonei (diffuse peritoneal adenomucinous type) is an uncommon syndrome with recurrent voluminous noninvasive accumulation of mucin in the peritoneal cavity, arising from a low-grade mucinous adenocarcinoma usually of the appendix. Mucin in the abdomen appears very much like ascites and cannot be distinguished from ascites on plain radiography. At US hypoechoic mucin contains nonmobile echoes and displaces small bowel loops due to mass effect. This mass effect of hypodense collections of mucin in the perihepatic and perisplenic spaces is seen as scalloping of the borders of the liver and spleen, without invasion or infiltration on CT and helps to distinguish it from ascites. Pseudomyxoma peritonei has a relatively better prognosis as it abuts and displaces small bowel and mesentery without invasion and therefore being more amenable to debulking (►Fig. 4D).

Mucinous tumor nodules disseminated from high grade, moderately or poorly differentiated mucin producing tumors of appendix, ovary, colon, pancreas, and gall bladder are hypodense lesions with or without calcification (►Fig. 4A, B)

#### Table 1  Cystic masses of the mesentery

<table>
<thead>
<tr>
<th>Common</th>
<th>Uncommon</th>
<th>Rare</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mucinous carcinomatosis</td>
<td>Pseudomyxoma peritonei</td>
<td>Nonpancreatic pseudocyst</td>
</tr>
<tr>
<td>Abscess</td>
<td>Mesenteric cysts</td>
<td></td>
</tr>
<tr>
<td>Pseudocyst of pancreas</td>
<td></td>
<td></td>
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<tr>
<td>Necrotic nodes</td>
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</tr>
</tbody>
</table>

Fig. 3  Classification of mesenteric diseases.

Fig. 4  Mucinous carcinomatosis: (A) A 51-year-old lady known carcinoma ovary, posthysterectomy presenting with hypodense mucinous metastatic deposits invading into the surface of right lobe of liver (arrows) and (B) deposits in the small bowel mesentery and serosa (arrows). (C) 21-year-old male with metastatic poorly differentiated mucinous adenocarcinoma of the rectosigmoid demonstrating mucinous deposits with punctate calcification (arrows). (D) Primary pseudomyxoma peritonei in a 74-year-old patient with past surgical history of mucinous appendiceal neoplasm on regular follow up, presenting with scolapping mucinous deposits on liver (long arrow) and recurrent mucin in the abdomen (short arrow).
deposited on mesentery, serosal, and peritoneal surfaces. Calcification within the mucinous deposits are likely to be amorphous, scattered punctate, or punctate clusters (Fig. 4C). This condition is sometimes termed high-grade pseudomyxoma peritonei or peritoneal mucinous carcinomatosis, indicating direct invasion and infiltration of peritoneal surfaces by cellular components of the deposits. At surgery, these deposits are prone to rupture with spillage into the peritoneal cavity, hence having a poorer prognosis. Multidetector CT with reformatted sections allows for detection of 5- to 10-mm hypodense nodules with or without soft-tissue attenuation which could represent solid tumor elements, compression of mesentery, or fibrosis. MRI allows for excellent depiction of bright hyperintense mucinous deposits on T2-weighted fat-suppressed and postcontrast T1-weighted images in hidden areas, having a greater sensitivity for smaller nodules but is often not the primary modality of choice due to cost constraints and longer time of study.

Mesenteric Abscess, Infections
Intra-abdominal abscesses are commonly associated with diverticulitis, appendicitis, or surgeries. Postoperative mesenteric abscess (Fig. 5A) and pancreatitis-related fat necrosis with mesenteric abscesses are diagnosed in the appropriate clinical setting. Mesenteric abscesses of all sizes are best seen at CECT as focal interloop/intra-abdominal irregular or well defined peripherally enhancing, thin- or thick-walled hypodense lesions with or without septations and with or without pockets of air within.

Extraintestinal manifestations of severe Crohn's disease with mesenteric abscesses, interloop fistulae (Fig. 5B) and enhancing enlarged nodes are also best seen at CECT/CT enterography. Findings of mesenteric hyperemia, comb sign, mesenteric fat proliferation, and bowel changes add to diagnostic confidence of Crohn's disease.

Manifestations of abdominal tuberculosis closely resemble primary and secondary mucinous/nonmucinous peritoneal carcinomatosis with conglomerate necrotic mesenteric nodal masses presenting as mesenteric abscesses and loculated fluid appearing as mesenteric cystic collections. Ancillary imaging features of tuberculosis, such as splenomegaly, calcified granulomata in the liver, spleen or nodes, and miliary microabscesses in the liver and spleen may help lead to diagnosis. These may be associated with diffuse peritoneal enhancement, adjacent solid and necrotic nodes, omental caking, and ascites with or without radiological features of ileocecal tuberculosis (Fig. 5C, D).

