

# Aortic Diseases

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## I. Abdominal Aortic Aneurysms

### A. Etiology

1. Atherosclerotic, degeneration
2. Genetic role due to high incidence in family members (11-fold risk in first-degree relative)
3. Pathogenesis unknown: theories include abnormal collagen and elastin, increased matrix metalloproteinases or decreased inhibitors (TIMP), increased plasmin activity, immunologic, flow disturbances, and inflammatory

### B. Risk factors

1. Smoking
2. Family history
3. Atherosclerosis
4. Male: Female 5:1
5. Hypertension
6. Decreased prevalence in blacks and diabetic

### C. Presentation

1. Asymptomatic in 75% at time of diagnosis
2. Local mass effects on the GI tract, lumbar spine, ureters
3. Thrombosis
4. Embolize
5. Rupture

### D. Risk of rupture (5 year)

1. 7 cm or larger – >75%
2. 6 cm – 35%
3. 5-5.9 cm – 25%

### E. Diagnosis

1. Physical exam, limited in obese patients and small aneurysms
2. Sometimes seen on plain x-ray by calcified rim
3. Ultrasound good for screening and followup
4. CT very accurate
  - Delineates extent, suprarenal or iliacs
  - Identifies inflammatory aneurysms
  - Shows abnormal anatomy (horseshoe kidney, duplicated vena cava, left sided vena cava, retroaortic renal vein (5%), tumors)
  - Sufficient preop imaging for most cases
5. Aortography
  - Does not accurately show size because of intraluminal thrombus
  - Not necessary preop unless suprarenal, suspect mesenteric and renal stenosis, iliac occlusive disease

6. Most located infrarenal

### **F. Operative repair**

1. Repair when 5 cm or larger
2. Expansion 5 mm/6 month period
3. Elective Cardiac evaluation: Stress test, thallium, dobutamine echo, or coronary cath.
4. Dacron or PTFE graft replacement
5. Retroperitoneal approach for hostile abdomen suprarenal involvement, may have decreased pulmonary complications, not good when R iliac involved
6. IMA usually ligated but reimplant if poor back-bleeding, low pressure, previous colectomy, neither internal iliac patent
7. Remember: Cancer and AAA, treat the more severe/symptomatic problem first. AAA it's the size, Cancer it's the obstruction/bleeding problem

### **G. Early complications**

1. Post Op MI
2. Renal failure from embolization or hypotension
3. Trash foot from embolization
4. Ischemic colitis
  - Suspect when early BM, diarrhea
  - Sigmoidoscopy to assess degree of ischemia and need for colectomy
7. Ureteral injury
8. Sexual Dysfunction
  - Injury to sympathetic plexus over left common carotid artery
  - Retrograde ejaculation 66%, Impotence 33%

### **H. Late complications**

1. Anastomotic aneurysm: should be repaired
2. Graft infection: requires removal and extra-anatomic bypass, some advocate in situ replacement with cryovein
3. Aortoenteric fistula
  - Usually to third/fourth part of duodenum from proximal anastomosis
  - Presents with GI bleeding, suspect if previous graft
  - Requires prompt endoscopy, in OR if massive bleeding
  - CT scan to look for perigraft air and fluid in stable patient if endoscopy negative
  - Treat as for infected graft with bowel repair.

### **I. Ruptured AAA**

1. 50% mortality before hospital, 50% mortality for those arriving to hospital
2. Presents with abdominal and back pain, shock, pulsatile abdominal mass
3. Prompt operation, CT only in stable patients with unclear diagnosis

4. Prep and drape before induction.
5. Resuscitate after supraceliac aortic control.

**J. Endovascular stent graft repair:**

1. Recognized, safe option
2. Needs suitable anatomy
3. Should be considered in high risk patient, esp. elderly
4. Definitely decreases hospital stay, blood loss, and post op complications
5. Needs frequent followup and imaging
6. Conversion open rate 5%
7. Mortality 1-5 %

