Case 204: Shortness of Breath

- **Chief complaint**
  - 35-year-old male presents by private vehicle with shortness of breath

- **Vital signs**
  - **HR: 146** BP: 105/54 **RR: 28** **Sat: 93% on RA** **T: 38.5°C** Wt: 80 kg

- **Patient Appearance**
  - Patient appears in mild respiratory distress

- **Primary survey**
  - Airway: speaking in full sentences
  - Breathing: mild respiratory distress, tachypneic, minimal accessory muscle use, bibasilar rales
  - Circulation: warm skin, 2+ distal pulses, trace bi-pedal edema

- **Action**
  - Place patient on the monitor
  - Oxygen by NC or NRB
  - Advanced airway equipment and crash cart to the bedside
  - Two large bore peripheral IV lines (draw rainbow top)
  - Order stat ECG
  - Order stat portable CXR (delayed interpretation)
  - POCUS: Cardiac and Lung
    - **Figure 204.3 (A) –** Apical 4 Chamber view, no pericardial effusion, normal relative chamber size (no R heart strain)
    - **Figure 204.3 (B) –** Parasternal Long view, E-Point Septal Separation (EPSS): Distance > 1 cm = Poor LV Function
    - Thoracic views with normal lung sliding bilaterally, +B lines (verbal report)

- **History**
  - Source: Patient
  - HPI: This is a 35-year-old male who presents with worsening shortness of breath over the last 1.5 weeks. His dyspnea is worse on exertion and with lying flat. Today he felt intermittent substernal chest discomfort that was not clearly exertional. He has had frequent palpitations. Two weeks ago he had flu-like symptoms that have resolved with the exception of his fatigue and fever. ROS is otherwise negative.
  - PMHx: negative

- PSHx: none
- Allergies: none
- Meds: none
- Social: denies alcohol, smoking, or drugs
- PCP: none

- **Physical Exam**
  - General: alert and oriented, mild distress
  - HEENT: Normal
  - **Neck:** +jugular venous distension
  - **Lungs:** tachypneic, bibasilar rales
  - **Heart:** tachycardic, no murmurs
  - Abdomen: normal
  - Neuro: normal
  - **Extremities:** bilateral trace pedal edema
  - Skin: normal
  - Back: normal

- **Instructor Prompt:** learners should discuss differential diagnosis

- **Action**
  - Order Labs
    - CBC, BMP, troponin, BNP, LFT, magnesium, PT/INR, PTT, lactate, urinalysis, blood cultures
    - Consider D-dimer
  - Order Imaging
    - CXR
  - Order Meds
    - Furosemide 20 mg IV
    - Acetaminophen 1000 mg PO
    - Learner may consider empiric antibiotics with differential including sepsis

- **Response/Results**
  - Patient reevaluation and repeat vitals:
    - Vitals after tylenol, IV furosemide and O_2:
      HR: 110 BP: 104/53 RR: 18 Sat: 98% T: 37.8°C
    - If Tylenol not given: **HR: 125 T: 38.5°C**
    - If furosemide not given: **HR: 125 RR: 25**
    - If O2 not given: **RR: 25 Sat: 91%**
    - Prompt: given tylenol, O_2 and furosemide as needed
  - **Case 204 Lab Results** (significant for WBC 14.2)
  - Additional Lab Results: **Troponin 0.55 ng/mL** (HS 550 ng/L)**, BNP 350, Lactate 2.3**, D-dimer 450 ng/mL if ordered

    - EKG (**Figure 204.1** - sinus tachycardia)
    - CXR (**Figure 204.2** - pulmonary venous congestion, Kerley B lines)

- **Instructor Prompt:** discuss diagnostic clues for myocarditis

- **Action**
    - Consult cardiology
        - Express concern for new-onset heart failure and need for urgent formal transthoracic echocardiogram
    - Update patient and/or family on presumed diagnosis and plan
    - Admit patient to telemetry unit

- **Diagnosis**
    - Primary Diagnosis: Myocarditis
    - Secondary Diagnoses: Acute Decompensated Heart Failure

- **Critical actions**
    - Obtain IV access
    - Obtain stat ECG
    - Begin antipyretic
    - Begin treatment for new-onset heart failure with diuretics
    - Admit to telemetry unit

- **Instructor Guide**
    - This is a case of myocarditis in a patient who was previously healthy. The patient presents with a history of an influenza-like syndrome, ongoing fever, tachycardia out of proportion to fever and hypoxia. Critical historical clues include dyspnea on exertion, orthopnea, and vague chest discomfort. Important early actions include obtaining IV access and an ECG. Patients with myocarditis with evidence of volume overload should be treated with IV loop diuretics. Fever should be treated with antipyretics. The patient must be dispositioned to a telemetry unit.