Congenital and Acquired Mesenteric Cysts
Cystic mesenteric lesions can be congenital or acquired. Prenatal diagnosis of intra-abdominal simple cysts raises the possibility of multiple differentials. Postnatal US assessment allows further characterization with respect to location (relationship to bowel or ovary), cyst wall morphology (gut signature in duplication cyst), internal debris (mesenteric, ovarian, and duplication cyst) (Fig. 6A-C), fat content or calcification (dermoid), and wall calcification (meconium pseudocyst).

A mesothelial cyst is uncommon and demonstrates a single layered wall with no internal content or septations. Anatomically, an enteric duplication cyst occurs commonly along the mesenteric border of the ileum, and is characterized by a multilayered wall at US demonstrating the “bowel signature.” However, differentiation of duplication cyst from mesenteric and ovarian cysts on US remains challenging, especially when they are large. In a female neonate, an abdominopelvic cyst with debris needs to be considered ovarian in origin if one ovary cannot be separately visualized. There may be a “daughter cyst” within an ovarian cyst which represents a smaller cyst within a larger cyst.

Calcification in the wall of a unilocular cyst in a neonate should raise the possibility of a meconium pseudocyst (Fig. 6D, E). They commonly have an irregular shape and insinuate into peritoneal contours.

A multiloculated intra-abdominal “infiltrative” cystic lesion is more commonly a lymphangioma. Lymphangioma may appear like an ascites in neonates on US. Absence of free fluid in the Morrison's pouch, paracolic gutters, and pelvic recesses allows differentiation from ascites. Further imaging with MRI/CT is indicated only as a problem-solving tool. These lesions demonstrate variable appearances on CT and MR depending on the presence of proteinaceous content, hemorrhage, or fat. These lesions may be closely adherent to the bowel, necessitating bowel resection.

In adults, the differentials of mesenteric cystic lesions include congenital and acquired cysts (Table 2). In addition to the cysts described above, the possibilities of pancreatic pseudocyst (Fig. 7A), nonpancreatic pseudocyst, and hydatid cyst need to be considered.

Nonpancreatic pseudocysts (Fig. 7B, C) appear as thick walled septate lesions which may contain hemorrhage or...
pus. They are remnants of prior mesenteric abscess or hematoma, often detected incidentally and may be mistaken for a hydatid cyst.19

Intra-abdominal hydatid cysts (►Fig. 7D) may be single or multiple. While CT demonstrates extent of intra-abdominal dissemination of hydatids, US plays a key role in diagnosis of hydatid cysts with characteristic findings of daughter cysts, spoke wheel pattern, water lily sign (complete detachment of endocyst from pericyst), and floating membrane sign (partial separation of endocyst from pericyst).23 MRI is used for

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**Table 2** Classification of mesenteric cysts

<table>
<thead>
<tr>
<th>Type</th>
<th>Wall</th>
<th>Loculation</th>
<th>Enhancement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple cystic</td>
<td>Enteric duplication cyst</td>
<td>All layers of the gut</td>
<td>Unilocular ± debris</td>
</tr>
<tr>
<td>Enteric cyst</td>
<td>Enteric epithelium</td>
<td>Unilocular</td>
<td>Thin non enhancing wall</td>
</tr>
<tr>
<td>Mesothelial cyst</td>
<td>Mesothelium</td>
<td>Unilocular</td>
<td>Thin non enhancing wall</td>
</tr>
<tr>
<td>Mildly complex cystic lesion</td>
<td>Lymphangioma</td>
<td>Lymphatic/endothelial lining</td>
<td>Multilocular-chylous, serous/hemorrhagic</td>
</tr>
<tr>
<td>Pseudomyxoma peritoneum/mucinous deposits</td>
<td>Avascular mucinous deposits</td>
<td>Multilocular ± internal echoes</td>
<td>No enhancement</td>
</tr>
<tr>
<td>Nonpancreatic pseudocyst</td>
<td>Fibrotic thick wall ± wall calcification</td>
<td>Unilocular</td>
<td>Thick wall ± enhancement</td>
</tr>
<tr>
<td>Pancreatic pseudocyst</td>
<td>Fibrotic thick wall</td>
<td>Unilocular</td>
<td>Uniform wall enhancement</td>
</tr>
<tr>
<td>Infectious tuberculosis/Crohn’s disease</td>
<td>Loculated ascites, necrotic nodal mass Abscesses</td>
<td>Multiunilocular</td>
<td>Rim enhancement</td>
</tr>
<tr>
<td>Infectious hydatid</td>
<td>Outer pericyst, middle laminated membrane and inner germinal layer26</td>
<td>Multiloculated ± rim calcification or total calcification27</td>
<td>Enhancing wall if infected</td>
</tr>
<tr>
<td>Cystic with solid components</td>
<td>Cystic mesenchymal tumors, GIST</td>
<td>Necrotic tumor mass</td>
<td>Unilocular with rim ± solid component</td>
</tr>
</tbody>
</table>

