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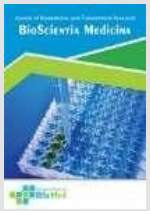

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Colchicine as a Strategic Therapeutic Alternative for Dengue-Associated Acute Pericarditis: Navigating the Hemorrhagic Risk

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A B S T R A C T

Background: Dengue infection remains a pervasive arboviral disease in tropical regions, manifesting with a spectrum of clinical severities ranging from undifferentiated fever to life-threatening shock and severe hemorrhage. While cardiac involvement such as myocarditis is documented, acute pericarditis is an underrecognized complication that poses a unique therapeutic dilemma. The standard first-line anti-inflammatory treatment for pericarditis, specifically non-steroidal anti-inflammatory drugs (NSAIDs), is relatively contraindicated in dengue due to the inherent coagulopathy and thrombocytopenia associated with the disease. **Case presentation:** We report a case of a 53-year-old male presenting with high-grade fever, retro-orbital pain, and severe chest discomfort characteristic of pleuritis. Physical examination revealed a distinct pericardial friction rub and relative bradycardia (56 bpm). Laboratory analysis confirmed dengue infection with significant leukopenia (1.7 x 10^3/µL), thrombocytopenia (49 x 10^3/µL), and elevated liver transaminases. Electrocardiography (ECG) demonstrated diffuse ST-segment elevation, while echocardiography showed preserved ejection fraction (67.7%) without pericardial effusion. Diagnosed with dengue-associated acute pericarditis, the patient was ineligible for NSAIDs due to the high risk of gastrointestinal hemorrhage. He was successfully managed with low-dose Colchicine (0.5 mg daily) alongside standard supportive care. Rapid resolution of chest pain and normalization of ECG findings were observed within 48 hours without hemorrhagic complications. **Conclusion:** This case underscores the utility of Colchicine as a safe, effective, and strategic alternative to NSAIDs for managing acute pericarditis in thrombocytopenic dengue patients. Early recognition of the pericarditis triad in dengue is crucial to prevent mismanagement, and Colchicine should be considered a cornerstone of therapy in this specific clinical context.

1. Introduction

Dengue infection, an acute febrile illness caused by the dengue virus (DENV), stands as one of the most formidable public health challenges in the modern era. Belonging to the Flaviviridae family, DENV has entrenched itself as a pervasive pathogen across the tropical and subtropical belts of the world, with nations like Indonesia serving as critical epicenters for transmission and hyperendemicity.[1] The epidemiological trajectory of dengue has been relentless; the World Health Organization (WHO) estimates that millions of infections occur annually, a figure that continues to climb due to unplanned urbanization, climate variability, and the expanding geographic footprint of the Aedes mosquito vectors.[2]

Clinically, dengue is a chameleon. Its presentation spans a vast spectrum, ranging from a benign, self-limiting undifferentiated fever to the catastrophic manifestations of severe dengue.[3] This severe form is historically defined by its hallmark systemic failures: profound plasma leakage resulting in shock, severe hemorrhage due to coagulopathy, and potentially fatal

organ impairment. For decades, the clinical gaze has been fixed almost exclusively on these hemodynamic crises—managing fluid shifts and preventing hypovolemic shock. However, as our understanding of dengue pathophysiology deepens, it has become increasingly clear that the virus is not merely a vascular pathogen but a systemic one, capable of inflicting damage on extra-dengue targets. Among these, the cardiovascular system has emerged as a critical, yet frequently underappreciated, determinant of morbidity and mortality.[4]

While the classic pathophysiological hallmarks of dengue involve endothelial permeability and thrombocytopenia, the concept of the dengue heart is gaining necessary recognition in clinical literature.[5] Cardiac involvement in dengue is no longer considered a medical curiosity but a statistically significant complication. Reports from hospitalized cohorts indicate that cardiac abnormalities occur with varying frequency, ranging from 11.4% to nearly 20% of cases. This wide variance likely reflects the diversity in diagnostic tools used—from simple electrocardiography to advanced biomarkers—but the signal is consistent: the heart is a target.