- **Case Teaching Points**
    - The differential for adult myocarditis should include pericarditis, acute coronary syndrome, acute exacerbation of congestive heart failure, congenital heart disease, and pulmonary disease. Pulmonary embolism may be considered but can be ruled out in this case with reassuring POCUS and negative D-dimer. This patient's young age and absence of risk factors decreases the likelihood of ischemic heart disease or congestive heart failure. Pericarditis may occur concurrently with myocarditis, and bedside ultrasound may reveal a thickened pericardium or pericardial effusion in addition to the myocardial hypokinesis often seen with myocarditis. Workup should include early ECG, electrolyte and troponin testing, chest x-ray, and formal echocardiogram. Most patients with

myocarditis will require admission with telemetry monitoring, often to an ICU level of care.

- **What is the incidence and prevalence of myocarditis?**
    - Overall incidence of myocarditis is unknown and potentially underdiagnosed.
    - The leading cause of myocarditis worldwide is Chagas disease (*Trypanosoma cruzi*), while viruses are the leading cause in the United States.
    - Mortality at 1 year is 20%, at 5 years is 50%.

- **What is the pathogenesis of myocarditis?**
    - The pathogenesis of myocarditis occurs from direct invasion of pathogens into myocytes with subsequent toxicity from a pathologic host response to the invasion.
    - Three stages of myocarditis: acute (viral cytotoxicity and focal necrosis), subacute (subsequent autoimmune injury to myocytes), and chronic (ongoing myocardial fibrosis and cardiac dysfunction).
    - Coxsackievirus, Epstein-Barr virus, cytomegalovirus, HHV-5, and parvovirus are just a few of the known viral causes of viral and post-viral myocarditis. The list of viruses that can potentially cause myocarditis, however, is extensive.
    - *Borrelia burgdorferi* (Lyme disease) and *Trypanosoma cruzi* (Chagas disease) are known bacterial and protozoan causes.
    - Cardiac sarcoidosis and giant cell myocarditis are rare but important forms of myocarditis given their poor prognosis and lack of response to usual care.

- **How does myocarditis typically present?**
    - Paradoxically, patients with abrupt onset and fulminant myocarditis (with hemodynamic compromise) have the best prognosis, while patients with more insidious forms of myocarditis fare the worst and may even require heart transplantation.
    - Historical clues to the diagnosis include recent or ongoing malaise, fever, fatigue, myalgias, vomiting, and/or diarrhea. Classic myocarditis symptoms are typical of other viral syndromes: fever, myalgias, malaise, and GI and respiratory symptoms. Nearly 3 in 4 patients report breathlessness and 1 in 3 report chest pain.
    - Examination may reveal tachycardia out of proportion to fever, signs of heart failure, and a pericardial friction rub.
    - The most common adult presentations include dyspnea, chest pain, and dysrhythmias; the most common pediatric presentation is dyspnea.

- **What are some clues that could lead to the diagnosis of myocarditis?**
    - Emergency diagnosis is complex given that the gold standard for diagnosis is endocardial biopsy, meaning that the diagnosis is most commonly based on symptoms with supportive testing.
    - ECG changes can include sinus tachycardia, widened QRS, low electrical activity, prolonged QT interval, AV blocks, changes typical for ACS, or changes typical of

pericarditis. The ECG may be normal as well, although it remains unclear how often this might be the case.
    - Given the overlap of clinical symptoms and ECG changes in myocarditis, maintain a low threshold to treat as ACS (including cath lab activation for STE) in patients with diagnostic uncertainty.
  - Troponin may be elevated in acute myocarditis but is poorly sensitive (34%) and moderately specific (89%).
  - Echocardiography is critical in diagnosing acute myocarditis and fulminant myocarditis in particular; it may reveal a non-dilated and hypo-contractile left ventricle in fulminant myocarditis. Presence of pericardial effusion and pericardial tamponade should also be identified in patients with suspected pericarditis or myocarditis.
  - Cardiac MRI is useful to help confirm diagnosis but is unlikely to be obtained in the ED setting. Historically, endocardial biopsy has been the gold standard mechanism for diagnosis confirmation despite poor sensitivity and risk.

