M2 TEACHING FROM PATHOLOGY TO CLINICAL EXAM USING CVS SYSTEM AS AN EXAMPLE

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WHAT YOU LEARN NOW

Example: Myocardial Infarction

Epidemiology
Normal anatomy & physiology
Pathology – gross and microscopic
Pathophysiology
Clinical presentation
Complications
Pharmacology
IN CLINICAL PRACTICE...

Example: Myocardial Infarction

Presentation: “Doctor, my chest pain”

History

Examination

Investigations

Management

• Acute vs follow-up
• Pharmacological vs non pharmacological

Diagnosis & Differential diagnosis

Confirm diagnosis, rule out Ddx
IN CLINICAL PRACTICE...

What are the causes of chest pain?
What do I need to consider in particular for this patient?
How do I tell the causes of chest pain apart?

• On history
• On examination: what to look out for?
• What investigations to order and how to interpret?
COPING WITH THE CONTENT

“There are too many microorganisms to memorize”

Classify the microorganisms

• By characteristics (gram + vs – vs does not stain)
• By disease caused (causes of pneumonia: community vs hospital-acquired, broncho- vs lobar-, etc)


Focus on things that are common and things that are dangerous
COPING WITH THE CONTENT

“These drugs have so many funny side effects that do not make sense”

**Link** to mechanism, physiology: why does the drug cause this side effect? Work it out step by step. Do not hesitate to revisit M1 content.

**Focus** on things that are common and things that are dangerous

**Explain** it to yourself and others. If you can understand, you do not need to memorize. Study in groups and test each other.

**Think of a patient:** if you give this drug, what do you need to look out for and why?
COPING WITH THE CONTENT

“I study hard but nothing stays”

Revise soon and often.
Find something interesting about what you study.
Active vs passive knowledge – how do you make the knowledge yours?

- Digest what you read not simply sift through
- Summarize ± make notes (but don’t overdo)
- Classification schemes: e.g. “The causes of respiratory tract infection include…”
- Compare and contrast: e.g. B cells vs T cells
- Make links
TODAY’S OBJECTIVE

To better make sense of M2 content, and at the same time prepare for M3.

• To draw the link between clinical presentation and a strong foundations in preclinical science
• To integrate physiology, pathology, pharmacology, and clinical exam.
• To illustrate the advice of ‘understand not memorize’ and ‘think of a patient’

The best way to learn your M2 content well, is to think about preparing for M3 and beyond.
CASE 1

What does this mean to you?
QUESTIONS TO ASK

Atherosclerosis

• So what? What’s going to happen next?
• How does it develop?
• Why does *this patient* have atherosclerosis
• What can we do about it?
• How could it have been prevented?
CONNECTING CONCEPTS

Hypertension → Hyperlipidemia → Atherosclerosis → Plaque rupture → Stroke

Hyperlipidemia → Atherosclerosis → Myocardial infarct → Congestive cardiac failure

Atherosclerosis → Chronic stenosis → Angina → Peripheral vascular disease

Diabetes → Atherosclerosis
CASE 2

Mdm Tan is a 65-year old Chinese lady presenting to the GP with a flu. She also has a history of hypertension, hyperlipidemia, and diabetes; these were first diagnosed 5 years ago on routine screening.

- **Hypertension**: Clinic BP 145/90 (target 130/80). On amlodipine, enalapril
- **Hyperlipidemia**: LDL cholesterol 5.1 mmol/L (target: 4.1 mmol/L). On simvastatin
- **Diabetes**: HbA1c 8% (target 7%). On metformin and glipizide

*What does this mean to you?*
COMPLICATIONS OF DIABETES

Learn a list:

- Cerebrovascular accident
- Diabetic foot ulcers
- Diabetic ketoacidosis or hyperglycemic hyperosmolar state
- Diabetic nephropathy
- Diabetic retinopathy
- Hypoglycemia due to over-treatment
- Ischemic heart disease
- Peripheral vascular disease
- Peripheral “glove and stocking” neuropathy
COMPLICATIONS OF DIABETES

Acute
- Hypoglycemia
- DKA, HHS

Chronic
- Microvascular
  - Nephropathy
  - Neuropathy
  - Retinopathy
- Macrovascular
  - Peripheral Vascular disease
  - Cerebrovascular disease
  - Ischemic Heart disease

Much easier than learning the list!
COMPLICATIONS OF DIABETES

Acute
- Hypoglycemia
- DKA, HHS

Chronic
- Microvascular
  - Nephropathy
  - Neuropathy
  - Retinopathy
  - Kidney Failure
  - Foot ulcers
  - Amputation
  - Blindness

- Macrovascular
  - Peripheral Vascular disease
  - Cerebrovascular disease
  - Ischemic Heart disease

Complications
- Dialysis
- Hyperlipidemia
- Hypertension

Dialysis → Kidney Failure
Kidney Failure → Complications

Peripheral Vascular disease → Blindness
CASE 3

Mr Raju is a 78-year old Indian gentleman with a prosthetic aortic valve, fitted 7 years ago for aortic stenosis. What does this mean to you?