The spectrum of dengue-associated cardiac manifestations is broad. It includes rhythm disturbances such as relative bradycardia (a classic autonomic sign) and atrial fibrillation, as well as functional impairments like myocarditis. Myocarditis, inflammation of the heart muscle itself, has been the primary focus of cardiac dengue research. However, a less common but equally debilitating complication is acute pericarditis—inflammation of the fibro-serous sac surrounding the heart. Often overshadowed by the drama of plasma leakage or the severity of myocarditis, pericarditis in dengue presents a unique diagnostic challenge. It can mimic the chest discomfort of pulmonary congestion or the agony of ischemic heart disease, leading to confusion in the high-pressure environment of an emergency department.[6]

Understanding why the pericardium becomes inflamed during dengue requires navigating a complex, multifactorial pathogenesis. Current hypotheses suggest a double-hit mechanism. First, there is the potential for direct viral invasion. DENV is known to exhibit tropism for muscle cells, and while direct isolation from pericardial tissue is rare, the virus's presence in myocardial fibers suggests it can directly breach cardiac structures. Second, and perhaps more dominant, is the indirect, exuberant immune response—often termed the cytokine storm. The host's defense against DENV involves the massive release of pro-inflammatory mediators. Monocytes and macrophages, upon detecting the virus, secrete high levels of tumor necrosis factor-alpha (TNF-α) and Interleukins, specifically IL-1 and IL-6. These cytokines are the architects of dengue's systemic damage; they degrade the endothelial glycocalyx, causing plasma leak, but they also spill over into the pericardial space.[7] Recent insights have specifically implicated the NLRP3 inflammasome in this process. When activated by viral danger signals, the inflammasome triggers a cascade that releases Interleukin-1 beta (IL-1β), a potent pyrogen and inflammatory mediator that recruits neutrophils to the pericardium, resulting in the classic friction rub, chest pain, and electrocardiographic changes seen in pericarditis.

It is at the intersection of this cardiac inflammation and the systemic hematological defects of dengue that clinicians encounter a profound therapeutic gap. The management of acute pericarditis, when it occurs in isolation, is well-codified. International guidelines, such as those from the European Society of Cardiology (ESC), advocate unequivocally for high-dose non-steroidal anti-inflammatory drugs (NSAIDs)—typically aspirin or ibuprofen—as the first-line backbone of therapy. These drugs act by inhibiting cyclooxygenase (COX) enzymes, thereby reducing the production of prostaglandins that drive pain and inflammation. However, in the context of dengue, the administration of NSAIDs is fraught with peril. Dengue infection is inherently characterized by thrombocytopenia (a plummeting platelet count) and platelet dysfunction.[8] The virus suppresses bone marrow production of

megakaryocytes and increases peripheral destruction of platelets. Furthermore, the critical phase of dengue often involves a fragile coagulopathy where the risk of spontaneous bleeding—from the gums, nose, or gastrointestinal tract—is extraordinarily high. This creates a dangerous contradiction. The standard treatment for the heart (NSAIDs) is toxic to the blood. NSAIDs, particularly non-selective ones like aspirin, irreversibly inhibit COX-1, which is essential for the formation of Thromboxane A2, a key molecule for platelet aggregation. Giving high-dose aspirin to a patient with a platelet count of 40,000/μL is clinically akin to "adding fuel to the fire." It significantly exacerbates the risk of catastrophic gastrointestinal mucosal bleeding and can precipitate the transition from dengue fever to severe dengue hemorrhagic fever (DHF).[9]

Consequently, clinicians are left paralyzed. They cannot use the gold-standard anti-inflammatory drugs due to bleeding risks. Corticosteroids, another potential alternative, are often viewed with skepticism in acute viral infections due to fears of increasing viral replication or inducing metabolic derangements like hyperglycemia, which can complicate the fluid management of dengue shock. This leaves the patient in a state of unmanaged suffering: enduring the sharp, pleuritic pain of pericarditis while the medical team waits for the platelets to recover, a process that can take days. There is an urgent, unmet need for a therapeutic strategy that can suppress pericardial inflammation without disarming the coagulation cascade.[10]

This case report seeks to bridge this therapeutic gap by detailing the successful management of a 53-year-old male who presented with this precise dilemma: confirmed dengue infection complicated by acute pericarditis. In the absence of viable NSAID options, we turned to Colchicine. Colchicine is an ancient anti-inflammatory agent, traditionally used for gout, but recently repurposed for cardiovascular protection. Unlike NSAIDs, Colchicine does not inhibit arachidonic acid metabolism or platelet aggregation. Its mechanism is distinct and sophisticated: it binds to tubulin, inhibiting microtubule polymerization, which hampers the migration of neutrophils to sites of inflammation. More importantly, in the context of viral-induced inflammation, Colchicine has been shown to inhibit the assembly of the NLRP3 inflammasome. By blocking this specific pathway—the very pathway driven by dengue viremia—Colchicine acts as a precision tool. It dampens the IL-1β mediated cytokine storm affecting the heart without touching the clotting mechanism.

