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Divergent clinical phenotypes of post-inflammatory interventricular septal injury in young military personnel

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A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of the article

Post-inflammatory myocardial injury in young, physically active populations like military personnel presents significant diagnostic and prognostic challenges. Inflammation triggered by infections, systemic stress, or thoracic trauma may escape detection by standard electrocardiography (ECG) and biomarkers. When localized within the interventricular septum (IVS), it may impair both conduction and contractility, increasing the risk of adverse cardiac events.

Aim. To demonstrate the clinical heterogeneity of post-inflammatory interventricular septal injury and highlight the role of cardiac magnetic resonance (CMR) imaging in diagnosis and risk stratification.

Materials and methods. A comparative analysis of myocardial changes was conducted in two young male servicemembers who developed significant cardiac manifestations following presumed inflammatory triggers (infection and trauma). Clinical evaluation included ECG, ambulatory rhythm monitoring, serum biomarkers, and CMR.

Results. Both patients demonstrated preserved or only minimally elevated biomarkers, underscoring the limitations of laboratory screening alone. Case 1: a 40-year-old patient developed presyncope after pneumonia and blast-related trauma. Monitoring revealed high-grade atrioventricular block (Mobitz Type II). CMR identified focal intramural late gadolinium enhancement within the basal IVS, necessitating permanent pacemaker implantation. Case 2: a 39-year-old patient presented with acute dyspnea and fatigue after a viral infection. Despite a normal ECG, CMR revealed segmental fibrosis of the IVS with a moderately reduced left ventricular ejection fraction (45 %) without conduction disturbances.

Conclusions. Post-inflammatory septal injury in young military personnel manifests across a broad clinical spectrum, ranging from malignant conduction failure to early-onset heart failure. These cases underscore the pivotal diagnostic value of CMR in detecting structural changes that remain occult on echocardiography. Early comprehensive evaluation and risk stratification are essential to prevent sudden cardiac death and preserve operational readiness.

Keywords: myocarditis, ventricular septum, atrioventricular block, cardiac magnetic resonance imaging (cardiac MRI), heart failure, sudden cardiac death.

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Різні клінічні фенотипи постзапального пошкодження міжшлуночкової перетинки у молодих військовослужбовців

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Постзапальне ураження міокарда у молодих людей, які ведуть активний спосіб життя, зокрема у військовослужбовців, часто становить складну діагностичну та прогностичну проблему. Запальне ураження міокарда, спричинене інфекціями, системним стресом або травмою грудної клітки, може не бути виявлене за даними стандартної електрокардіографії (ЕКГ) та шляхом визначення біомаркерів пошкодження міокарда. За локалізацією у міжшлуночковій перетинці воно може порушувати і провідність, і скоротливість, підвищуючи ризик несприятливих кардіальних подій.

Мета роботи – показати клінічну гетерогенність постзапального ураження міжшлуночкової перетинки та висвітлити роль магнітно-резонансної томографії серця (МРТ серця) у діагностиці та стратифікації ризиків.

Матеріали і методи. Здійснили порівняльний аналіз міокардіальних змін у двох молодих чоловіків-військовослужбовців, у яких зафіксовано значущі кардіальні прояви після впливу ймовірних запальних тригерів (інфекція та травма). Клінічне обстеження передбачало виконання ЕКГ, амбулаторного моніторування ритму, визначення біомаркерів пошкодження міокарда, МРТ серця.