What if:

• He now presents with bruising over both legs
• He now presents with a 2-week history of fever. There are no localizing symptoms e.g. cough, dysuria, diarrhoea, etc.
• He now presents with pitting edema of bilateral lower limbs
• He is well but has a BP of 160/70.
CASE 3

Mr Raju is a 78-year old Indian gentleman with a prosthetic aortic valve, fitted 7 years ago for aortic stenosis.

What questions do you need to ask?

• Why:
  - Why did he have AS
  - Why did he need a new valve?

• So what?
  - What does this mean for him?
  - What must be done for him now?
  - What must we be careful / look out for?
CONNECTING CONCEPTS

For every disease, ask:

- **Who gets this disease?** *(Epidemiology, risk factors)*
- **What can cause it? What caused it in this patient?** *(Etiology)*
- **How does the cause lead to this disease?** *(Pathogenesis)*
- **How does a patient present?** *(Presentation)*
- **How do we tell how bad is the disease?** *(Severity)*
- **What are the complications?**
- **How do we treat / prevent these complications?** *(Management)*
  (At the moment you only learn part of the pharmacology)

Asking these questions NOW helps you to both better learn M2 content, and prepare for M3 and beyond.
CASE 3

Mr Raju is a 78-year old Indian gentleman with a prosthetic aortic valve, fitted 7 years ago for aortic stenosis.

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• He is well but has a BP of 160/70.
Cardinal presentations in CVS:

• Chest pain
• Breathlessness
• Palpitations
• Edema
• Syncope
• Congenital: cyanosis, breathlessness, failure to thrive
• Asymptomatic with murmur or arrhythmia
• Cardiovascular risk factors: asymptomatic vs symptomatic
• Vascular disease in other organs: stroke, limb ischemia etc

However: They all also have non-cardiac causes!
A 70-year old Indian lady presents to A&E with sudden chest pain. She says that she has had this before “a few times” but this is the worst episode. She is in distress and is drenched in sweat.

• How can we help her?
• What is the diagnosis?
• What questions do we need to ask?
CHEST PAIN

What is pain?

• Somatic pain
• Visceral pain. Specific patterns:
  - Myocardial ischemia
  - Referred pain
  - Abdominal colic
• Neuropathic pain

Make sense of the ‘story’ of ‘chest pain’ by understanding its mechanism. The same principles apply to all other “pain”
CHEST PAIN

History:
• **Site:** Retrosternal
• **Onset:** Acute onset. ? Prior episodes
• **Character:** Vague, crushing
• **Radiation:** To left shoulder, arm, jaw
• **Associated symptoms:** diaphoresis, dyspnoea, orthopnoea
• **Time course:** Relieved <30min vs lasts >30 min
• **Exacerbating and relieving factors:** relieved by GTN
• **Severity:** mild vs 10/10
### CHEST PAIN

<table>
<thead>
<tr>
<th></th>
<th>Ischemic</th>
<th>Dissection</th>
<th>Respiratory</th>
<th>GERD</th>
<th>MSK</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Character</strong></td>
<td>Crushing</td>
<td>Tearing</td>
<td>Pleuritic</td>
<td></td>
<td>Localized</td>
</tr>
<tr>
<td><strong>Radiation</strong></td>
<td>Left arm, jaw</td>
<td>Interscapular</td>
<td>Lateral, back</td>
<td>Epigastrium</td>
<td>-</td>
</tr>
<tr>
<td><strong>Assoc symp</strong></td>
<td>Diaphoresis</td>
<td></td>
<td>e.g. cough, LOW, trauma</td>
<td>Sour taste, regurgitation</td>
<td>-</td>
</tr>
<tr>
<td><strong>Dyspnoca</strong></td>
<td>Orthornoca</td>
<td>±</td>
<td>+</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td><strong>Exacerbating</strong></td>
<td>Exertion (consistent)</td>
<td>Inspiration</td>
<td>Meals, lying down</td>
<td>Motion, coughing</td>
<td>-</td>
</tr>
<tr>
<td><strong>Relieving</strong></td>
<td>Nitrates</td>
<td>Time (pain max at onset)</td>
<td>Antacids</td>
<td></td>
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</tbody>
</table>

*Also consider:* epidemiology (age), risk factors (e.g. vascular risk factors), acute vs subacute vs chronic onset of symptoms.
DIFFERENTIATING CONCEPTS

Not all ischemic chest pain is the same.

Compare and contrast:
Acute myocardial infarction vs Stable Angina

• Single disease process of ischemic heart disease: Same pathogenesis and risk factors. AMI – superimposed acute precipitant, e.g. plaque rupture.

• Heart muscle dies when there is ‘infarct’. Hence diagnostically, cardiac enzymes (troponin, CK-MB) will be positive

• Clinical presentation: Chest pain or breathlessness, short-lived in angina but persistent and more severe in AMI.

