Unusual presentations of fatal conditions in the ED

Dr. Vu Kiet Tran, MD, FCFP, MHSc, MBA
Example 1

• Visit 2 (seen by NP #1): 31yo male presenting with “confusion” and persistent headache
• Was snowboarding 1 ½ months ago. Wearing helmet
• Fell
• LOC for about 1 min.
Example 1

• PMHx: GERD
• Meds: Prevacid
• Allergies: Tetracycline
• Drugs: no
• ETOH: occ
Example 1

- Visit 1 (seen by NP#1): Seen in the ED 5 days after the fall because of confusion and headache
- c/o dizziness, nausea
- Headache is constant since the fall
- Numbness and tingling in left hand
- Taking Advil/ASA ever 4h prn and “muscle relaxant”
- No CT head done at the time of the fall
Example 1

- Visit 1:
- CT head N
- D/C with diagnosis of “Concussion” and head injury instructions, Advil/Tylenol prn
Example 1

- Visit 2:
- Story is same
- Persistent headache
- Wakes up from headache. Relieved by Tylenol #3
- Difficulty concentrating at work
- Intermittent blurred vision
- Intermittently cannot recall his colleagues’ names
- Today, difficulty typing and making proper sentences
Example 1

- Visit 2:
- O+A x 4
- Pupils equal, PERL
- Gait N, tandem N, heels/toes N
- Finger-nose N
- RAM N
- CN N
- Romberg N
- Pronator drift negative
Example 1

- Initial impression?
- Investigations?
- Interventions?
- Disposition?
Example 1

• Impression: “Post-concussion Syndrome” vs slow space-occupying lesion?
• Follow-up by Family doctor
• Get MRI in Buffalo the next day (was driving to Buffalo tomorrow for meeting and wanted to avoid CT head if possible)
Example 1 – Subdural Hematoma

- Visit 3 (2 days after visit 2)
- MRI (from Buffalo): Left subdural hematoma, 16.9cm x 2.8cm x 8.7cm with shift of 1.5 cm.
- Neuro exam normal
- Neurosurgery called
- Left burr hole
- Discharged 2 days after visit 3
Mild head injury

• 80% of cases of head injury
• 1% will have a neurosurgical correctable problem
Post-concussive syndrome

• 29-90% of patients develop post-concussive syndrome as a result of blunt traumatic brain injury
  – 50% have symptoms at 1 month
  – 15% have symptoms at 1 year
Post-concussive syndrome

• Definition: 3 of the following
  – Headache
  – Dizziness
  – Cognitive deficit
  – Fatigue
  – Irritability
  – Insomnia
  – Sensitivity to light and sound
Chronic subdural hematoma

• Symptoms occur insidiously (developing weeks after the injury)
  – Headache
  – Light-headedness
  – Cognitive impairment
  – Apathy
  – Somnolence
  – seizures
Comparison

**Post-Concussive Syndrome**
- Headache
- Dizziness
- Cognitive deficit
- Fatigue
- Irritability
- Insomnia
- Sensitivity to light and sound

**Chronic Subdural**
- Headache
- Light-headedness
- Cognitive impairment
- Apathy
- Somnolence
- seizures
“Acute on chronic” subdural Hematoma

• Dural collagen synthesis is induced and fibroblasts spread over the inner surface of the dura to form a thick outer membrane, resulting in an encapsulation of the clot.
• Over time, the chronic SDH can liquify and form a hygroma.
• At any time, the hematoma may expand due to re-bleeding or from osmotic draw of water into the hygroma (high protein content)
“Acute on chronic” subdural Hematoma

- Jan 2008-Dec 2010
- 9 cases (8% of chronic SDH) of “acute on chronic subdural hematoma”
- Most common cause of re-bleed: slip and fall (after ETOH intoxication)


**“Acute on chronic” subdural Hematoma**

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**Table 1. Clinical features of patients with acute-on-chronic subdural hematoma**