Abbreviation: GIST, gastrointestinal stromal tumor.
reviewing complications of hydatids such as infection, cyst rupture, and intra-abdominal dissemination, in which case it may appear as a multiloculated mass (known as encysted peritoneal hydatidosis).23,24

Classification of cystic lesions as simple cystic (mesothelial and duplication cysts), mildly complex (lymphangioma, pseudomyxoma peritonei, pseudocyst, hydatid, and abscess), and cysts demonstrating solid components (malignancies) may help characterize mesenteric cystic lesions in adults on imaging.22

**Solid Lesions of Mesentery (Nonnodal)**

Solid nonnodal masses of the mesentery encompass several entities, the most common being metastases.25,26

> **Table 3** lists nonnodal solid mesenteric masses.

<table>
<thead>
<tr>
<th>Common</th>
<th>Uncommon</th>
<th>Rare</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peritoneal metastases</td>
<td>Carcinoid</td>
<td>Inflammatory pseudotumor</td>
</tr>
<tr>
<td>Primary peritoneal carcinoma</td>
<td>Desmoid tumor</td>
<td>Soft tissue sarcoma</td>
</tr>
<tr>
<td>Desmoid tumor</td>
<td>Sclerosing mesenteritis</td>
<td>Desmoplastic round cell tumor</td>
</tr>
<tr>
<td>Gastrointestinal stromal tumor</td>
<td></td>
<td>Sclerosing mesenteritis</td>
</tr>
</tbody>
</table>

**Metastases**

Mesenteric invasion by metastases may be by blood, lymph, peritoneal seeding, and direct spread. Breast cancer, lung cancer, and melanoma spread to the mesentery by hematogeneous and lymphatic routes. Ovarian, pancreatic, breast, and colonic cancers spread commonly by peritoneal seeding, while direct extension into the mesentery occurs from pancreas and stomach.25

Calciﬁed mesenteric deposits from ovary and mucinous tumors of the colon and stomach are usually associated with similar deposits in the peritoneum and omentum, and may thus be differentiated from calciﬁed retractile mesenteritis and mesenteric carcinoid.26

Metastatic and lymphomatous nodal masses of the mesentery are rather common and will be discussed under nodal masses.

Sclerosing mesenteritis is a relatively uncommon inﬂammatory lesion of the mesentery (Fig. 8A, B) seen predominantly in men in the sixth and seventh decades.26,27 Causation is yet undeﬁned, but a strong association with prior abdominal surgery and immunoglobulin (Ig)-G4 related disease is documented. Sclerosing mesenteritis as a harbinger of lymphoma, urogenital, and gastrointestinal malignancies is yet unproven.26

There are three histologic subtypes of sclerosing mesenteritis, or rather stages, namely, (1) mesenteric lipodystrophy (representing the ﬁrst stage of fat necrosis and more of a pathological diagnosis), (2) mesenteric panniculitis, and (3) retractile mesenteritis.28 Imaging with CECT for abdominal pain and discomfort with or without fever demonstrates a mass-like soft-tissue attenuation inﬁltrating fat in the root of mesentery, referred to as mesenteric panniculitis in radiology parlance. The extent of inﬂammatory activity is deﬁned by a thin line of soft tissue of less than 3-mm thickness, the tumoral pseudocapsule (Fig. 8A, B). The fat ring sign (Fig. 8B) is seen as a halo of fat sparing around mesenteric vessels within.29 In addition, the inﬂammatory mass may contain multiple soft tissue nodules, usually less than or equal to 5 mm. Any nodule greater than 10 mm is concerning for malignancy and should be biopsied.25,30 These signs are key in differentiation of mesenteric panniculitis from “misty mesentery” in mesenteric edema or hemorrhage.31

Rearranged mesenteritis occurs when inﬂammation progresses to ﬁbrosis and a desmoplastic reaction results in a spiculated lobulated soft-tissue mass with or without internal cavitation or calcification. This causes shortening of the

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Fig. 7 Adult mesenteric cysts. (A) A young male with prior history of pancreatitis on follow up. CT demonstrates a pancreatic pseudocyst (arrows). (B, C) Incidentally detected well defined thick-walled mesenteric root cyst with wall calcification (blue arrow). Patient had no prior pancreatitis but gives history of prior trauma which is corroborated by old fracture D12 (yellow arrow) seen on sagittal reconstruction. In this context, a diagnosis of nonpancreatic pseudocyst was suggested and followed up. (D) Adult presented with suspected abdominal mass. CT demonstrated a well-defined lesion with rim calcifications and subtle internal vesicular cysts, hydatid cyst. CT, computed tomography.

