HARRISON

EDITION 22nd

CARDIOLOGY

PART 2

Cardiology

Chapter 268 Acute And Chronic Myocarditis — Completed

Chapter 269 Dilated Cardiomyopathies — Completed

Chapter 270 Amyloidosis And Other Restrictive Cardiomyopathies — Completed

Chapter 271 Cardiac Transplantation And Prolonged Assisted Circulation — Completed

Chapter 272 Aortic Stenosis — Completed

Chapter 273 Aortic Regurgitation — Completed

Chapter 274 Mitral Stenosis — Completed

Chapter 275 Mitral Regurgitation — Completed

Chapter 276 Mitral Valve Prolapse — Completed

Chapter 277 Tricuspid Valae Disease — Completed

Chapter 278 Pulmonic Valae Disease — Completed

Chapter 279 MIXED And MVHD — Completed

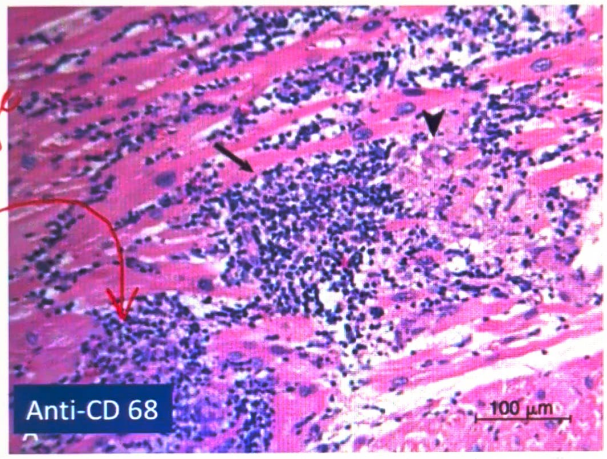
Chapter 280 Congenital Heart Disease In The Adult Part 1 — Completed

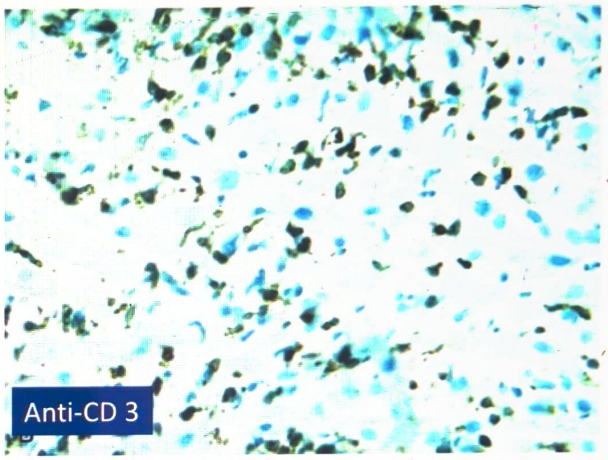
Cardiology

Chapter 282 Atrial Myxoma And Other Cardiac Tumors	Completed
Chapter 283 Cardiac Trauma	Completed
Chapter 281 Pericardial Diseases	Completed
Chapter 284 Ischemic Heart Disease	Completed
Chapter 285 Non-ST-Segment Elevation Acute Coronary Syndrome (NSTEMI-UA)	Completed
Chapter 286 STEMI	Completed
Chapter 287 Percutaneous Coronary Interventions And Other Interventional Procedures	Completed
Chapter 288 Hypertension	Completed
Chapter 289 Renovascular Hypertension	Completed
Chapter 290 Deep-Venous Thrombosis And Pulmonary Thromboembolism	Completed
Chapter 291 Diseases Of The Aorta	Completed
Chapter 292 Arterial Diseases Of The Extremities	Completed
Chapter 294 Pulmonary Hypertension	Completed

THE STANDARD DALLAS CRITERIA

Idiopathic myocarditis as an inflammatory infiltrate of the myocardium with necrosis and/or degeneration of adjacent myocytes not typical of the ischemic damage associated with coronary artery disease





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CLASS I INDICATION FOR ENDOMYOCARDIAL BIOPSY INCLUDES

a. New-onset heart failure of <2 weeks' duration associated with a normal-sized or dilated left ventricle and hemodynamic compromise

b. Heart failure associated with unexplained restrictive cardiomyopathy

c. Heart failure associated with dilated cardiomyopathy of any duration and suspected allergic reaction and/or eosinophilia

d. Heart failure >3 months' duration associated with a dilated left ventricle and new ventricular arrhythmias, second- or third-degree heart block, or failure to respond to usual care within 2 weeks