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>GCS</th>
<th>Cause</th>
<th>Symptom</th>
<th>Past History</th>
<th>PT(INR)</th>
<th>Anticoagulants</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>66</td>
<td>9</td>
<td>?</td>
<td>Discovered on the road</td>
<td>Craniotomy 10 yr pta</td>
<td>1.12</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>48</td>
<td>3</td>
<td>Slip</td>
<td>Discovered on the road</td>
<td>HI 10 D, 1 M pta; warfarin for atrial fibrillation</td>
<td>2.44</td>
<td>Aspirin, warfarin</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>52</td>
<td>10</td>
<td>?</td>
<td>Discovered on the road</td>
<td>Traffic accident 15 yr pta</td>
<td>0.94</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>52</td>
<td>15</td>
<td>?</td>
<td>Headache for 9d</td>
<td>Aspirin</td>
<td>1.01</td>
<td>Aspirin</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>63</td>
<td>15</td>
<td>Slip</td>
<td>Hemiparesis for 3d</td>
<td>Alcoholic LC; slip 2 M pta</td>
<td>0.95</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>65</td>
<td>15</td>
<td>Slip</td>
<td>Hemiparesis for 3d</td>
<td>Craniotomy 12 yr pta</td>
<td>0.97</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>69</td>
<td>15</td>
<td>Slip</td>
<td>Hemiparesis for 1d</td>
<td>Slip 3 M pta</td>
<td>0.99</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>80</td>
<td>11</td>
<td>?</td>
<td>Hemiparesis for 4d</td>
<td>Diabetes, COPD, alcoholic LC</td>
<td>1.12</td>
<td>Aspirin</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>83</td>
<td>15</td>
<td>Slip</td>
<td>Hemiparesis for 2w</td>
<td>Stomach CA 7 yr pta; femur fracture 5 yr pta</td>
<td>1.06</td>
<td>No</td>
</tr>
</tbody>
</table>
“Acute on chronic” subdural Hematoma

Findings:

- Alcoholism with multiple episodes of trauma was one of the prominent features
- Spontaneous bleeding could be due to rupture of fragile thin walls from the neomembrane
Delayed ICH after minor head injury (and shock)

- 10,095 consecutive head injury from 1977-1987
- 237 presented with shock (multi-system injury)
- 10 had initial normal CT head scan, then eventually developed ICH
## Delayed ICH after minor head injury

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td>8 male, 2 female</td>
</tr>
<tr>
<td><strong>GCS</strong></td>
<td></td>
</tr>
<tr>
<td>GCS 13-15</td>
<td>4</td>
</tr>
<tr>
<td>GCS 9-12</td>
<td>3</td>
</tr>
<tr>
<td>GCS &lt; 9</td>
<td>3</td>
</tr>
<tr>
<td><strong>Presence of skull fracture</strong></td>
<td>6</td>
</tr>
<tr>
<td><strong>Primary surgery for extra-cranial systemic injury</strong></td>
<td>4</td>
</tr>
<tr>
<td><strong>Time interval between initial CT scan and the development of delayed ICH</strong></td>
<td>4-12 hours</td>
</tr>
</tbody>
</table>
Delayed ICH after minor head injury

• Postulation
  – Hypotension may provide a protective mechanism to prevent the initial ICH.
  – Once the shock corrected, the ICH then becomes evident
Delayed ICH in minor head injury in children

- 8-year, retrospective, cohort study of children
- < 14 yo
- Between April 1992-March 2000
- Definition of delayed ICH: > 6h after initial injury
- Total 17,962 children with uncomplicated minor head injury
Delayed ICH in minor head injury in children

• Incidence of delayed ICH represented 2.5% of all ICH

• Incidence of delayed ICH *with* deterioration: 0.14 cases per 100,000 children per year

• Incidence of delayed ICH *without* deterioration: 0.57 cases per 100,000 children per year
Delayed ICH in minor head injury in children