The primary aim of this study is to highlight the diagnostic and therapeutic challenges inherent in managing dengue-associated acute pericarditis. We aim to dismantle the one-size-fits-all approach to pericarditis and advocate for a tailored strategy in thrombocytopenic hosts. The novelty of this report lies in its proposal of Colchicine not merely as a second-line agent, but as the strategic first-line alternative in this specific clinical niche. While Colchicine is known for recurrent pericarditis, its use during the acute, viremic, and thrombocytopenic phase of dengue is rarely documented. By focusing on the shared pathophysiology of the NLRP3 inflammasome in both dengue and pericarditis, this paper provides a biological rationale for Colchicine's use. Ultimately, this report emphasizes the necessity of looking beyond the classic signs of plasma leakage to identify cardiac phenotypes in dengue, providing clinicians with a validated roadmap for managing inflammation when the body's coagulation system is compromised.

2. Case Presentation

Written informed consent was obtained from the patient for the publication of this case report and any accompanying clinical images. The patient was fully briefed on the educational intent of this report, and all personal identifiers have been anonymized to protect patient confidentiality in accordance with international ethical standards for medical publishing.

A 53-year-old Asian male presented to the Emergency Department of Payangan Hospital, a secondary care center in a dengue-endemic region of

Indonesia. He reported a chief complaint of an acute, debilitating febrile illness that had persisted for five days. The fever was described as high-grade and continuous, refractory to standard home antipyretics, and was accompanied by the classic breakbone constellation of dengue symptoms: severe frontal headache, intense retro-orbital pain characteristic of flavivirus ocular involvement, and generalized, disabling arthralgia and myalgia (Table 1a).

Significantly, the clinical picture evolved on day 4 of the illness with the emergence of a new and alarming symptom: chest discomfort. The patient described the pain as sharp, retrosternal, and distinctly pleuritic in nature, worsening with deep inspiration. Unlike the vague, oppressive heaviness typical of myocardial ischemia, this pain exhibited classic positional variability. It was notably exacerbated by supine positioning, particularly when the patient lay on his left lateral decubitus side, and was substantially alleviated when he adopted a sitting position and leaned forward—a maneuver that typically reduces friction between the inflamed pericardial layers. Review of systems was positive for profuse night sweats and persistent nausea, though he denied any episodes of active hemorrhage such as epistaxis, gingival bleeding, or melena. His past medical history was unremarkable, with no documented evidence of prior cardiovascular disease, autoimmune pathologies, or recent travel outside the local endemic zone.

Upon admission to the Emergency Department, the patient was alert and oriented (Glasgow Coma Scale E4V5M6). An assessment of vital signs revealed a patient who was hemodynamically stable yet exhibiting a distinct dissociation between his temperature and heart rate. His blood pressure was 110/70 mmHg, and his respiratory rate was 20 breaths per minute. However, despite a sub-febrile temperature of 37.6°C (axillary), he exhibited a relative bradycardia with a heart rate of 56 beats per minute. This finding is clinically significant, often suggestive of autonomic dysfunction or direct involvement of the sinoatrial node, commonly observed in arboviral infections. Oxygen saturation was preserved at 96% on room air. Head and neck examination revealed no evidence of scleral icterus or conjunctival pallor, though the tourniquet test was positive, signaling underlying capillary fragility characteristic of the critical phase of dengue. The cardiovascular examination provided the most pivotal diagnostic clue. Cardiac auscultation demonstrated a regular rhythm with distinct S1 and S2 heart sounds; however, a high-pitched, scratchy, and superficial sound was clearly audible at the left sternal border. This sound, consistent with a pericardial friction rub, persisted even during breath-holding maneuvers, a critical semiological test that definitively distinguished it from a pleural friction rub. Pulmonary auscultation remained clear bilaterally with no clinical evidence of pleural effusion, and the abdominal examination was benign, showing no palpable hepatosplenomegaly or ascites. Peripheral perfusion was maintained, with warm extremities, palpable pulses, and an absence of pedal edema.

Comprehensive laboratory evaluation confirmed a diagnosis of severe dengue with warning signs, characterized by marked hematological and biochemical derangements (Table 1b). The complete blood count revealed profound bone marrow suppression. The patient exhibited significant leukopenia (WBC 1.7 x $10^3/\mu L$) and severe thrombocytopenia with a platelet count of 49 x $10^3/\mu L$. Concurrently, there was evidence of hemoconcentration—a surrogate marker for plasma leakage—indicated by a hemoglobin level of 16.2 g/dL and a hematocrit of 46.6%. Hepatic involvement was evident, with a Grade 2 elevation in liver transaminases. Aspartate Aminotransferase (SGOT) was elevated to 156 U/L, and Alanine Aminotransferase (SGPT) to 120 U/L. Renal function and electrolyte panels remained within normal limits. The dengue profile confirmed an acute secondary infection. The Dengue NS1 Antigen was positive, and both IgM and IgG anti-dengue antibodies were reactive. The systemic inflammatory burden was high, with a C-reactive protein (CRP) of 45 mg/L and an

erythrocyte sedimentation rate (ESR) of 60 mm/hr. High-sensitivity Troponin I was measured at 0.04 ng/mL. While this level ruled out massive myocardial necrosis typical of infarction, it represented a borderline elevation suggestive of minor myopericardial irritation.