Answer: A

ENDOMYOCARDIAL BIOPSY

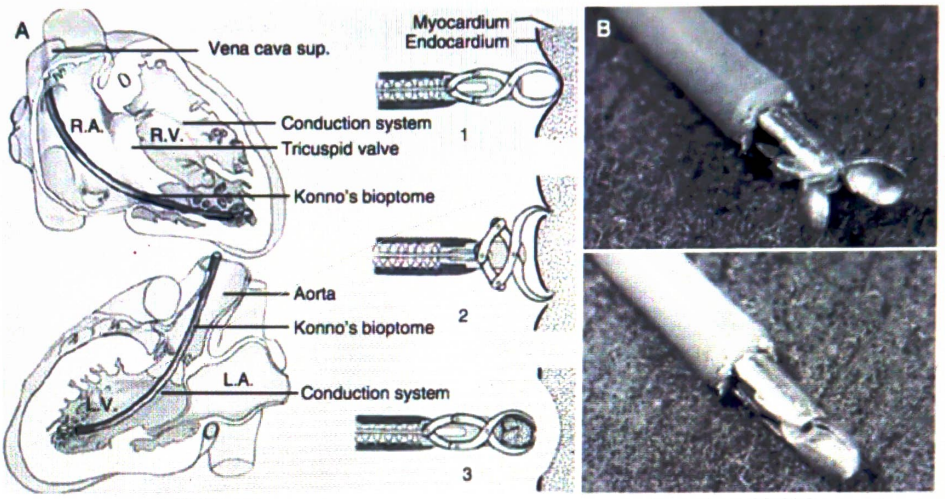
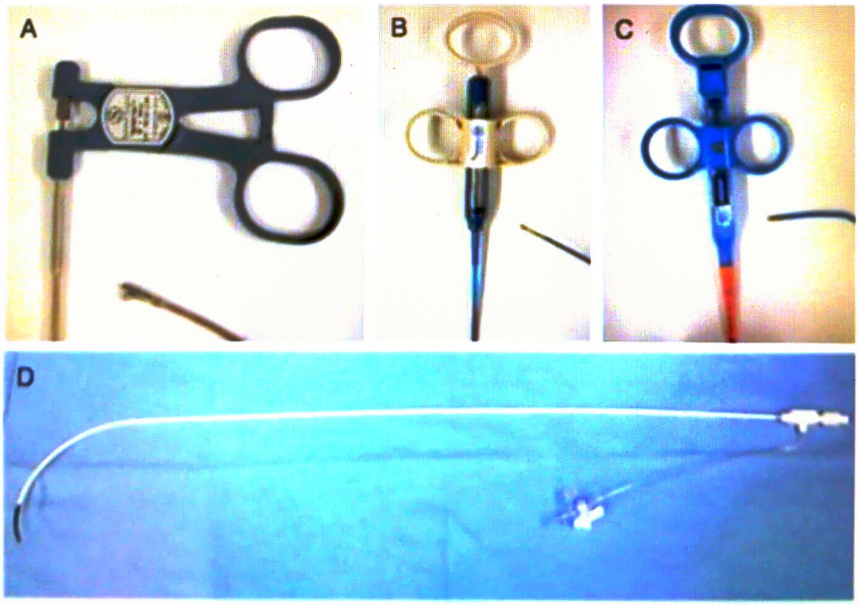


FIGURE 19.15 A, Original illustration by Konno and Sakakibara of the percutaneous technique of endocardial and myocardial specimen collection by use of a bioptome. **B,** Biopsies are taken by opening and closing the cutting claw at the tip of the catheter. (From Konno S, Sakakibara S. Endo-myocardial biopsy. Dis Chest 1963;44:345.)

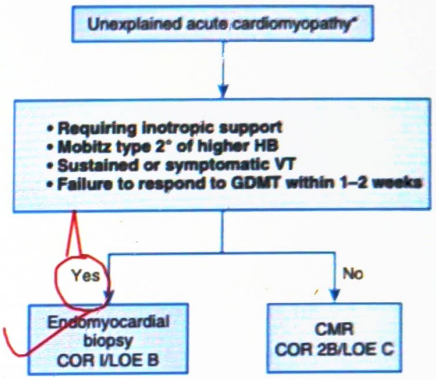
ENDOMYOCARDIAL BIOPSY



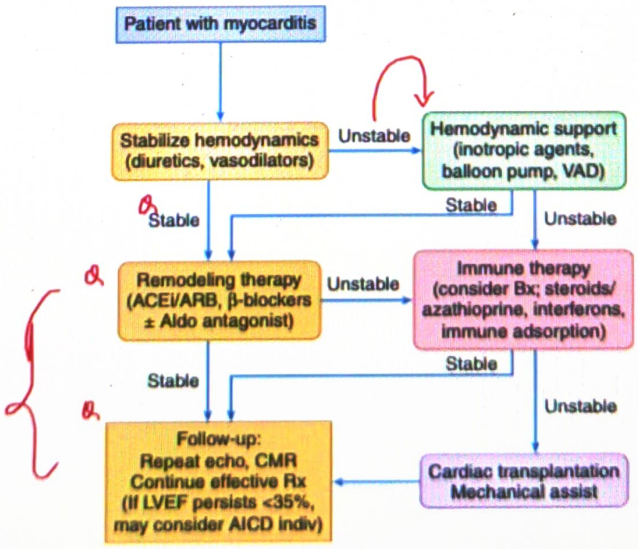
INDICATIONS FOR ENDOMYOCARDIAL BIOPSY

CLINICAL SCENARIO	CLASS OF RECOMMENDATION	LEVEL OF EVIDENCE
New-onset heart failure of <2 weeks' duration associated with a normal-sized or dilated left ventricle and hemodynamic compromise	I	B
New-onset heart failure of 2 weeks' to 3 months' duration associated with a dilated left ventricle and new ventricular arrhythmias, second- or third-degree heart block, or failure to respond to usual care within 2 weeks	I	B
Heart failure >3 months' duration associated with a dilated left ventricle and new ventricular arrhythmias, second- or third-degree heart block, or failure to respond to usual care within 2 weeks	IIa	C
Heart failure associated with dilated cardiomyopathy of any duration and suspected allergic reaction and/or eosinophilia	IIa	C
Heart failure associated with suspected anthracycline-induced cardiomyopathy	IIa	C
Heart failure associated with unexplained restrictive cardiomyopathy	IIa	C
Suspected cardiac tumors	IIa	C
Unexplained cardiomyopathy in children	IIa	C
New-onset heart failure of 2 weeks' to 3 months' duration associated with a dilated left ventricle, without new ventricular arrhythmias or second- or third-degree heart block, that responds to usual care within 2 weeks	IIb	B
Heart failure >3 months' duration associated with a dilated left ventricle, without new ventricular arrhythmias or second- or third-degree heart block, that responds to usual care within 2 weeks	IIb	C
Heart failure associated with unexplained hypertrophic cardiomyopathy	IIb	C
Suspected arrhythmogenic RV dysplasia	IIb	C
Unexplained ventricular arrhythmias	IIb	C
Unexplained atrial fibrillation	III	C

