


**INFLAMMATION,
INFILTRATION &
INVESTIGATION**



OREGON CARDIOVASCULAR SYMPOSIUM
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50 F WITH ALTERED SENSORIUM

Presents to the emergency department.

- Lethargic, mental slowness, fatigue
- Duration: 5 days, waxing and waning

Reports 3 months of cough, rhinorrhea, dyspnea, intermittent wheezing and chest tightness

ROS (+) Unsteady gait, diplopia, intermittent headache

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PRECEDING 3 MONTHS

- Multiple presentations to urgent care and PCP office
- Treated for recurrent bronchitis/sinusitis with multiple courses of antibiotics
- Multiple courses of prednisone with only transient improvement in symptoms
- Trial of ipratropium-albuterol inhaler with minimal relief

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PAST MEDICAL HISTORY

- 1. Hypothyroidism
 - Hashimoto's thyroiditis s/p thyroidectomy
- 2. COPD
 - Gold 1
- 3. PTSD
- 4. Anxiety
- 5. Migraine headaches
 - No history of complex migraines

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ADDITIONAL MEDICAL HISTORY

SOCIAL HISTORY

- Former Tobacco use: 1 ppd x 30 years; quit 3 months ago
- Alcohol use: none
- Drug use: none

MEDICATIONS

- Albuterol inhaler q6h PRN
- Levothyroxine 112 mcg
- Sumatriptan 100 mg PRN
- Prednisone 10 mg daily (most recently)

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ADDITIONAL MEDICAL HISTORY

SURGICAL HISTORY

- Thyroidectomy due to Hashimoto's thyroiditis (1997)
- Hernia repair in childhood

FAMILY HISTORY

- No family history of premature coronary artery disease

ALLERGIES

- Codeine: causes nausea

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OBJECTIVE:

Vitals:

BP 125/75 | Pulse **111** | T 36.7 °C (98.1 °F) | RR 20 | SpO2 99% R/A
 Height 5' 7" | Weight 139 lb | BMI 21.8 kg/m²

Physical Exam:

HEENT/Neck: PERRL, EOMI. No scleral icterus. Oral mucosa moist. JVP ~7cm of H2O.

Cardiovascular: Tachycardic. Regular rhythm. No murmur. 2+ radial and dorsalis pedis pulses bilaterally.

Respiratory: CTAB. No wheezes or crackles.

Neurologic: A&O x 3. No facial droop. 5/5 strength in bilateral upper and lower extremities. No sensory changes. Smooth finger to nose and heel to shin.

Extremities: No cyanosis, clubbing or edema. No rash.

Psych: Slow to answer questions and has hard time recalling history. Flat affect.

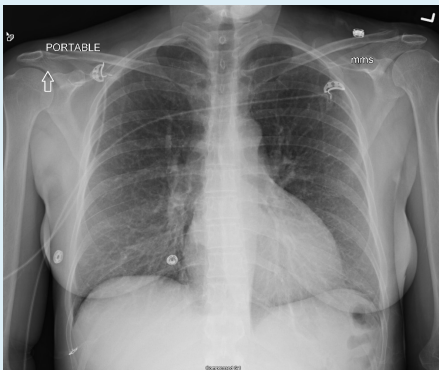
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INITIAL LABORATORY VALUES

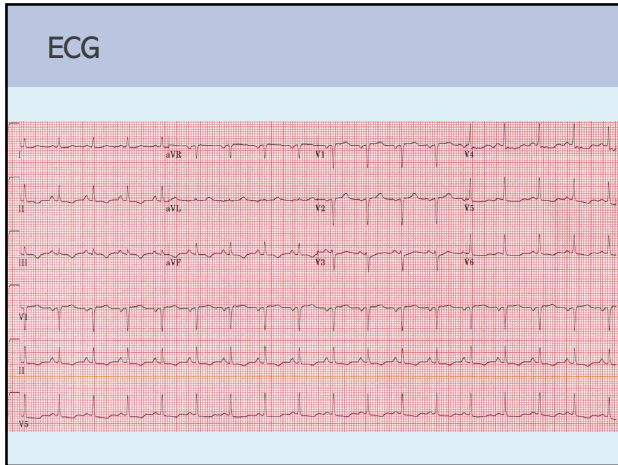
Sodium	136 mmol/L	WBC	22.48
Potassium	4.1 mmol/L	Hemoglobin	13.2
Chloride	103 mmol/L	Hematocrit	40.6
Bicarbonate	22.1 mmol/L	Platelets	170
Glucose	75 mg/dL	Total protein	7.5
BUN	18.7 mg/dL	Albumin	3.4
Creatinine	0.9 mg/dL	Total Bilirubin	0.8
Calcium	9.1 mg/dL	AST	28
Lipase	20 U/L	ALT	30
Ethanol	< 10 mg/dl	Alkaline phosphatase	157
Creatinine Kinase	130 U/L	TSH	1.35
Troponin T	1.21 ng/mL	Free T4	1.18