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Identified Cases in Which Diagnosis of Intracranial Hemorrhage Was Delayed (&gt;6 Hours After Reported Injury)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
<td>Age</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----</td>
</tr>
<tr>
<td>DDIH with decreased level of consciousness</td>
<td>7 y</td>
</tr>
<tr>
<td></td>
<td>3 y</td>
</tr>
<tr>
<td></td>
<td>13 y</td>
</tr>
<tr>
<td></td>
<td>6 y</td>
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<td>11 y</td>
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<tr>
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<td>7 y</td>
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<td>3 y</td>
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<td></td>
<td>17 d</td>
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<tr>
<td></td>
<td>4 y</td>
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<tr>
<td>NAT*</td>
<td>3 y</td>
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<tr>
<td></td>
<td>2 y</td>
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<tr>
<td></td>
<td>7 y</td>
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<td></td>
<td>6 mo</td>
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<tr>
<td></td>
<td>2 y</td>
</tr>
<tr>
<td></td>
<td>4 y</td>
</tr>
<tr>
<td>Di*</td>
<td>2 y</td>
</tr>
<tr>
<td></td>
<td>5 y</td>
</tr>
</tbody>
</table>

Pediatrics 2010; 126: e33-e39
Delayed ICH and pre-injury warfarin or Clopidogrel use

• Study objective
  – To understand the prevalence of immediate traumatic (blunt) intracranial hemorrhage and the cumulative incidence of delayed traumatic intracranial hemorrhage in patients receiving warfarin or clopidogrel

Ann Emerg Med, June 2012; 59 (6): 460-468
Delayed ICH and pre-injury warfarin or Clopidogrel use

- A prospective, observational study at 2 trauma centers and 4 community hospitals
- Emergency department (ED) patients with blunt head trauma and preinjury warfarin or clopidogrel
- April 2009 through January 2011
- Patients were followed for 2 weeks
- Delayed traumatic intracranial hemorrhage was defined as traumatic intracranial hemorrhage within 2 weeks after an initially normal CT scan result and in the absence of repeated head trauma.
Delayed ICH and pre-injury warfarin or Clopidogrel use

• A total of 1,064 patients were enrolled
  – 768 warfarin patients [72.2%]
  – 296 clopidogrel patients [27.8%]

• The prevalence of immediate traumatic intracranial hemorrhage
  – clopidogrel 12.0% (33/276; 95% CI 8.4% to 16.4%)
  – warfarin 5.1% (37/724; CI 3.6% to 7.0%)
  – relative risk 2.31 (95% CI 1.48 to 3.63)

• Delayed traumatic intracranial hemorrhage
  – Warfarin 0.6% (4/786; CI 0.2% to 1.5%)
  – Clopidogrel 0% (0/243, CI 0% to 1.5%).
Key red flags

- Wake up from headache
- New or worsening cognitive impairment
- Seizures
Example 2

- 84yo female presents with chest pain
- While getting dressed today at 1000am (seen in the ED 1.5 hours later), felt CP across both scapula.
- No irradiation
- No pleuretic
- No pain with movement of shoulders
- Now, CP anterior of the chest and abdomen
Example 2

• No SOB, but “cannot take deep breaths”
• No Fever/nausea/vomiting
• Cough for 6 weeks
• Some phlegm
Example 2

• VS: 128/81, 96, 18, 36.5°, 95% on RA
• Looks well
• Not in any discomfort at all
• Heart N, no murmurs
• Lungs clear
• T-spine N
• Chest N, no tenderness
• Right and left scapula and rhomboid muscles N
Example 2

• ECG: NSR, No ST-T changes
• Trop neg
• LFTs, Amylase N
• CXR: unrolled Aorta, cardiac silhouette N, no pneumothorax
Example 2

• What is your preliminary diagnosis?
• What investigations would you do?
• What would be her disposition?
Example 2

• BP (L): 163/90
• BP (R): 137/92
Example 2

- CT chest: Type B intramural hematoma with compression of the true lumen to 8mm in the descending Aorta. No intimal flap.
Aortic dissection