Результати. В обох пацієнтів встановлено нормальні або лише незначно підвищені рівні біомаркерів, що підтверджує обмеженість лише лабораторного скринінгу. Випадок 1 описує 40-річного пацієнта, у якого виникло пресинкопе на фоні синусової брадикардії після пневмонії та мінно-вибухової травми. За даними амбулаторного моніторингу визначено атріовентрикулярну блокаду високого ступеня (Мобітц II) та тривалі вентрикулярні паузи (>2200 мс). Під час МРТ серця виявлено осередкове інтрамуральне пізнє накопичення гадолінію у базальному відділі перетинки; це зумовило необхідність імплантації постійного кардіостимулятора. Випадок 2 стосується 39-річного пацієнта з симптомами серцевої недостатності, що виникли гостро (задишка та втома) невдовзі після вірусної інфекції. Незважаючи на те, що за даними ЕКГ зміни не виявлено, під час МРТ серця визначено сегментарний фіброз міжшлуночкової перетинки та помірно знижену фракцію викиду лівого шлуночка (45 %) без порушень провідності.

Висновки. Постзапальне ураження перетинки має широкий клінічний спектр – від злоякісних порушень провідності до ранньої серцевої недостатності. Описані випадки підтверджують критичну цінність МРТ серця для виявлення структурних

Ключові слова: міокардит, міжшлуночкова перетинка, атріовентрикулярна блокада, магнітно-резонансна томографія серця (МРТ серця), серцева недостатність, раптова серцева смерть.

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змін, що залишаються непомітними при ехокардіографії. Раннє комплексне обстеження та стратифікація ризику необхідні для запобігання раптовій серцевій смерті та забезпечення оперативної готовності особового складу.

Atrioventricular (AV) conduction disturbances in young and middle-aged adults represent a profound diagnostic and prognostic challenge, particularly when they occur in the absence of known structural heart disease. While atherosclerotic coronary artery disease and age-related degenerative changes are the primary drivers of conduction failure in older populations, non-ischemic myocardial injury, specifically of inflammatory or post-infectious origin, has emerged as a critical and often underrecognized etiology in younger, physically active individuals [1,2,3].

Viral infections are well-documented triggers of myocardial inflammation, initiating a spectrum of cardiac involvement that ranges from transient, asymptomatic edema to permanent, focal fibrosis [4,5,6]. A critical clinical caveat is that post-inflammatory myocardial injury may remain subclinical. Patients often present with unremarkable surface electrocardiography (ECG), preserved or only marginally reduced systolic function, and negative cardiac biomarkers, such as Troponin I [1,3,4]. Despite this clinically "silent" profile, the underlying structural substrate can predispose patients to malignant ventricular arrhythmias, sudden cardiac death (SCD), or progressive ventricular dysfunction [7,8,9].

Young, physically active populations, most notably military personnel, occupy a unique risk category [1,2,9]. The requirement to maintain operational duties and high-intensity physical exertion during the acute or subacute phases of a viral illness can exacerbate myocardial inflammation [3,10]. This "double hit" of inflammatory stress and hemodynamic load may convert focal injury into high-risk electrical instability. When localized to the interventricular septum (IVS), where the His–Purkinje system resides, even minor scarring can disrupt infra-Hisian conduction, manifesting as high-grade AV block (Mobitz Type II) or intermittent complete heart block [5,10,11].

Conventional diagnostic tools, including transthoracic echocardiography and routine laboratory testing, frequently lack the sensitivity to detect focal, non-ischemic lesions [12,13,14]. Consequently, cardiac magnetic resonance (CMR) imaging has emerged as the gold-standard reference for non-invasive tissue characterization [3,10]. Through multiparametric techniques, specifically late gadolinium enhancement (LGE) and T1/T2 mapping, CMR can visualize intramural septal fibrosis that remains occult on other modalities [15,16,17,18,19,20].

Despite the increasing use of CMR, the clinical spectrum of post-inflammatory injury remains remarkably heterogeneous [3,16]. Comparative data regarding how similar septal injury can produce vastly different clinical phenotypes, ranging from life-threatening bradyarrhythmias to isolated heart failure, remain limited [11].

Aim

To demonstrate the clinical heterogeneity of post-inflammatory interventricular septal injury and highlight the role of cardiac magnetic resonance imaging in diagnosis and risk stratification.

