

The two-tier safety net: subacute pericarditis and the ethics of diagnostic access in Aotearoa New Zealand

Omid Delfi,¹ and Christopher Yee.²

Affiliations:

¹School of Medicine, University of Auckland, Auckland, New Zealand

²Department of General Medicine, Auckland City Hospital, Auckland, New Zealand

Correspondence to: odel967@aucklanduni.ac.nz | ORCID 0000-0002-0183-116X

Abstract

Subacute pericarditis presents a distinct clinical challenge because its gradual onset can mask progression to cardiac tamponade. In Aotearoa New Zealand, public health services meet international safety guidelines for follow-up imaging, yet geographic location and access to private medical insurance generate variations in diagnostic turnaround. We present the case of a 55-year-old male presenting with a large pericardial effusion (22 mm). Although public follow-up occurred within the recommended three-month safety window, private insurance in a metropolitan centre enabled echocardiographic confirmation of effusion resolution at three weeks. Large effusions (>20 mm) carry higher baseline risks of delayed complications, creating a structural reliance on intermediate outpatient surveillance. Where private insurance provides rapid confirmation, individuals residing in rural areas or lacking private insurance face extended waitlists without an intermediate diagnostic buffer, relying on primary care red-flag triage to identify acute deterioration. To address these structural gaps, we evaluate workforce expansion, specialist retention, and community-based diagnostics as pathways to improve regional access across Aotearoa New Zealand.

Keywords: Access; Cardiac tamponade; Diagnostic guidelines; Subacute pericarditis; Treatment guidelines

Introduction

Pericarditis is an inflammatory syndrome of the pericardium that carries a significant risk of progression to cardiac tamponade, a state of obstructive shock.^{1,2} Subacute pericarditis features a gradual onset, where progressive fluid accumulation initially causes minimal symptoms due to pericardial compliance, explaining why patients may initially remain asymptomatic. When fluid volume exceeds pericardial compliance, rising intrapericardial pressure restricts diastolic filling, precipitating symptoms such as exertional dyspnoea, chest discomfort, elevated venous pressure, presyncope, and clinical red flags of hemodynamic compromise.³ International guidelines, including the European Society of Cardiology (ESC)¹ and the 2025 American College of Cardiology (ACC) Expert Consensus Statement⁴, suggest repeat imaging within three to six months for subacute cases to monitor for resolution or deterioration.

In Aotearoa New Zealand, public health services maintain clinical safety by triaging low-risk effusions to guideline-compliant three- to six-month follow-up windows.⁵⁻⁶ System capacity limits prevent routine public services from offering early intermediate surveillance for large effusions (>20 mm), which carry higher baseline risks of delayed complications. This report examines a 55-year-old male presenting with a large pericardial effusion. While public follow-up occurred within guideline thresholds, private insurance in a metropolitan centre enabled echocardiographic confirmation of effusion resolution at three weeks. By analysing this case, we explore how geographic location and private sector access interact to dictate diagnostic turnaround across Aotearoa New Zealand, examining the reliance on primary care red-flag triage to maintain patient safety while on public waitlists.⁷⁻⁹

To evaluate these clinical and structural dynamics, this analysis addresses two central questions:

1. What do current international and national guidelines recommend for the medical management and surveillance of subacute pericarditis, and how do these timelines align with clinical risk dynamics?
2. How do regional health infrastructure, diagnostic workforce distribution, and private sector availability interact to influence diagnostic turnaround across Aotearoa New Zealand?

