Mesenteric Arterial Disease

13

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Anatomy, Normal Arterial and Venous Anatomy: Collateral Circulation

Celiac Artery

- Arises at level of L1, bordered by the median arcuate ligament (MAL) at aortic hiatus superiorly and superior border of pancreas inferiorly
- 3 branches:[1] left gastric, splenic, common hepatic arteries.

SMA

- Inferior to celiac trunk, origin is crossed by neck of pancreas and splenic vein. Comes off superior to uncinate process and 3rd portion of duodenum

[1]Multiple variations exist (Most common-common hepatic arises from SMA or directly from abdominal aorta).

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- SMV runs parallel and usually along its right border
- 1st Branch: usually inferior pancreaticoduodenal artery—supplies collateral circulation with celiac via gastroduodenal and superior pancreaticoduodenal
- 2nd Branch: middle colic artery—arises at inferior border of pancreas.

IMA

- 3–4 cm above aortic bifurcation and just left of midline at level of L3.
- Main trunk divides into sigmoidal branches and left colic artery.
- Collateral circulation: ascending left colic artery forms inferior marginal artery of Drummond (major collateral arcade between SMA and IMA)
- Meandering mesenteric artery (of Moskowitz) known as "arc of Riolan": runs centrally medial to mesenteric border of colon and through middle mesenteric arcade near inferior mesenteric vein. Sigmoidal branches lead to left and right superior rectal arteries: collateralize with hypogastric arteries in pelvis.

Diagnosis and Management of Mesenteric Disease

Clinical Presentation/History

Acute mesenteric ischemia (AMI): associated with sudden abdominal pain and often rapid clinical deterioration.

- Pain out of proportion to physical examination.
- Guarding and rebound tenderness early in disease course
- Peritonitis with diffuse pain at later stages with signs of intestinal ischemia

Nonocclusive mesenteric ischemia (NOMI) or mesenteric venous thrombosis (MVT): slower more insidious course. Associated with critical illness, hospitalization, often on pressors.

– Abdominal pain and distension with nausea/vomiting, and bloody stools.

Chronic mesenteric ischemia (CMI): associated with postprandial pain and progressive weight loss.

– Dull/crampy pain located in epigastric region 15–45 min after meals, severity varies according to size and type of meal.
– "Food fear" and decreased oral intake.
– Frequently in elderly women (70% of patients).
– Physical exam: nonspecific, undernourished and cachetic, abdominal bruit may be heard, guarding and rebound tenderness often absent.

Imaging

Duplex US-color Doppler to assess flow velocities and resistance index in splanchnic arterial beds [1].

- SMA: PSV ≥295 cm/s associated with 50% stenosis,
  - PSV ≥400 cm/s associated with ≥70% stenosis
- Celiac: PSV ≥240 cm/s associated with 50% stenosis
  - PSV ≥320 cm/s associated with ≥70% stenosis.
- Celiac or SMA with stent:
  - PSV ≥445 cm/s associated with ≥70% stenosis in SMA
  - PSV ≥289 cm/s associated with ≥70% stenosis in celiac.

CTA: 93% sensitivity and 96% specificity for detecting AMI.

– Ease and speed of performance and ability to simultaneously image mesenteric arteries, veins, and visceral organs.

- CT findings in AMI—bowel wall thickening, dilation, attenuation.
- Pneumatosis intestinalis, portal venous gas, mesenteric edema, and ascites can be seen on CT as well associated with AMI.
- Venous engorgement, "target sign" in SMV with thrombus in center of the lumen can be seen in patients with MVT.

Magnetic Resonance Angiography (MRA): longer to perform than CTA but avoids radiation.

- Poor visualization of the IMA, peripheral splanchnic vessels, calcified plaque, previously placed stents vs. CTA.

Conventional angiography: "gold standard."

- AP and lateral views of the visceral aorta, and selective catheterization of the celiac, SMA, IMA provides most accurate localization of lesions
- Allows for therapeutic options: balloon angioplasty, stenting, thrombolysis, and percutaneous thrombus extraction.

Management

Medical treatment: usually not effective alone in patients with symptomatic mesenteric ischemia.

- High intensity statins recommended
- Aggressive fluid resuscitation with restoration of urine output, correction of electrolyte abnormalities and acidosis should be done/started before operation.
- Patients with chronic mesenteric ischemia who are malnourished should receive TPN or enteral nutrition.
- Broad spectrum IV antibiotics recommended in patients with AMI
- After stent placement—300 mg clopidogrel load followed by 1–3 months of 75 mg daily and maintained on aspirin and high dose statin indefinitely.

