Evaluation of mesenteric ischemia at multidetector ct

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Learning objectives

1. To demonstrate the CT features of venous and arterial mesenteric ischemia
2. To try and determine the etiology of ischemia
3. To illustrate the vascular and non vascular findings in occlusion of mesenteric vessels
Background

Vascular supply to the intestine and mesentery arises from the branches of abdominal aorta i.e. celiac axis, superior mesenteric artery (SMA) and inferior mesenteric artery (IMA) with significant collateral channels and anastomoses between the branches of these vessels, and due to these collateral channels the intestine does not suffer substantial injury until there is significant vascular compromise. Mesenteric ischemia can present clinically in acute or chronic manner and at times the diagnosis could be made at the imaging only, due to nonspecific clinical presentation. A critical factor in the survival of these patients is prompt diagnosis and appropriate intervention thereof. And the role of imaging becomes all the more important to diagnose it early, as the clinical diagnosis is usually delayed and can result in irreversible injury to the bowel.

There have been revolutionary advancements in the diagnostic imaging techniques in the last decade resulting in proper and timely management of vascular pathologies. One of these is the multidetector computed tomography (MDCT) which allows very fast scanning covering the entire abdomen in few seconds. There are practically no motion artifacts; appropriate timing of arterial and portal phases is achieved thus reducing the dose of intravenous contrast. Thin collimation and faster scan allows maximum opacification of the mesenteric arteries and veins and with multiplanar reformations (MPR) the distal branches are adequately visualized. Volume rendered (VR) and maximum intensity projections (MIP) provide excellent overall representations of the mesenteric vasculature. Ultrasound and color Doppler provides real time imaging of the mesentery, bowel and the major vessels, flow velocities and the spectral waveform pattern could be nicely evaluated, however it suffers from various drawbacks including: operator dependency, obscuration of the vessels due to bowel gases and inability to assess the distal vessels. Contrast enhanced phase resolved magnetic resonance (MR) angiography also provides the desired information on the mesenteric vessels and is comparable to MDCT angiography.
IMAGING FINDINGS

The modality of choice to demonstrate the mesenteric vasculature is undoubtedly the MDCT augmented with MPR and 3 dimensional renderings as and when required. The radiologist should be well versed with the vascular anatomy of the mesentery and bowel, their variants and the various pathologies. It will be helpful to recapitulate the same at this point.

Normal Anatomy

Arterial

Three branches of abdominal aorta that supplies the entire bowel are:

1. Celiac artery
2. SMA
3. IMA

CELIAC ARTERY is the first major branch of abdominal aorta and gives rise to three branches which are

1. left gastric artery
2. Splenic artery
3. Common hepatic artery

SMA is the most important artery arising just distal to the celiac axis and it supplies the bowel from lower part of lower part of duodenum to the proximal two thirds of transverse colon. Branches of SMA include:

1. Inferior pancreaticoduodenal artery
2. Intestinal arteries (jejunal and ileal arteries)
3. Ileoceleal artery
4. Right colic artery
5. Middle colic artery

IMA is the third branch supplying the left one third of transverse colon, descending colon, sigmoid colon and upper rectum. Its branches are:

1. Left colic artery
2. Sigmoid branches
3. Superior rectal artery

Venous
Venous drainage occurs via superior mesenteric vein which joins with the splenic vein to form portal vein.

**PATHOPHYSIOLOGY OF MESENTERIC ISCHEMIA**

Insufficient perfusion of the small bowel and mesentery may result from arterial occlusion by embolus or thrombosis, venous thrombosis, or nonocclusive processes such as vasospasm or low cardiac output. Severity of the injury is inversely proportional to the mesenteric blood flow and is influenced by the number of vessels involved, systemic mean blood pressure, duration of ischemia, and collateral circulation. The superior mesenteric vessels are involved more frequently than the inferior mesenteric vessels, with blockage of the latter often being silent because of better collateral circulation. Damage to the affected bowel portion may range from reversible ischemia to transmural infarction with necrosis and perforation. The injury is complicated by reactive vasospasm in the SMA after the initial occlusion. Arterial insufficiency causes tissue hypoxia, leading to initial bowel wall spasm. This leads to gut emptying by vomiting or diarrhea. Mucosal sloughing may cause bleeding into the gastrointestinal (GI) tract\(^1\).