To further investigate the chest pain and friction rub, advanced cardiac testing was performed. The 12-lead ECG provided the electrical confirmation of pericardial inflammation. It demonstrated sinus bradycardia at 55 bpm alongside diffuse, concave-upward ST-segment elevations in leads I, aVL, and V2 through V6. Notably, PR-segment depression was observed in Lead II, with reciprocal ST depression in lead aVR. These diffuse changes, lacking the reciprocal depressions typical of ischemic coronary distinct territories (except in aVR), met the standard electrical criteria for acute pericarditis. Bedside echocardiography was utilized to assess hemodynamic function and rule out tamponade. The imaging revealed a normal left ventricular (LV) size with preserved global systolic function (Ejection Fraction [EF] = 67.7%). Right Ventricular function was also normal (TAPSE 2.25 cm). Crucially, the scan showed no pericardial effusion, classifying the presentation as dry pericarditis. This finding was instrumental in management, as it eliminated the immediate need for pericardiocentesis. Synthesizing the clinical, laboratory, and imaging data, a multifaceted diagnosis was established: (i) Dengue Infection (Day 5) with Warning Signs: Supported by the presence of persistent vomiting and laboratory evidence of hemoconcentration; (ii) Acute Pericarditis (Viral Etiology): The patient fulfilled at least two of the four diagnostic criteria mandated by international guidelines: typical pleuritic chest pain, a pathognomonic pericardial friction rub, and specific ECG changes; and (iii) Sinus Bradycardia: Attributed to transient autonomic dysfunction or intrinsic nodal involvement secondary to the viral infection.

TABLE 1A. SUMMARY OF CLINICAL FINDINGS ON ADMISSION (CLINICAL PRESENTATION)

1. Demographics & Clinical History	
Patient Profile	53-year-old Asian Male
Chief Complaint	Acute Febrile Illness (Day 5) with New Onset Chest Discomfort (Day 4)
Pain Characteristics	Sharp, retrosternal, **pleuritic**; Positional (exacerbated by supine/left lateral decubitus, alleviated by sitting forward)
Associated Symptoms	Severe frontal headache, retro-orbital pain, arthralgia, myalgia, night sweats, nausea
2. Vital Signs & Hemodynamics	
Blood Pressure	110/70 mmHg
Heart Rate	**56 bpm (Relative Bradycardia)**
Temperature	37.6°C (Axillary)
Respiratory Rate / SpO2	20 breaths/min | 96% on Room Air
3. Physical Examination Findings	
Cardiovascular	Regular rhythm, distinct **Pericardial Friction Rub** audible at left sternal border (persisted during breath-holding)
Dermatological	**Positive Tourniquet Test** (indicating capillary fragility)
General/Other	Alert (GCS E4V5M6); No scleral icterus; Lungs clear bilaterally (no effusion); Abdomen unremarkable

Abbreviations (Table 1a): *bpm: beats per minute; GCS: Glasgow Coma Scale; SpO2: Peripheral Oxygen Saturation.*

TABLE 1B. SUMMARY OF CLINICAL FINDINGS ON ADMISSION (DIAGNOSTIC INVESTIGATIONS)

4. Laboratory Investigations	
Hematology	Leukocytes: **1.7 x 10^3/µL** (Leukopenia) Platelets: **49 x 10^3/µL** (Severe Thrombocytopenia) Hemoglobin: 16.2 g/dL (Hemoconcentration)
Biochemistry (Liver)	SGOT (AST): **156 U/L** SGPT (ALT): **120 U/L** (Grade 2 Elevation)
Serology	**Dengue NS1 (+), IgM (+), IgG (+)**
Inflammatory Markers	CRP: 45 mg/L | ESR: 60 mm/hr
Cardiac Biomarkers	Hs-Troponin I: 0.04 ng/mL (Borderline elevation)
5. Cardiac Imaging & Diagnostics	
Electrocardiogram (ECG)	Sinus bradycardia (55 bpm); **Diffuse concave-upward ST-segment elevation** (I, aVL, V2-V6); PR-segment depression (Lead II); Reciprocal ST depression (aVR)
Echocardiography (TTE)	LVEF 67.7% (Preserved function); Normal RV function (TAPSE 2.25 cm); **No pericardial effusion** ("Dry" Pericarditis)