• Defined as separation of the layers within the aortic wall
  – Tears in the intimal layer result in the propagation of dissection (proximally or distally)

• Mortality is still high despite advances in diagnostic and therapeutic modalities
Example 2

• The diagnosis of acute aortic dissection requires a high index of suspicion
  – History and physical examination
  – Electrocardiography
Presentation

- No one sign or symptom can positively identify acute aortic dissection
  - Sudden onset of severe chest pain that often has a tearing or ripping quality (classic symptom)
    - Chest pain may be mild
    - No pain in about 10% of patients
  - Neck or jaw pain: With aortic arch involvement and extension into the great vessels
  - Tearing or ripping intrascapular pain: May indicate dissection involving the descending aorta
  - Syncope
  - Stroke symptoms
  - Altered mental status
  - Numbness and tingling, pain, or weakness in the extremities
  - Horner syndrome (i.e., ptosis, miosis, anhidrosis)
  - Dyspnea or Hemoptysis
  - Dysphagia
  - Fever
  - Anxiety and premonitions of death
The painless dissection

- Aortic dissection is painless in about 10% of patients
- Painless dissection is more common in
  - Neurologic complications
  - Marfan syndrome.
## Classification

<table>
<thead>
<tr>
<th>Standford</th>
<th>DeBakey</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Ascending Aorta is involved (DeBakey I and II)</td>
</tr>
<tr>
<td>B</td>
<td>Descending Aorta is involved (DeBakey III)</td>
</tr>
<tr>
<td>C</td>
<td>Only the ascending Aorta is involved</td>
</tr>
</tbody>
</table>
Imaging

• Chest radiography:
  – Widening of the mediastinum is the classic finding
  – Hemothorax may be evident if the dissection has ruptured

• Computed tomography with contrast:
  – The definitive test
  – Useful only in hemodynamically stable patients

• Echocardiography:
  – TEE is more accurate than TTE
  – TTE is most useful in ascending aortic dissections
  – TEE is as sensitive and specific as CT scanning and MRI
  – TEE is strongly dependent on operator experience

• MRI:
  – The most sensitive method for diagnosing aortic dissection
  – Specificity is similar to that of CT scanning

• Aortography:
  – Is being replaced by newer, safer imaging modalities
## Management

<table>
<thead>
<tr>
<th>Type A (DeBakey I and II)</th>
<th>Type B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emergency surgical correction</td>
<td></td>
</tr>
<tr>
<td>• Intimal tear is resected and replaced by Dacron graft</td>
<td></td>
</tr>
<tr>
<td>• Endovascular repair</td>
<td></td>
</tr>
<tr>
<td>Medical management</td>
<td></td>
</tr>
<tr>
<td>• Decreasing the blood pressure and the shearing forces of myocardial contractility</td>
<td></td>
</tr>
<tr>
<td>• Antihypertensive therapy</td>
<td></td>
</tr>
<tr>
<td>• Pain management</td>
<td></td>
</tr>
<tr>
<td>Stenting</td>
<td></td>
</tr>
</tbody>
</table>
Key messages

• 10% of dissections do not have pain
• Some patients present with only mild pain, often mistaken for a symptom of MSK conditions in the thorax, groin, or back
• Consider aortic dissection in all patients presenting with chest pain
• Beware of pain across the scapula
• Always perform BP in bilat arms in ALL patients with CP
Example 3

- 35yo female presents with “dental abscess”
- c/o left wisdom tooth “coming up” 2 months prior to the ED visit
- Was given Levaquin by her dentist with some improvement
- Then pain recurred, and was given Clidamycin by her dentist again.
- Complicated by thrush
- Now, on a course of Flagyl
- Still c/o dental pain and difficulty swallowing
Example 3

• PMHx: Iron deficient anemia
• Medication: Flagyl
• Allergies: Erythromycin
Example 3

- VS: 109/71, 71, 18, 37.6°, 99% on RA
- In pain
- Airway N
- Tongue N
- Floor of mouth N
- Neck: mild tenderness at Left mandibular angle, no mass
Example 3