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PORTABLE 1-VIEW CXR



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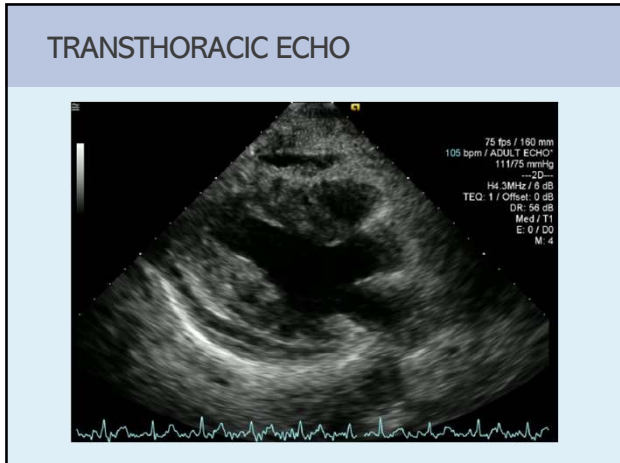
WHAT IS YOUR INITIAL DIFFERENTIAL?

WHAT ADDITIONAL LABS WOULD YOU LIKE TO HAVE?

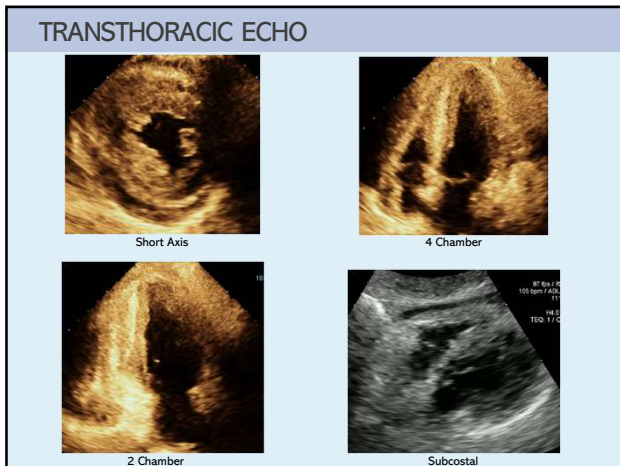
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DIFFERENTIAL		ADDITIONAL LABS	
Neutrophils	34.2%	ESR	76
Lymphocytes	12.3%	CRP	4.96
Monocytes	6%	Rapid COVID test	Negative
Eosinophils	46.7%	Lactate	1.4
Basophils	0.4%		
Immature granulocytes	0.4%		

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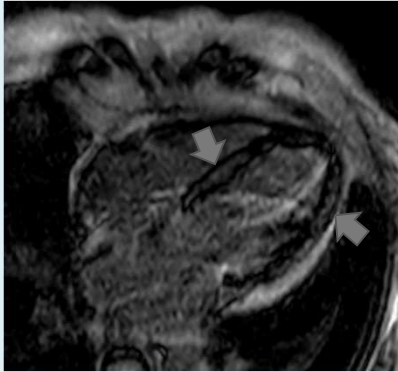
CARDIOLOGY CONSULT

At this point Cardiology was consulted.

Cardiac MRI was recommended for further investigation.

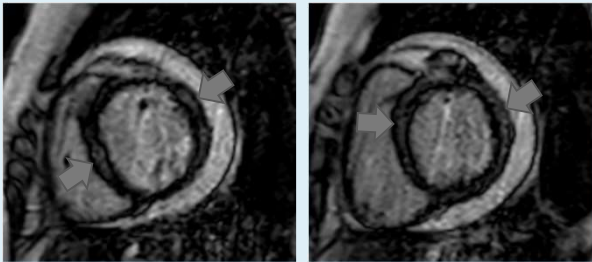
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CARDIAC MRI – 4 CHAMBER



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CARDIAC MRI – SHORT AXIS



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WHAT UNIFYING DIAGNOSIS WOULD PUT ALL OF THIS TOGETHER?