Case Presentation

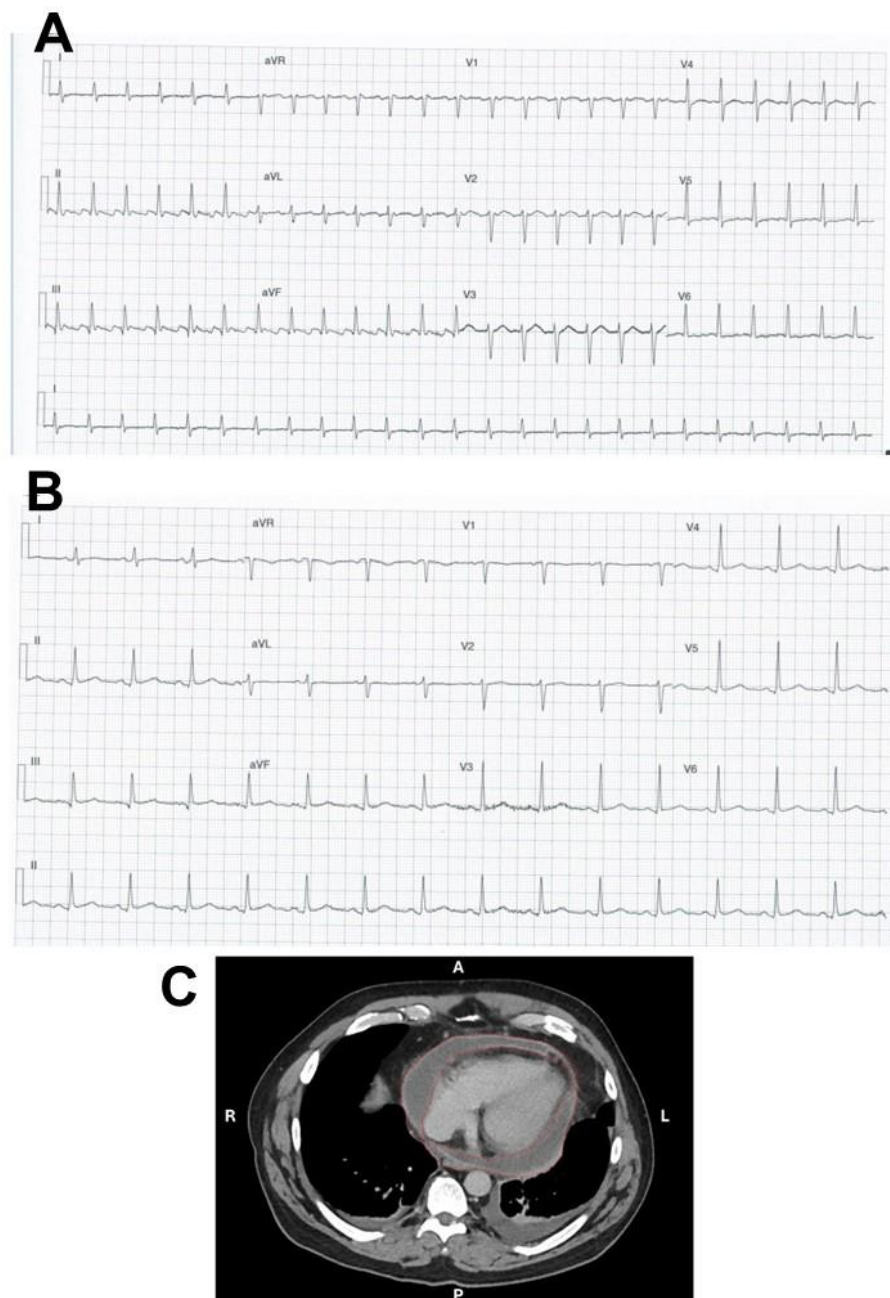
Patient History and Examination

A 55-year-old male presented in early 2026 with a two-month history of abdominal distension and pleuritic chest pain. On admission, physical examination revealed tachycardia (109 bpm), tachypnoea (22 breaths/min), dual heart sounds without pulsus paradoxus, and marked abdominal distension.

Diagnostic Findings and Clinical Course

Admission electrocardiogram (ECG) demonstrated new-onset atrial flutter with 2:1 conduction (Figure 1A), stabilised to 88 bpm following pharmacological rhythm control (Figure 1B). Contrast-enhanced chest CT identified a 22 mm complex pericardial effusion (Figure 1C). Initial C-reactive protein (CRP) measured 196 mg/L, peaking at 256 mg/L on day two. CT mesenteric angiogram excluded gastrointestinal pathology. Rheumatological and infectious screens (Supplemental Tables 2 and 3) were non-diagnostic for secondary aetiologies, establishing a working diagnosis of idiopathic subacute pericarditis. Pleural fluid aspiration was negative for malignancy and tuberculosis.