From Cooper LT et al. The role of endomyocardial biopsy in the management of cardiovascular disease. J Am Coll Cardiol 2007;50:1914



TREATMENT ALGORITHMS FOR PATIENTS WITH MYOCARDITIS

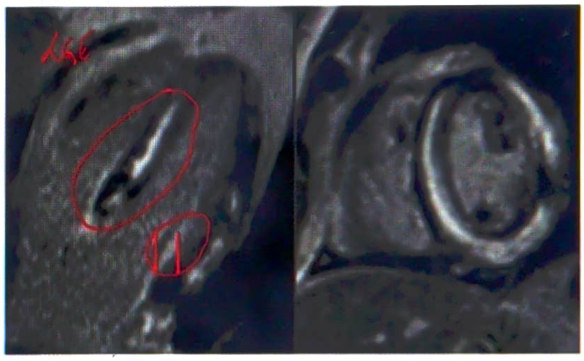


PROGNOSIS

- The prognosis is good for "uncomplicated" acute myocarditis, presenting with LVEF ≥0.50 without ventricular arrhythmias or circulatory shock.
- In the largest series, this low-risk group accounted for about three-fourths of patients with acute myocarditis, in which spontaneous recovery was common without specific therapies other than diuretics and nonsteroidal analgesics for chest pain.
- Almost all of these patients still had LVEF ≥0.50 on follow-up imaging and survived without trans- plantation at 5 years of follow-up. In the one-fourth of patients with complicated presentations, ~18% died or underwent heart transplantation in the next 5 years.

The LGE image shown below is consistent with?

a. Myocarditis
b. Sarcoidosis
c. Fabry's disease
d. Amyloidosis



Answer: A

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CARDIAC MRI IN MYOCARDITIS

- Cardiac MRI is being used with increasing frequency for noninvasive assessment of patients with suspected myocarditis.
- Cardiac MRI can evaluate 3 markers of tissue injury –intracellular & interstitial edema **(T2W)** hyperemia & capillary leakage **(EGE)** and necrosis & fibrosis **(LGE)**.
- A combination of T1-weighted and T2-weighted images had the best combination of sensitivity and specificity.

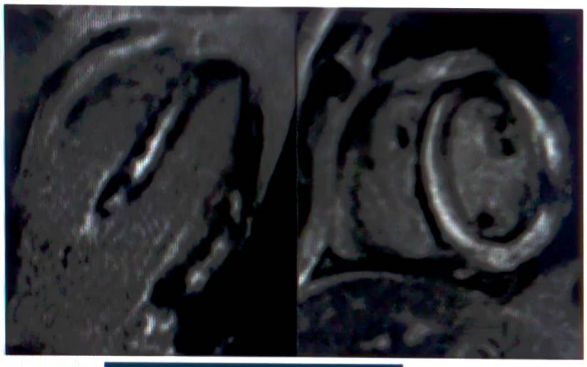
LAKE LOUISE CRITERIA
AT-LEAST 2 OF THE 3

- Regional or global myocardial signaling intensity increase in T2-weighted images.
- Increased global myocardial early gadolinium enhancement ratio b/w myocardium and skeletal muscle in gadolinium enhancement T1W images.
- At least one focal lesion with nonischemic regional distribution in inversion recovery prepared gadolinium enhanced T1W images (late gadolinium enhancement).

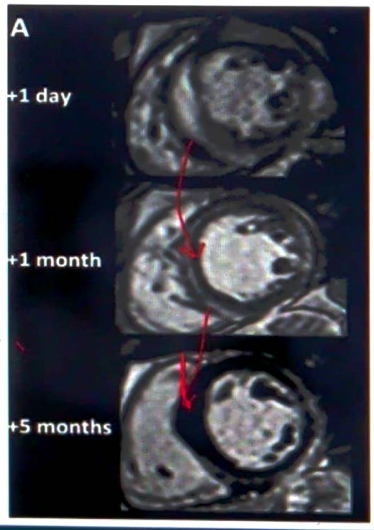
- Combination of EGE, LGE and T2 weighed imaging
- Excellent sensitivity and specificity (67%, 91%)

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MYOCARDITIS



Sparing sub-endocardium



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GIANT CELL MYOCARDITIS

- Predominated by rapid progressive HF, ventricular arrhythmias and heart blocks
- Giant cell myocarditis have survival less than 6 months and is improved with the use of immunosuppressive therapy
- Giant cell myocarditis often have other autoimmune disorders including
  - –Thymoma
  - –Thyroiditis
  - –Pernicious anemia

A 55-year old male presents to you with history of syncope. Physical examination reveals erythema nodosum. He undergoes underwent TBLN biopsy which had revealed non caseating granuloma. You plan a Holter which is unremarkable except for NSVT. What is the next step in management?

a. ACE levels and FDG PET scan of heart f/b primary prevention ICD

b. ACE levels and CMR f/b primary prevention ICD

c. ACE levels and FDG PET scan of heart f/b secondary prevention ICD

d. ACE levels and CMR f/b secondary prevention ICD

Answer: C

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GRANULOMATOUS MYOCARDITIS

STEP 1. SUSPICION OF MONOMORPHIC VT DUE TO GRANULOMATOUS DISEASE

MOMOMORPHIC VT OTHER THAN LV POSTERIOR FASCICULAR MORPHOLOGY
NORMAL ECHOCARDIOGRAM
ABSENCE OF OBSTRUCTIVE CAD/CHANNELOPATHIES/REVERSIBLE CAUSES