• Mouth: edema and erythema of left lower gums (proximal to the wisdom tooth)
• Slight trismus
Example 3

• What is your preliminary diagnosis?
• What investigations would you do?
• What would be her disposition?
Example 3

• Consulted dentistry at Mount-Sinai
• Seen that same evening at Mount-Sinai?
• Discharged home?
• 3-4 days later, collapsed at her mother’s home
• Brought back to the ED
Example 3

- Hb: 30
- WBC: 1.4
- Neutro: 0.06
- Platelets: 76
- Film: presence of blasts
Example 3

- Dx: Acute Myeloid Leukemia
Acute Myeloid Leukemia

- AML is a malignant disease of the bone marrow in which hematopoietic precursors are arrested in an early stage of development.
- This developmental arrest results in 2 disease processes:
  - First, the production of normal blood cells markedly decreases:
    - Anemia
    - Thrombocytopenia
    - Neutropenia
  - Second, the rapid proliferation of these cells, along with a reduction in apoptosis, results in their accumulation in the bone marrow, the blood, the spleen and liver.
Acute Myeloid Leukemia

• Most AML subtypes are distinguished from other related blood disorders by the presence of more than 20% blasts in the bone marrow.
Epidemiology

- The prevalence of AML increases with age.
- The median age of onset is 70 years
  - However, AML affects all age groups
- AML is more common in men than in women
### Presentation

<table>
<thead>
<tr>
<th>Symptoms of bone marrow failure</th>
<th>Symptoms of organ infiltration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia – fatigue, decreased energy, dyspnea, dizziness, angina/STEMI</td>
<td>Organ infiltration – spleen, liver, gums (hypertrophy), skin (rash), bone (pain)</td>
</tr>
<tr>
<td>Neutropenia – fever, URTI that does not improve</td>
<td>Swollen gums due to gingivitis (neutropenia)</td>
</tr>
<tr>
<td>Thrombocytopenia – bleeding gums, ecchymosis, lung hemorrhage, GI bleeds, hemorrhagic strokes</td>
<td>Leukostasis – respiratory distress, altered mental status</td>
</tr>
</tbody>
</table>
Treatment

• Current standard chemotherapy regimens cure only a minority of patients with AML
• Patients should avoid exposure to crowds and people with contagious illnesses, especially children with viral infections
• Any patient with neutropenic fever or infection should immediately be treated with broad-spectrum antibiotics.
• Appropriate transfusion support must be provided to patients with AML
  – Transfusion of platelets and clotting factors (FFP, cryoprecipitate) as needed
  – Blood products must be irradiated to prevent transfusion-associated GVHD
Key messages

• High index of suspicion
• Beware of patients with repeated infections that do not improve
• Gum hypertrophy/pain/bleeding is a “classic” sign of AML
• Shit happens!
Example 4

- 39yo male presents with Earache, sore throat, and body aches
- “pus coming out of both ears” x 2 days
- Nasal congestion, rhinorrhea, sore throat, dry cough, nausea for 1 week
- Vomiting
- Lots of coughing
- CP because of cough
- Chills
Example 4

- BP 120/79, Pulse 130, resp 20, Temp 37.3, Sat 98%RA
- Nasal congestion +++
- Cough +++
- Ear (R): reddish TM, bulging TM, watery secretions. No pus
- Ear (L) N
- Throat N
- Heart N
- Lungs clear
- Abdo N
- Skin N
Example 4

- Diagnosis?
- Management?
Example 4

- **CXR**: Heart and mediastinum are normal. Lungs and pleura are clear
Example 4

• Disposition?
Example 4

- 2 days later...
- Presents with flu-like symptoms and fever
- Lethargy and SOB
- Cannot keep food down
- BP 101/61, pulse 143, Resp 20, Temp 37.7, Sat 95%
- Jaundiced
Example 4

• BW
  – HB 149
  – WBC 34.3
  – Na 129
  – Bic 23
  – AST, ALT N
  – Bili 126
  – ALP 110
Example 4