**ETIOLOGY**

**ARTERIAL OCCLUSION**

Embolic occlusion:

- Cardiac emboli - Mural thrombus after myocardial infarction, auricular thrombus associated with mitral stenosis and atrial fibrillation, septic emboli from valvular endocarditis
- Emboli from fragments of proximal aortic thrombus due to a ruptured atheromatous plaque
- Atheromatous plaque dislodged by arterial catheterization

Thrombotic occlusion:

- Atherosclerotic vascular disease
- Aortic aneurysm
- Aortic dissection
- Arteritis
- Dehydration from other causes

**NON OBSTRUCTIVE MESENTERIC ISCHEMIA**

- Hypotension from congestive heart failure, myocardial infarction, sepsis, aortic insufficiency, severe liver or renal disease, or recent major cardiac or abdominal surgery
- Vasopressive drugs
• Ergotamines
• Cocaine
• Digitalis

VENOUS OCCLUSION

• Hypercoagulability from protein C and S deficiency, antithrombin III deficiency, dysfibrinogenemia, abnormal plasminogen, polycythemia vera (most common), thrombocytosis, sickle cell disease, pregnancy, and oral contraceptive use
• Tumor causing venous compression or hypercoagulability (paraneoplastic syndrome)
• Infection, usually intra-abdominal (eg appendicitis, diverticulitis, or abscess)
• Venous congestion from cirrhosis (portal hypertension)
• Venous trauma from accidents or surgery, especially portocaval surgery
• Increased intra-abdominal pressure from pneumoperitoneum during laparoscopic surgery
• Pancreatitis
• Decompression sickness (1,2)

PROCEDURE DETAILS

In this study we present the findings of mesenteric ischemia on CT scans performed on 16 slice MDCT. Noncontrast scans were obtained in all cases followed by contrast enhanced scans covering the entire abdomen from the dome of diaphragms to the level of perineum. Plain scans should be done, as they help in demonstrating vascular calcifications, hyperattenuating intravascular thrombus and intramural hemorrhage in the bowel (Fig 1,2).

For contrast enhanced scans 100-150ml of nonionic iodinated contrast was given at the rate of 2-5ml/sec and images obtained in arterial and venous phase. A collimation of 0.5-0.25mm was used with 5mm section thickness and images reconstructed at thin interval of 1-2mm for multiplanar reformations.

IMAGING FINDINGS

Mesenteric Vessels:

Thrombo-embolic occlusion of the SMA is the most commonly encountered finding in cases of bowel and mesenteric ischemia. Embolic phenomena account for approximately 50% of all clinical cases, arterial thrombosis for about 25%, nonocclusive for roughly 20%, and venous thrombosis for less than 10%. Rarely,
isolated spontaneous dissections of the SMA have been reported\(^{(3,4,5,6)}\). In our study three cases of SMA thrombosis due to atherosclerosis and one case due to embolic occlusion were seen (Fig.3,4)

Thrombotic occlusion usually takes place with underlying atherosclerotic disease in the aorta and its branches. Intimomedial calcifications and thickening can be seen in the aorta and SMA (Fig. 3-8) with the findings of thrombosis/narrowing of SMA. IMA is less commonly involved than SMA (Fig.9). The thromboembolic occlusion are seen as filling defects in the mesenteric vessels and their branches.

The most common cause of mesenteric arterial occlusion is emboli dislodged from the heart. The emboli usually wedge at the branching points. Myocardial infarction, infective endocarditis and valvular diseases are few of the predisposing conditions (Fig.10).

Prothrombotic conditions such as deficiency of Protein C and S are rare causes of mesenteric vascular thrombosis and one such case is presented in this study, who was a middle aged female patient presented in the emergency department with severe acute abdominal pain and symptoms of intestinal obstruction. CECT reveal thrombosis of SMA, and some jejunal loops were nonviable. Her thorough investigations reveal deficiency of protein S. The necrosed bowel was resected and appropriate anticoagulant therapy was administered resulting in clinical and radiological improvement (Fig. 11,12,13).