Endovascular treatment: Balloon angioplasty and stenting in CMI, generally accepted as primary therapy now.

Surgical treatment—laparotomy with visceral revascularization.

- Bowel should be reassessed after vascular reconstruction and reperfusion.
- Intraoperative angiography can be done to assess mesenteric flow.
- Second look laparotomy in 24 h recommended to reassess bowel viability.

Acute Mesenteric Ischemia (AMI)

Embolus—arterial emboli are the most common cause of AMI (40–50% of cases) [2].

- Source is frequently an intracardiac mural thrombus (from afib, MI, cardiomyopathy, structural heart defects, cardiac tumors).
- Other sources: septic embolic from endocarditis, mural thrombus from proximal aneurysm in abdominal or thoracic aorta, atheromatous plaque.
- SMA is most common source for emboli in aorta—usually lodges several cm from origin, distal to middle colic artery.

Thrombosis—second most common cause of AMI (20–35% of cases).

- Preexisting plaque is most common.
- Hypercoagulability syndromes can predispose to thrombosis as well.
- Affected segment is usually at the origin at the level of the aorta.
- Mortality for AMI patients higher with mesenteric thrombosis vs. emboli.

NOMI—due to impaired perfusion in the absence of thromboembolic disease. Five to 15% of cases.

- Can be due to a low-flow state which is made worse by presence of atherosclerotic disease.
- Most commonly occurs secondary to cardiac disease (e.g. sever CHF patients undergoing cardiac surgery, afib causing reduced left ventricular function).
- Risk factors: older age, hypovolemia, systemic vasoconstrictors, vasoactive drugs (e.g. Digoxin, alpha-adrenergic agents, beta-blocking agents, cocaine), aortic insufficiency, cardiopulmonary bypass, abdominal compartment syndrome, liver failure, patients on hemodialysis (incidence 40× higher).

Treatment selective mesenteric angiography and catheter based infusion of intra-arterial vasodilators (papaverine and prostaglandin E1 [PGE1]) as well as angioplasty and stenting if needed.

- Intravenous PGE1 infusion has been shown to treat vasospasm associated with NOMI as well.
- Low doses of intra-arterial iloprost (inhibitor of platelet aggregation with fibrinolytic activity) have shown significant vasodilatory effect on mesenteric blood flow.

Mesenteric Venous Thrombosis and Portal Vein Thrombosis 5–15% of cases, involvement usually limited to superior mesenteric vein but can also involve IMV, splenic vein, and portal veins [3].

- Primary (idiopathic).
- Secondary—when underlying disease process is present
  - 3 main categories: direct injury (surgery, trauma), local venous stasis/congestion (due to increased intraabdominal pressure, hypersplenism, CHF, obesity), thrombophilia (protein C/S deficiency, antithrombin 3 deficiency, factor V leidin, OCP use, polycythemia vera, HIT, antiphospholipid syndrome, CMV infection)

- *Treatment*: prompt initiation of systemic anticoagulation (improves survival and reduces risk of recurrence).
  - Abdominal exploration with laparotomy if peritonitis/severe gastrointestinal bleeding/or intestinal stricture.
  - Lifelong anticoagulation with vitamin K antagonists recommended.

Chronic Mesenteric Ischemia

Most common cause: atherosclerosis.

- Hx of smoking, HTN, HLD, and atherosclerotic disease in other areas common. Can also be associated with vasculitis and inflammatory conditions (e.g. lupus, Buerger disease, radiation arteritis)

Medical treatment: high intensity statins and risk factor modification.

Surgical treatment: Revascularization is indicated in all patients with symptoms of CMI.

- Goal is to relieve symptoms, restore normal weight and prevent bowel infarction.
- Primary endovascular stenting is first choice in treatment in >80% of patients
- SMA is the primary target for revascularization.
  - Ideal lesion for endovascular treatment: short, focal stenosis or occlusion with minimal to moderate calcification or thrombus.
  - Celiac lesions have high rates of restenosis with angioplasty and stenting.
- Open revascularization: type of open reconstruction based on patient's anatomy and risk assessment.
  - 2-vessel reconstructions (CA and SMA) with a bifurcated polyester graft from supraceliac aorta accounts for >80% of mesenteric reconstructions [4].

