ScienceInnovation
INTERNATIONAL SCIENTIFIC JOURNAL

ISSN:
2181-3337

INTERNATIONAL SCIENTIFIC JOURNAL

SCIENCE AND INNOVATION

VOLUME 4 / ISSUE 10

D:
BIOLOGY
AGRICULTURE
MEDICINE
PHARMACETICS
VETERINARY

OCTOBER 2025

International Scientific Journal
SCIENCE AND INNOVATION
Series D
Volume 4 Issue 10
October 2025

Tashkent 2025

CLINICAL AND INSTRUMENTAL CRITERIA FOR EARLY DIAGNOSIS OF CARDITIS IN PEDIATRICS: AN INTEGRATED, STEPWISE FRAMEWORK

G.K. Yusupova[1], D.T. Ashurova[2]
PhD[1]
Tashkent State Medical University[1,2]
https://doi.org/10.5281/zenodo.17434044

Abstract. *To develop an integrated, stepwise framework for early recognition of pediatric carditis—including myocarditis, rheumatic carditis, and cardiac involvement in MIS-C/Kawasaki phenotypes—by combining clinical "red flags," laboratory markers, electrocardiography (ECG), echocardiography (echo), and cardiac magnetic resonance (CMR) into practical decision pathways for different care settings. Early diagnosis of pediatric carditis remains challenging due to heterogeneous etiologies, age-dependent presentations, and variability in test availability across primary, secondary, and tertiary care. Delayed verification risks progression to heart failure, adverse remodeling, and chronic valvular pathology. Narrative synthesis of contemporary pediatric and cardiology guidance (SERVQUAL omitted; focus on medicine), emphasizing the 2018 updated Lake Louise CMR criteria for myocarditis, echocardiographic standards from the World Heart Federation (WHF) for rheumatic heart disease (2012; 2023 update), the revised Jones criteria (2015), and MIS-C frameworks (post-COVID era). The article translates these into implementable checklists, tables, and a stepwise diagnostic route with escalation triggers. We propose a triad-based workflow—Clinical profile → Core triage tests (troponin/NT-proBNP, ECG, echocardiography) → Etiologic confirmation (CMR for myocarditis; echo-based WHF/Jones pathways for rheumatic disease; MIS-C criteria with cardiac focus)—supported by standardized thresholds, minimum datasets, and quality checkpoints. Tables summarize age-specific red flags, biomarker thresholds and interpretation, ECG and echo findings by etiology, CMR markers per Lake Louise, and differential diagnoses. A structured, context-aware diagnostic route reduces missed early presentations and accelerates therapy initiation while aligning with ethical/operational constraints (radiation minimization, sedation avoidance, and informed consent). The framework is adaptable to resource-variable settings and supports consistent documentation for longitudinal follow-up.*

Keywords: *pediatric carditis; myocarditis; rheumatic carditis; MIS-C; Kawasaki disease; echocardiography; cardiac MRI; Lake Louise criteria; Jones criteria; troponin; NT-proBNP; early diagnosis.*

Abbreviations

ARF — Acute Rheumatic Fever; **BNP/NT-proBNP** — B-type natriuretic peptide/N-terminal pro-BNP; **CMR** — Cardiac Magnetic Resonance; **CRP** — C-reactive protein; **ECG** — Electrocardiogram; **EF** — Ejection Fraction; **EMB** — Endomyocardial Biopsy; **ESR** — Erythrocyte Sedimentation Rate; **LGE** — Late Gadolinium Enhancement; **LV** — Left Ventricle; **MIS-C** — Multisystem Inflammatory Syndrome in Children; **RHD** — Rheumatic Heart Disease; **WHF** — World Heart Federation.

1. Introduction

Carditis in the pediatric population encompasses inflammatory involvement of myocardium, pericardium, and/or valves from diverse etiologies: viral (myocarditis), autoimmune (acute rheumatic fever—ARF), and hyperinflammatory post-infectious entities (MIS-C, Kawasaki-like disease). The clinical imperative is early recognition: pediatric myocarditis can present as a subtle viral syndrome with disproportionate tachycardia, while rheumatic carditis may debut with subclinical valve regurgitation only detectable by echocardiography. MIS-C adds hemodynamic instability and coronary involvement risks. Despite advances in imaging, diagnostic inertia persists due to non-specific complaints, variable expertise, and access disparities. This paper synthesizes current standards into an operational, escalation-ready diagnostic route intended for clinicians working across the pediatric care continuum.