Figure 1: Electrocardiographic and radiographic findings in subacute pericarditis. (A) 12-lead ECG on admission. Demonstrates new-onset atrial flutter with characteristic sawtooth waves and 2:1 conduction resulting in a ventricular rate of 150 bpm; **(B) 12-lead ECG post-rate control.** Shows successful stabilisation of the ventricular rate at 88 bpm following pharmacological intervention; **(C) Contrast-enhanced axial CT chest.** The red dashed lines delineate a large, complex 22 mm pericardial effusion surrounding the heart. Anatomical markers indicate anterior (A), posterior (P), right (R), and left (L) orientations.



Nonsteroidal anti-inflammatory (NSAIDs) were withheld due to active duodenitis and gastric ulceration. Treatment was initiated with colchicine monotherapy (0.5 mg twice daily) alongside amiodarone for rhythm control.^{4,10,11} Inflammatory markers improved, with CRP declining to 96 mg/L prior to discharge on day six. On post-discharge day 13, outpatient laboratory testing demonstrated a transient CRP increase to 69 mg/L (following a transient value of 4 mg/L on post-discharge day four). The patient remained clinically asymptomatic without exertional dyspnoea, chest pain, fever, or signs of elevated central venous pressure. Private echocardiography accessed three weeks post-discharge confirmed complete effusion resolution. Routine public sector follow-up echocardiography occurred at just under three months, confirming sustained resolution within standard guidelines.^{1,4,12}

Discussion

Diagnostic Physiology, Imaging Modalities, and Clinical Kinetics in Pericardial Disease

To address the first question regarding current guideline recommendations for subacute pericarditis, we evaluated international and national management frameworks alongside clinical risk dynamics. International guidelines emphasise distinguishing primary from secondary aetiologies in pericarditis management.^{1,4} In this patient's case, comprehensive diagnostic evaluation (detailed in Section 2.2 and Supplemental Tables 2 and 3) systematically excluded autoimmune, infectious, and malignant causes, confirming an idiopathic aetiology. It is important to note that although systemic inflammatory markers such as CRP provided diagnostic utility during this patient's acute presentation, recent consensus guidelines, including the 2025 ACC Expert Consensus Statement,⁴ note their limited specificity and recognise that non-inflammatory phenotypes can present with normal biomarker levels. While inflammatory markers reflect systemic inflammation, they cannot assess disease progression or the anatomical extent of pericarditis. Integrating patient symptoms with multimodality cardiac

imaging is therefore essential to quantify pericardial fluid volume, evaluate spatial distribution, and detect hemodynamic compromise.^{1,4,13}

Transthoracic echocardiography (TTE) serves as the primary imaging modality for evaluating pericardial disease.^{4,5,12,13} TTE enables dynamic assessment of cardiac structures, chamber collapse, and real-time hemodynamic without ionising radiation.^{2,4,12,13} When TTE is delayed by regional sonographer shortages or limited by poor acoustic windows, contrast-enhanced chest CT provides a reliable secondary alternative.^{2,4,13} CT accurately quantifies effusion volume, evaluates pericardial thickening, identifies loculated collections, and detects static signs of elevated intrapericardial pressure such as vena caval distension or chamber compression.^{2,4,13} While CT lacks dynamic Doppler flow assessment, rapid acquisition and widespread availability in secondary health centres make it a practical diagnostic option when ultrasound access is constrained.^{2,13} Cardiac MRI delivers detailed tissue characterisation for active pericardial inflammation, though routine access remains constrained by scanner availability, acquisition time, and cost.^{4,13} A detailed comparison of these modalities appears in Supplemental Appendix 1.^{4,13}

