What are the special clues in symptoms & lab findings that will help you determine which disease you're dealing with?

1. **Rheumatic Heart Disease:**
    - Group A Beta-Hemolytic Streptococcal infection.
    - If no infection was mentioned yet a lab test was done that shows an elevated antistreptolysin O (ASO) That is your clue of a previous Streptococcal infection hence → RHD.

    What if you had both acute and chronic RHD?
    - You have to know that Aschoff bodies are only in ACUTE RHD.
    - In CHRONIC RHD → Fibrosis

    But what if RHD wasn't even a choice, Even though all first three lines are basically screaming at your face that it's RHD, what to do then?

    → If it tells you there are Aschoff bodies. What should you diagnose this with? **Myocarditis.**

    → If it tells you it's in the posterior wall of left atrium. What is it? **Endocarditis**

    → If it tells you McCallum Plaque. → **Endocarditis**

    → If it tells you there are Fibrinous secretions in pericardium. What ever could that be? Pericarditis. But if it tells you fibrinous deposition → **Endocarditis**

    → If it tells you it's on a point where valve usually closes (but giving it a fancy name to through you off of course) then it's **Endocarditis.**

    ** If there is a past history of RHD, chances are it's Chronic not acute.

2. **Congestive heart failure:**
    - If the patient was said to have repeated attacks of RHD.
    - Or one severe attack.
    - Or past history of Chronic RHD.
3. **Acute endocarditis:**
    - STAPH. AUREUS
    - Normal or diseased valves .
        → If he was a drug abuser → Tricuspid valve is mostly affected.
        → Even if they didn't mention his heart was normal, given that he's a drug abuser is enough to know it's acute,
4. **Sub-acute endocarditis:**
    - STREPT. VIRIDENCE
    - Diseased valves.
    - An abnormal heart in general.

5. **Marantic endocarditis:**
    - No infectious agent.
    - Sterile.

    What's its therapy? Anticoagulant.
6. **LSE (Libman-Sack endocarditis):**
    - Dystrophic calcification.
    - SLE (systemic lupus erythematous)
7. **Atherosclerosis:**
    - Morphology:
        - → Intimal thickening and lipid accumulation.
        - → Fatty streaks.
    - LDL or VLDL in the blood.
8. **Ischemic heart disease:**

    If it was one of the choices then they probably will tell you there's a past history of atherosclerosis.

    Moreover, the most common one is coronary Artery atherosclerosis.
9. **Angina pectoris:**
    1. **Stable angina or typical angina pectoris:**
        - 70% to 75% narrowing of **lumen.**
        - The chest pain is **episodic and associated with exertion.**
        - Is usually relieved by rest (thereby decreasing demand) or with a **strong vasodilator like nitroglycerin**.
    2. **Unstable or crescendo angina:**
        - 90% narrowing (fixed) of **lumen**
        - Pain occurs with **progressively increasing frequency**, and is precipitated with progressively less exertion, even at rest.
        - It is induced by disruption or rupture of an **atheroma plaque** with **superimposed partial thrombosis**.
        - **Unstable angina** is often the precursor of subsequent acute MI.
    3. **Prinzmetal or variant angina:**
        - An uncommon pattern of episodic angina that occurs **at rest** **due to coronary artery spasm.**
        - Prinzmetal angina generally responds promptly to vasodilators, such as **nitroglycerin** and **calcium channel blockers**.
        - It is **not** related to **atherosclerotic disease.**

10. Myocardia infarction:

- Troponin (7-10 days in blood) (peaks at 48)
- CK-MB (72 hrs. in blood) (peaks at 24 -48)
- If the patient died suddenly even after being rescued from MI , what's probably the cause?
- What if no labtests were mentioned, how to diagnose it?

  Severe crushing sub-sternal chest pain, which may radiate to the neck, jaw, epigastrium, shoulder or left arm.

  But note that if he was diabetic, old of hypertensive **no pain is present.**

  So what if he was diabetic, how can we now for sure if it's MI?

  If he has difficulty in breathing, weak rapid pulse, sweating and ischemia.

