Thoracic Emergencies in the Oncology Patient
Richard M. Gore, MD

**THORACIC EMERGENCIES IN THE ONCOLOGY PATIENT**

Richard M. Gore, MD
North Shore University Health System
University of Chicago
Evanston, IL

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**NO DISCLOSURES**
rgore@uchicago.edu

**THORACIC COMPLICATIONS**

- SVC SYNDROME
- PULMONARY EMBOLISM
- CARDIAC TAMPONADE
- MALIGNANT TENSION HYDROTHORAX

**TOPICS**

- SVC SYNDROME
- PULMONARY EMBOLISM
- CARDIAC TAMPONADE
- MALIGNANT TENSION HYDROTHORAX

**SUPERIOR VENA CAVA SYNDROME**
**SVC SYNDROME: DEFINITION**

- Symptom complex caused by obstruction of blood flow in the SVC limiting return of blood from the head, neck, and upper trunk to the right heart

**SVC OBSTRUCTION: CLINICAL FEATURES**

- Engorged veins of neck and upper chest wall-multiple collaterals in chest and upper abdomen
- Laryngeal edema, cyanosis, papilledema, MS changes, stupor, coma, LAD
- Bending forward worsens the venous engorgement

**SVC OBSTRUCTION: ETOLOGY**

- Small cell lung cancer
- Squamous cell lung cancer
- Lymphoma
- Metastases from breast, melanoma

**SVC OBSTRUCTION: ETOLOGY**

- Indwelling catheters
- Pacemaker wires
- Mediastinitis: TB, histoplasmosis
- Thoracic aortic aneurysm

**SVC OBSTRUCTION: CLASSIFICATION**

- LUMEN OBSTRUCTION: malignant thrombus or bland thrombus with pacemaker leads or catheter related thrombosis
- EXTRINSIC COMPRESSION: CA, fibrosing mediastinitis, aneurysm, goiter

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SUPERIOR VENA CAVA SYNDROME:

- Pre or supra-azygos
- Post or infra-azygos

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COLLATERAL CIRCULATION BETWEEN SVC AND IVC

- Internal mammary veins
- Vertebral veins
- Lateral thoracic veins
**MALIGNANT SVC OBSTRUCTION: TREATMENT**

- STEROIDS
- RADIATION THERAPY (lung cancer, lymphoma, germ cell tumors)
- CHEMOTHERAPY
- STENTING
- SURGERY – BYPASS GRAFTING

**BENIGN SVC OBSTRUCTION: TREATMENT**

- ANTICOAGULATION-THROMBOLYSIS
- CATHETER REMOVAL, LEAD EXPLANTATION
- STENTING
- TREATMENT OF INFECTIOUS ETIOLOGY
- SURGERY – BYPASS GRAFTING
**SVC OBSTRUCTION: PROGNOSIS**

- Poor prognosis for malignant conditions
- NSCLC resistant to CXRT < 6 mo survival
- Benign etiology, stents or surgery have a 90% patency rate; need anti-coagulation

**SVC OBSTRUCTION: DIAGNOSTIC YIELD FOR CA**

- Bronchoscopy: 50-70%
- TTNB: 75%
- Mediastinoscopy or mediastinotomy: >90%

**TOPICS**

- SVC SYNDROME
- PULMONARY EMBOLISM
- CARDIAC TAMPONADE
- MALIGNANT TENSION HYDROTHORAX
INCIDENTAL PULMONARY EMBOLISM

- 1.8% overall
- 3.3% progressive cancer
- 2.5% stable cancer
- 0.7% NED
- 1.0% non-oncology patients

Hu GC JCAT 32: 783-787, 2008

INCIDENTAL PULMONARY EMBOLISM IN INPATIENTS

- PE in 5.7%
- 9.2% > 70 years
- 16.7% > 80 years
- Most are peripheral, >30% missed initially


INCIDENTAL PULMONARY EMBOLI ON NON PE MDCT

- 4.0% inpatient prevalence
- 0.9% outpatient prevalence
- 70.0% with unsuspected emboli had cancer
- Wide window settings allow for better embolus detection

Shetty AJR 184: 264-2167, 2005

INCIDENTAL PULMONARY EMBOLISM

- PE in 3.4%
- 4% in inpatients
- 0.9% in outpatients

Storto AJR 62: 464-467, 2005
TOPICS

- SVC SYNDROME
- PULMONARY EMBOLISM
- PERICARDIAL EFFUSION-TAMPONADE
- MALIGNANT TENSION HYDROTHERAX

CARDIAC TAMPONADE
Accumulation of pericardial fluid, blood, tumor, or air that increases intrapericardial pressure, restricting cardiac filling, and decreasing cardiac output.