Abbreviations (Table 1b): *WBC: White Blood Cell; SGOT: Serum Glutamic Oxaloacetic Transaminase; SGPT: Serum Glutamic Pyruvic Transaminase; CRP: C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; LVEF: Left Ventricular Ejection Fraction; TAPSE: Tricuspid Annular Plane Systolic Excursion.*

The therapeutic management of this patient represented a complex clinical exercise, necessitating the navigation of two competing physiological imperatives. The primary challenge lay in a distinct therapeutic dichotomy: the management of severe dengue requires aggressive fluid resuscitation to counteract plasma leakage and hemoconcentration, whereas the management of acute pericarditis traditionally demands fluid restriction to prevent pericardial strain and the administration of anti-inflammatory agents that are inherently hazardous to the coagulation system. Consequently, the treatment strategy was bifurcated into a meticulous supportive regimen for the systemic viral infection and a targeted, hemorrhage-sparing anti-inflammatory protocol for the cardiac complications.

The cornerstone of dengue management involved judicious intravenous fluid resuscitation. Given the patient's hemoconcentration (Hematocrit 46.6%) and clinical evidence of dehydration, Ringer's Lactate was selected as the crystalloid of choice. The infusion was initiated at a rate of 30 drops per minute, a calculation designed to maintain intravascular volume without precipitating volume overload—a complication that could prove disastrous in a patient with compromised diastolic filling due to pericardial inflammation. The fluid rate was not static; it was dynamically titrated based on serial hematocrit monitoring, ensuring that the rate was tapered as plasma leakage resolved. Concurrent with fluid management, a robust prophylactic strategy was employed to protect the gastrointestinal mucosa. In the setting of severe thrombocytopenia (Platelets 49,000/µL), the gastric lining is exquisitely vulnerable to stress ulceration and spontaneous bleeding. To mitigate this risk, the patient was started on Omeprazole 40 mg intravenously every 12 hours, supplemented by Sucralfate syrup to provide a physical cytoprotective barrier. Furthermore, recognizing the hepatic involvement (Grade 2 transaminitis), a

hepatoprotective agent, Heparmin, was administered to support liver function recovery and prevent progression to acute liver failure.

The management of the patient's pericardial inflammation required a significant deviation from standard cardiological guidelines due to the hematological profile of dengue. Standard protocols for acute pericarditis advocate for high-dose aspirin or ibuprofen as first-line therapy. However, in this clinical context, these agents were deemed strictly contraindicated. With a platelet count critically low at 49,000/µL, the administration of non-steroidal anti-inflammatory drugs (NSAIDs)—which irreversibly inhibit Cyclooxygenase-1 (COX-1) and permanently disable platelet aggregation—would have exponentially increased the risk of gastrointestinal hemorrhage or progression to Dengue Hemorrhagic Fever. The decision to withhold NSAIDs was absolute, prioritizing hemostatic integrity over conventional pain management. To bridge the therapeutic gap, Colchicine was selected as the primary disease-modifying agent. It was initiated at a dose of 0.5 mg orally once daily. This specific low-dose regimen was chosen with strategic intent. Pharmacologically, it provided sufficient blockade of tubulin polymerization and NLRP3 inflammasome assembly to dampen pericardial inflammation. Clinically, this conservative dosing minimized the risk of gastrointestinal toxicity, specifically diarrhea—a known side effect of Colchicine that would be detrimental in a patient already battling the fluid imbalances of dengue. For symptomatic relief of fever and residual pain, Paracetamol was administered intravenously at a dose of 1 gram every 8 hours. This provided safe analgesia without interfering with platelet function. Isosorbide Dinitrate (ISDN) was kept as a rescue medication for refractory chest pain but was ultimately not required. Additionally, given the patient's admission bradycardia (56 bpm), atropine was prepared at the bedside for immediate administration should the heart rate fall below 50 bpm, ensuring readiness for potential autonomic collapse.