Materials and methods

The cases were conducted at the Department of Propaedeutics of Internal Medicine, Radiation Diagnostics and Radiation Therapy, Zaporizhzhia State Medical and Pharmaceutical University, in the Cardiology Department of the Municipal Non-Profit Enterprise "City Hospital of Emergency and First Aid", Zaporizhzhia, Ukraine, in accordance with the Declaration of Helsinki and Good Clinical Practice standards. The study protocol was approved by the Bioethics Commission of Zaporizhzhia State Medical and Pharmaceutical University (Protocol No. 6 dated 16.04.2026). All participants provided written informed consent prior to inclusion.

This report utilizes a comparative clinical case analysis of two active-duty male military personnel presenting with new-onset cardiovascular symptoms. To ensure a comprehensive diagnostic profile, both patients underwent an escalated evaluation protocol consisting of a detailed retrospective analysis of infectious, inflammatory, and environmental exposures, with specific focus on the temporal relationship between viral illness or trauma and the onset of cardiac symptoms.

Serial 12-lead ECG and 24-hour ambulatory Holter monitoring were utilized to quantify arrhythmic burden and characterize conduction stability. We prioritized the detection of infra-Hisian markers such as Mobitz Type II patterns and prolonged ventricular pauses [7,11].

Serum cardiac Troponin I and inflammatory markers were assessed at multiple time points to detect acute myocardial necrosis or ongoing systemic inflammation [1,4].

Transthoracic echocardiography was performed using an Esaote MyLab Eight system (Italy) with M-mode, B-mode, Doppler imaging, tissue Doppler imaging in accordance with the current clinical guidelines [12,13,14]. Clinical reporting was standardized according to the ASE guidelines to ensure accuracy in describing myocardial echogenicity and wall motion abnormalities [21,22].

Both patients were scanned using a 1.5-Tesla MRI system following a standardized multiparametric protocol designed to detect non-ischemic myocardial injury. The protocol adhered to the updated Lake Louise Criteria for myocardial inflammation [1,5,15].

Standard segmented steady-state free precession cine sequences were acquired in long-axis (2-chamber, 3-chamber, and 4-chamber) and short-axis views [13,15] to calculate left ventricular end-diastolic and end-systolic volumes, left ventricular ejection fraction (LVEF), regional wall motion abnormalities, with specific attention to the IVS.

To enhance the detection of subclinical myocardial injury, comprehensive tissue characterization was performed using advanced parametric mapping techniques in conjunction with conventional CMR sequences [17,18].

Myocardial edema was assessed using T2-weighted imaging and T2 mapping, which are sensitive markers of acute inflammatory activity [17].

LGE imaging was acquired 10–15 minutes following intravenous administration of a gadolinium-based contrast agent (0.1–0.2 mmol/kg) using inversion-recovery sequenc-

Fig. 1. Episodes of atrioventricular block documented on 24-hour ambulatory Holter monitoring.

es. LGE was used to assess the presence, distribution, and extent of myocardial replacement fibrosis [23,24].

A non-ischemic pattern of myocardial injury was defined by mid-wall or subepicardial LGE distribution with sparing of the subendocardial layer [19]. Given the clinical relevance of conduction system involvement, particular attention was paid to the IVS. Septal LGE, especially when localized to the basal or mid-septal segments, was interpreted in the context of the known anatomical course of the His bundle and proximal bundle branches [5,10,19].

The cases were analyzed side-by-side to contrast the "arrhythmic phenotype" (Case 1) against the "myopathic phenotype" (Case 2). Therapeutic decisions ranging from guideline-directed medical therapy (GDMT) to the surgical implantation of a permanent pacemaker were based on the integration of CMR-detected structural substrates and the clinical risk of SCD [1,2,22].

Results

The series comprise two male active-duty military personnel, aged 40 and 39 years, with previously unremarkable cardiovascular histories. Prior to the index events, both individuals demonstrated high levels of physical fitness and operational readiness, with no documented coronary artery disease, congenital cardiac anomalies, or systemic comorbidities. In both cases, onset of clinical symptoms was temporally associated with specific inflammatory triggers encountered during the performance of military duties.