Fig. 8 Sclerosing mesenteritis (A) showing tumoral pseudocapsule (yellow arrows) (B) fat ring sign (curved red arrow) and nodules (blue arrows).
mesentery, kinking, and fixation of small bowel leading to small bowel obstruction.\textsuperscript{26,29}

MRI in sclerosing mesenteritis demonstrates mildly hyperintense T1-enhancing soft-tissue mass, heterogeneously hyperintense, or diffusely hypointense on T2 depending on the amount of fibrosis.\textsuperscript{25} They do not demonstrate fluorodeoxyglucose (FDG) positron emission tomography (PET) avidity and thereby can be differentiated from lymphoma and other malignancies.\textsuperscript{32}

Carcinoid tumor forms a very close differential for sclerosing mesenteritis. Primary mesenteric carcinoid is very rare.\textsuperscript{25,33} The mesentery is involved as a result of direct extension or lymphatic spread from a bowel primary, forming a hyper enhancing spiculated soft-tissue mass (►Fig. 9A–C) with a stellate pattern as a result of proliferative desmoplastic reaction to the neuroendocrine substances released.\textsuperscript{26} The distal ileum, rectum, appendix, colon, and stomach are the most common sites of origin.\textsuperscript{34} Calcification is seen within this metastatic mass in approximately 70\% of cases.\textsuperscript{35} Encasement of vessels and mesenteric infiltration may result in small bowel obstruction and ischemia.\textsuperscript{36}

The fat ring sign is absent in metastatic mesenteric carcinoid. Often the primary carcinoid tumor in the bowel is undetectable, often being less than 2 cm in size. On CECT, the primary tumor enhances vividly with contrast. Octreotide scan is the most sensitive imaging modality for the diagnosis of neuroendocrine tumors and its metastases to lung, liver, peritoneum, and nodes.\textsuperscript{26} They are often associated with multiple endocrine neoplasia, neurofibromatosis type 1, and other coexisting abdominal adenocarcinomas\textsuperscript{37,38} (►Fig. 9A).

Desmoid tumors are made of proliferating fibroblasts with intracellular collagen, sporadically seen in women in the third and fourth decades but also seen in patients with prior abdominal surgery or trauma. Other associations include Gardner’s syndrome and familial adenomatous polyposis syndrome (FAPS).\textsuperscript{39}

The site of origin of desmoid may be from abdominal wall, extra-abdominal or intra-abdominal. Within the abdomen, the mesentery is the preferred site of origin of these slow growing desmoids, with a majority of these lesions arising from near the SMA origin.\textsuperscript{40} Though they do not metastasize, they are known to be locally aggressive, infiltrating adjacent bowel, and vessels with stubborn recurrences.\textsuperscript{26}

The propensity for recurrence and the tendency to aggression are, however, unpredictable. On CT and MRI, imaging and enhancement characteristics (►Fig. 9D) are subject to the proportions of fibrous and cellular components. They can therefore be T2 hyperintense (myxoid components) to T2 hypointense (dense collagenous stroma) with mild-to-moderate enhancement patterns reflecting similar heterogeneity. Preoperative MRI can be particularly helpful in delineating soft-tissue character and planes. Notably fluid signal intensity with heterogeneous T2 hyperintensity and vivid postcontrast enhancement seen in postoperative follow-up MRI are indicators of growth as opposed to T2 hypointense residual bland fibrous residue.\textsuperscript{40,41}

Inflammatory myofibroblastic tumors are uncommon tumors of mesenchymal origin seen in young adult girls and children, largely in lung, mesentery, and omentum. Though

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{fig9.png}
\caption{Coronal arterial phase CT abdomen: avidly enhancing solid mass along the medial wall of the duodenum (red arrows), duodenal carcinoid with similar avidly enhancing nodal metastases at root of mesentery (blue arrows). There is an associated gall bladder adenocarcinoma (yellow arrows). (B, C) Axial portal phase contrast enhanced CT abdomen: Mildly enhancing irregular spiculated lesion in the mesentery (curved yellow arrow), associated with bowel wall edema in adjacent small bowel loops (blue arrows) due to venous ischemia, mesenteric carcinoid. (D) Axial arterial phase contrast enhanced CT: Solid hypodense mass in the mesentery with no hyperenhancement (blue star) - mesenteric desmoid. (E) Axial portal-venous phase contrast enhanced CT of a 7-year-old girl shows a hypodense circumscribed mass with internal heterogeneous as well as peripheral enhancement (arrows), inflammatory myofibroblastic pseudotumor. CT, computed tomography.}
\end{figure}
considered to be neoplastic, they are often associated with elevated inflammatory markers.42-44