2. Epidemiology and Clinical Spectrum

Incidence estimates for pediatric myocarditis vary globally due to case definition and ascertainment differences, and many cases remain unrecognized until significant LV dysfunction develops. ARF remains prevalent in regions with constrained primary prevention and delayed streptococcal treatment; subclinical RHD is not rare when screened systematically by echo. MIS-C peaked during and after major SARS-CoV-2 waves but continues to present sporadically, with cardiac involvement a key determinant of severity and resource use.

Age effects. Infants may present with poor feeding, lethargy, and non-specific respiratory signs rather than chest pain. School-age children more often report chest discomfort and exercise intolerance; adolescents resemble adults with chest pain, palpitations, or syncope.

3. Pathophysiology (Brief Overview)

Myocarditis involves immune-mediated myocyte injury triggered by viral or other agents; edema and necrosis lead to conduction abnormalities and systolic/diastolic dysfunction. In ARF, molecular mimicry post-Group A Streptococcus infection targets valve tissue, especially the mitral and aortic valves. MIS-C features dysregulated post-infectious inflammation affecting myocardium and coronaries, with variable shock physiology.

4. Clinical “Red Flags” and Early Triage

A careful history (recent infection, sore throat, epidemiological ARF risk, COVID-19 exposure), examination (vital signs, gallop rhythm, new murmurs, perfusion), and age-aware symptom weighting are the backbone of early suspicion.

Table 1. Age-linked clinical red flags prompting cardiac work-up

Age group	Typical red flags	Practical notes
Infants	Feeding refusal, lethargy, diaphoresis with feeds, tachypnea	Low symptom specificity; keep low threshold for ECG + biomarkers + bedside echo
School-age	Chest pain, fatigue, palpitations, exercise intolerance, fever	Pain reproducibility does not exclude myocarditis; assess disproportionate tachycardia
Adolescents	Chest pain, presyncope/syncope, palpitations	Consider myocarditis vs. arrhythmic disorders; review drugs/stimulants
Any age (ARF risk)	Migratory polyarthritis, chorea, erythema marginatum, subcutaneous nodules	Seek evidence of recent Group A strep; auscultation may be insensitive—echo is key

Age group	Typical red flags	Practical notes
MIS-C context	Persistent fever, GI symptoms, rash, hypotension/shock	Early echo and lab inflammation panel; consider ICU admission thresholds

5. Laboratory Markers in Early Diagnosis

Inflammation. CRP and ESR are supportive but non-specific; markedly elevated profiles, particularly in MIS-C, heighten suspicion. **Myocardial injury/strain.** Troponin I/T elevation supports myocardial involvement; NT-proBNP/BNP reflects stress and is useful in risk stratification.

Strep evidence (ARF). Antistreptolysin-O and anti-DNase B titers (paired if possible) support recent infection when aligned with Jones criteria. **MIS-C panels.** Include ferritin, D-dimer, fibrinogen, and IL-6 where available—cardiac markers (troponin/BNP) are central.

Biomarker panel and interpretation in pediatric carditis

Marker	Early signal	Interpretation caveats
CRP/ESR	↑ supports inflammation	Non-specific; integrate with clinical picture
Troponin I/T	↑ suggests myocyte injury	Mild rises may occur in non-carditis states; trend with ECG/echo
NT-proBNP/BNP	↑ indicates myocardial stress/dysfunction	Age-dependent reference ranges; correlate with echo EF/strain
ASO/anti-DNase B	Evidence of recent strep A	Need clinical context per Jones criteria
Ferritin/D-dimer (MIS-C)	Hyperinflammation/coagulopathy	Helpful for severity; not diagnostic of carditis alone

6. Electrocardiography

Sinus tachycardia, ST-T abnormalities, and AV conduction delays (first-degree to higher-degree block) are frequent in myocarditis and can be seen in ARF carditis. Ventricular ectopy or non-sustained VT warrants close monitoring. A normal ECG does not exclude myocarditis; repeat testing can improve sensitivity.

Common ECG findings and suggested actions

Finding	Possible implication	Action
Sinus tachycardia out of proportion to fever	Early myocarditis or hemodynamic stress	Add troponin/BNP; proceed to echo
ST-T changes (diffuse)	Myopericardial inflammation	Exclude ischemia; trend markers; echo
PR prolongation/AV block	Conduction system involvement (myocarditis/ARF)	Telemetry; consider ICU if high-grade block
Ventricular ectopy/NSVT	Myocardial irritability	Telemetry, echo, consider CMR escalation

7. Echocardiography: First-Line Imaging

Myocarditis. Look for LV global/segmental hypokinesia, reduced EF, chamber dilation, pericardial effusion; speckle-tracking strain may precede EF decline. **Rheumatic carditis.** Apply WHF pathological regurgitation criteria (color Doppler jets, jet length/velocity, pansystolic/holodiastolic persistence, morphological leaflet abnormalities). Subclinical carditis is

diagnosable by echo even without audible murmurs. **MIS-C.** Assess LV function, pericardial fluid, and coronary artery Z-scores; repeat serially in acute and subacute phases.