- Antegrade mesenteric bypass: bypass from distal thoracic or supraceliac aorta (often spared from disease) via transperitoneal upper midline or bilateral subcostal incision [4].

Key Steps

– Supraceliac aorta exposed after division of diaphragmatic crura.
– systemic heparinization and clamp using two aortic clamps or a Satinsky clamp
– Aortotomy and proximal anastomosis with graft to aorta
– Graft is gently beveled and right graft limb anastomosed end-to-end to the celiac axis or hepatic artery
– Left limb of graft is tunneled behind pancreas and end-to-side anastomosis done with SMA.

Retrograde mesenteric bypass: inflow from infrarenal aorta/prior infrarenal aortic grafts/or the iliac arteries.

- Usually only reconstruct one artery (SMA) in retrograde bypasses.
  – Common iliac artery is most commonly used as source of inflow (avoids aortic cross clamp).
  – Large 8 or 10 mm graft should be used and prefer "C" shaped graft when iliac is used as inflow.
  – Either right or left iliac may be used, but right iliac usually lays better when used for inflow.

Retrograde Open Mesenteric Stenting (ROMS) [2]—hybrid approach done via midline laparotomy to expose SMA.

– Used in selected patients with AMI due to in situ thrombosis when there is an indication for laparotomy, or if severe aortic/iliac calcifications and there is no good inflow source for bypass.

SMA Dissection

– SMA is most commonly affected mesenteric artery where spontaneous dissection occurs (followed by celiac). Still rare with 0.06% incidence [5]. Typically older men in 50s-60s. Can be caused by connective tissue diseases (Marfans, Ehlers Danlos syndrome, Loeys-Dietz syndrome), but also associated with obesity, tobacco use, atherosclerosis, alcohol abuse, obesity, heavy weight lifting, pregnancy
– Usually originates 1–3 cm from SMA origin (where SMA transitions from a fixed retropancreatic position to a mobile mesenteric root).
– Symptomatic vs. Asymptomatic presentation

Symptomatic: abdominal pain 90% of cases [5] (severe, tearing, mid-epigastric area, occasional radiation to back) or abdominal pain out of proportion to exam (suggestive of mesenteric ischemia)

Diagnosis CTA (gold standard) and contrast enhanced CT, less commonly (Duplex Ultrasound, MRI)

Sakamoto Classification based on CT [5–7]:

Type 1: patent false lumen with both entry and reentry tears
Type 2: a "cul de sac" shaped false lumen without reentry
Type 3: thrombosed false lumen with ulcer defect
Type 4: completely thrombosed false lumen without ulcer

Type 1—can be treated conservatively with anticoagulation and medical management

Type 2—follow closely for signs of ischemia, if present treat endo vs. open

Type 3—high likelihood of requiring intervention either endo or open

Type 4—do not require any intervention

Management

Asymptomatic—medical management with anticoagulation/antiplatelet agents

Symptomatic—initial Tx: bowel rest and fluid resuscitation, anticoagulation

If patients fail to improve or signs of ischemia-

Surgery: (if hemorrhage or concern for bowel necrosis)

Visceral bypass (aortavisceral bypass, extraanatomic bypass (gastroepiploic or hepatic artery to SMA bypass), intimectomy/fenestration combined with vein patch, or autogenous graft with saphenous vein

Endovascular

- SMA stent via transfemoral or transbrachial access
- Self-expanding nitinol bare metal stent preferred (or covered stent graft if aneurysmal degeneration present)
- Recommended lifelong antiplatelet
- CTA or duplex surveillance at 1 month, every 6 months for first year, then annual surveillance

Splanchnic Artery Aneurysm

Incidence/Etiology

Dilation of enlargement of artery to 1.5–2× normal diameter. 1/3 of patients will have associated aortic, renal, iliac, lower extremity, or cerebral artery aneurysm. Overall incidence 0.1–2%. Etiology: atherosclerosis, medial degeneration, collagen vascular disease.

– Risk factors: multiparity, portal hypertension, inflammatory conditions (associated with splanchnic pseudoaneurysms).

– Genetic disease and connective tissue disorders have also been implicated.