They may be echogenic, iso-, or hypoechoic on US with internal vascularity and demonstrate heterogeneous enhancement patterns (►Fig. 9E), sometimes with early peripheral enhancement and delayed fill in on CECT owing to fibrous components. They are FDG PET avid and on MRI are iso- or hypointense to muscle depending on the internal fibrous content.40

Gastrointestinal stromal tumor can arise from anywhere along the gut, from the esophagus to the anus (►Fig. 10A–C), forming the most common mesenchymal tumors. They arise from the interstitial cells of Cajal in the muscularis propria, usually during middle age. In the gut, their most common locations are the stomach and small intestine.26,45

They appear as circumscribed enhancing soft-tissue masses without mesenteric edema or invasion of adjacent organs or vessels, and are unlikely to present as small bowel obstruction.26 Their slow indolent growth means they are often detected when large with intraluminal necrosis and hemorrhage seen within. Poor prognostic factors include size > 5 cm, lobulated contour, heterogeneous enhancement, ulceration, mesenteric fat infiltration, regional lymphadenopathy, or an exophytic growth pattern. They are not associated with ascites but do metastasize to the liver and peritoneum.46 Diagnosis is straightforward due to their expression of CD117 antigen (also called the c-KIT). Associations are known with neurofibromatosis 1 and the Carney’s triad.45

A differential diagnosis of leiomyosarcoma (►Fig. 10 D, E) may be entertained when presented with a large solid heterogeneously enhancing mass in the seventh decade. The latter tumors will be negative for c-KIT and CD34.46

Neurogenic tumors: primary neurogenic tumors of the mesentery are very rare. Neurogenic tumors are more common in the retroperitoneum are usually seen in the abdomen among children and young adults (42–60% of cases). Retroperitoneal ganglioneuromas (benign) and ganglioneuroblastomas (intermediate) are lobulated soft tissue masses, involving the root of the small bowel mesentery by extension, where they are seen to encase the vessels without vascular compromise (►Fig. 11).47 Mesenteric schwannomas have been reported but are extremely rare.48

Nodal Masses of the Mesentery

These are the most common solid masses of the mesentery and have a varied differential. Mesenteric nodes < 5 mm
(4.6 mm) in short axis diameter⁴⁹ are routinely seen in asymptomatic patients on multidetector CT. Size and morphological characterization of abnormal nodes facilitates differentiation of a etiology (→ Table 4).⁵⁰,⁵¹

Metastases to mesenteric nodes commonly arise from pancreas and gastrointestinal tract: colon and small bowel, adenocarcinoma, and carcinoma. However, they can also be involved in breast malignancy and small cell cancer of lung, as well as melanoma, bladder tumor, leukemia, and Kaposis sarcoma.⁴⁹

Metastatic nodes are of soft tissue attenuation with diffuse homogeneous enhancement, sometimes showing central necrosis (→ Fig. 12A, B).⁴⁹,⁵¹

Lymphoma is a common cause of malignant mesenteric lymphadenopathy, usually seen at clustered at the root, sometimes distributed in the distal mesentery or as a combined pattern.⁴⁹ The mesentry is most commonly involved by Non-Hodgkin’s lymphoma (45%) as opposed to 3 to 5% in Hodgkin’s disease.⁵²-⁵⁴ The nodes may be discrete or grow to coalesce and form bulky uniformly enhancing nodal masses encasing the mesenteric vessels forming the “sandwich sign” (→ Fig. 12C–E).⁵⁵ Post radiochemotherapy, the nodes may demonstrate cavitation or calcification. FDG PET scans are used staging and end of treatment response depending on the type of lymphoma.⁵⁶

Mesenteric adenitis presents in adults and children with abdominal pain, often with a clinical diagnosis of suspected appendicitis (Table 5). The presence of round ileocolic nodes of 5 to 10 mm in the right iliac fossa with a normal appendix (→ Fig. 13A, B) and a terminal ileal thickness of <5 mm is then called primary mesenteric adenitis.⁵⁷,⁵⁸

Enlarged mesenteric nodes in the presence of appendicitis, inflammatory bowel disease, diverticulitis, or systemic lupus erythematosus are termed secondary mesenteric adenitis.⁴⁹ These nodes are enlarged enhancing and usually oval. *Yersinia enterocolitica* infection involves right lower quadrant