DE-CMR | ^{18}FDG PET-CT | HRCT

MEDIASTINAL ADENOPATHY, WITH/WITHOUT EXTRAMEDIASTINAL ADENOPATHY
(only nodes of ≥ 1 cm diameter considered significant) +
MID-MYOCARDIAL SCAR, WITH/WITHOUT ENDO/EPICARDIAL EXTENSION,
AND/OR FOCAL EXCESSIVE MYOCARDIAL ^{18}FDG UPTAKE

HIGH SUSPICION OF MMVT DUE TO GRANULOMATOUS DISEASE

STEP 2. DEMONSTRATION OF GRANULOMA

ABSENCE OF HIGH ^{18}FDG UPTAKE IN EXTRAMEDIASTINAL NODES → SAMPLING OF MEDIASTINAL NODES
SUGGESTED METHODOLOGY:
VATS-GUIDED BIOPSY/
TRANS-ESOPHAGEAL USG-GUIDED FNAC

HIGH EXTRAMEDIASTINAL NODE ^{18}FDG UPTAKE IN THE ABSENCE OF OTHER CAUSES → EXCISION BIOPSY OF EXTRAMEDIASTINAL NODE WITH HIGH ^{18}FDG UPTAKE
SUGGESTED NODE: SUPRACLAVICULAR

GRANULOMA

STEP 3. EVALUATION FOR ETIOLOGY OF THE GRANULOMA

TESTS DONE

TESTS DONE ON SAMPLED NODES
Stain for AFB
Culture for AFB
DNA-PCR for M.tuberculosis

TUBERCULIN SKIN TEST

INTERPRETATION OF TESTS

Stain or Culture positive for *M.tuberculosis*

- PCR positive
  ? ACTIVE TUBERCULOSIS
  ? SARCOIDOSIS
  → MMVT DUE TO ACTIVE TUBERCULOSIS
- PCR negative, SKIN TEST positive
  ?SARCOIDOSIS, with latent tuberculosis
  ?MMVT due to tuberculosis

Stain and Culture negative for *M.tuberculosis*

- PCR and SKIN TEST negative
  → MMVT DUE TO SARCOIDOSIS

STEP 4. TREATMENT

(all patients received both disease-specific treatment & anti-arrhythmic treatment)

DISEASE-SPECIFIC TREATMENT	ANTI-TUBERCULOSIS TREATMENT	ANTI-TUBERCULOSIS Rx + STEROIDS ± METHOTREXATE	CORTICOSTEROIDS ± METHOTREXATE
ANTI-ARRHYTHMIC TREATMENT	ANTI-ARRHYTHMIC DRUGS + ICD (recommended in all); RFA after 6-12 weeks of disease-specific therapy, if frequent recurrences of VT occur		

FULMINANT MYOCARDITIS

- Acute form of myocarditis, whose main characteristic is a rapidly progressive clinical course with the need for hemodynamic support
- Patients are often able to recall a distinct time of the onset of symptoms, usually within the preceding 2–4 weeks
- Life-threatening arrhythmia and aborted sudden cardiac death represent the most dramatic clinical presentations
- Abnormalities include nonspecific ST segment changes and ST-T elevation mimicking acute coronary syndromes and conduction disturbances

EOSINOPHILIC MYOCARDITIS: DRUGS CAUSING

Acetazolamide	Oxyphenylbutazone
Amitriptyline	Para-aminosalicylic acid
Amphotericin B	Penicillin
Ampicillin	Phenindione
Carbamazepine	Phenobarbital
Cefaclor	Phenylbutazone
Chloramphenicol	Phenytoin
Chlorthalidone	Spironolactone
Desipramine	Streptomycin
Hydrochlorothiazide	Sulfadiazine
Indomethacin	Sulfisoxazole
Interleukin-4	Sulfonylureas
Isoniazid	Tetanus toxoid
Methyldopa	Tetracycline

- The syndrome usually emerges within 8 weeks of the initiation of a new drug but can occur at any time after drug consumption.
- Corticosteroids and drug withdrawal usually resolve this syndrome; however, some patients may display a prolonged and relapsing course.

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DIPTHERITIC MYOCARDITIS







DIPTHERITIA

- R, 7 years girl, unimmunized, presented with history of
- fever, dysphagia, and swelling of neck for 5 days
- Physical examination shows
  - – Bull- neck appearance
  - – **Graybrown leather-like adherent membrane over the posterior oropharynx and hypopharynx.**
- At 2 hours of admission, child developed respiratory distress, audible stridor with severe subcostal and suprasternal retraction.

COMPLICATIONS- TOXIC CARDIOMYOPATHY

- Seen in 10-25% of patients with oropharyngitic diphtheria
- Responsible for 50-60% of deaths
- Typically, myocarditis is seen at the end of the second week of illness but may be seen as early as 5 days
- 'Myocarditis - most important predictor of mortality'

ECG abnormalities
T wave inversion in precordial leads
Sinus tachycardia
A-V block 2nd degree
"Sickle-shaped sagging" of ST segment
RBBB
Multiple Atrial Ectopic
Pseudo infarction pattern
Sinus bradycardia

PREDICTORS OF MYOCARDITIS IN DIPTHERIA

Independent predictors of development of myocarditis :

1. Bull neck
2. Pseudomembrane score of >2
3. Elevated aspartate aminotransferase level
4. Urinary ketone levels

Elevation of the serum aspartate aminotransferase concentration closely parallels the severity of myonecrosis

MANAGEMENT OPTIONS?

- Temporary pacemaker
- Carnitine
- Steroids

A 48-year-old woman with a history of seizure disorder and uveitis 1 year ago presents after 1 episode of loss of consciousness lasting a few seconds following exercise. She has experienced lightheadedness with exertion on 2 previous occasions. Her heart rate is 42 bpm, and her blood pressure is 80/50 mm Hg. ECG shows Mobitz type II second-degree heart block, and an echocardiogram shows a reduced left ventricular ejection fraction of 30% with no regional wall motion abnormalities. What is the best study to diagnose this patient's suspected condition?