Pancreatic carcinoma can invade the superior mesenteric vessels by direct invasion or can simply lead to their narrowing by extrinsic mass effect (Figure 14-17). Tear drop sign in SMV is considered specific sign of its invasion by the pancreatic carcinoma. Optimal results are achieved when axial source images and post processed images are combined for the assessment of vascular narrowing and perivascular cuff\(^{(7)}\).

Narrowing of the superior mesenteric vein at the level of confluence with the splenic vein is seen in one case which gave history of pancreatitis i (Fig.18,19). The narrowing could result due to fibrosis in the peripancreatic region involving the SMV.

Pancreatitis is one of the leading causes of thrombotic occlusion of the SMV and splenic vein, the arterial complications of pancreatitis include disruption of the vessel wall and pseudo aneurysm formation. Splenic vein is more frequently
involved than the SMV because of its close proximity to the body of pancreas (Fig. 20-23).

Bowel ischemia and infarction can occur with a reduction of mesenteric blood supply without vascular occlusion, which is called nonocclusive mesenteric ischemia or infarction. A reduction of the mesenteric blood supply is the result of mesenteric arterial vasoconstriction due to hypotension or administration or abuse of vasoconstrictive agents\(^{(8,9,10,11)}\). CT reveals signs of bowel ischemia without evidence of thrombotic occlusion of vessels (Fig 24,25).

OTHER FINDINGS

Bowel

The most frequently encountered finding in mesenteric ischemia is bowel wall thickening caused by edema, hemorrhage or super infection (Fig 26). However in pure arterial occlusive ischemia there is thinning (paper thin) rather than thickening of bowel loops due to absence of blood flow (Fig.27)\(^{(12,13,14,15,16,17)}\).

There is absence of enhancement of the wall of involved loops in acute arterial ischemia (Fig 27). Dilatation of the bowel loops occurs due to interrupted peristaltic waves and the loops are fluid filled due to increased intestinal secretions (Fig.28).

Mesenteric fat stranding and ascites due to transudation of fluid in the mesentery are associated nonspecific findings in ischemia\(^{(12,13,14)}\).(Fig 29). The presence of pneumatosis intestinalis and the portovenous air indicated transmural infarction in the setting of mesenteric ischemia (Fig. 24,25).
Fig. 1: Noncontrast enhanced section shows complete ring like mural calcification in the SMA giving the false appearance of thrombosed vessel with patent lumen in the periphery.

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Fig. 2: After contrast administration (same patient as in Fig 1) a large hypodense thrombus is seen in SMA causing almost complete obliteration of its lumen, obtained at a proximal level than figure 1.

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Fig. 3: Sagittal reformatted image of a 70 year old man presenting with nonspecific gradually progressive abdominal pain with acute worsening (same patient as in Figure 1). This contrast enhanced sagittal image reveal completely occluding hypodense thrombus in the SMA.

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**Fig. 4:** Coronal image (same patient as in Fig 3) demonstrates the thrombus in main trunk of SMA and the jejunal branches. Atherosclerotic calcifications in the trunk of SMA is seen on coronal image.

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**Fig. 5:** This 55 year old male patient presented with complaints of intermittent abdominal pain, especially after 2-3 hours postprandial. Coronal contrast enhanced image reveals almost completely occluding thrombosis in ileocolic and in one of the jejunal branches of SMA.

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Fig. 6: Axial image (same patient as in fig 5) shows the thrombus as filling defect in ileocolic branch of SMA

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Fig. 7: Another patient with atherosclerotic narrowing of SMA. Sagittal image shows marked intimomedial thickening in aorta and SMA. There is approximately 50% luminal narrowing in the SMA due to the markedly thickened intimomedial complex

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**Fig. 8:** Axial image of the same patient as in Fig 7 with atherosclerotic narrowing of SMA.

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Fig. 9: A partially occluding thrombus is seen in the IMA

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Fig. 10: Contrast enhanced scans in a young adult male who gives history of valvular heart disease reveal a large hypodense thrombus in abdominal aorta extending into the origin of SMA causing severe luminal narrowing. One of the proximal jejunal loops show thin and nonenhancing walls. Bowel resection and embolectomy was performed and the patient was discharged in good condition.