### Table 4 Nodal masses of the mesentery

<table>
<thead>
<tr>
<th>Malignant</th>
<th>Infectious</th>
<th>Inflammatory</th>
</tr>
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<tbody>
<tr>
<td>Lymphoma</td>
<td>Tuberculosis</td>
<td>Castleman’s disease</td>
</tr>
<tr>
<td>Nodal metastases</td>
<td>Appendicitis</td>
<td>Whipple’s disease</td>
</tr>
<tr>
<td>Carcinoid</td>
<td>Enteritis</td>
<td>Crohn’s disease</td>
</tr>
<tr>
<td>Kaposis’s sarcoma</td>
<td>Mesenteric adenitis</td>
<td>Sclerosing mesenteritis</td>
</tr>
<tr>
<td></td>
<td>Diverticulitis</td>
<td>Sprue</td>
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<td></td>
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<td>Sarcoidosis</td>
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### Table 5 Mesenteric adenitis

<table>
<thead>
<tr>
<th>Secondary</th>
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<tbody>
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<td>Mesenteric adenitis &gt;5 mm</td>
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<td>Secondary Appendicitis</td>
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<td>Ulcerative colitis</td>
<td></td>
</tr>
<tr>
<td>Diverticulitis</td>
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<tr>
<td>Crohn’s disease</td>
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<tr>
<td>Systemic lupus erythematosus</td>
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<tr>
<td>Primary</td>
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<tr>
<td>Right lower quadrant nodes with ileal thickness of &lt;5 mm</td>
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![Fig. 12](A, B) Axial and coronal reformat of contrast enhanced CT in a patient with abdominal discomfort and loss of weight demonstrates necrotic metastatic nodal mass in the mesentry from a heterogeneously enhancing periampullary carcinoma. (C) Bulky conglomerate mass of lymph nodes in the root of the mesentry, on either side of the mesenteric vessels forming the mesenteric ‘sandwich sign’. (D, E) Axial and coronal contrast enhanced CT abdomen in a patient with rigor and left upper quadrant pain demonstrates circumferential wall thickening and aneurysmal dilatation of a loop of small bowel (curved blue arrow) with an associated enlarged mesenteric lymph nodes (yellow arrows). Small bowel lymphoma. CT, computed tomography.
nodal enlargement with associated thickening of the terminal ileum resembling Crohn’s disease.\(^{49,50}\)

Granulomatous diseases causing mesenteric adenopathy include tuberculosis and sarcoidosis. Tuberculous mesenteric adenopathy (►Fig. 13C) may be isolated or associated with gastrointestinal, omental, and peritoneal disease with or without ascites. The nodes are cystic, discrete, or clustered in character but usually do not attain the massive sizes seen in lymphoma. They often show rim enhancement due to central cavitation or caseation. Sarcoidosis does not affect the mesentery alone and is usually associated with retroperitoneal nodes (►Fig. 13D). The black pearl sign of sarcoid nodes has been described as a characteristic finding.\(^{60}\)

Unusual causes include Castleman’s disease and Whipple’s disease. Whipple’s disease is seen in young white males, is caused by a bacterium called Tropheryma whippelli, and is seen as enlarged mesenteric nodes of very low attenuation (10–20 HU) on CT, due to internal fat content. Intestinal manifestations include mucosal nodularity in the jejunal loops, thickened mucosal folds, and prominent small bowel loops.\(^{61}\)

Hypoenhancing and cavitating bulky upper mesenteric lymph nodes demonstrating internal fat content or cystic components are also noted in some patients with gluten-related enteropathy (celiac disease).\(^{62,63}\) Other imaging features of celiac disease are fluid filled prominence of small bowel loops with dilution of oral contrast, flocculation of barium, telescoping, intussusception, and conformation (bowel loops apposed to each other with their walls conforming to one another without an intervening space) of small bowel loops.\(^{63}\)

Castleman’s disease is a nonneoplastic lymphoproliferative disease presenting with significantly enlarged hyperenhancing abdominal or mesenteric nodes (►Fig. 13E), commonly unicentric, and solitary nodal, sometimes multinodal.\(^{54,64}\) Some patients develop systemic symptoms and lymphoma is a top differential.\(^{53}\)

**Fatty Lesions of the Mesentery**

Fatty lesions of the mesentery (►Fig. 14) may be neoplastic and nonneoplastic. Among the nonneoplastic fatty masses, mesenteric panniculitis, and sclerosing mesenteritis have already been discussed. Fat appears hyperechoic on US and on CT it has an attenuation of 10 to 100 HU.\(^{65}\)

MRI is exemplary in detection of microscopic fat, well depicted on in-phase and out-phase sequences. Macroscopic fat appears hyperintense on T1 and hypointense on T1 fat-suppressed series.\(^{65}\) However, most fatty tumors of the mesentery are easily characterized on CT, MRI being used only for problem solving, when the fraction of fat within a lesion is very small.