Surgical Treatment

Open Repair

Ligation of proximal and distal branches without reconstruction in cases of frank rupture and hemodynamic collapse during elective cases if there is adequate collateralization

Celiac and hepatic artery can be ligated if there is collateralization from gastroduodenal and pancreaticoduodenal arteries and portal vein is patent

Endovascular Therapy

Associated with shorter hospital stay, lower cost, faster recovery

- Treatment: coil and glue embolization, injection of particles or gelfoam, placement of covered stents or flow diverting stents, injection of ethyl alcohol or thrombin.

Splenic artery aneurysms Most common (60%), females:males 4:1 [8]. Most are saccular and located in the mid or distal splenic artery and its bifurcations. Risk factors: multiparity, fibromuscular dysplasia, portal hypertension, blunt trauma, infection, pancreatitis.

Lower rupture risk in patients who are not pregnant (>50% rupture during pregnancy)

"Double rupture" phenomenon—occurs after initial tamponade in lesser sac followed by free rupture into retroperitoneum

Treatment: Any patient with symptoms should undergo intervention, women who are pregnant or are of childbearing age should undergo treatment as well. If there is a pseudoaneurysm it should be treated as well. Otherwise treatment indicated if >2 cm in size.

Open surgery: complete resection with splenectomy, proximal and distal ligation of the aneurysm, or ligation with arterial reconstruction.

– Splenectomy is not always needed due to collaterals from short gastritis (unless ligation is done at splenic hilum).

Endovascular: coil or glue embolization associated with 96% technical success

- Post-embolization syndrome (fever, abdominal pain, nausea/vomiting) can occur after embolization but usually resolves.
- Covered stent repair limited to proximal lesions (limited ability to get adequate wire support due to tortuosity)

Hepatic artery aneurysms Second most common (20%) [6]. Most hepatic aneurysms found in extrahepatic vasculature (75–80%) and most commonly in the common hepatic artery, right hepatic artery is second most common. Male to Female ratio 3:2. No association with pregnancy with incidence or rupture risk. Percutaneous and endoscopic interventions have led to increased rise of hepatic artery aneurysms.

Treat any symptomatic aneurysm, pseudoaneurysm, and aneurysms >2 cm.

Open surgery: Via right subcostal or midline laparotomy, can use intraoperative ultrasound to identify aneurysm.

– Common HAA can be ligated or endovascular embolization if GDA is patent. HAA distal to GDA can be treated by arterial reconstruction after ligation.
– Aneurysms within the liver can be treated with resection, ligation, or embolization.
– Should not do hepatic artery ligation in presence of cirrhosis. If Right hepatic artery is ligated or embolized must do a cholecystectomy.

SMA aneurysms 6% of cases, slight male predominance. Almost exclusively occur in the first 5 cm of the artery [6].

Etiology—commonly infection, mycotic etiology accounts for 60% of SMA aneurysms. Other causes: atherosclerosis, connective tissue disease, pancreatitis with trauma.

Presentation: Nonrutured cases likely to cause colicky pain, intestinal angina, weight loss—in 70–90% of patients. High association with rupture as well compared to other splanchnic aneurysms (38–50%).

Treatment: recommend observation of small (<2.5 cm) aneurysms.

- All pseudoaneurysms and those with a mycotic etiology should be repaired.
- Treatment operations: surgical aneurysmectomy, arterial reconstruction, simple ligation (in rare cases).
- If mycotic or inflammatory in etiology then do NOT use PTFE or Dacron bypass grafts.
- Open approach via midline or retroperitoneal, proximal dissection may extend underneath renal vein and if necessary, the adrenal vein may be ligated.
- Must confirm presence of a replaced or aberrant right hepatic branch.
- Endovascular management can be done with either coil embolization or stent grafts.

Celiac artery aneurysms: 4% of splanchnic aneurysms [6]

Etiology—infectious, atherosclerosis, medial degeneration, median arcuate ligament syndrome implicated in formation of some celiac aneurysms.

Presentation—rupture rates of 10–20%, history of colicky abdominal pain, or intestinal angina. Acute abdominal pain and hemorrhagic shock in cases of rupture.

Treatment: recommend repair for aneurysms >2.5 cm.

Surgical options: ligation with or without reconstruction, aneurysmectomy, aneurysmorrhaphy.

- Arterial reconstruction depends on collaterals.
- Endovascular options include coil or glue embolization, percutaneous thrombin or ethanol injection, stent graft placement.