• CXR: the mediastinum is now prominent most consistent with mediastinal lymphadenopathy. Chest CT should be done for further evaluation

• Abdo US: Mildly enlarged liver and spleen without focal lesions. No intra-abdominal lymph nodes. GB and bile ducts are normal
Example 4

- CT chest:
  - Extremely worrisome finding of abnormal infiltrating soft tissue throughout the mediastinum suggestive of acute mediastinitis.
  - Abnormal thickening of the esophageal wall, while no perforation is identified
  - No PE or aortic dissection
Example 4 – Acute mediastinitis

• Mediastinitis is an infection involving the mediastinum
• Surgical emergency
• High mortality rate
  – Overall lifetime mortality rate of 19-47%
Example 4 – Acute mediastinitis

• May begin primarily from structures in the mediastinum, or it may be the result of an infection extending downward from the oropharynx (which case it is called descending necrotizing mediastinitis)

• Typically polymicrobial in nature resulting from a disruption of normal mucosal and tissue barriers
  – Rupture of the esophagus or trachea
  – From surgical intervention
Acute mediastinitis - etiology

- Esophageal rupture
  - the most common cause of mediastinitis currently
- Descending necrotizing infection
  - Head and Primary neck infections
  - Foreign body
  - Pharyngitis
  - Tonsillitis
  - Sinusitis
  - Otitis media
- Dental infections
- Sialadenitis
- Suppurative thyroiditis
- Endotracheal intubation
- Post-thoracic surgery/instrumentation
- TB
- Fungal infections
Presentation

• Common symptoms of patients with mediastinitis include:
  – History of an upper respiratory tract infection or a recent dental infection (common), or thoracic surgery/instrumentation
  – Fever, chills
  – Pleuritic, retrosternal chest pain radiating to the neck or interscapular pain
  – Shortness of breath
  – Confusion
  – Sore throat
  – Swelling in the neck
  – Odynophagia
Presentation

• A complete examination of the head and neck, including the oral cavity, is essential
  – Ill appearance
  – Fever
  – Edema of the neck and face
  – Crepitus of chest or neck
  – Hamman sign (crunching sound upon auscultation of the heart)
  – Stridor
  – Trismus
  – Cranial nerve deficits
Management

• Immediate surgical consultation.
  – Extensive and aggressive debridement of necrotic tissues with exploration of all mediastinal fascial spaces

• Wide antimicrobial coverage is required
Our patient

- No esophageal perforation
- Pleural effusion culture negative
- Fungal culture negative
- HIV Neg
- Mycobacteria acid-fast negative
- EBV PCR Positive initially
- EBV PCR undetectable at follow-up
Infectious Mononucleosis Necrotizing Mediastinitis

• EBV-related acute suppurative mediastinitis
  – Initial development of a pharyngeal abscess
    • Development of septic thrombophlebitis of the jugular veins resulting in septic emboli (LeMierre’s syndrome)
    • Abscess can invade the space between the alar and the prevertebral fasciae of the neck (“the danger space”). Once an infection enters this danger space, it has direct access into the mediastinum

• Only 2 cases of EBV-related descending necrotizing mediastinitis have been reported in the literature

Intensive CareMed 2002; 28: 663-664
Chest 1997; 112: 833-835
Key messages

- Consider mediastinitis in all chest pain, especially after an URI or dental infection
- Beware of CP and odynophagia (and Hamman’s sign)
- Shit happens!
Summary

- Among the many benign conditions we see, we are bound to find/miss the fatal ones
- Maintain a healthy level of paranoia
- Always keep a high index of suspicion
- Be thorough in the history (and pay attention!)
  - Be a good listener!
- Keep your differentials wide
Summary

• Beware of red flags (or weird symptoms or things that do not make sense)
• Beware of repeat visits (or repeated antibiotic use)
Summary

Shit happens!...

(just make sure you leave an open door for the patient to return when you screwed up!)