Cardiac emergency that can be fatal.

**CARDIAC TAMONADE: ACUTE**
- Rapid onset seen in cardiac/great vessel trauma or s/p invasive procedure
- Beck triad: hypotension, jugular venous distention, and distant heart sounds
- Effusion may be small, given the relative inelasticity of the pericardium

**CARDIAC TAMONADE: SUBACUTE**
- More gradual process of fluid accumulation
- Allows for stretching of pericardium and much larger effusions than seen acutely
- The most common type of tamponade seen in malignancy, TB, uremia
- S+S more subtle, some or all of Beck’s triad may be absent

**CARDIAC TAMONADE: EFFUSIVE CONSTRUCTIVE PERICARDITIS**
- Related to scarred pericardium and most often occurs in patients with malignancy or prior radiation exposure

**CARDIAC TAMONADE: PROGNOSIS**
- Patients with underlying malignancy have highest mortality
- In penetrating chest trauma patients, tamponade associated with better outcomes because tamponade acts as a stabilizing force
**CARDIAC TAMPOANADE: ECHO FINDINGS**

- Inversion free wall RA > ½ systole
- RV diastolic collapse
- Pulsus paradoxus
- CXR: pericardium can hold > 200cc of fluid before an enlarged silhouette is noted

**CARDIAC TAMPOANADE: MR FINDINGS**

- Can see as little as 30 cc fluid
- Limited role due to emergent nature
- Swinging heart and paradoxical septal bounce

**CARDIAC TAMPOANADE: MR FINDINGS**

- Simple transudative effusions exhibit low signal intensity on T1W and high signal intensity on T2W images. The presence of septations and debris suggests a complex effusion.

**CARDIAC TAMPOANADE: CT FINDINGS**

- Enlarged SVC ≥ adjacent thoracic aorta
- Enlarged IVC > 2X adjacent aorta
- Contrast reflux into IVC, azygos vein
- Enlarged hepatic and renal veins

**CARDIAC TAMPOANADE: CT FINDINGS**

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**CARDIAC TAMPONADE: CT FINDINGS**

- <0 HU: chylopericardium- CA, infection
- 0-20 HU: simple serous effusion- CHF, renal failure, or non-hemorrhagic CA
- > 20 HU hemopericardium, CA, purulent exudates, or myxedematous effusion

**TOPICS**

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- MALIGNANT TENSION HYDROTHORAX

**MALIGNANT TENSION HYDROTHORAX**

- Pleural effusions develop in 50-70% of all cancer patients
- Tension hydrothorax: unusual complication
- Marked mediastinal shift and compression of lung causes severe hypoventilation and respiratory acidosis
- Pressure on heart and great vessels inhibits central venous return causes decreased cardiac output, metabolic acidosis, and circulatory collapse
TRADITIONAL CHEMOTHERAPY

- Cytotoxic in nature and acts primarily by eliminating neoplastic cells. Change in tumor size, which is an indicator of change in the number of neoplastic cells, evolved into the radiologic biomarker of treatment response.

CYTOTOXIC CHEMOTHERAPY

- Classified by their mechanism of action
- ALKYLATING AGENTS (cyclophosphamide): form DNA cross links
- ANTIMETABOLITES (MTX, 6-MP): inhibit folic acid and purine synthesis respectively
- VINCA ALKYLOIDS (vincristine): inhibit microtubulin formation

- Final effect of all these drugs is to inhibit cell division in rapidly dividing cells
- Reduce cell turnover in cancer cells
- Unfortunately these drugs can also affect normal cells especially those with rapid division
- Why the gut and bone marrow, organs with rapid cell turnover are very susceptible

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**MOLECULAR TARGETED THERAPY (MTT)**

- Significant advances in molecular cytogenetics has led to the development of MTT which selectively target tumor cells and modify their biologic characteristics, act on various targets: growth factor receptors, signaling molecules, cell-cycle proteins, molecules that direct apoptosis and angiogenesis.