Medical management of subacute pericarditis depends on inflammatory activity and underlying aetiology. Current consensus guidelines recommend a minimum three-month course of colchicine combined with NSAIDs or aspirin for initial episodes, accompanied by exercise restriction (maintaining heart rate below 100 bpm) until clinical remission.^{1,4,10,11} When NSAIDs are contraindicated due to peptic ulcer disease or active gastrointestinal bleeding, colchicine monotherapy controls pericardial inflammation.^{1,26} Extended colchicine regimens (six to 12 months) are indicated for disease recurrence, which occurs in 15% to 30% of idiopathic cases.^{1,4}

Cardiac tamponade occurs in approximately 3% of acute pericarditis presentations and up to one-third of large chronic effusions.^{2,4,14} Hemodynamic compromise is driven by the rate of fluid accumulation relative to pericardial compliance rather than absolute volume alone.^{1,2,4} Although our patient demonstrated declining CRP values prior to discharge, the initial 22 mm effusion represented an elevated baseline volume (>20 mm) associated with higher baseline risk for delayed complications, establishing the need for ongoing outpatient surveillance.

The post-discharge CRP increase to 69 mg/L on day 13 illustrates the challenge of biomarker monitoring. Under the 2025 ACC Expert Consensus Statement,⁴ isolated CRP fluctuations without accompanying clinical symptoms do not signify disease progression or warrant emergency repeat imaging. Patient safety relies on monitoring for specific clinical red flag features: worsening exertional dyspnoea, orthopnoea, presyncope or syncope, chest pain exacerbation, recurrent fever, or physical examination signs of elevated central venous pressure such as jugular venous distension.^{1,2,4,12,14} Because the patient remained asymptomatic without red flag features, conservative management was appropriate. Demonstrating complete effusion resolution on the three-week private scan provided psychological reassurance; it did not alter the mandatory three-month colchicine course or eliminate recurrence risk.^{4,10} Both international consensus and the CSANZ Echocardiography Appropriateness Guidelines confirm that in stable patients without clinical red flags, early routine repeat imaging is not indicated.^{4,12}

Geographic and Socioeconomic Inequities in Diagnostic Access

Addressing the second question regarding systemic and geographic diagnostic access, we examined the structural dynamics of healthcare delivery in Aotearoa New Zealand. The healthcare system operates under a dual-system model where the public sector manages acute stabilisation while the private sector absorbs elective demand.^{5,13} Public health services face

strict capacity ceilings, operating under resource management thresholds that triage routine non-urgent surveillance to three- to six-month windows.^{7,12} The fact that 13.1% of public secondary referrals are currently declined due to capacity constraints demonstrates that public facilities must restrict access to manage baseline demand.⁸ To bypass these waitlists, approximately one-third of New Zealanders maintain private medical insurance.^{5,15} As analysed by Fenton and Gauld,²⁷ this reliance on private care creates workforce competition between sectors, drawing specialist capacity away from public hospitals and reinforcing public wait times.

These structural capacity limits are further compounded by geographic location across regional and rural Aotearoa New Zealand. Data from the SCANZ Workforce Survey confirm that public hospital imaging volume is directly dictated by regional cardiac sonographer staffing density.¹⁶ Table 1 demonstrates how regional variations in sonographer FTE per capita mirror lower overall diagnostic throughput across non-metropolitan health districts.¹⁶

Table 1: Regional variation in echocardiography provision in New Zealand (source: Buckley et al.¹⁶).

Metric	National Mean	Regional Mean
Echocardiograms per 100,000 population	1,748	1,658
Sonographer FTE per 100,000 population	1.4	1.3
Waitlist Duration (Average)	~3 months	Not reported

While nationwide published datasets specifically detailing cardiac sonographer staffing numbers remain sparse post-SCANZ, contemporary reporting confirms that geographic disparities in diagnostic imaging persist. The 2025 Office of the Auditor-General (OAG) report

on planned care demonstrates that access to planned medical care and diagnostic imaging varies according to regional health district and geographic location.⁷ The report notes that patients in rural communities face extended wait times for specialist diagnostic services due to workforce shortages and regional capacity limits.⁷ Official reporting from Health New Zealand Te Whatu Ora documents public waitlists for non-urgent echocardiography ranging between three to six months depending on the district.¹⁷