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Fig. 11: Sagittal reformatted arterial phase contrast enhanced image reveal partially occluding thrombosis in main trunk of SMA extending into the jejunal branches of SMA in a patient with protein S deficiency.

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Fig. 12: coronal reformatted arterial phase contrast enhanced images reveal partially occluding thrombosis in main trunk of SMA extending into the jejunal branches of SMA (same patient as in fig 11).

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**Fig. 13:** Same patient as in figure 11, after proper anticoagulant therapy reveal complete resolution of thrombosis.

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Fig. 14: Thirty five year old male presented with vague abdominal discomfort and loss of 20kg of weight in last four month. Ultrasound was unremarkable, hence CECT was requested. Axial image of contrast enhanced CT reveals an ill-defined ‘cuff’ of soft tissue around the proximal SMA and its proximal branches. SMV is not visualized and is probably compressed/invaded by the lesion.

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Fig. 15: Sagittal reformatted images of the same patient as in figure 14 reveals the ill-defined 'cuff' of soft tissue around the proximal SMA and its proximal branches.

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**Fig. 16:** Coronal reformation of same patient reveal the poorly circumscribed soft tissue encasing the SMA.

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Fig. 17: Endoscopic guided ultrasonography (EUS) was performed in the same patient as in Fig 14 which reveal an ill-defined hypoechoic area in the pancreatic uncinate process. An FNAC sample was obtained through the lesion. It came out to be pancreatic adenocarcinoma

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Fig. 18: Abrupt luminal narrowing is seen in the SMV just before the portal vein confluence, the portal vein is intact. Patient had an attack of acute pancreatitis in the recent past, and the narrowing in the SMV is assumed to be due to focal fibrosis induced by pancreatitis.

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Fig. 19: Coronal image of same patient as in fig 18 reveal the narrowing of SMV in this follow up case of pancreatitis

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**Fig. 20:** Acute pancreatitis with SMV thrombosis. SMV is distended by hypodense thrombus. Streakiness seen around SMV.

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Fig. 21: Coronal reformat in the same patient reveal the SMV thrombosis extending into the jejunal tributaries. Splenic vein is patent. pancreas appears bulky and there is streakiness in peripancreatic tissues suggesting acute pancreatitis.

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Fig. 22: Another follow up case of pancreatitis with thrombosis of SMV. Thrombus in SMV is visualized with partial thrombosis in few of its tributaries.

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Fig. 23: Axial scan at a lower level in the same patient as in fig 22 reveal Collateral channels seen in right perirenal region suggesting portal hypertension.

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Fig. 24: A case of nonocclusive mesenteric ischemia reveals air in the portal system.

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Fig. 25: Same patient as in fig 24 reveal thickened bowel walls with mucosal hyper enhancement. Foci of air are seen in the walls of bowel loops (pneumatosis intestinalis), a sign of bowel ischemia.

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Fig. 26: Same patient as in figure 11 reveal thickened jejunal loops in left lumbar region which show target appearance in this case of acute SMA thrombosis.

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Fig. 27: There is paper thinning of two jejunal loops in this patient of acute embolic narrowing of SMA (same patient as in Fig 10) with luminal dilatation and loss of normal valvulae conneventis. There is absence of normal mural enhancement in these loops (suggesting nonviable loops) whereas the normal mucosal enhancement is seen in one of the adjacent loops.

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Fig. 28: Multiple dilated small bowel loops are seen showing air-fluid levels in a case of acute embolic occlusion of SMA. Mesentery appears hazy

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**Fig. 29:** Contrast enhanced CT images reveal ascites, stranding in the mesentery and thickened bowel loops in this case of mesenteric ischemia.

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Conclusion

Due to the vague clinical signs and symptoms the diagnosis of mesenteric ischemia is usually delayed and can adversely affect the prognosis. With the use of MDCT the findings of mesenteric and bowel ischemia are nicely demonstrated and it is an essential tool in the evaluation of these cases. There is variety of etiologies of mesenteric ischemia and thus the CT findings can vary. CECT not only aids in early diagnosis, but may also help in preventing the permanent damage to the bowel by prompting early intervention.
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