Case 1: the arrhythmic phenotype. A 40-year-old male presented for evaluation following a complex clinical course initiated by blast-related thoracic trauma (July 2025), subsequently complicated by post-traumatic pneumonia. Over a three-week period, the patient experienced escalating symptoms, including recurrent presyncope, orthostatic dizziness, exertional headaches, and a subjective sensation of "cardiac pauses". Upon admission, the patient was hemodynamically stable but presented with significant resting bradycardia (heart rate 44–46 bpm).

Initial 12-lead ECG demonstrated sinus bradycardia and a rightward axis deviation with captured episodes of intermittent second-degree AV block, specifically Mobitz Type II.

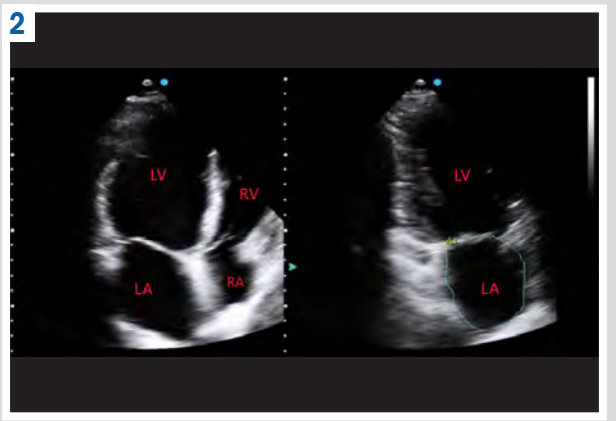
To further characterize the arrhythmic burden, 24-hour ambulatory Holter monitoring was performed, which confirmed high-risk conduction system instability *(Fig. 1)*.

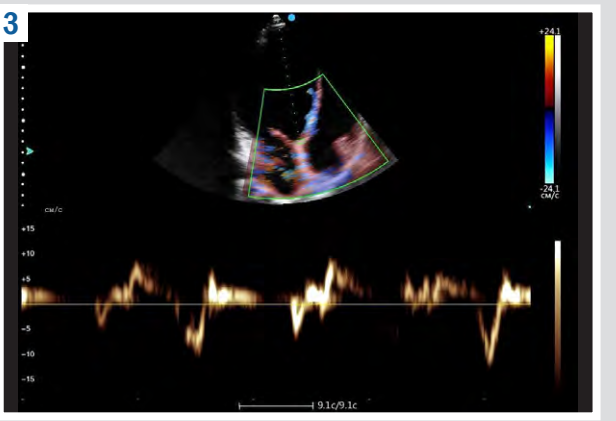
Holter monitoring revealed a heart rate ranging from 30 bpm to 107 bpm, 36 distinct episodes of ventricular pauses exceeding 2000 ms (with the longest R–R interval reaching 2289 ms), and 119 isolated polymorphic premature ventricular complexes.

Laboratory analysis demonstrated minimal Troponin I elevation (0.1 ng/mL), suggesting low-grade ongoing myocardial injury.

Transthoracic echocardiography revealed significant adverse remodeling, characterized by dilation of both the left atrium (left atrial volume index – 35 mL/m^2) and the left ventricle (LV) (end-diastolic volume index – 93.8 mL/m^2). Valvular assessment identified mild-to-moderate functional mitral regurgitation and mild tricuspid regurgitation. The overall LV geometry remained within normal limits (LV mass index – 113 g/m^2, relative wall thickness – 0.29). The IVS exhibited heterogeneous echogenicity, highly suggestive of post-fibrotic structural changes. Diastolic assessment indicated Grade I dysfunction with elevated filling pressures. Global systolic function was mildly reduced (LVEF 50 %), and a small pericardial effusion was visualized *(Fig. 2, 3)*.