• Management differs: In AMI emergency reperfusion is key, in angina treatment may be medical or revascularization. Risk factor control is important in both
PHYSICAL EXAM - CARDIAC

The physical exam sessions may not seem so important at this point, but wait till you are thrown into the wards!

Better to pick up what you can along the way

• **It’s not simply going through a routine.** At higher levels you may vary the steps depending on what you have picked up / suspect
• **Examine with a purpose; know what you are looking for**
• **You can only find what you look for, you can only look for what you know**
• **Know the steps and be familiar – so you can free up your mind to interpret the signs**
1. Wash your hands
2. Introduce yourself to the patient and explain what you are about to do
3. Expose the patient and position him/her at 45 degree
4. Inspect the chest from the end of the bed, asking the patient to take in a deep breath, look out for scars (CABG, Mitral valvotomy)
5. Look at the hands for pallor, cyanosis, clubbing
6. Measure pulse rate over 30 seconds.
   - Check for collapsing pulse.
   - Check for radial-radial delay.
   - Check for radio-femoral delay
7. Look at face, conjunctiva, sclera, mouth and tongue – for jaundice, pallor, cyanosis
8. Look at the neck for Jugular Venous Pulsation
9. Palpate Carotid pulses, one at a time
10. Feel for apex beat
11. Feel for palpable thrills, parasternal heave, palpable P2
12. Auscultate the heart in sequence – Mitral, Tricuspid, Pulmonary, Aortic. Time the heart sounds by palpating carotid pulse simultaneously.

13. Turn the patient to the left side and reconfirm the position of the apex beat. Listen with the bell at the apex for MS murmur. (Can be done before or after the rest of auscultation)

14. Sit the patient up and leaning forward and listen to the left lower sternal edge in full expiration for AR murmur.

15. Auscultate carotid arteries for carotid bruits

16. Auscultate the chest posteriorly for crackles

17. Palpate lower limbs for pitting oedema

18. Thank the patient and cover up

19. Request to palpate the remaining peripheral pulses (Brachial, Femoral, Popliteal, Dorsalis Pedis and Posterior Tibial)

20. Request to check the blood pressure, look at temperature chart, perform dipstick urinalysis (for haematuria).
MAKE SENSE OF THE STEPS

Introduction
- Wash hands
- Introduction
- Position
- Exposure

Body
- Inspection
  - Interventions
  - Patient condition
  - Clues

Precordium
- Hands – both sides
- Pulse: HR, delays, collapse
- Eyes
- Mouth
- Neck – JVP, Carotids

Peripheries
- Apex beat, parasternal heave
- Ascultate all areas
- Dynamic maneuvers

End
- Carotids
- Chest
- Liver
- Lower limbs

Requests
CASE 6

Your end point:

Sir, this is an elderly Indian gentleman who is alert at rest and on oxygen via nasal prongs. He has a midline sternotomy scar and a right saphenous vein graft scar suggesting past coronary artery bypass.

On peripheral examination, there is no clubbing and no stigmata of infective endocarditis. He is tachycardic at HR 110 and tachypnoic at RR 30. With an irregularly irregular pulse he is in atrial fibrillation. There is no scleral icterus, conjunctival pallor, or central cyanosis.

He has an elevated jugular venous pressure and bilateral pedal edema. The apex beat is displaced in the 6th intercostal space in the anterior axillary line. First and second heart sounds are heard with no murmur or additional heart sounds. Basal lung crepitations are heard.

In summary, this is an elderly Indian gentleman with biventricular heart failure. The most likely etiology is ischemic heart disease.
HEART FAILURE

Physiology:

\[ BP = CO \times TPR \]

\[ CO = HR \times SV \]

Preload \quad Contractility \quad Afterload

The body defends cardiac output
HEART FAILURE

Its clinical presentation directly follows from its pathophysiology: Consider Left Heart Failure (simplified schema)

Old infarct

↓ Contractility

Valve regurgitation

↑ Preload

Aortic stenosis

↑ Afterload

Hypertension

Myocardial remodelling

↓ Cardiac Output

RAAS activation, fluid retention

Backward failure – congestion in Lungs, Liver, Peripheries

Right heart failure

Forward failure – tissue hypoperfusion
HEART FAILURE

Clinical signs:
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HEART FAILURE

Possible etiology:

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HEART FAILURE

Management principles: inhibit the vicious cycle that causes heart failure to progressively worsen.

Old infarct  ↓  Contractility

Valve regurgitation  ↑  Preload

Aortic stenosis  ↑  Afterload

Hypertension  →  Myocardial remodelling

RAAS activation, fluid retention

Forward failure – tissue hypoperfusion  ↓  Cardiac Output

Backward failure – congestion in Lungs, Liver, Peripheries  →  Right heart failure
SUMMARY OF TOOLS

This session is about the tools, not about the content

• Ask the right questions: especially ‘why’ and ‘so what’
• Draw the links from pathophysiology to clinical presentation
• Understand stories by making sense of things.
• Think of a patient
• Group your concepts
• Clarify by distinguishing between similar concepts.