- **What are the critical aspects of ED treatment of myocarditis?**
  - The goal of treatment is to preserve left ventricular function and is directed at the stage of disease.
  - Patients should be treated similarly to other causes of acute decompensated heart failure:
    - If BP is normal or elevated, use nitrates for afterload reduction (sublingual or gtt)
    - It the patient is volume overloaded, use diuretics (start with Furosemide 20-40 mg IV if naive)
    - Use supplemental oxygen and NIPPV as needed for respiratory support
  - Patients who present in cardiogenic shock may require gentle IV fluids, inotropes and vasopressors.
  - Mechanical support should be considered for refractory cardiogenic shock including ventricular assist devices and extracorporeal membrane oxygenation (ECMO).
  - ED providers must anticipate that patients with acute myocarditis may have dysrhythmias and should provide usual care (e.g., amiodarone for ventricular tachycardia).
  - Severe presentations require cardiology and/or cardiac surgery consultation.
  - Routine use of IV immunoglobulin is not recommended in adults but may have a role in pediatric myocarditis.
  - Antivirals and immunomodulators also have no role in the emergent setting.
  - Treatment of pericarditis (related):
    - Ibuprofen (600-800 mg q8 hours) or ketorolac
    - Colchicine (0.5-1.2 mg daily or divided BID) -> if given for 3 mo can reduce recurrence of non-bacterial causes

- **What are the indications for mechanical circulatory support (e.g. ECMO or Impella)?**
  - Low cardiac output despite conventional medical treatment (vasopressors/inotropes) and intra-aortic balloon pump

- Refractory systemic hypotension, cardiac index < 2.0 L/min/m^2, persistent hyperlactatemia, worsening end-organ failure

- **POCUS Pearls**
  - The echocardiographic findings seen in myocarditis are non-specific; bedside echo may demonstrate impaired systolic and diastolic function and evidence of new valvular regurgitation.
  - A key use of echo in the patient with suspected myocarditis is to exclude other causes of chest pain and shortness of breath, such as pericardial effusion or thrombus.
  - The main method of measuring LV systolic function is the ejection fraction (EF); a reduced EF supports the clinical diagnosis of myocarditis.
  - EF can be measured in a variety of ways, some more labor-intensive than others.
  - Normal EF = 50-85%
    - E-Point septal separation (EPSS) is the closest vertical distance of the anterior mitral leaflet tip to the septal wall during diastole using a parasternal long axis view. > 1 cm distance = poor LV function.
    - Fractional shortening (FS) uses an M-mode tracing through mid-LV in a parasternal short or long axis view to measure the percentage of the LV diameter change between diastole to systole. FS = (LVEDD- LVESD)/LVEDD x 100% where LVEDD = LV End-diastolic diameter and LVESD = LV End-systolic diameter. Normal range is 30-45%.
    - Visual gestalt, even with limited training, is reasonably accurate in categorizing EF into normal, mild-moderately decreased, and severely impaired LV function categories. Websites such as http://www.cardiacejectionfraction.com provide a small amount of free training online.
    - Simpson's method requires tracing the LV endocardial surface in an apical 2 or 4 chamber view to calculate LV volume in systole and diastole, and then calculating the EF using the formula EF = (End-diastolic volume – end-systolic volume)/end-diastolic volume. The other methods above are less time-consuming.