CMR imaging provided the definitive diagnosis, demonstrating intramural non-ischemic late gadolinium enhancement within the interventricular septum, consistent with post-inflammatory myocardial injury rather than subendocardial ischemic damage. T2-weighted imaging revealed no evidence of active myocardial inflammation,





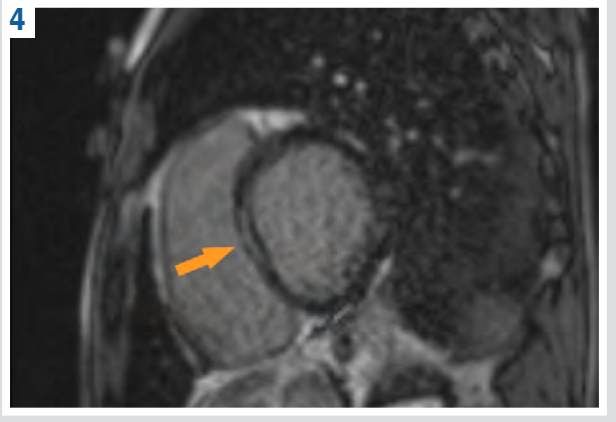


Fig. 2. Transthoracic echocardiography: apical four- and two-chamber views. **LA:** left atrium; **LV:** left ventricle; **RA:** right atrium; **RV:** right ventricle.

Fig. 3. Tissue Doppler imaging of the interventricular septum.

Fig. 4. Cardiac magnetic resonance imaging of the heart. Arrow indicates intramural non-ischemic late gadolinium enhancement.

Fig. 5. Cardiac magnetic resonance of the heart: apical four-chamber view. Arrow demonstrates intramural non-ischemic late gadolinium enhancement.

edema, or infiltration (T2 ratio <2.0), suggesting a chronic fibrotic stage rather than acute myocarditis.

Mildly reduced left ventricular systolic function (LVEF 49 %) and trace pericardial effusion were additionally observed *(Fig. 4, 5)*.

eThe patient was stabilized with GDMT for subclinical heart failure, including ACE inhibitors and spironolactone; however, beta-blockers were strictly contraindicated due advanced conduction disease. Given the high risk of progression to complete heart block, a permanent pacemaker (Vivatron G70A) was implanted.

The post-procedural course was uncomplicated. Subsequent ECG monitoring demonstrated stable pacing without recurrence of prolonged pauses or high-grade AV block.

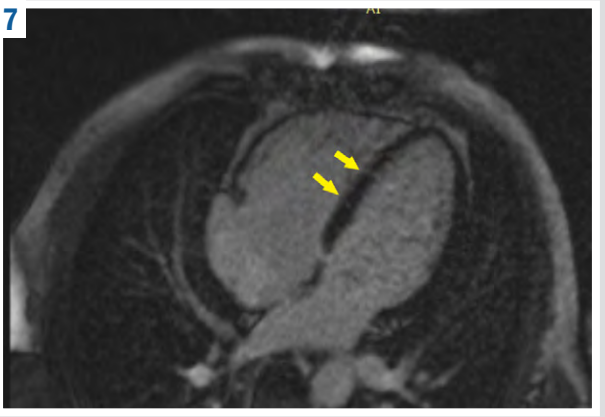
Following device implantation and optimization of medical therapy, the patient reported resolution of neurological symptoms and no episodes of dizziness or presyncope and was advised temporary restriction from intense physical activity, with planned gradual return to duty under cardiology supervision.

Case 2: the myopathic phenotype. A 39-year-old male active-duty servicemember was admitted with a 14-day history of progressively worsening exertional dyspnea and pronounced generalized weakness, leading to a reduction in physical performance. The onset of symptoms followed a documented mild viral upper respiratory tract infection treated conservatively while on duty. Unlike Case 1, the patient denied episodes of syncope, presyncope, palpitations, or dizziness, suggesting a predominantly mechanical rather than electrical cardiac manifestation.