**MOLECULAR TARGETED THERAPIES**

- **TKIs:** sunitinib, sorafenib inhibit VEGF are used for renal cell carcinoma
- **MCAs:** rituximab vs CD20 antibody in NHL bevacizumab vs met RCC, CRC

**TYROSINE KINASE INHIBITORS**

- TARCEVA (erlotinib): NSCLC, pancreatic cancer [TKI]
- ERBITUX (cetuximab): CRC, H+N cancer [monoclonal AB vs epidermal growth factor]
- YERVOY (ipilimumab): melanoma [monoclonal AB activates immune system]
**GIST TREATMENT CHANGES: PET-CT**

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**MTT RELATED PNEUMATOSIS**

- Anti-VEGF activity compromises mural integrity
- Wall disruption due to necrosis of serosal tumor implants or mural lesion
- Impaired healing of surgical anastomosis
- Ischemia due to mesenteric venous thrombosis
70.8% of patients were asymptomatic.

Mean duration of MTT 3 months

25% just after 1 month of therapy

70.8% of pneumatosis or perforation found incidentally in asymptomatic patients on routine follow-up studies.

90% pneumatosis involved normal bowel with or without tumor deposits

Most perforations occurred at tumor sites or at a surgical anastomosis

Only 40% of patients had pneumoperitoneum

Mesenteric venous gas seen in 40% of pneumatosis patients - no PVG seen

Most patient recovered with drug cessation

All patients with pneumatosis successfully treated conservatively - all recovered with D/C medication

Surgery avoided in most patients with perforation

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PSEUDOMEMBRANOUS COLITIS

PSEUDOMEMBRANOUS COLITIS RISK FACTORS
- ANTIBIOTICS
- S/P SURGERY, SHOCK, BURNS, CARDIAC ARREST, SCI
- PROXIMAL TO COLON OBST
- HUS, ISCHEMIC COLITIS, UREMIA
- LEUKEMIA, LYMPHOMA, AIDS

PSEUDOMEMBRANOUS COLITIS: CT FEATURES
- CIRCUMFERENTIAL AND DIFFUSE MURAL THICKENING
- PROMINENT HAUSTRA
- ECCENTRIC POLYPOID WALL THICKENING
- SHAGGY LUMINAL CONTOUR
- INHOMOGENEOUS BOWEL WALL
- BA TRAPPED BETWEEN THICK FOLDS SIMULATE SINUS TRACTS
PSEUDOMEMBRANOUS COLITIS: CT FEATURES

- Circumferential and diffuse mural thickening
- Prominent haustra
- Eccentric polypoid wall thickening
- Shaggy luminal contour
- Inhomogeneous bowel wall
- BA trapped between thick folds simulate sinus tracts

TYPHLITIS

TYPHLITIS: CLINICAL FEATURES

- Necrotizing inflammation of the cecum
- Transmural edema and ulceration can cause perforation
- Pseudomonas, Candida, CMV, E coli
- ALL, AIDS, aplastic anemia
- Fever, neutropenia, watery-bloody diarrhea

TYPHLITIS: CT FEATURES

- Circumferential and diffuse mural thickening
- Low density intramural areas: edema/necrosis
- Pericolonic fluid
- Pneumatosis
- Thickening of fascial plane
- CT to follow wall thickness and detect pneumoperitoneum

INFECTIOUS COLITIDES

RIGHT COLON:
Shigella, Salmonella

DIFFUSE COLITIS:
E Coli, CMV, Crypto

RECTOSIGMOID:
HSV, LGV, Gonorrhea

LEFT COLON+RS:
Schistosomiasis

CMV COLITIS
GI RADIATION INDUCED TOXICITY

- S+S: diarrhea, rectal bleeding, fecal incontinence
- Acute: occurs within 3 months of end of therapy
- Chronic: occurs after 3 month period

GI RADIATION INDUCED TOXICITY: SITES IN PORTAL

- RECTAL CANCER: 9.5-20%
- PROSTATE CANCER: 6-15%
- ANAL CANCER: 3-7%
- CERVICAL CANCER: 9.5-3.6%

GVHD

GVHD
GVHD

- Functioning T lymphocytes of the graft are introduced into an immunocompromised recipient and damage the epithelial and vascular endothelial structures of the host’s skin, liver, and gastrointestinal tract
- Not only a primary complication but acts as a predisposing factor for other complications

Maculopapular rash
Hyperbilirubinemia
Hepatic dysfunction
Diarrhea
Malabsorption
GI hemorrhage