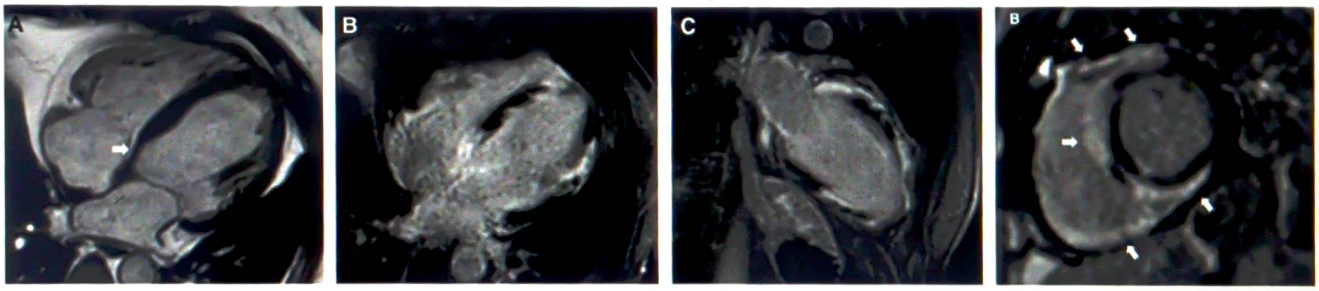
a. FDG-PET
b. Cardiac MRI
c. Ambulatory rhythm monitoring
d. Genetic testing

- Cardiac MRI is the first-line imaging study to diagnose cardiac sarcoidosis.
- Echocardiography can show left ventricular dysfunction, thinning of the basal septum, and regional wall abnormalities in a noncoronary distribution. However, these findings are neither highly sensitive nor specific for cardiac sarcoidosis.

Answer: B

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CARDIAC MR IN SARCOIDOSIS



Basal septal thinning | LGE showing patchy left ventricular involvement | 'Hook sign' pattern

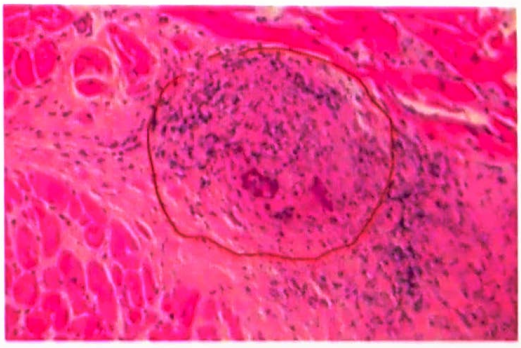
ENDOMYOCARDIAL BIOPSY

Characteristic findings

- Non-necrotic granulomas & isolated giant cells
- ± Lymphocytic/granulocytic infiltration combined with myocardial fibrosis
- Sharply demarcated areas of involvement
- No extensive eosinophilia or myocyte necrosis

Indirect EM biopsy signs of CS

- Small collection of histiocytes (Microgranulomas)
- Lymphangiogenesis
- Confluent fibrosis with fatty change



Non-necrotic granuloma in CS

A 53-year-old woman presents to the office with a history of syncope. She also notes occasional dizziness, which is non-exertional, lasting a few seconds each time, with spontaneous resolution. She does not have a family history of cardiac disease. She has a normal physical examination. She wore a rhythm monitor for 14 days that showed 1 episode of sustained ventricular tachycardia. She has a cardiac MRI that shows late gadolinium enhancement at the base of the interventricular septum. What is the next best treatment

a. Catheter ablation
b. Amiodarone
c. Corticosteroids
d. Tafamidis

Answer: C

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TREATMENT ALGORITHM FOR CLINICALLY MANIFEST CS PATIENTS

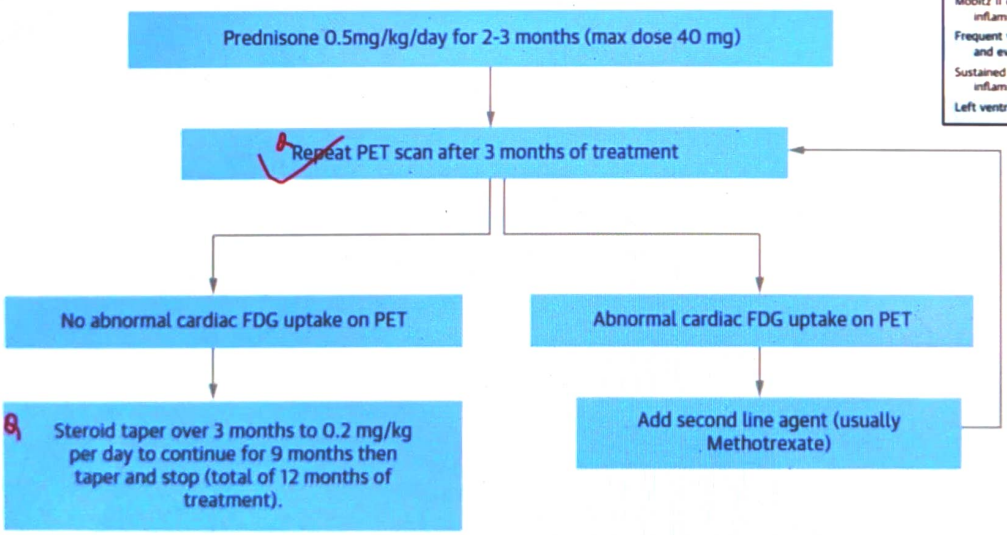


TABLE 4 Clinical Situations Where Immunosuppression Should Be Considered in Patients With Cardiac Sarcoidosis

- Mobitz II or 3rd degree heart block and evidence of myocardial inflammation
- Frequent ventricular ectopy or nonsustained ventricular arrhythmias and evidence of myocardial inflammation
- Sustained ventricular arrhythmias and evidence of myocardial inflammation
- Left ventricular dysfunction and evidence of myocardial inflammation