Serial 12-lead ECGs consistently demonstrated normal sinus rhythm at a rate of approximately 90 bpm, with preserved QRS morphology, normal AV conduction, and no intraventricular conduction delays. Importantly, repeated measurements of cardiac Toponin I remained within the normal reference range throughout hospitalization, supporting a subacute or clinically silent inflammatory myocardial process rather than ongoing myocyte necrosis.

Transthoracic echocardiography revealed fibro-sclerotic thickening of the aortic root and the leaflets of both aortic and mitral valves without hemodynamically significant valvular stenosis. Mild (Grade I) mitral and tricuspid regurgitation were present. LV geometry was preserved (LV mass index 93 g/m^2, relative wall thickness 0.33); however, diastolic function assessment demonstrated Grade II diastolic dysfunction with elevated LV filling pressures, indicating reduced myocardial compliance.

Notably, the IVS exhibited marked echogenicity heterogeneity, raising suspicion of post-inflammatory



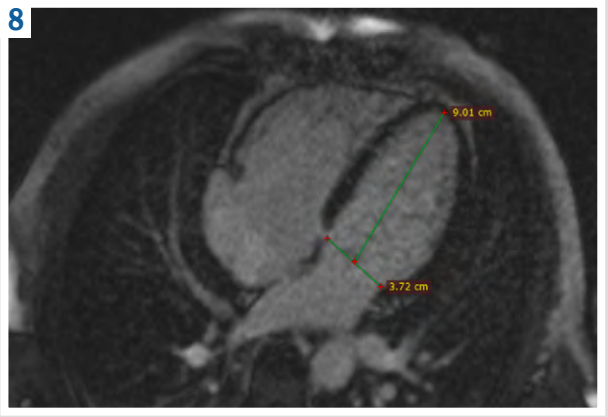


Fig. 6. Transthoracic echocardiography showing marked echogenicity heterogeneity within the interventricular septum. **LA:** left atrium; **LV**: left ventricle; **AO**: aorta; **IVS**: interventricular septum.

Fig. 7. Cardiac magnetic resonance of the heart. Arrow indicates segmental non-ischemic late gadolinium enhancement in the mid-wall of the interventricular septum.

Fig. 8. Cardiac magnetic resonance of the heart: apical four-chamber view illustrating left ventricular size and ejection fraction.

Table 1. Comparison of arrhythmic and myopathic phenotypes of septal non-ischemic myocardial injury

Parameter	Case 1 (arrhythmic phenotype)	Case 2 (myopathic phenotype)
Primary trigger	Blast trauma / pneumonia	Viral infection
Dominant symptom	Presyncope / ventricular pauses	Dyspnea / generalized weakness
Biomarker profile	Minimal Troponin I elevation (0.1 ng/mL)	Consistently normal Troponin I
LV systolic function (EF)	49 % (mildly reduced)	45 % (moderately reduced)
Conduction status	Mobitz II / transient grade III block	Normal sinus rhythm
Septal involvement (CMR)	Intramural fibrosis (diffuse septal)	Focal segmental fibrosis (segments 8, 9)
Definitive treatment	Permanent Pacemaker + GDMT	Conservative GDMT

structural remodeling *(Fig. 6)*. In contrast to Case 1, this patient demonstrated regional contractile impairment with septal hypokinesia and moderately reduced global systolic function, consistent with subcompensated left-sided heart failure. The probability of pulmonary hypertension was low.

CMR revealed focal, segmental non-ischemic LGE localized to the mid-wall of the IVS, predominantly involving segments 8 and 9 *(Fig. 7)*.

These findings were consistent with post-inflammatory myocardial fibrosis rather than ischemic or infiltrative pathology. LVEF was moderately reduced at 45 %, corroborating echocardiographic evidence of systolic dysfunction *(Fig. 8)*. No CMR features of infiltrative cardiomyopathy (including sarcoidosis) or primary dilated cardiomyopathy were identified.