The clinical response to this tailored therapeutic strategy was rapid and validated the decision to utilize Colchicine as a monotherapy for inflammation. The impact of Colchicine on the inflammatory process was evident within the first 48 hours of therapy. The patient, who initially presented with debilitating, pleuritic retrosternal pain, reported a dramatic improvement in symptoms. The Visual Analog Scale (VAS) for pain plummeted from a severe 7/10 on admission to a mild 2/10, allowing the patient to lie supine without significant distress. This rapid analgesic response is characteristic of Colchicine's effect on crystal-induced and cytokine-mediated inflammation. Concurrently, the systemic signs of dengue infection began to abate. The high-grade fever, which had persisted for nearly a week, finally subsided on Day 7 of the illness, marking the transition from the critical phase to the recovery phase. The patient's hemodynamic profile also stabilized; the relative bradycardia observed on admission resolved, with the heart rate normalizing to a physiological range of 60–70 beats per minute by Day 8. This normalization likely reflected both the resolution of autonomic dysfunction and the abatement of pericardial irritation.

Serial laboratory monitoring demonstrated a robust recovery of the hematopoietic system, confirming that the therapeutic interventions did not impede marrow recovery. The platelet count showed a consistent upward trajectory, rising from the critical nadir of 49,000/µL to 85,000/µL, and finally reaching a safe level of 120,000/µL prior to discharge. This platelet recovery was achieved without any episodes of clinical bleeding, vindicating the strict avoidance of NSAIDs. Electrocardiographically, the dry pericarditis showed complete resolution. A follow-up 12-lead ECG performed on Day 10 (discharge day) revealed the disappearance of the diffuse ST-segment elevations and PR-segment depressions that characterized the admission tracing. The return of the ST segments to the isoelectric baseline signaled the cessation of active myocardial and pericardial injury.

The patient was discharged in a stable condition on Day 10. To consolidate the treatment success and prevent the complication of recurrent pericarditis—which occurs in 15-30% of cases treated with short courses—the patient was prescribed to continue oral Colchicine for a total duration of two weeks. This extended course serves as a prophylactic measure, ensuring complete suppression of any residual inflammasome activity within the pericardial space. The successful outcome of this case underscores the utility of Colchicine as a safe, effective, and sophisticated alternative for managing the cardiac complications of dengue in the thrombocytopenic host.

TABLE 2. DIAGNOSIS, TREATMENT, FOLLOW UP, AND OUTCOME

1. Final Diagnosis	
Primary Diagnosis	1. **Dengue Infection** (Secondary, Day 5) with Warning Signs (Vomiting, Hemoconcentration). 2. **Acute Pericarditis** (Viral etiology; Dry type). 3. **Sinus Bradycardia** (Autonomic dysfunction).
2. Therapeutic Management Strategy	
Dengue Supportive Care	• **Fluid Resuscitation:** Ringer's Lactate (IV), initiated at 30 drops/min, titrated strictly to hematocrit levels. • **Gastric Protection:** **Omeprazole** 40 mg IV q12h + Sucralfate syrup (to prevent stress ulcers in thrombocytopenia). • **Hepatoprotection:** Heparmin administered for Grade 2 transaminitis.
Pericarditis Protocol *(The Hemorrhage-Sparing Strategy)*	• **CONTRAINDICATION:** **Aspirin & Ibuprofen strictly avoided** due to platelet count of 49,000/µL. • **Disease-Modifying Agent:** **Colchicine** 0.5 mg PO daily. *(Rationale: Low dose selected to minimize GI toxicity/diarrhea while targeting NLRP3 inflammasome).* • **Analgesia:** Paracetamol 1g IV q8h. • **Rescue/Standby:** Atropine (for HR < 50 bpm); ISDN (for refractory pain - not required).
3. Clinical Course & Timeline	
Response to Therapy	**48 HOURS POST-COLCHICINE** Significant pain reduction; Visual Analog Scale (VAS) dropped from **7/10 to 2/10**. **DAY 7 OF ILLNESS** Complete defervescence (Fever subsided). **DAY 8 OF ILLNESS** Heart rate normalized to physiological range (60-70 bpm). **HEMATOLOGICAL TREND** Platelets recovered: **49,000** → 85,000 → **120,000/µL**.
4. Outcome & Discharge Plan	
Discharge Status (Day 10)	• **Hemodynamics:** Stable, normal sinus rhythm. • **ECG Findings:** **Resolution of ST-segment elevations**; return to isoelectric baseline. • **Bleeding:** No hemorrhagic complications observed throughout admission.
Home Medication	**Oral Colchicine** to complete a total 2-week course (Prophylaxis against recurrent pericarditis).

Abbreviations: IV: Intravenous; PO: Per Os (Orally); q12h: Every 12 hours; VAS: Visual Analog Scale; HR: Heart Rate; ISDN: Isosorbide Dinitrate; GI: Gastrointestinal.