**II. Iliac Aneurysms**

- A. Usually extension of AAA (20% involve iliacs), uncommon as an isolated aneurysm**
- B. Occurs in common or internal iliac, rarely in external iliac**
- C. Internal may be palpable on rectal exam**
- D. Replace common iliac aneurysm with graft**
- E. Ligate and oversew branches of internal iliac aneurysm.**
- F. Can also be safely repaired with covered stent-graft**

**III. Splenic Artery Aneurysms**

- A. Most common visceral aneurysm (60%) but still rare (<1%), most common in women (4: 1), especially multiparous**
- B. Medial degeneration of arterial wall**
  1. Associated with multiple pregnancies, fibromuscular dysplasia, portal hypertension, pancreatitis
  2. Calcification in up to 70%
- C. Risk of rupture**
  1. 95% of splenic aneurysms diagnosed during pregnancy are ruptured, with 70% maternal mortality and up to 95% fetal mortality.
  2. 2% of asymptomatic aneurysms in nonpregnant patients, 25% mortality
  3. Double rupture phenomenon, first into lesser sac, then peritoneal cavity
- D. Indications for treatment**
  1. All symptomatic aneurysms, usually LUQ or epigastric pain, radiation to L subscapular area
  2. Aneurysms discovered in women who are pregnant or plan to be
  3. Aneurysms > 2-3 cm in very good risk patients with expected mortality < 1%

**E. Treatment options**

1. Aneurysmectomy or ligation (reconstruction rarely indicated), with splenectomy if distal
2. If associated with pancreatitis, may require distal pancreatectomy or pseudocyst drainage
3. Percutaneous transcatheter embolization in high-risk patients

**IV. Hepatic Artery Aneurysms**

**A. Second most common visceral aneurysm (20%), male to female ratio 2:1**

**B. 80% extrahepatic, 20% intrahepatic (most are traumatic)**

**C. Rupture in 20%, mortality rate 35%**

1. Rupture into bile duct may cause hematuria and GI hemorrhage.
2. Free intraperitoneal rupture with shock
3. Rupture into stomach or duodenum uncommon

**D. Treatment**

1. All should be treated unless extremely high risk.
2. Ligation and aneurysmectomy for common hepatic aneurysm
3. Reconstruction (vein preferred) may be necessary for proper hepatic aneurysm.
4. Intrahepatic aneurysm may require liver resection, percutaneous embolization may be an option.

**V. Other Visceral Aneurysms**

**A. SMA aneurysms**

1. 5.5% of visceral aneurysms, majority are mycotic.
2. May rupture or occlude
3. All should be repaired.

**B. Celiac aneurysms**

1. 4% of visceral aneurysms
2. 13% rupture rate, mortality 40%
3. All should be repaired or ligated.

**C. Gastric or gastroepiploic aneurysms**

1. 90% of reported cases present with rupture, 70% into GI, 30% intraperitoneal.
2. All should be treated with ligation, can be embolized.

## **VI. Renal Artery Aneurysms**

- A. Incidence 0.1% general population, 0.7% hypertensive patients, 9% in patients with renal artery fibromuscular dysplasia**
- B. Most are saccular, extrarenal, at branch points.**
- C. Rarely rupture, higher risk of rupture during pregnancy, with maternal mortality 50%, fetal mortality 80%, often requiring nephrectomy**
- D. Indications for repair**
  - 1. Pregnant or planning pregnancy
  - 2. > 2 cm
  - 3. Causing embolization, renovascular hypertension, flank pain, or hematuria
  - 4. Dissecting aneurysm

## **VII. Renovascular Hypertension**

### **A. Pathophysiology**

- 1. Renin produced by juxtaglomerular cells and secreted into renal veins in response to decreased renal blood flow
- 2. Renin converts angiotensin in plasma to angiotensin I
- 3. Angiotensin I converted to angiotensin II by angiotensin converting enzyme in lung
- 4. Angiotensin II is a vasoconstrictor and stimulates aldosterone secretion by adrenal cortex.
- 5. Aldosterone elevates blood pressure by increasing resorption of sodium and water.
- 6. If contralateral kidney normal, compensatory diuresis normalizes volume-, hypertension due to vasoconstriction
- 7. With bilateral disease, hypertension due to volume expansion