Echocardiographic pointers by etiology

Etiology	Key echo features	Notes
Myocarditis	↓ EF, regional WMA, pericardial effusion, strain impairment	Strain may be earliest abnormality
Rheumatic carditis	Pathologic MR/AR per WHF, leaflet thickening, chordal elongation	Subclinical disease detectable; follow standardized WHF criteria
MIS-C	LV dysfunction, pericardial effusion, coronary dilation (Z-scores)	Repeat studies to track recovery

8. Cardiac MRI (CMR): Updated Lake Louise Criteria (2018)

Multiparametric CMR is the non-invasive reference standard when myocarditis is suspected and echo/ECG/biomarkers are inconclusive or severity must be clarified. The **2018 Lake Louise** update requires **≥1 T1-based marker** of non-ischemic injury (elevated native T1, increased ECV, non-ischemic LGE) **and ≥1 T2-based marker** of edema (elevated T2 or regional T2-weighted abnormalities). In pediatric practice, protocols should minimize sedation and scan time; gadolinium safety and renal function must be considered.

CMR markers and practical read-outs (Lake Louise 2018)

Domain	Marker	Practical interpretation
T2 (edema)	Elevated global/regional T2; T2-weighted hyperintensity	Active inflammation/edema
T1 (injury/fibrosis)	Elevated native T1; increased ECV; non-ischemic LGE	Myocyte injury; scar if LGE persists
Combined rule	≥1 T2 + ≥1 T1 marker	Supports myocarditis diagnosis

9. Endomyocardial Biopsy (EMB)

EMB is reserved for fulminant or refractory cases or when diagnosis will change management and non-invasive tests are non-diagnostic. Pediatric risk-benefit must be carefully weighed; many centers prioritize CMR-guided management.

10. Differential Diagnosis

Chest pain and troponin elevations are not myocarditis-specific. Consider pericarditis, anomalous coronaries (rare but critical), congenital/acquired channelopathies, hypertrophic or dilated cardiomyopathy, pulmonary embolism (adolescents), sepsis-related myocardial dysfunction, and non-cardiac etiologies (costochondritis, GERD).

Differential diagnosis highlights

Condition	Distinguishing features	First tests
Pericarditis	Pleuritic pain, pericardial rub, diffuse ST elevation with PR depression	ECG, echo (effusion), inflammatory markers
Coronary anomalies/ischemia	Exertional pain/syncope, ischemic ECG	Echo (origins), CMR/CT if needed
Cardiomyopathies	Family history, chronic symptoms, abnormal wall thickness	Echo, ECG, genetics if indicated

Condition	Distinguishing features	First tests
Sepsis-related dysfunction	Systemic infection, elevated lactate	Labs, echo (global dysfunction)
Non-cardiac chest pain	Reproducible tenderness, GI symptoms	Focused exam, reassurance, selective testing

11. Etiology-Specific Frameworks

11.1. Rheumatic Carditis and Jones Criteria

The **2015 Jones criteria** integrate clinical major/minor criteria with evidence of recent Group A strep infection; in moderate/high-risk populations, echocardiography is emphasized to detect **subclinical carditis**. Early identification enables secondary prophylaxis to prevent RHD progression.

11.2. MIS-C and Coronary-Focused Echo

MIS-C requires prompt evaluation of LV function, arrhythmic risk, and coronaries (proximal segments by Z-scores). Cardiac markers guide severity; shock physiology mandates ICU care and consideration of inotropes alongside immunomodulatory therapy.

12. Stepwise Diagnostic Pathways (Setting-Specific)

12.1. Primary/ED Setting (First 2–6 Hours)

1. **Clinical screen:** red flags (Table 1).
2. **Core labs:** troponin, NT-proBNP, CRP/ESR; plus CBC, electrolytes.
3. **ECG** with telemetry if arrhythmias suspected.
4. **Bedside echo** (focused if comprehensive is unavailable).

Escalate to tertiary care if high-grade AV block, sustained VT, hypotension/shock, or severe LV dysfunction.