HEMATOPOIETIC STEM CELL TRANSPLANTATION

- Acute and chronic myeloid leukemia
- Myelodysplastic syndrome
- Acute lymphoblastic leukemia
- Multiple myeloma
- Hodgkin’s disease
- Non-Hodgkin’s lymphoma

PRE-ENGRAFTMENT PHASE (DAY 10-30)

- The period immediately after the myeloablative therapy
- Severe immune compromise due to pancytopenia
- Host defense barriers are further weakened by mucositis and other chemotherapy related complications

Marrow aplasia
Typhlitis
Pseudomembranous colitis
**EARLY POST-TRANSPLANT PHASE**

- After successful engraftment of donor stem cell there is resumption of hematopoiesis however lymphocyte recovery lags

**LATE POST-TRANSPLANT PHASE (> 100 days)**

- Lymphocyte levels return to normal but recovery of humoral immunity lags behind gradually improving through the first year following transplantation

**GVHD: CT FINDINGS**

- Mural thickening of SB (100%), colon (59%), stomach (9%), distal eso (9%)
- **NO** mesenteric adenopathy
- Mucosal hyperenhancement 54%
- Discontinuous mural thickening 41%
- Serosal hyperenhancement 31%
- Dilated bowel 21%

Kalantari  *BN AJR 181: 1621-1625, 2003*

**GVHD: CT FINDINGS**

- Engorged vasa rectae 91%
- Mesenteric fat stranding 73%
- Ascites 45%
- Splenomegaly 36%
- Periportal edema 36%

Kalantari  *BN AJR 181: 1621-1625, 2003*

**GVHD: CT FINDINGS**

- GB wall hyperenhancement 23%
- Biliary sludge 18%
- Pericholecystic fluid 18%
- Thick GB wall 9%
- Hepatomegaly 9%

Kalantari  *BN AJR 181: 1621-1625, 2003*
Steatosis and steatohepatitis are side effects of chemotherapy and are most often seen in patients with breast and colon CA receiving tamoxifen, irinotecan, 5FU and leucovorin. Mediated by production of reactive oxygen species (ROS) resulting in oxidative stress in hepatocytes. Steatosis is reversible if chemotherapy is discontinued.

Steatohepatitis affects hepatic reserve for regeneration leading to increased risk of 90 day post op mortality. Fatty liver should be reported in all chemo therapy patients as it may necessitate a change in therapy.
STEATOSIS: CT FEATURES

- LOW ATTENUATION < Portal Vein
- LIVER DENSITY < Spleen
- "HYPERDENSE" INTRAHEPATIC VESSELS

ON NON-CONTRAST CT
NORMAL LIVER 60HU
LIVER DENSITY = SPLEEN
**SINUSOIDAL OBSTRUCTION SYNDROME (SOS)**

HEPATIC VENO-OCCCLUSIVE DISEASE

- Endothelial injury is the initiating event
- Produces a hypercoaguable state
- Venular and sinusoidal lumen reduced due to edema, partial to complete fibrosis of venular lumina
- Cyclophosphamide most common agent

**SOS: SINUSOIDAL OBSTRUCTION SYNDROME**

- 70% of patients recover spontaneously
- Rapidly increasing transaminases and bilirubin, portal vein thrombosis, multiorgan failure
- Treat with anti-thrombic therapy
- Long term survivors at risk for elevation of LFTs

**PORTAL HYPERTENSION**

- Hepatic vein
- Coronary vein
- Sinusoid
- Liver
- Inferior vena cava
- Portal vein
- Splenic vein
- Superior mesenteric vein
- Inferior mesenteric vein
- Hepatic artery
PRE-HEPATIC PORTAL HYPERTENSION

Hepatic vein

Portosystemic collaterals

Sinusoid

Portal vein

Splenomegaly

Thrombus

Liver

PRE-SINUSOIDAL PORTAL HYPERTENSION

S. mansoni eggs

Portosystemic collaterals

Sinusoid

Portal vein

Splenomegaly

POST-SINUSOIDAL PORTAL HYPERTENSION

Centrilobular fibrosis

SOS

Sinusoid

Portal vein

Splenic vein

POST-HEPATIC PORTAL HYPERTENSION

Thrombus

Sinusoid

Portal vein

Splenomegaly

Liver

SOS

- Periportal edema
- Ascites
- RHV < 4.5 mm
- Thick GB wall
**SOS vs GVHD**

- SOS: periportal edema, ascites, narrow right hepatic vein
- GVHD: mural thickening of small bowel

Erturk SM AJR 186: 1497-1501, 2006