3. Discussion

This case report serves as a clinical paradigm for a rare but increasingly recognized intersection between two major medical disciplines: infectious disease and acute cardiology. The presentation of acute pericarditis complicating the critical phase of dengue infection exemplifies a specific extra-dengue manifestation that challenges traditional management protocols. Historically, the clinical lens on severe dengue has been myopically focused on hemodynamic stability, specifically the management of plasma leakage and the prevention of hypovolemic shock. While these remain the primary determinants of mortality, this case highlights the necessity of expanding that vigilance to include the cardiovascular system. The successful resolution of symptoms in this thrombocytopenic patient using Colchicine provides a compelling, evidence-based argument for its utility as a standard-of-care alternative when the conventional pharmacological arsenal (NSAIDs) is rendered dangerous by the disease process itself.[11]

The mechanisms by which the dengue virus (DENV) insults the pericardium are not merely collateral damage but represent a complex, likely biphasic pathophysiological process.[12] To understand the rationale for our treatment, one must first dissect the two primary theories of cardiac injury in dengue: direct viral cytotoxicity and immune-mediated damage. The first phase involves the virus itself. DENV is an arbovirus with a known capability to invade muscle cells. While the virus's specific tropism for pericardial mesothelial cells is less extensively mapped than its affinity for hepatocytes or leukocytes, DENV has been isolated from myocardial tissue in autopsy studies.[13] This suggests that the virus can breach the cardiac barriers, leading to direct cellular injury. In this context, the pericarditis is a primary viral infection of the pericardial sac, necessitating a host response that clears the pathogen but leaves inflammation in its wake. The second, and perhaps more dominant mechanism, is the hyper-inflammatory response, often termed the cytokine storm. Upon infection, the host's innate immune system—specifically monocytes and macrophages—mounts an aggressive defense, releasing high titers of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), Interleukin-18 (IL-18), and Interleukin-1 beta (IL-1β).[14]

Current research has drawn a specific and critical link between DENV infection and the NLRP3 inflammasome. The inflammasome is an intracellular multiprotein complex that acts as a sensor for danger signals or damage-associated molecular patterns (DAMPs) triggered by the virus.[15] When activated, the NLRP3 inflammasome catalyzes the cleavage of pro-IL-1β into its active form. This cytokine is a potent mediator of vascular permeability—contributing to the plasma leakage seen in severe dengue—but it is also a powerful chemotactic agent. It recruits neutrophils to the pericardial space, where their activation leads to local inflammation, fibrin deposition (audible clinically as a friction rub), and the stimulation of pain receptors.[16] Understanding this pathway is vital, as it identifies the specific molecular target for our therapeutic intervention.

In the high-pressure environment of a tropical emergency department, a middle-aged male presenting with chest pain and ST-segment elevation on ECG triggers an almost reflex activation of acute coronary syndrome (ACS) protocols. This case illustrates the ischemic mimic nature of dengue pericarditis and the danger of misdiagnosis. Administering antiplatelets or anticoagulants (standard for ACS) to a dengue patient could be fatal. Several nuanced features helped steer the diagnosis away from ischemia. First, the ECG morphology demonstrated diffuse, concave-upward ST elevations, contrasting with the localized, convex elevations typically seen in myocardial infarction. Second, the presence of a friction rub was a high-specificity sign for pericardial inflammation. Third, the patient exhibited significant bradycardia (56 bpm), a known autonomic complication of dengue infection resulting from increased vagal tone or direct sinus node involvement. Finally, distinguishing this presentation from viral myocarditis was crucial for prognosis. The

patient's preserved left ventricular ejection fraction (67.7%) and the absence of overt heart failure symptoms allowed us to isolate the pathology to the pericardium, ruling out fulminant myocardial necrosis.[17]