Mesenteric lipoma is a rare benign tumor, sometimes presenting in children, appearing as soft echogenic lesion on US, giving in to probe compression with no vascularity and displacing...
bowel loops around it. They resemble well-differentiated fatty tumors on CT and are usually excised if large or symptomatic.56

Liposarcomas include well differentiated, myxoid, pleomorphic, and round cell types are very unusual in the mesentery but is discussed here in view of a case that presented in our practice.56-58 They are commonly seen in the retroperitoneum and displace the small bowel and mesentery by mass effect. The myxoid type often resembles a cystic lesion on plain CT (Fig. 15A, B), while diffuse enhancement with contrast demonstrates the solid nature of the lesion on CT and MRI.56-58 Thick enhancing septations and solid components are seen in well-differentiated liposarcomas, often with internal calcification. The pleomorphic and round cell varieties are largely solid enhancing tumors on CT, difficult to differentiate from other sarcomas.57

Mesenteric fat proliferation is a manifestation of chronic inflammation of small bowel. Classically, described as proliferation of fat in the mesentery of abnormal inflamed small bowel usually along the mesenteric side, it can extend circumferentially.17 It is an important marker of Crohn’s disease, appropriately called “creeping fat” (Fig. 15C, D).61 Considerable perirectal fat proliferation can also be seen in ulcerative colitis,49,70 bearing testimony to the chronicity of the disease, but is not a specific indicator for ulcerative colitis, since it can also be seen in Crohn’s involving large bowel (Fig. 15E, F).

Mesenteric lymphangiomas have already been described and are thin-walled fluid or fat attenuation lesions.52 They may be multiloculated and typically demonstrate transcompartmental extension, insinuating into areas of least resistance.

Nonmass Miscellaneous Mesenteric Diseases
Nonneoplastic diseases of the mesentery are very often overlooked because they are soft markers of disease. However, they often provide important clues to underlying systemic events.

Mesenteric Edema
Non inflammatory mesenteric edema has been described as misty mesentery. It could be a result increased water logging due systemic causes such as chronic kidney disease, hypoproteinemia, cardiac (Fig. 16A, B), and hepatic failure.25,31 Misty mesentery can be differentiated from mesenteric panniculitis by the presence of associated ascites and by absence of fat rim sign and mesenteric nodes.

Localized causes may be vascular as in portal or SMV thrombosis (Fig. 16C) or vasculitis, as well as contusion, trauma, or surgery. Enteric infections, hepatic and other malignancies, contraceptive pills, and hypercoagulopathies can result in portal and SMV thrombosis. Pancreatitis and cirrhosis may also predispose to thrombosis leading to mesenteric edema.21

Mesenteric Trauma
It is difficult to distinguish mesenteric blood from edema, but increased densities of 40 to 90 HU are noted in hematoma and clinical history is crucial to make the diagnosis. The “sentinel clot” sign representing a focus of high density may indicate the source of hemorrhage.25 Contrast extravasation from mesenteric vessels may be elicited on delayed imaging (Fig. 16D, E).72

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Fig. 15 (A, B) Elderly lady being evaluated for abdominal pain, incidentally detected left paraenteric well circumscribed cystic appearing lesion (arrows) with internal fat content, liposarcoma. (C, D) Mesenteric fat proliferation displacing inflamed small bowel loops along the mesenteric border is very specific as in this case of Crohn’s disease. (E, F) Pericolonic fat proliferation is nonspecific and may be seen in Crohn’s or ulcerative colitis.
In trauma, a high index of suspicion needs to be maintained, because if there is localized mesenteric edema or fluid sans bowel wall abnormalities, there is often an associated mesenteric tear or laceration of a mesenteric vessel.\(^7\)

**Mesenteric Inflammation**

Pancreatitis is the most widespread cause of mesenteric inflammation all over the world. Inflammatory fat stranding, fluid pockets, and fat necrosis extend and evolve with the severity and chronicity of inflammation. Splenic and SMA pseudoaneurysm are a complication of pancreatitis.

Inflammatory bowel disease is another important cause of mesenteric inflammation and encompasses haziness and stranding in the mesenteric fat and congested vasa recta (in the acute setting), also known as the “comb” sign. Fibrofatty proliferation and fat wrapping of the bowel is seen as a chronic manifestation. Mesenteric thickening may also be seen in chronic cases and may cause technical difficulties at surgery.\(^1\)\(^,\)\(^7\)

Other common causes include diverticulitis, appendicitis and cholecystitis. Inflamed jejunal and sigmoid diverticula may extend into their anchoring mesentery, with formation of abscesses if they progress.\(^1\)\(^,\)\(^2\)

**Mesenteric Nonrotation**

Traditionally, as a result of embryological intestinal rotation, the small bowel mesentery is attached to the posterior abdominal wall via a wide mesenteric root which extends from the duodenojejunal flexure on the left of the vertebral column to the right iliac fossa\(^7\) with the large bowel subtended around the periphery. Malrotation or nonrotation results in the small bowel on the right (\(\text{Fig. 17A, B}\)) and the large bowel on the left with a short small bowel mesentery, which forms a pivot around which the small bowel can volve.\(^7\) There are many variations of abnormal rotation, as a result of which clinical presentation may be in infancy or adulthood.