ETIOLOGIES OF DILATED CARDIOMYOPATHY

Inflammatory Myocarditis
Infective
Viral (coxsackie,[a] adenovirus,[a] HIV, hepatitis C)
Parasitic (*T. cruzi*—Chagas' disease, trypanosomiasis, toxoplasmosis)
Bacterial (diphtheria)
Spirochetal (*Borrelia burgdorferi*—Lyme disease)
Rickettsial (Q fever)
Fungal (with systemic infection)
Noninfective
Granulomatous inflammatory disease
Sarcoidosis
Giant cell myocarditis
Eosinophilic myocarditis
Polymyositis, dermatomyositis
Collagen vascular disease
Checkpoint inhibitor chemotherapy
Transplant rejection

Toxic
Alcohol
Catecholamines: amphetamines, cocaine
Chemotherapeutic agents (anthracyclines, trastuzumab)
Interferon
Other therapeutic agents (hydroxychloroquine, chloroquine)
Drugs of misuse (emetine, anabolic steroids)
Heavy metals: lead, mercury
Occupational exposure: hydrocarbons, arsenicals
Metabolic[a]
Nutritional deficiencies: thiamine, selenium, carnitine
Electrolyte deficiencies: calcium, phosphate, magnesium
Endocrinopathy
Thyroid disease
Pheochromocytoma
Diabetes
Obesity
Hemochromatosis

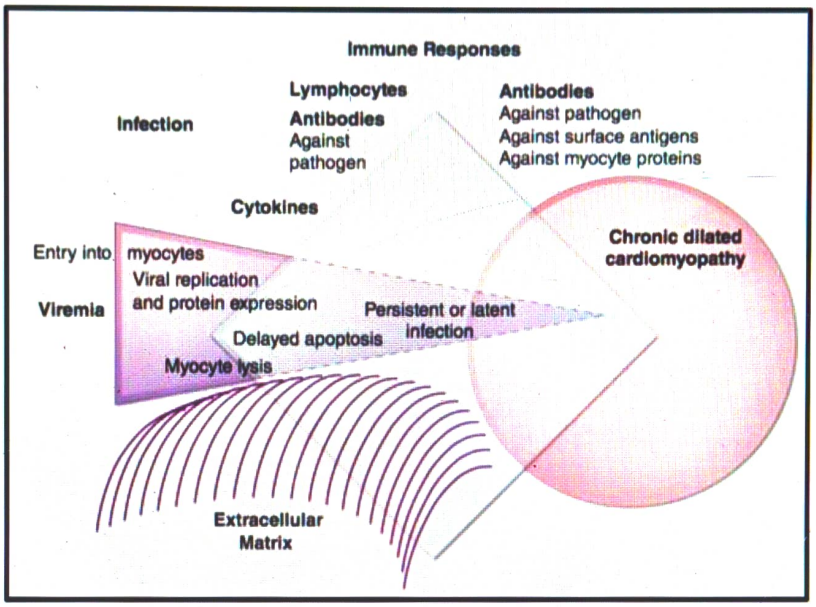
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ETIOLOGIES OF DILATED CARDIOMYOPATHY

Inherited Metabolic Pathway Defects[a]
Familial[a] (See Table 254-3)
Skeletal and cardiac myopathy
Dystrophin-related dystrophy (Duchenne's, Becker's)
Mitochondrial myopathies (e.g., Kearns-Sayre syndrome)
Arrhythmogenic ventricular cardiomyopathy
Hemochromatosis
Associated with other systemic diseases
Susceptibility to immune-mediated myocarditis
Overlap with Nondilated Cardiomyopathy
"Minimally dilated cardiomyopathy"
Hemochromatosis[a]
Amyloidosis[a]
Hypertrophic cardiomyopathy[a] ("burned-out")
"Idiopathic"[a]

"Idiopathic"[a]
Miscellaneous (Shared Elements of Above Etiologies)
Peripartum cardiomyopathy
Left ventricular noncompaction[a]
Tachycardia-related cardiomyopathy
Supraventricular arrhythmias with uncontrolled rate
Very frequent nonsustained ventricular tachycardia or high premature ventricular complex burden

NATURAL COURSE OF ILLNESS LEADING TO DCMP



GENES AND CARDIOMYOPATHY

- 30% cases of DCMP are familial
- Most familial DCMP are AD in inheritance (Exception??)
  - –Age dependent and incomplete penetrance
- Mis-sense, Deletions, duplications
  - –Mis-sense: Most common
- The most commonly recognized genetic causes of DCM?
  - –Truncating mutations in TITIN gene (25% Familial cases)

Acquired Dystrophin mutation?

DILATED CARDIOMYOPATHY



	DILATED
Ejection fraction (normal >55%)	Usually <30% when symptoms severe
Left ventricular diastolic dimension (normal <55 mm)	≥60 mm
Left ventricular wall thickness	Normal or decreased
Atrial size	Increased, may also be primarily affected
Valvular regurgitation	Related to annular dilation; mitral appears earlier during decompensation; tricuspid regurgitation with right ventricular dysfunction
Common first symptoms	Exertional intolerance / ↓ ATTENTION
Congestive symptoms[a]	Left before right, except right prominent in young adults
Arrhythmias	Ventricular tachyarrhythmia; conduction block in Chagas' disease, and some families. Atrial fibrillation.