Rare splanchnic aneurysms (gastric/gastroepiploic, pancreaticoduodenal and gastroduodenal, IMA, jejunal/ileal/colic artery aneurysms)

Gastric Artery and Gastroepiploic Artery Aneurysms

– 4% of all splanchnic aneurysms, usually due to atherosclerosis, trauma, infection [6]. Usually along left or right gastric artery.
– 90% are ruptured on initial presentation. Any GAA or GEAA discovered before rupture should be treated.
– Open surgical aneurysmectomy or exclusion. Simultaneous revascularization usually not required.
– If endovascular: must embolize both distal and proximal (aneurysm may recruit a new vascular supply in a retrograde manner if proximal embolization is done without distal embolization).

Pancreaticoduodenal/Gastroduodenal Aneurysms Extremely rare, ~2% of all splanchnic aneurysms [6].

– Usually associated with pancreatic pathology, celiac occlusion or stenosis, or abdominal trauma or iatrogenic injury.
– Male to female ratio 4:1.
– *Presentation*: vague epigastric abdominal pain, may radiate to back, gi bleeding, hypotension, emesis, diarrhea, jaundice.
– GI or biliary tract is site of rupture in 65% of cases.
– No correlation between size and rupture risk.
– Treat regardless of size.

– Endovascular management is ideal—coil embolization techniques.
– Open repair with ligation may require partial pancreatectomy or pancreaticoduodenectomy.

IMA/Jejunal/Colic Artery Aneurysms extremely rare, usually small <1 cm.

– Usually symptomatic and often ruptured on presentation.
– Recommend treatment for any lesion seen, most ruptured are <1 cm.
– Open surgical treatment by aneurysmectomy, or ligation.
– Endovascular treatment by coil embolization.

Celiac Artery Compression Syndrome (aka Median Arcuate Ligament Syndrome [MALS]) anatomic and clinical illness resulting from extrinsic compression of the celiac axis.

Presentation postprandial and exercise induced abdominal pain, nausea, vomiting, weight loss, "food fear." Female:Male 3:1, nausea/vomiting, unintentional weight loss, exercise induced pain, second-sixth decade of life, autonomic dysfunction, fibromyalgia.

– Median arcuate ligament comes from diaphragmatic crura from L1 to 4 which projects cephalad to join the anterior longitudinal ligament of the spine overlying the celiac axis.
– Composed of the edge of crura that crosses the aorta at the level of the celiac artery. Splays over the aorta and laterally to the suspensory ligament and fourth portion of the duodenum.
– In most cases: the ligament does not encroach the celiac artery and causes no compression. 10–24% may have no symptoms.
– Underlying pathophysiology unknown, possible hypothesis is that compression of the celiac plexus nerve fibers may lead to pan and alter gastric myoelectrical activity/impair motility.

- CTA/MRA to evaluate structural elements of celiac artery, median arcuate ligament and viscera.
- Abdominal duplex ultrasound.
- Normally distance between celiac artery and median arcuate ligament increases during inspiration and decreases with expiration. PSV >200cm/s, no flow, or retrograde common hepatic arterial flow during inspiration or expiration consistent with 70% stenosis of celiac artery. Elevated PSV with expiration which normalize or decrease with inspiration and standing suggestive of MALS.

Angiography—asymmetric focal narrowing of proximal celiac axis with poststenotic dilation; narrowing increases with expiration. Increased collaterals in celiac distribution means significant stenosis.

Gastric Tonometry: blood gases and PCO_2 measured during peak exercise and recovery after 10 min bicycle test.

- Positive result: gastric arterial PCO_2 gradient >0.8 after exercise, increase in gastric PCO_2; arterial lactate levels <8 mmol/L.

Management: open or laparoscopic release of the median arcuate ligament. Percutaneous celiac ganglion block perioperatively can be done as a diagnostic and potentially therapeutic test.

- Endovascular therapy with percutaneous transluminal angioplasty (PTA) or stenting alone is NOT recommended (does not address the issue of extrinsic compression by the ligament).

Middle aortic syndrome developmental aortic narrowing from coarctations in the aorta.

- Can be distinguished based on the most cephalad extent of the involved portion (suprarenal 69%, suprarenal-infrarenal 23%, infrarenal 8%) [3]

Etiology overfusion of the two dorsal aortas during 4th week of gestation.

Most frequent genetic diseases in utero associated with abdominal aortic coarctations: neurofibromatosis-1 (NF1), and tuberous sclerosis; maternal rubella during 1st trimester.