**Internal Mesenteric Hernia**

An internal mesenteric hernia occurs across a congenital or acquired defect in the mesentery, usually in the vicinity of the ligament of Treitz or the terminal ileum. These may be of a transmesenteric subtype, involving both the peritoneal layers, or an intramesenteric type, involving a single layer of peritoneum.\(^2\) They are the most common internal hernia in children, with pediatric cases accounting for 35%.\(^7\) Acquired causes may be related to prior trauma, surgery, inflammation, or ischemia. They tend to have a rather acute presentation of bowel obstruction.\(^2\)

On imaging, there is small bowel obstruction with dilatation of small bowel loops described to be abutting the abdominal wall without intervening omental fat and abnormal location of the bowel loops lateral or posterolateral to the colon. Associated findings of stretched mesenteric vessels converging at the hernial orifice and rightward displacement of mesenteric vessels may be noted.\(^2\)

The “SMV beaking” sign indicating attenuation and beak-like configuration of the SMV, as well as reversal of the SMA–SMV relationship, in the distal aspect (“crisscross appearance”) have been described in relation to postoperative mesenteric hernias after the laparoscopic Roux-en-Y gastric bypass.\(^7\)

These hernias may be challenging to detect on imaging because these loops are not covered by a sac and may be present anywhere in the peritoneal cavity.\(^2\) Complications such as volvulus, ischemia, and strangulation may ensue due to the small size of the mesenteric aperture (2–3 cm).\(^2\)
Mesenteric Volvulus
Mesenteric volvulus is a twisting of the mesentery around itself (►Fig. 17C) which may be a result of congenital nonrotation, congenital bands, idiopathic, or due to adhesions. Often in closed loop obstruction, there is a cone-shaped engorged mesentery with small bowel at the periphery and fluid in the mesentery due to mesenteric ischemia. If unaddressed, this leads to bowel wall ischemia, nonenhancing, paper thin bowel wall, progressing to necrosis, and gas in the mesenteric veins.79

Superior Mesenteric Artery Disease
Thrombosis of the SMA is a relatively common cause of small bowel ischemia in patients with extensive atherosclerotic disease. Embolism and dissection extending into the SMA are rarer causes of acute mesenteric ischemia that should not be overlooked. It may be seen as an intravascular filling defect on contrast CT or hyperattenuating thrombus on plain CT, in addition to the findings of segmental bowel wall edema and bowel dilatation due to adynamic ileus with or without mesenteric edema in acute arterial occlusions.80 Dissection in the SMA is rare cause of small bowel ischemia. The extent of luminal compromise influences clinical presentation and management and is therefore classified (►Fig. 18A, B). Among vasculitic diseases, Takayasu's vasculitis is seen in younger individuals and usually results in formation of collateral arcades which are well depicted at CECT. However, they are also predisposed to thrombosis and mesenteric ischemia.80 Rapid and accurate diagnosis is crucial for implementation of medical or surgical management.

New Concepts and Trends
The traditional anatomical description of the mesentery has been questioned and reevaluated in recent years. The
mesentery has been given the exalted position of an organ. The discovery is credited to J Calvin Coffey, at the University Hospital Limerick. According to Coffey et al., the mesentery is contiguous from the stomach to the anal canal expunging the theory that some segments of the small and large bowel are retroperitoneal. In his article, he validates the finding of anatomist Toldt and Rosa, way back in 1879. He also presents a new understanding of diseases involving the mesentery, categorizing them into primary, and secondary mesenteropathies.

Conclusion

The mesentery is indeed an extraordinary structure, with an intriguing relationship with the bowel. Within the abdomen, it is a flexible, pliant shield of the adipose tissue acting as a tether, yet allowing for mobility of the bowel. The diseases of the mesentery as described above are intrinsic to the nature of the mesenteric structure. An understanding of the mesenteric structure is imperative to assess the radiological features of infective, inflammatory, vascular, and neoplastic processes within. As in any other organ, correlation with presentation and clinical findings are essential to the formation of a sound primary and differential diagnosis. In addition, there is new and exciting research on the mesentery with promise to unravel more mysteries of the mesentery.

Conflict of Interest

None declared.

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