Management focused on optimization of GDMT for heart failure with mildly reduced ejection fraction. Continuous in-hospital telemetry revealed no episodes of high-grade AV block, significant bradyarrhythmias, or malignant ventricular arrhythmias. The patient demonstrated gradual clinical improvement with medical therapy alone, including improved exercise tolerance and reduced dyspnea, obviating the need for invasive rhythm-management interventions.

To facilitate comparison between the arrhythmic and myopathic phenotypes, the main clinical and imaging features of both cases are summarized in *Table 1*.

Taken together, these two cases illustrate distinct clinical phenotypes arising from non-ischemic, post-inflammatory myocardial involvement of the IVS in young, physically active military servicemembers. Despite similar structural substrates detected by CMR, the clinical expression differed substantially.

Case 1 was dominated by malignant conduction system disease, characterized by Mobitz type II AV block, intermittent progression to complete heart block, and prolonged ventricular pauses, necessitating permanent pacemaker implantation.

In contrast, Case 2 exhibited a predominantly myopathic phenotype, with regional septal hypokinesia, diastolic

dysfunction, and moderately reduced systolic function leading to symptomatic heart failure, but without clinically significant conduction disturbances.

These observations underscore the heterogeneous clinical impact of post-inflammatory septal myocardial injury, which may selectively impair either the cardiac conduction system or myocardial contractile function. Advanced imaging with CMR proved essential in both cases for uncovering the underlying structural substrate, guiding risk stratification, and informing individualized management strategies.

Discussion

The present case series illustrates the heterogeneous clinical manifestations of post-inflammatory non-ischemic myocardial injury in young, physically active servicemembers. Despite a shared inflammatory background and similar localization of myocardial involvement within the IVS, the two patients exhibited markedly divergent clinical phenotypes: intermittent high-grade AV block with prolonged ventricular pauses in Case 1, and new-onset heart failure with systolic and diastolic dysfunction in Case 2.

This divergence highlights a fundamental principle of myocardial pathology: the clinical phenotype is determined not only by the etiology of injury, but by its precise anatomical and cellular distribution. Inflammatory myocardial processes are inherently heterogeneous, affecting conduction tissue, contractile myocardium, or both to varying degrees.

In the arrhythmic phenotype (Case 1), inflammation and subsequent fibrosis were localized predominantly to the IVS, a region that houses the His bundle and proximal components of the His-Purkinje system. These structures represent a narrow and highly specialized electrical pathway, rendering them exquisitely vulnerable to even small foci of edema or fibrosis. Consequently, minimal structural disruption may result in abrupt infra-Hisian conduction failure, manifesting as Mobitz type II AV block or transient complete heart block [5]. Such disturbances are universally considered organic rather than functional, do not respond to vagolytic maneuvers, and are associated with a substantial risk of asystole and SCD due to the absence of reliable escape rhythms [7,11,25].

By contrast, the myopathic phenotype (Case 2) demonstrates that septal myocardial injury may preferentially involve the contractile syncytium while sparing the specialized conduction tissue. In this scenario, inflammation affects cardiomyocytes more diffusely, leading to impaired relaxation, reduced compliance, and eventually depressed systolic performance [2,14]. The result is a clinical picture dominated by exertional dyspnea and heart failure rather than arrhythmia. This presentation aligns with the increasingly recognized entity of “silent myocarditis”, in which significant myocardial dysfunction develops despite a normal surface ECG, absence of syncope or palpitations, and negative cardiac biomarkers [3,4].

A unifying factor in both cases is the presence of sustained physical and environmental stress during or shortly after an inflammatory trigger. Military personnel frequently operate under conditions of extreme physical exertion, sleep deprivation, thermal stress, and sustained sympathetic activation [9]. These factors amplify myocardial oxygen demand and catecholaminergic stimulation, potentially exacerbating inflammatory myocardial injury and electrical instability [3,8,23].