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HYPEREOSINOPHILIC SYNDROME WITH EOSINOPHILIC MYOCARDITIS

Causes of hypereosinophilic syndrome:

- Primary neoplastic syndrome (PDGFRA, FGFR1, or JAK2 mutations)
- Acute eosinophilic leukemia, CML, various T cell leukemia/lymphomas, B cell lymphoma, solid tumors
- Parasites (most commonly helminths)
- Drug hypersensitivity reactions
- Eosinophilic granulomatosis with polyangiitis (EGPA)
- Solid tumors (adenocarcinomas in particular)
- Idiopathic

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ADDITIONAL WORKUP

Parasitic Infection: Negative parasitic workup

Eosinophilic granulomatosis with polyangiitis (formerly Churg-Strauss syndrome): Negative ANA and ANCA x2

Hematologic: Negative flow cytometry and bone marrow biopsy to exclude lymphoma/leukemia

Malignancy: No malignancy evident on CT C/A/P or MRI brain

MRI/MRA Brain: Multiple small infarcts and microhemorrhages throughout the cerebellum and cerebrum in addition to a small subdural hematoma.

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EOSINOPHILIC MYOCARDITIS

- Rare form of myocardial inflammation. Characterized by eosinophilic infiltration of the myocardial tissue
- Identifying the underlying eosinophilic process is important for constructing a tailored treatment approach

PREVALENCE

- Rare disorder. True prevalence unknown
- 151 biopsy proven cases published from 2000-2017

Brambatti M, Matassini MV, Adler ED, Klingel K, Camici PG, Ammirati E. Eosinophilic Myocarditis: Characteristics, Treatment, and Outcomes. J Am Coll Cardiol. 2017; 70 (19): 2363-2375.

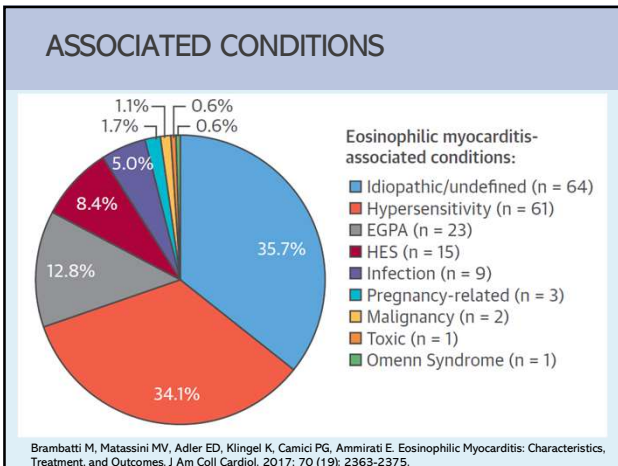
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DIAGNOSIS OF EOSINOPHILIC MYOCARDITIS

- Gold standard: Endomyocardial biopsy
 - Limitations: Patchy involvement decreases biopsy sensitivity and specificity

- Supportive:
 - Serum eosinophilia
 - Troponin elevation
 - Late gadolinium enhancement on CMR in non-vascular territory

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TREATMENT

The underlying etiology determines the treatment modality.

- Eosinophilic granulomatosis with polyangiitis → Cyclophosphamide
- Myeloproliferative variant → Imatinib
- Parasitic infection → Albendazole/Ivermectin
- Idiopathic hypereosinophilic syndrome → Steroids

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OUTCOMES

- In a 2017 meta-analysis of biopsy proven cases of eosinophilic myocarditis 22.3% of patients died during their index hospitalization, while 2.8% of patients received a long-term VAD
- However, the majority of patients survive eosinophilic myocarditis with supportive care through the acute phase

Brambatti M, Matassini MV, Adler ED, Klingel K, Camici PG, Ammirati E. Eosinophilic Myocarditis: Characteristics, Treatment, and Outcomes. J Am Coll Cardiol. 2017; 70 (19): 2363-2375.

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HOSPITAL DISCHARGE & FOLLOW UP

- Mild residual vision and memory deficits remain
- Titrated to prednisone 10mg daily with no worsening or recurrence of symptoms
- Eosinophils decreased to normal range
- Repeat echocardiogram with resolution of pericardial effusion and preserved EF

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THANK YOU!

Questions?

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