While a three- to six-month timeframe aligns with routine guideline surveillance for low-risk effusions, large effusions (>20 mm) carry up to a 33% risk of tamponade or delayed complication.^{2,4,14} Public health services schedule routine follow-up within this three- to six-month window to maintain baseline safety, yet this leaves no outpatient diagnostic buffer for intermediate re-evaluation if subacute or ambiguous symptoms arise post-discharge. Consequently, workforce limits translate into unequal access to intermediate diagnostic assurance for high-volume effusions.

This disparity is further compounded by geography and sector access. Patients with private health insurance in major metropolitan centres can readily secure rapid outpatient echocardiography (as seen in this case at three weeks) for early diagnostic confirmation. Conversely, rural patients face a compound disadvantage: private diagnostic infrastructure is virtually absent, and alternative cross-sectional modalities such as chest CT encounter identical regional equipment and staffing shortages.^{8,13,14} Individuals residing in rural areas or lacking private health insurance thus remain tied to public waitlists, illustrating how geographic location and financial resources intersect to dictate diagnostic turnaround without altering underlying medical management.^{5,17,18}

Pathways Toward Workforce and Capacity Resilience

To address regional diagnostic equity, we propose two complementary pathways: refining primary care triage protocols and expanding diagnostic capacity. Standardised clinical prioritisation protocols, such as national echocardiography appropriateness criteria, help identify high-risk presentations and optimise imaging utilisation.¹² Primary care clinicians serve as vital first-line assessors in subacute pericarditis, evaluating patient recovery through combined clinical assessment and inflammatory marker tracking. While CRP remains a widely used biomarker to monitor inflammatory activity, modern guidelines emphasise correlating laboratory values with clinical symptoms. An isolated CRP fluctuation in an asymptomatic patient typically does not require acute re-imaging; conversely, a rising CRP accompanied by clinical red flags (such as worsening exertional dyspnoea, presyncope, orthopnoea, or jugular venous distension) indicates the need for rapid public hospital evaluation and urgent echocardiography.^{1,4,12} Clear triage pathways support effective escalation during acute deterioration, though clinical prioritisation alone cannot fully resolve public sector capacity limits for routine non-urgent surveillance. Scan throughput remains closely linked to sonographer workforce density.¹⁷ Addressing these capacity constraints requires combining clinical vigilance in primary care with targeted resource mobilisation.⁵

To expand workforce capacity, we propose targeted training initiatives and scope expansion for clinical staff. We highlight the value of increasing graduate intake through university programs, such as the University of Auckland's cardiac ultrasound pathway,¹⁹ alongside postgraduate training for clinical nurse specialists.^{20,21} Developing advanced scopes of practice for cardiac sonographers and nurse specialists provides a practical framework to manage ongoing diagnostic demand.^{22,23} We believe adapting established international protocols for primary care point-of-care ultrasound (POCUS) enables trained clinicians to perform preliminary rule-out assessments for pericardial effusions during routine primary care follow-up.^{20,21,24} This will help with decentralising preliminary screening to primary care offers a way to reduce secondary

hospital waitlists and support clinical decision-making for stable outpatients.^{22,23} It should be noted that training expansion requires parallel retention strategies for specialised staff. Attrition among cardiac sonographers and diagnostic technologists, influenced in part by trans-Tasman remuneration differences, contributes to workforce migration.^{5,25,26} We emphasise that supporting competitive remuneration, workload sustainability, and structured career development provides a practical pathway to improve public sector retention.^{25,26}

Finally, physical infrastructure investments complement workforce retention efforts to bring care closer to patients. We propose establishing dedicated community diagnostic centres and deploying mobile ultrasound units to rural health districts as practical options to expand regional access.²⁷ Decoupling routine elective surveillance from acute hospital pressures helps protect outpatient scanning capacity during periods of high emergency demand. Combining structured primary care triage, workforce development, retention initiatives, and regional diagnostic infrastructure offers a balanced strategy to improve access equity across Aotearoa New Zealand.⁵

