

## SUPERIOR MESENTERIC VEIN THROMBOSIS

- Superior mesenteric vein (SMV) thrombosis causes 5%-10% of acute mesenteric ischemia.
- Intestinal ischemia following mesenteric vein thrombosis is due to resistance in the mesenteric venous blood flow causing profound wall edema, fluid efflux into the bowel lumen with resulting systemic hypotension and increase in blood viscosity.
- As a result, arterial flow is diminished leading to submucosal hemorrhage and bowel infarction.
- Up to 75% of patients with mesenteric venous thrombosis have an inherited thrombotic disorder. However, the true prevalence of these disorders in patients with mesenteric venous thrombosis is difficult to estimate because most studies include patients with all forms of deep venous thrombosis.
- The clinical presentation is often subtle early in the disease and becomes characteristic only when advanced and severe, when ischemia progresses to necrosis.
- The mortality of SMV thrombosis is approximately 25%

Inherited Hypercoagulable States	Acquired Hypercoagulable States
Factor V leiden mutation (20-40%)	Malignancy
APC resistance (10%)	Surgery
Prothrombin gene mutations (8%)	Trauma
Protein C deficiency	Pregnancy
Protein S deficiency	Oral contraceptives
Antithrombin deficiency	IBD
Antiphospholipid Ab (4%)	Immobilization
	Hormone replacement therapy
	Pregnancy,smoking,h/o thromboembolism

### Signs & Symptoms

- abdominal pain in 85%-usually severe and disproportionate to physical findings
- anorexia in 50%
- GI bleeding in 45%
- nausea and vomiting in 45%
- Physical signs depend on the severity and stage of intestinal injury.
- Peritoneal signs are late manifestations that indicate bowel infarction.
- Laboratory abnormalities include: leukocytosis, neutrophilia, elevated serum lactate, and high amylase.
- Metabolic acidosis and hypoxia suggest severe intestinal insult.

### Diagnosis

- The diagnosis is usually made by CT or angiography.
- **Plain films**
  - Plain x-rays are usually normal or demonstrate nonspecific abnormalities, ie ileus.
  - Significant x-ray abnormalities, such as pneumoperitoneum or pneumatosis intestinalis, usually reflect bowel infarction but rarely differentiate between arterial and venous occlusion.

- **CT scan**
  - diagnostic in approx. 80% of cases
  - demonstrates thrombus in the mesenteric, portal, or splenic vein as a central area of low density surrounded by an enhanced peripheral vascular rim.
  - dilated SMV, collateral mesenteric vessels, intestinal mural thickening, vascular engorgement, dilated bowel, thickened and streaky mesentery
  - polypoid intraluminal projections (corresponds to the thumbprints present on abdominal x-ray)
- **Angiography**
  - slow or absent filling of SMV
  - persistent filling defect in the SMV from the thrombus
  - reconstitution of flow around the thrombus by collateral vessels (if chronic component of obstruction is present)
  - resistance to venous flow permits visualization of refluxed contrast into aorta
  - contrast extravasation into bowel lumen, indicating active bleeding
  - less sensitive in detecting superior mesenteric venopathy than arteriopathy
  - SMV may fail to opacify (technical factors), requiring selective injections with higher contrast volumes or during papavarine infusion

### **Treatment**

- IV anticoagulation with heparin
- Prolonged anticoagulation with coumadin post acute heparinization (6months - longer if thrombophilic condition present)
- Surgery to resect nonviable bowel if peritoneal signs present
- Thrombolytic therapy (streptokinase, urokinase) - success in small number of patients though still considered experimental

### **SMV Thrombosis and IBD**

- Recent studies have shown that the risk of venous thrombotic events and pulmonary embolus in IBD is three times higher than in the general population, and the relative risk is even higher in the younger age group.
- These findings support evidence that an intrinsic prothrombotic state is present in IBD.
- Occlusive fibrinoid lesions have been described in intestinal arteries in the early stages of Crohn's, and submucosal thrombosis has been reported in UC (even in normal bowel).
- There are histologic similarities between Crohn's disease and intestinal models of embolization.
- Resistance to Factor V leiden deficiency, PT G20210A mutation, deficits in Anti-Thrombin III and protein C/S, anticardiolipin Ab have all been examined in IBD patients, though not more frequently than in the general population.
- However, when these inherited hypercoagulable states are present in IBD patients, they put patients at a higher risk for developing thrombotic complications.
- The tendency of IBD patients to form arterial and venous clots is not fully understood.

Fichera, et al, Diseases of Colon and Rectum 2003

- evaluate incidence of symptomatic postoperative SMV thrombosis in IBD patients undergoing colonic resections
- retrospective study of 83 patients undergoing total colectomy for IBD
- 14 patients (16.9%) with new onset postoperative acute abdominal pain (6 - 90 days, median interval 10 days) were evaluated by CT of abdomen
- 4 patients (4.8% of entire patient population & 28.6% of the CT group); {3 females; 3 patients with UC, 1 with Crohn's} developed symptomatic postop SMV thrombosis
- patients with diagnosis of SMV thrombosis had complete coagulation profile
- 2 patients with extension of clot into portal vein
- hematologic work-up negative in 3 patients, 1 patient heterozygous for prothrombin G20210A mutation
- all patients successfully treated with systemic anticoagulation (median f/u-18months)
- study concluded that postop SMV thrombosis is more frequent than previously recorded in patients with IBD, possibly resulting from direct surgical trauma in the setting of a borderline intrinsically hypercoagulable state

#### References

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