The crux of this case report lies in the management of the therapeutic gap. The European Society of Cardiology (ESC) guidelines are unequivocal: the first-line therapy for acute pericarditis is high-dose aspirin (750-1000 mg) or ibuprofen (600 mg). These agents work by inhibiting Cyclooxygenase-1 (COX-1).[18] However, in the physiological context of dengue, COX-1 inhibition is catastrophic. It permanently disables platelet aggregation in a host already suffering from severe thrombocytopenia (platelet count 49,000/µL). Using NSAIDs in this scenario would likely convert a stable dengue case into dengue hemorrhagic fever (DHF), precipitating severe gastrointestinal mucosal bleeding. This left us with a distinct clinical paradox: how to suppress life-altering cardiac inflammation without disarming the body's coagulation mechanism. Colchicine emerged as the strategic solution because its mechanism of action is distinct from the arachidonic acid pathway.[19] Colchicine binds to tubulin, inhibiting microtubule polymerization. This prevents the migration of neutrophils toward the inflamed pericardium, effectively halting the cellular component of inflammation. Crucially, Colchicine inhibits the assembly of the NLRP3 inflammasome. By blocking this specific complex, Colchicine prevents the release of IL-1β and IL-18. Since the NLRP3 inflammasome is implicated in both the systemic pathogenesis of Dengue and the local pathogenesis of pericarditis, Colchicine acts as a form of precision medicine, targeting the exact cytokine pathway driving the disease. Most importantly, Colchicine has no impact on platelet function or coagulation factors. It allows the clinician to treat the heart without endangering the blood.[20] In our case, a conservative dose of 0.5 mg daily was sufficient to induce clinical remission. This low dose also mitigated the risk of diarrhea—a common side effect of Colchicine that would have been detrimental in a dengue patient requiring meticulous fluid management. The rapid resolution of symptoms aligns with recent data suggesting Colchicine is highly effective in reducing symptom duration and preventing recurrence in viral pericarditis (Figure 1).

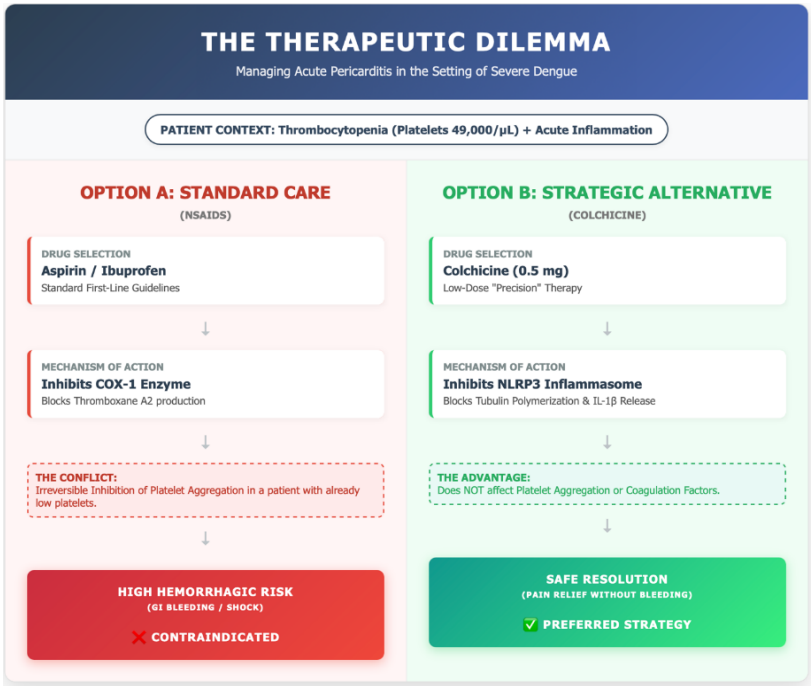

Figure 1. Managing acute pericarditis in dengue.

We acknowledge the limitations inherent in this report. No pericardial fluid was available for viral isolation as the pericarditis was dry (without effusion), and cardiac MRI (CMR) was not performed to definitively rule out sub-clinical myocardial edema. However, the diagnosis was robustly established using validated clinical criteria. Future research should prioritize large-scale observational studies to quantify the true incidence of dry pericarditis in dengue cohorts. Furthermore, randomized controlled trials are needed to establish the optimal dosing of Colchicine in thrombocytopenic populations to standardize this strategy.

4. Conclusion

Dengue infection must be reconceptualized not merely as a hemorrhagic fever, but as a systemic disease with the potential for significant multi-organ involvement, including the heart. This case report challenges the plasma-centric view of dengue management, urging clinicians to maintain a high index of suspicion for acute pericarditis in any patient presenting with chest pain, bradycardia, or ECG abnormalities, even in the absence of a pericardial effusion. The most significant contribution of this report is the validation of Colchicine (0.5 mg/day) as a safe, effective, and physiologically sound therapeutic strategy for dengue-associated pericarditis. By utilizing Colchicine, we successfully navigated the hemorrhagic risk, bypassing the contraindications associated with NSAIDs. This strategy effectively dampened the inflammasome-mediated pericardial inflammation while protecting the vulnerable, thrombocytopenic host. For clinicians practicing in endemic regions, this report provides a clear directive: include pericarditis in the differential diagnosis of chest pain in dengue, avoid NSAIDs rigorously in the presence of thrombocytopenia, and consider Colchicine as the preferred first-line agent. This approach represents a shift towards mechanism-based therapeutics, ensuring that the treatment of one complication does not exacerbate the underlying lethality of the viral infection.

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