Conclusion

Managing subacute pericarditis requires balancing evidence-based surveillance timelines with structural diagnostic access. In Aotearoa New Zealand, public health services maintain clinical safety by triaging low-risk effusions to guideline-compliant three- to six-month windows. High-volume effusions (>20 mm) carry higher baseline risks of delayed complications, creating a reliance on intermediate outpatient surveillance. Where private health insurance in metropolitan centres enables rapid diagnostic confirmation, individuals residing in rural areas or lacking private insurance face extended waitlists without an intermediate diagnostic buffer. Maintaining patient safety within public capacity limits relies on primary care vigilance to identify clinical red flags and escalate acute deterioration. Long-term diagnostic equity requires

an updated national workforce survey to quantify sonographer staffing across health districts, alongside targeted investments in nurse specialist training, salary retention, and decentralised community diagnostic hubs.

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Supplemental Material

Supplementary table 1: Longitudinal haematology and inflammatory markers.

Clinical Timeline	WBC	Neutrophils	CRP	Status
Reference range	4.0-11.0	1.9-7.5	<5	
Admission (day 1)	22.51 (H)	19.13 (H)	196	Clinical presentation
Admission (day 2)	15.55 (H)	12.52 (H)	256	Peak inflammation
Admission (day 4)	10.26	8.00 (H)	118	Clinical improvement
Admission (day 5)	9.56	6.89	96	Pre-discharge
Post-discharge (day 4)	10.3	7.4	4	Community recovery
Post-discharge (day 13)	9.3	6.9	69	Transient flare

Supplementary table 2: Rheumatological panel (admission day three).

Investigation	Result	Interpretation
ANA (titre)	160	Low titre positive
DFS70 IgG	Detected	Rarely associated with SARD
dsDNA (RIA)	< 7 IU/ml	Negative
ANCA	Negative	No vasculitis
EENA panel	Negative	Includes Ro, La, Sm, RNP, Jo-1
Scl-70	Detected*	Likely false positive

Supplementary table 3: Infectious disease screening (admission day three).

Investigation	Result	Status
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HIV Antig/Antib screen	Negative	Negative
CMV (IgG/IgM)	Negative	Negative
EBV (EBNA-1 IgG)	Positive	Remote infection
Hepatitis serology	Negative	HBV/HCV negative

Supplemental appendix 1: Comparative analysis of diagnostic imaging modalities in pericardial disease.

Diagnostic Feature / Parameter	Transthoracic Echocardiography (TTE)	Contrast-Enhanced Chest CT	Cardiac Magnetic Resonance (CMR)
Primary Clinical Role	First-line diagnostic imaging & hemodynamic assessment.	Secondary / acute cross-sectional evaluation.	Advanced tissue characterisation & problem solving.
Effusion Volume & Localisation	Excellent for echo-free space measurement & overall distribution.	Precise volumetric quantification & detection of loculations.	Precise volumetric resolution across complex anatomical windows.
Hemodynamic Assessment	Gold Standard: Dynamic Doppler inflow, chamber collapse, IVC collapsibility.	Static signs only (e.g., IVC distension, atrial reflux).	Dynamic velocity mapping & pericardial restraint assessment.
Tissue Characterisation	Limited structural detail beyond fluid echogenicity.	Evaluates pericardial thickening, calcification, & density.	Gold-standard: Late gadolinium enhancement (LGE) & T2-weighted oedema.

Radiation & Contrast Exposure	None	Ionising radiation: Present May require contrast.	Ionising radiation: None May require contrast.
Regional Accessibility in NZ	Limited by sonographer staffing density.	Availability in secondary and regional hospitals.	Highly restricted; centralised in major tertiary centres.
Cost & Examination Time	Low to moderate cost; rapid acquisition (~20–30 min).	Moderate cost; fast scan time (~5–10 min).	High cost; extended examination duration (~45–60 min).