- **References**
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  - Editor(s): Dr. Andrew Ketterer
  - Editor-in-Chief: Dr. Kristen Grabow Moore
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    - ECG from “Life in the Fast Lane”: https://litfl.com/sinus-tachycardia-ecg-library/
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    - POCUS images courtesy of: Emory Emergency Medicine POCUS Archive

Case 204 Lab Results

Basic Metabolic Panel:

Na	135 mEq/L
K	4.5 mEq/L
Cl	98 mEq/L
CO_2	24 mEq/L
BUN	15 mg/dL
Cr	0.9 mg/dL
Gluc	86 mg/dL

Liver Function Panel:

AST	32 U/L
ALT	14 U/L
Alk Phos	90 U/L
T bili	1.1 mg/dL
D bili	0.3 mg/dL
Lipase	40 U/L
Albumin	4.5 g/dL

Complete Blood Count:

WBC	14.2 x 10^3/uL
Hb	14.1 g/dL
Hct	42.5%
Plt	150 x 10^3/uL

Coagulation Panel:

PT	13.1 sec
INR	1.0
PTT	26 sec

Urinalysis:

SG	1.018
pH	6.8
Prot	Neg
Gluc	Neg
Ketones	Neg
Bili	Neg
Blood	Neg
LE	Neg
Nitrite	Neg
Color	Yellow

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Figure 204.1- ECG

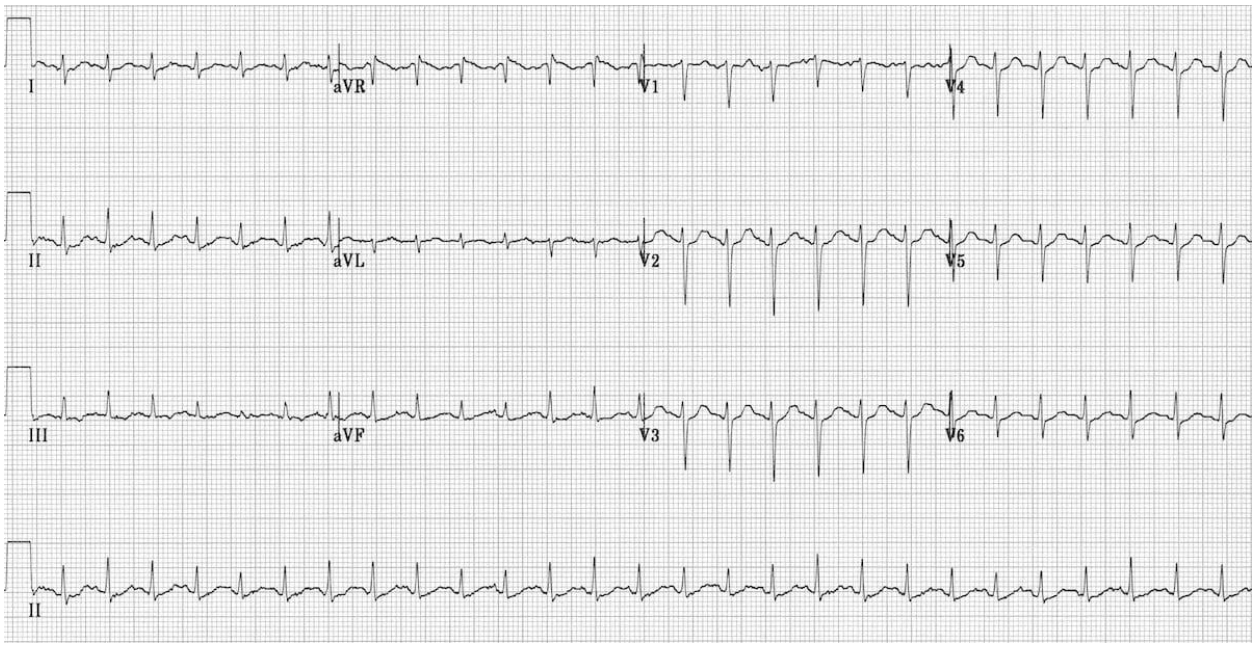

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Figure 204.2 – Portable Chest X-Ray