12.2. Secondary/Tertiary Setting (24–72 Hours)

1. **Comprehensive echo** (strain if available).
2. **CMR** if myocarditis remains suspected or to characterize severity.
3. **ARF pathway:** apply Jones criteria + WHF echo standards.
4. **MIS-C pathway:** inflammatory panel, cardiac markers, echo serially; ICU if shock.

Table 7. Minimal datasets by level of care

Level	Must-have	Nice-to-have
Primary/ED	ECG, troponin, NT-proBNP, CRP/ESR, focused echo	Strain echo; rapid strep tests if ARF suspected
Secondary	Comprehensive echo (with Doppler), telemetry	Strain, serial markers
Tertiary	CMR (Lake Louise), advanced labs, ICU protocols	EMB (selected), cardiac CT, genetics (when indicated)

13. Quality and Safety Considerations

- **Radiation minimization:** Prefer echo and CMR over CT where feasible.
- **Sedation strategies:** Pediatric CMR protocols to reduce need for sedation; consider feed-and-sleep in infants.
- **Consent and communication:** Age-appropriate explanations; parental counseling on trajectory and follow-up.

- **Documentation:** Structured templates for clinical, ECG, echo, and CMR findings improve continuity and auditability.

14. Implementation: Checklists and Triggers

Adopt a **diagnostic passport** for key metrics (owner, frequency, thresholds) and set **green/amber/red zones** with predefined actions: e.g., troponin > reference + abnormal ECG → echo within 2 hours; EF <40% or high-grade AV block → ICU; echo-defined pathological MR/AR (WHF) → ARF pathway and secondary prophylaxis.

Table 8. Example trigger map for early actions

Trigger	Action
Troponin ↑ + ECG abnormal	Comprehensive echo within hours; telemetry
EF <40% or high-grade AV block	ICU transfer; inotrope/readiness; rhythm management
Pathologic MR/AR (WHF)	ARF/RHD pathway; secondary prophylaxis plan
MIS-C suspected with hypotension	ICU; immunomodulatory protocol; serial echo

15. Discussion

A unified, stepwise framework reconciles heterogeneity in pediatric presentations with the reality of multi-level care. Its strengths are (i) **simplicity** at the front door (clinical triage + core markers + ECG + echo), (ii) **clarity** in escalation (CMR when needed; EMB selectively), and (iii) **specificity** for etiologies (Jones/WHF for ARF; MIS-C cardiac focus). The approach supports earlier therapy, better documentation, and standardized follow-up. Limitations include variability in access to CMR and pediatric echo expertise; the framework should be adapted to local resources (e.g., hub-and-spoke referral models). Future multicenter work should quantify how pathway adherence affects time-to-diagnosis, ICU transfers, and medium-term LV recovery.

16. Conclusions

Early pediatric carditis diagnosis is best achieved through a structured route: **clinical suspicion → core triage (troponin/BNP, ECG, echo) → targeted confirmation (CMR for myocarditis; WHF/Jones echo standards for ARF; MIS-C cardiac pathway)**. The framework is implementable across care levels, reduces missed cases, and expedites definitive therapy while balancing safety, consent, and resource constraints.

REFERENCES

1. Law YM, et al. Diagnosis and Management of Myocarditis in Children. *Circulation*. 2021.
2. Ferreira VM, et al. Cardiovascular Magnetic Resonance in Non-Ischemic Myocardial Inflammation. *J Am Coll Cardiol*. 2018.
3. Luetkens JA, et al. Comparison of Original and 2018 Lake Louise Criteria for CMR in Myocarditis. *Radiol Cardiothorac Imaging*. 2019.
4. Gewitz MH, et al. Revision of the Jones Criteria for the Diagnosis of Acute Rheumatic Fever. *Circulation*. 2015.
5. World Heart Federation. Echocardiographic Diagnosis of Rheumatic Heart Disease—Guidelines 2012; Update 2023–2024.
6. Marangou J, et al. Echocardiographic Diagnosis of Rheumatic Heart Disease. *Global Heart*. 2024.
7. CDC/CSTE. MIS-C Surveillance Case Definition & Reporting Guidance. 2023.
8. Shah SS, et al. Cardiac Findings in Multisystem Inflammatory Syndrome in Children (MIS-C). *Cardiol Young*. 2023.

9. Williams JL, et al. Pediatric Myocarditis: Evaluation and Management. *Curr Treat Options Pediatr*. 2023.
10. Henderson LA, et al. American College of Rheumatology Clinical Guidance for MIS-C. *Arthritis Rheumatol*. 2022.

Contents

03.00.00 BIOLOGICAL SCIENCES

06.00.00 AGRICULTURAL SCIENCES

14.00.00 MEDICAL SCIENCES