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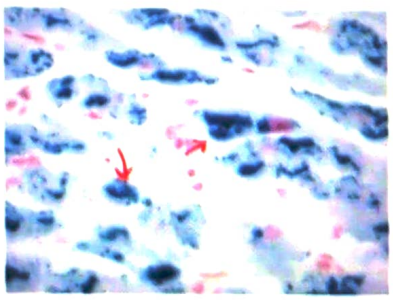
METABOLIC CAUSES OF CARDIOMYOPATHY

- Hypothyroidism and hyperthyroidism
- Pheochromocytoma
- Diabetes/Obesity
- Nutritional deficiencies: Thiamine, Selenium
- Dyselectrolytemia
  - –Hypocalcemia
  - –Hypomagnesemia (Thiamine dependent reactions)
- Hemochromatosis

HEMOCHROMATOSIS

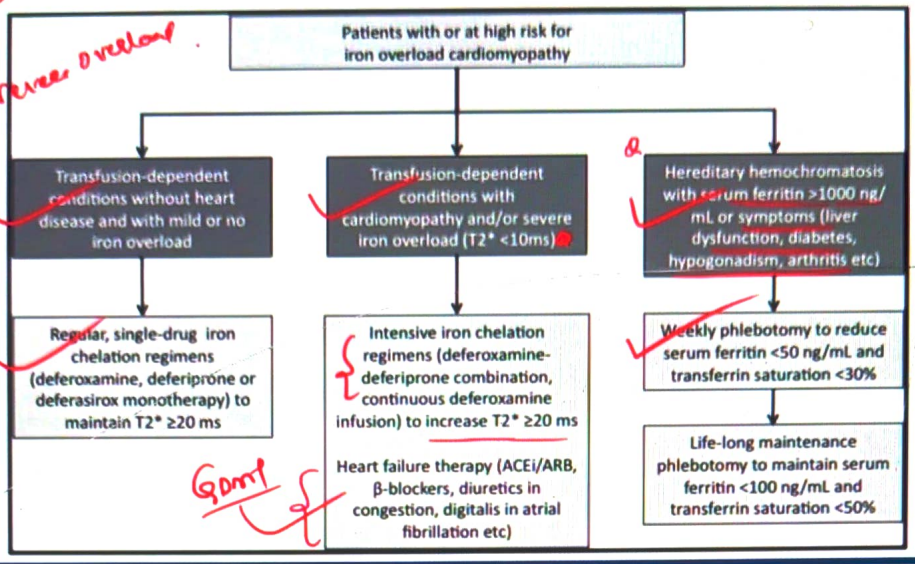
HFE gene Chromosome 6.

- Aquired or inherited
- The AR form is related to the *HFE* gene
- Up to 10% of the population heterozygous for one mutation, the clinical prevalence might be as high as 1 in 500
- Clinical presentation resembles DCM
- Diagnosis?
  - – Measurement of serum iron and transferrin saturation, with a threshold of >60% for men and >45–50% for women.



Prussian blue.

CARDIAC HEMOCHROMATOSIS



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A 12-year-old boy presents with severe burning pain in his extremities. Physical examination findings include numerous dark skin lesions over the lower trunk. A biopsy of the lesions reveals an excess of ceramide. He is diagnosed with a congenital disorder that eventually affects the heart and kidneys. What is the diagnosis?

A. Gaucher disease

B. Tay-Sachs disease

C. Lesch-Nyhan disease

D. Fabry disease

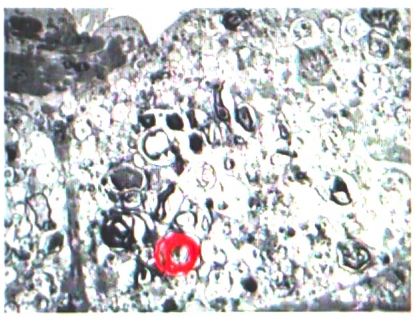
Fabry disease is a very rare, X-linked lysosomal storage disorder. There is a deficiency of the enzyme alpha-galactosidase, which leads to the accumulation of ceramide in blood vessels, nerves, and the heart.

- The disorder eventually affects the heart and kidneys. Angiokeratomas are common painless lesions around the umbilicus and lower abdomen. Fatigue and neuropathy are the most common presenting symptoms.

Answer: D

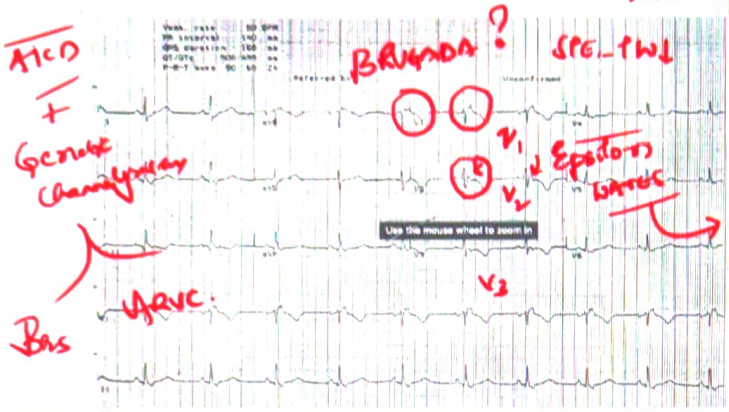
FABRY'S DISEASE

- Results from a deficiency of the lysosomal enzyme alpha-galactosidase (GLA gene)
- X linked recessive disorder
- Glycolipid accumulation may be limited to the cardiac tissues but usually also involves the skin, peripheral nerve, and kidney.
- Enzyme replacement therapy



Characteristic concentric lamellar inclusions of glycosphingolipids accumulating as a result of deficiency of the lysosomal enzyme alpha-galactosidase A

A 16-year-old ballet dancer presents with recurrent collapse that occurs during practice and when she stands up suddenly from a seated or a recumbent position. There is a family history of the sudden death of a 16-year-old sister during sleep. The patient's vital signs and the physical examination findings are unremarkable. An electrocardiogram is obtained (see image). Which of the following is considered the gold standard for making the suspected diagnosis?