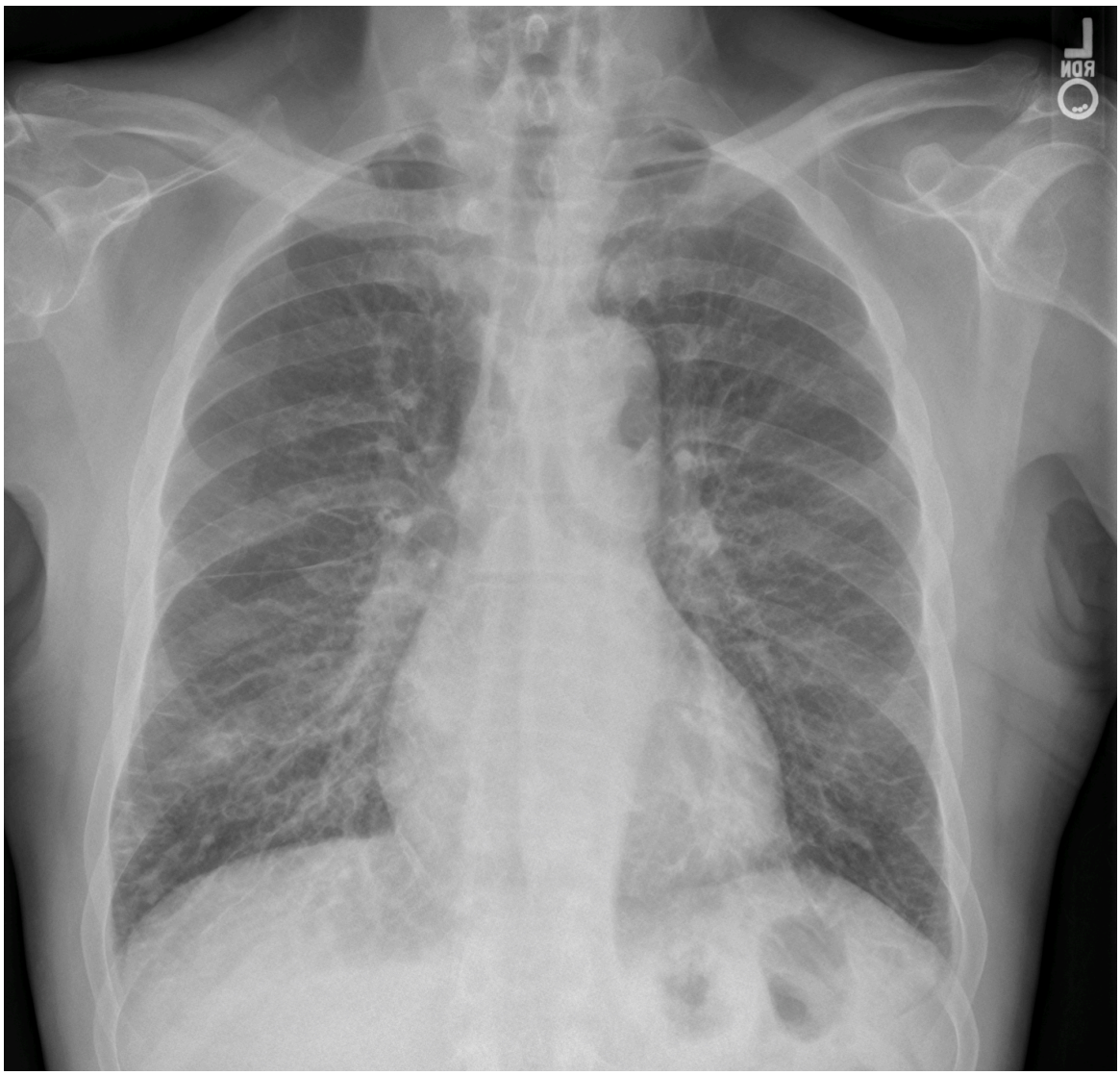

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Figure 204.3 (A&B) – POCUS Echo