### **B. Renovascular hypertension accounts for 5% of hypertension**

- 1. 2/3 by renal artery atherosclerosis
- 2. 1/3 by fibromuscular dysplasia (medial)
- 3. Rarely by renal artery aneurysm or dissection

### **C. Diagnosis**

- 1. High index of suspicion in young patient, recent onset, or severe, unresponsive to treatment
- 2. Duplex scanning ideal noninvasive screening
- 3. Arteriography to confirm renal artery stenosis
- 4. Functional significance by
  - Selective renal vein sampling (> 1.5 times)
  - Radionuclide renal scanning with captopril infusion

### **D. Indications for treatment**

- 1. All patients with severe, difficult to control hypertension
- 2. Young patients with moderate hypertension

### **E. Treatment options**

1. Aortorenal bypass, vein or prosthetic, hypogastric artery in children (vein becomes aneurysmal)
2. Endarterectomy
3. Hepatorenal (R) or splenorenal (L) bypass
4. Angioplasty, good for fibromuscular dysplasia (women) and short, nonorificial lesions; stent improves results for orifice stenosis.
5. Nephrectomy for poor-risk, non-reconstructable, or end-stage renal disease
6. Ex vivo repair

## **VIII. Acute Mesenteric Ischemia**

### **A. Presentation**

1. Abdominal pain out of proportion to physical findings
2. Vomiting or diarrhea
3. Peritoneal signs or acidosis are late signs.

### **B. Causes**

1. Mesenteric artery occlusion from embolism (1/3) or thrombosis (1/3)
2. Nonocclusive mesenteric ischemia (1/3)
3. Mesenteric vein thrombosis (uncommon)

### **C. Mesenteric artery embolism**

1. Embolism most commonly to superior mesenteric artery, usually distal to middle colic, so spares proximal jejunum
2. Underlying cardiac arrhythmia, myocardial infarction, or recent aortic catheterization (cholesterol emboli)
3. Angiogram if time permits
4. Embolectomy, resect non-viable bowel, remember second look in 48 hours.

### **D. Mesenteric artery thrombosis**

1. Underlying atherosclerotic stenosis, often with history of postprandial abdominal pain and weight loss (chronic mesenteric ischemia)
2. Occurs at origin of SMA, with poor collaterals from celiac and IMA disease, involves entire small bowel and proximal colon
3. Needs Angiogram, possible CT angio
4. Requires endarterectomy or aortomesenteric bypass to prevent rethrombosis, may consider thrombolysis and angioplasty if no infarction

### **E. Nonocclusive mesenteric ischemia**

1. Underlying moderate to severe mesenteric atherosclerosis, poor cardiac function, vasoactive agents, esp. digitalis
2. Angiography shows secondary and tertiary SMA branches with irregular narrowing
3. Intra-arterial papaverine, optimize cardiac output
4. Exploration if abdominal signs and symptoms persist

**F. Mesenteric venous thrombosis**

1. Underlying hypercoagulable state, CHF, Portal HTN, intra-abdominal sepsis/malignancy, DVT
2. Contrast CT can be diagnostic
3. Long term Anticoagulation, possible thrombolysis
4. Operation for infarction, possible thrombectomy of central veins
5. Second look

**IX. Chronic Mesenteric Ischemia**

- A. Significant atherosclerosis of at least 2 of the 3 mesenteric vessels, widely patent celiac or SMA unlikely to be found**
- B. All have weight loss due to postprandial pain (intestinal angina)**
- C. Abdominal bruit**
- D. Duplex scan: noninvasive screening**
- E. Biplanar aortography required before operation**
- F. Treat with aortoceliac and aortomesenteric bypass, endarterectomy, percutaneous angioplasty has been described.**
- G. Celiac compression by median arcuate ligament**
  1. Uncommon, some question clinical significance
  2. Treatment by division of median arcuate ligament