A. 24-Hour Holter monitoring

B. Exercise stress testing

C. Cardiac magnetic resonance imaging

D. Endomyocardial biopsy

Answer: C

A 54-year-old man is admitted for a newly diagnosed cardiomyopathy. His past medical history is notable for heavy alcohol use. Which of the following is true of an alcohol-induced cardiomyopathy?

a. Administration of thiamine can reverse the cardiomyopathy

b. A daily alcohol intake of > 80 grams per day (3 drinks) for five years prior to diagnosis is associated with cardiomyopathy

c. A hyper-trabeculated phenotype

d. Occurrence of atrial fibrillation is associated with poorer outcomes

Answer: B

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ALCOHOL CARDIOMYOPATHY

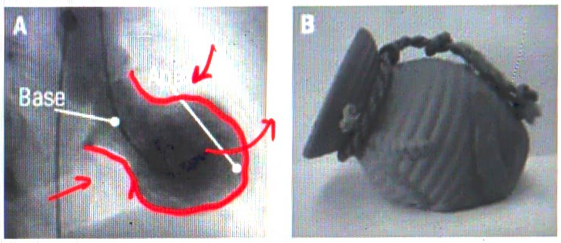
- The three criteria for diagnosing alcoholic cardiomyopathy include DCM phenotype, absence of other known and detectable causes of DCM, and a long history of heavy alcohol intake.
- The toxic effect is expected for daily alcohol consumption over 80 g (three or more standard-sized drinks per day) lasting 5 years or more before the onset or diagnosis.
- Atrial fibrillation, QRS width > 120 ms, and the absence of β-blocker therapy identify patients with a poor outcome.

CATECHOLAMINE INDUCED CARDIOMYOPATHY(TAKOTSUBO)

Typical Takotsubo

- Takotsubo Cardiomyopathy
- Apical Ballooning Syndrome
- Broken Heart Syndrome
- ampulla **cardiomyopathy**

First described in 1990 by Sato et al



2% of all troponin-positive patients presenting with suspected ACS

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CLINICAL FEATURES OF TAKOTSUBO CARDIOMYOPATHY

- Most common symptoms
  - Chest pain, 75.9 %
  - Dyspnea, 46.9 %
  - Syncope 7.7 %

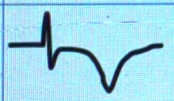
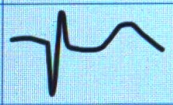
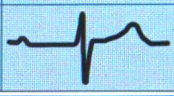
Some patients

  - Symptoms and signs of heart failure
  - Tachyarrhythmias
  - Bradyarrhythmias
  - Sudden cardiac arrest
  - Significant mitral regurgitation

- Approximately 10 percent of patients - cardiogenic shock

ELECTROCARDIOGRAM

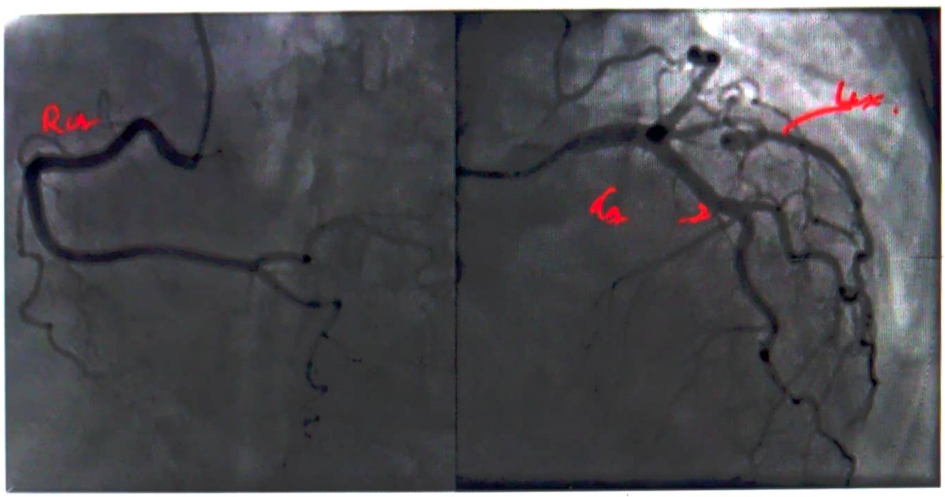
- ST segment elevation is frequent
- ST depression is a less common finding
- **Other findings**
  - QT interval prolongation,
  - T wave inversion,
  - abnormal Q waves,
  - non-specific abnormalities
  - VT

ECG change	Example	Prevalence in TTC
ST segment elevation, most commonly in praecordial leads		Common (46–100%)
Diffuse deep symmetric T-wave inversion. Resolves slowly and often partially		Common
Pathologic Q-waves that typically resolve before hospital discharge, with restoration of normal R-wave progression		37%
Prolonged QT interval (beginning of Q-wave to end of T-wave) which usually normalises in 1–2 days		
Prolonged PR (beginning of P-wave to beginning of QRS complex) interval		26%

ECHOCARDIOGRAM



ANGIOGRAM



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DIAGNOSIS - MAYO CLINIC DIAGNOSTIC CRITERIA

Proposed mayo clinic criteria for diagnosis of TC (All four are required for the diagnosis)

1. Transient hypokinesis, akinesis or dyskinesis of the left ventricular mid segments with or without apical involvement. The regional wall motion abnormalities typically extend beyond a single epicardial coronary distribution.
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
3. New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin.
4. Absence of pheochromocytoma and myocarditis.

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MANAGEMENT OF TAKOTSUBO

- **Generally a transient disorder**
- **Supportive management**
  - Heart failure
  - Hypotension
    - Mechanical circulatory support (eg, intra-aortic balloon counter-pulsation [IABP]) – preferred therapy
    - When there is marked LV dysfunction with severe hypotension or shock