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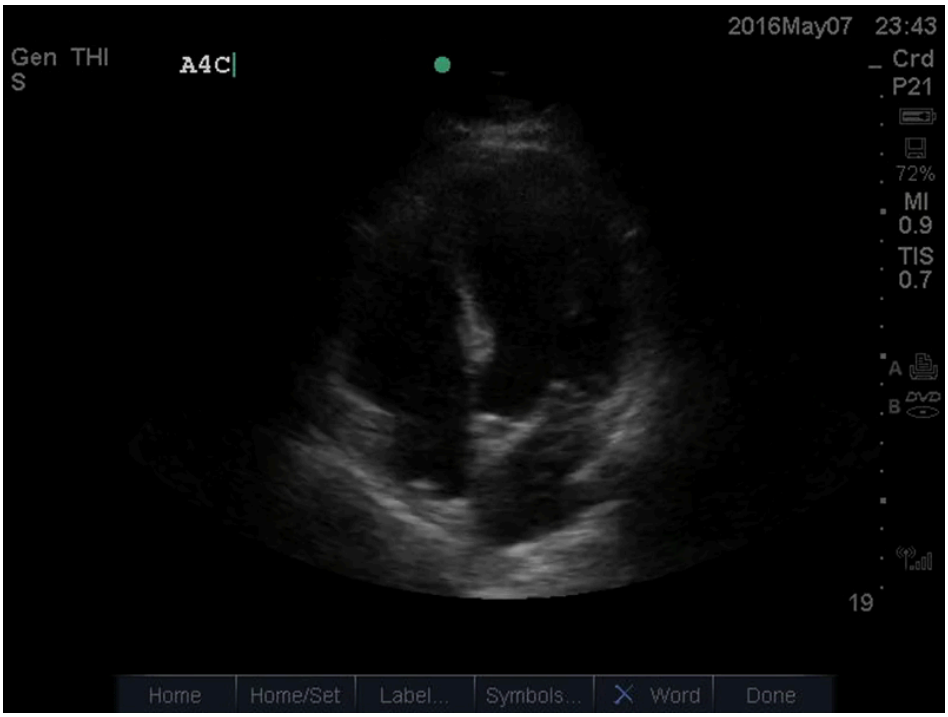
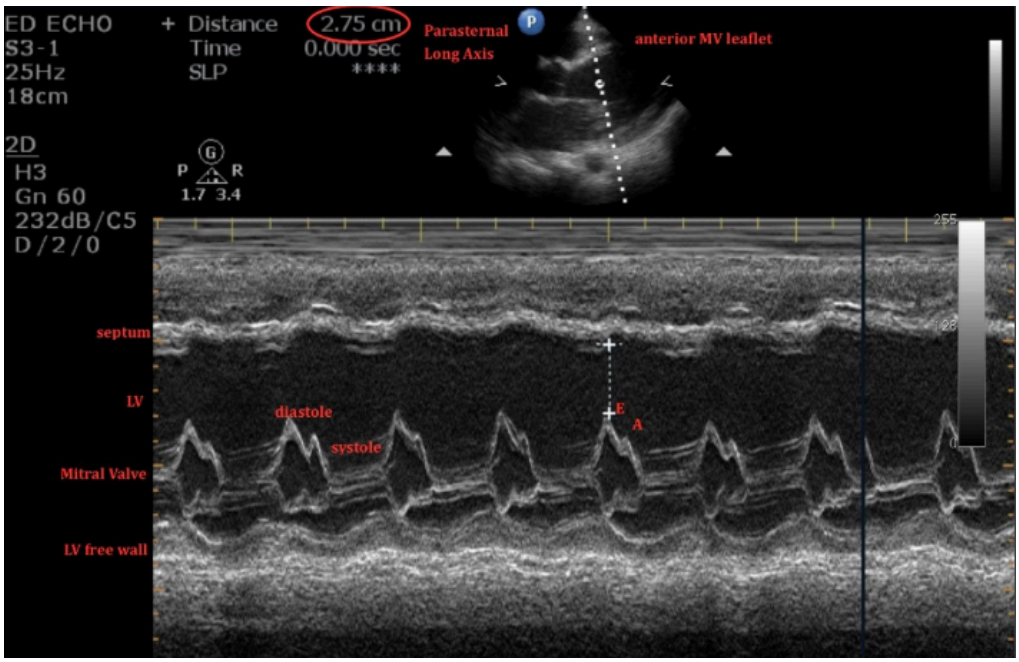
A - Apical 4 chamber view



B - Parasternal Long



EPSS (E-Point Septal Separation): Distance 2.5cm