– Nondevelopmental coarctation has been associated with umbilical artery catheterization during neonatal period.

Clinical symptoms hypertension from suprarenal or intrarenal aortic narrowing (often refractory to medication), rarely—lower extremity fatigue with exercise/or claudication, postprandial intestinal angina (6%) [3].

Evaluation CTA/MRA, Renal Doppler US (often used first line to assess renin mediated hypertension caused by aortic or renal artery disease), catheter based angiography

Medical management antihypertensive drugs (ACE inhibitors, ARBs), combined alpha and beta blockers (labetalol), beta-blockers, central alpha agonists (clonidine), diuretics, calcium channel blockers, peripheral alpha antagonists, vasodilators.

Surgical Treatment

Isolated abdominal aortic coarctations: patch aortoplasty.

- If segment of aorta is too narrow or with disease affecting the renal or splanchnic arteries:
  - Thoracoabdominal bypass with ePTFE.
  - Grafts positioned behind the left kidney with a gentle curve to the distal aorta.
  - Oversize grafts to recognize aortic growth in children. 8–12 mm grafts in young children, 12–16 mm grafts for early adolescents, 14–20 mm grafts in late adolescents and adults.

Questions

1. A 62-year-old female presents to the clinic with complaint of post prandial abdominal pain for the last 3 months associated with 5 lb of weight loss. A US mesenteric duplex was done showing a PSV of 200 cm/s in the celiac artery and 410 cm/s in the SMA; the IMA was unable to be visualized. Which of the following is consistent with the above clinical picture?
   (a) Mild-moderate SMA stenosis <70%
   (b) High grade SMA stenosis >70%
   (c) High grade celiac artery stenosis >70%
   (d) Aortoiliac occlusive disease
2. A 30-year-old female is found to have an incidental 3.2 cm splenic artery aneurysm on CT trauma "pan scan" after an MVC. She is afebrile, hemodynamically stable, denies any abdominal pain. What is the optimal management of her splenic artery aneurysm?
   (a) Admission for urgent operative resection and splenectomy
   (b) US surveillance in 1 month
   (c) US surveillance in 1 year
   (d) Elective splenic artery embolization
3. A 66-year-old female presents to the ED with complaint of post-prandial abdominal pain for 6 months, 10 lb weight loss, as well as abdominal pain during her spin classes causing her to need to stop. Her history is notable for DM2 and Fibromyalgia. Which are these would you expect to see on diagnostic imaging?
   (a) Angiography showing occlusion of the proximal SMA without flow seen in distal SMA
   (b) Angiography showing widely patent Celiac with moderate ostial stenosis of SMA <70%
   (c) US Mesenteric duplex showing celiac artery PSV >230 cm/s during expiration with normalizes with inspiration
   (d) US Mesenteric duplex showing elevated SMA velocities during inspiration >225 cm/s which normalizes with expiration

4. A 57-year-old male with chronic mesenteric ischemia and 20 lb weight loss over the past 5 months presents to your clinic. His PMH is notable for DM2, HTN, HLD, and CKD stage 4. On CTA there is circumferential calcification of the abdominal aorta, mild stenosis of the celiac origin, occlusion of the proximal SMA with retrograde filling distally, occlusion of the IMA, common iliac arteries are patent without significant disease, patent right EIA without significant disease, and circumferential calcification of the left EIA. What is the best operative plan?
   (a) Medical management
   (b) Retrograde SMA bypass with inflow from R CIA
   (c) Hybrid approach with Retrograde SMA stent placement
   (d) Antegrade bypass to SMA
5. A 40-year-old male with PMH notable for HTN, HLD, A-Fib, ESRD on HD, and PAD presents to the ED with acute abdominal pain starting 2 h ago. On abdominal exam he is diffusely tender throughout with light palpation. He is afebrile but his BP is 90/50 and HR is 110 s. Labs show WBC 11; Hgb 9.0; Lactate 6; SCr 1.4. What is your next best step in management.
   (a) Urgent mesenteric arterial duplex
   (b) NPO/NGT placement and Serial Abdominal Exams
   (c) IVF resuscitation and IV antibiotics with noncontrast CT abdomen/pelvis
   (d) IVF resuscitation/heparin drip/IV antibiotics with stat CTA abdomen/pelvis

Answers: 1 (b), 2 (d) 3 (c) 4 (b) 5 (d)

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