Myocarditis

Myocardial inflammation · Supportive care, rhythm watch, no exercise

CMR IS THE TEST
biopsy for etiology

RECOGNIZE IT *a chameleon — from chest pain to cardiogenic shock*

PRESENTATIONS

Chest pain mimicking ACS with a rise in troponin but **unobstructed coronaries** · new **heart failure** · **arrhythmia** or conduction block · **fulminant** disease with cardiogenic shock · sudden death in the young.

THE HISTORY THAT FITS

Recent viral illness in the preceding days to weeks. Ask specifically about **immune checkpoint inhibitors**, recent vaccination, cocaine, and new drugs. Fever, myalgia, and a pericardial component are common.

FIRST TESTS

Troponin (often high, does not track severity) · ECG: **nonspecific ST-T change, new block, or arrhythmia** · **echo** for wall motion, EF, effusion · inflammatory markers. **A normal ECG does not exclude it.**

CONFIRM THE DIAGNOSIS *2018 Lake Louise criteria on cardiac MRI*

CMR — THE NON-INVASIVE STANDARD

Diagnosis requires **one T2-based criterion PLUS one T1-based criterion**. **T2 (edema):** raised myocardial T2 relaxation time, or visual edema / increased T2 signal ratio. **T1 (injury):** raised T1 relaxation time, raised **extracellular volume fraction**, or **positive late gadolinium enhancement**. The 2018 criteria outperform any single CMR parameter and improved on the 2009 version.

ENDOMYOCARDIAL BIOPSY — STILL THE GOLD STANDARD FOR CAUSE

Uses histopathology, immunohistochemistry, and molecular criteria. Reserve for **selected cases**: unclear diagnosis, or when a specific subtype is suspected that changes treatment — **giant cell myocarditis**, **eosinophilic myocarditis**, cardiac sarcoidosis. Consider urgently in **fulminant disease or unexplained high-grade block**, where immunosuppression may be indicated.

ALWAYS EXCLUDE OBSTRUCTIVE CORONARY DISEASE

A troponin rise with ST change is an **ACS until proven otherwise**. Myocarditis is frequently a diagnosis made **after** angiography or CT coronary angiography shows unobstructed vessels. Do not anchor on a viral prodrome in a patient with risk factors.

MANAGEMENT *mostly supportive — the specifics are about what to avoid*

1. **Remove the inciting agent** and treat the underlying cause where one exists.
2. **Treat heart failure with standard GDMT** — ARNI or ACEi/ARB, beta-blocker, MRA, SGLT2 inhibitor, plus diuretics for congestion. Most recover LV function; continue therapy and reassess the EF.
3. **Telemetry for every admitted patient.** Arrhythmia and high-grade block are the early killers, and both can appear with a preserved EF.
4. **Restrict strenuous activity and competitive sport** temporarily — typically 3–6 months, with return guided by recovery of function, resolution of inflammation, and absence of arrhythmia.
5. **Immunosuppression is not routine.** It is indicated for specific histologies — giant cell, eosinophilic, sarcoid, and autoimmune-associated disease — and for **checkpoint-inhibitor myocarditis**, where **high-dose corticosteroids are started urgently**.
6. **Fulminant myocarditis:** ICU, inotropes and vasopressors, early consideration of **mechanical circulatory support or VA-ECMO** as a bridge. Survivors of the acute phase often recover substantial function, so aggressive support is justified.

SPECIAL CAUSES *the ones with specific therapy*

CHECKPOINT-INHIBITOR MYOCARDITIS

Rare but **high mortality**. Usually within weeks of starting therapy. **Stop the inhibitor, start high-dose steroids immediately**, escalate immunosuppression if refractory. Watch for concurrent myositis and myasthenia-like weakness.

GIANT CELL & SARCOID

Giant cell: rapidly progressive HF plus ventricular arrhythmia or block; needs biopsy, immunosuppression, and early transplant assessment. **Cardiac sarcoid:** suspect with high-grade block in a younger patient; FDG-PET and steroids.

THE DIFFERENTIAL *troponin up, coronaries clear — what else fits*

Diagnosis	How to tell it apart
ACS with unobstructed arteries	Plaque erosion, spontaneous coronary dissection, embolism, or vasospasm. **Intravascular imaging or provocation testing** may be needed; CMR shows an ischemic (subendocardial) LGE pattern.
Takotsubo syndrome	Emotional or physical stressor, **apical ballooning** with a circumferential pattern, troponin modest relative to the degree of dysfunction, and typically **recovers within weeks**.
Pericarditis / myopericarditis	**Pleuritic positional pain, rub, PR depression.** A troponin rise means the myocardium is involved — reclassify and change the exercise advice.
Cardiac sarcoidosis	**High-grade block or VT in a younger patient**, patchy LGE, extracardiac features. FDG-PET, and steroids rather than supportive care alone.
Stress or sepsis-related injury	Demand ischemia in critical illness. Modest troponin, no CMR inflammation, resolves with the underlying illness.

PROGNOSIS & FOLLOW-UP *most recover, a minority do not*

WHAT PREDICTS A WORSE COURSE

Reduced EF at presentation, hemodynamic instability, **sustained ventricular arrhythmia**, high-grade block, and **extensive late gadolinium enhancement** on CMR. Fulminant presentations that survive the acute phase often recover well; giant cell and immune-checkpoint disease carry the worst outlook.

THE FOLLOW-UP PLAN

Repeat echo at roughly 3–6 months to confirm recovery of function and guide device decisions. Continue GDMT while the EF is reduced, and do not stop it the moment numbers improve. **Consider repeat CMR** before clearing an athlete. Persistent dysfunction becomes a chronic cardiomyopathy pathway.

PEARLS & PITFALLS

- **Troponin height does not predict severity or prognosis** in myocarditis. The EF, the rhythm, and the hemodynamics do.
- **Exclude coronary disease first.** Calling myocarditis on a viral prodrome alone has missed many occlusions.
- **High-grade block in a young patient** should prompt thought of sarcoid, giant cell, or Lyme — not just idiopathic conduction disease.
- **Avoid high-dose NSAIDs** where there is myocardial involvement or heart failure; they promote sodium retention and may worsen injury.
- **Do not clear an athlete early.** Return to play is a staged decision after function, inflammation, and rhythm are reassessed.
- Fulminant presentations can recover remarkably — escalate support rather than withdrawing early.

References: 2018 Lake Louise Criteria, JACC Expert Recommendations on CMR in Non-Ischemic Myocardial Inflammation (2018) · 2022 AHA/ACC/HFSA Heart Failure Guideline · AHA Scientific Statement on Fulminant Myocarditis.