- **Later complicated with Arrhythmia.**

11. Hypertension:

- Most of the time it's idiopathic, so you'll need to check his history, is he suffering from hypertension risk factors? Like:
  - → **Hereditary,** Genetics- family history
  - → **Race.** African-Americans
  - → **Gender.** Men & postmenopausal women
  - → **Age**
  - → **Obesity**
  - → **Diet, particularly sodium intake**
  - → **Lifestyle-stressful**
  - → **Heavy alcohol consumption**
  - → **Diabetes**
  - → **Use of oral contraceptives**
  - → **Sedentary or inactive lifestyle**
  - → **Smoking**
- But what if he didn't have any of the mentioned risk factors, yet he was presented with a disease, how could I know?
  - Is it a renal, endocrine, vascular or neurogenic disease? Chances are its hypertension.
  - What if she was a woman with no underlying disease? Is she pregnant? Yes. Then it's hypertension.
- When there's a defect in sodium excretion you know its hypertension.
- If it lead to blindness, left ventricular hypertrophy or infarction its HTN.

12. Hyaline arteriosclerosis:

- Small blood vessels
- Benign hypertension
- Can be seen in elderly with HTN
- Leads to benign nephrosclerosis

13. Hyperplastic arteriosclerosis:

- Small blood vessels
- ONIONSKIN APPEARANCE

14. Left ventricular cardiac hypertrophy:

- Longstanding poorly treated HTN leads to left sided hypertensive heart disease.
- HTN induces left ventricular pressure overload, which leads to:
  - Hypertrophy of the left ventricle
  - Increase in the weight of the heart.
- The free LV wall is > 2cm and the weight of the heart is > 500 grams

15. Arterial Thrombi:

Most common is coronary artery, then cerebral then femoral.

- ❖ How to know its arterial thrombi though?
  - Begins at the sight of endothelial injury or turbulence.
  - It is usually superimposed on an atherosclerotic plaque and are firmly adherent to the injured arterial wall (mural).
  - Gray-white and friable.

16. Venous Thrombi:

- Usually begins in stasis.
- Contains more enmeshed erythrocytes.
- Most commonly affects the veins of the lower extremities (90% of cases).

17. Deep vein Thrombosis:

- Venous thrombosis.
- If it was caused by immobilization, CHF, pregnancy, age.
- If It gave rise to pulmonary infarction
- Causes edema and local pain but asymptomatic in 50%.

18. Pulmonary thromboembolism:

- 95% originates from DVT.
- Saddle embolus.
- Hypoxia, hypotension and right-sided heart failure (cor pulmonale)

19. Fat thromboembolism:

- After fracture of long bones.

20. Fat embolism syndrome:

- Pulmonary insufficiency.
- Neurologic symptoms
- Anemia
- Thrombocytopenia.

21. Air embolism:

- Obstetric procedure
- Chest wall injury
- An excess of 100 cc.

22. Decompression sickness:

- Gas embolism
- Sudden changes in the atmosphere
- Scuba divers or unpressurized aircraft
- Grecian bend.

23. Caisson disease:

- More chronic form of decompression sickness.
- Skeletal system
- Foci of ischemic necrosis
- Heads of femur, tibia and humeri.

24. Amniotic fluid embolism:

- Labor and immediate postpartum period.
- Mostly in the lungs
- Sudden severe dyspnea, cyanosis and hypotensive shock.
- Squamous cells and fetal skin or hair can be seen under the microscope.

25. Giant cell arteritis:

- Most common type
- Large to small arteries
- Women more than men, patients over 50
- Thickened painful temporal artery, jaw pain.
- Vision loss can also be a sign (ophthalmic artery)
- Treated by? Corticosteroids or anti-TNF therapy
- In morphology: Giant cells

26. Polyarteritis nodosa:

- Medium to small vessels
- Specially kidney and skin but never in lungs.
- Associated with hepatitis b & c
- Abdominal pain is the most special clinical feature
- Treated by? Corticosteroids or anti-TNF therapy
- Morphology: Segmental inflammation.

27. Wegener granulomatosis:

- Necrotizing granuloma of Upper and lower respiratory tracts
- Small to medium sized vessels
- Renal disease = glomerulonephritis.
- Positive C-ANCA (PR3-ANCA)
- Males more than females, age 40
- Clinical features are RT related + renal disease
- Treatment? Steroids, TNF-inhibitors, anti-B cell antibody (Rituximab) and cyclophosphamide
- Morphology? URT and renal lesions.

28. Buerger disease:

- Smokers
- Legs and hands
- Pain even at rest
- Treatment is to stop smoking (prevents further attacks)

29. HSP:

- Disease of small vessels
- IgA mediated – high levels of it.
- Causes skin purpura, abdominal pain, GI bleeding, orchitis and nephritis.
- Childhood
- IgA and C3 are deposited.

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Good Luck.