In Case 1, the pathophysiological cascade was further compounded by blast-related thoracic trauma. Exposure to high-pressure shock waves can result in myocardial contusion, microvascular injury, focal hemorrhage, and myocardial edema, even without overt chest wall injury. When such mechanical myocardial injury is followed by systemic inflammation, as in post-traumatic pneumonia, the heart is subjected to a “double hit” phenomenon [26]. Circulating pro-inflammatory cytokines, including interleukin-6 and tumor necrosis factor-α, are known to impair ion channel function, alter gap junction coupling, and depress conduction velocity within the His–Purkinje system [8,27]. Experimental and clinical data suggest that myocardium previously primed by focal septal injury is particularly susceptible to malignant bradyarrhythmias under these conditions [11].

The diagnostic trajectories of both cases underscore the limitations of traditional diagnostic modalities. While ECG remains indispensable for assessing chamber size, valvular function, and global systolic performance [12,13,21], it lacks the spatial resolution and tissue specificity required to distinguish active inflammation from chronic fibrosis. Similarly, cardiac biomarkers may remain normal in subacute or localized myocardial injury, as observed in Case 2.

In contrast to conventional imaging, CMR provides unparalleled non-invasive tissue characterization, serving as an alternative to endomyocardial biopsy in selected clinical scenarios [15,17,18]. LGE allows precise identification of intramural or mid-wall scar patterns typical of non-ischemic injury [23,24].

Importantly, septal LGE has been associated with a particularly malignant substrate; multiple studies have demonstrated that patients with septal fibrosis carried a significantly higher risk of malignant arrhythmias, SCD, and the need for permanent pacing compared to those with non-septal involvement [5,8,19,22].

While Case 1 exhibited a borderline LVEF of 49 %, evidence by Y. Chen et al. (2022) suggests that in patients with suspected myocarditis and preserved systolic function, conventional volumetric parameters may fail to capture subtle myocardial impairment. Their findings underscore that CMR-derived strain parameters provide superior diagnostic and prognostic accuracy compared to LVEF alone, particularly in identifying subclinical contractile dysfunction predisposing to electrical instability [16].

Furthermore, the incorporation of parametric mapping techniques, specifically native T1, T2 mapping, and extracellular volume (ECV) quantification, enables detection of diffuse or occult myocardial inflammation that can precede irreversible fibrosis [17,18,20]. This capability is especially relevant in patients such as Case 2, in whom conventional testing fails to explain the etiology of new-onset heart failure. Employing the revised Lake Louise criteria enhances diagnostic certainty and facilitates early intervention, potentially avoiding invasive endomyocardial biopsy, which is inherently limited by sampling error and procedural risks [1,2].

These observations carry direct implications for the management of high-risk occupational populations. First, Mobitz Type II AV block or ventricular pauses exceeding 2000 ms in physically fit individuals must not be attributed

to high vagal tone without urgent CMR evaluation to exclude organic septal disease [5,11,25]. Second, normal cardiac biomarkers do not rule out evolving myocardial injury; as shown in Case 2, reliance on troponin alone may delay necessary intervention in myopathic phenotypes [4,6]. Finally, strict activity restriction is mandatory during the inflammatory phase to prevent edema from transitioning into permanent fibrotic tissue. Return-to-duty decisions should be guided by comprehensive reassessment of both electrical and mechanical stability [1,9,22].

In summary, this case series demonstrates that post-inflammatory non-ischemic septal myocardial injury in young military servicemembers can manifest across a broad clinical spectrum, ranging from malignant conduction system disease to early-onset heart failure. As illustrated by the two contrasting phenotypes observed, the decisive determinant of clinical phenotype is the precise anatomical and cellular localization of injury within the IVS, whether preferentially involving the His–Purkinje system or the working myocardium. These mechanistic differences directly translate into divergent management strategies, from urgent permanent pacing to conservative guideline-directed heart failure therapy. Advanced CMR tissue characterization is therefore indispensable for uncovering the underlying substrate, enabling accurate risk stratification, and informing timely, potentially life-saving clinical decisions. Recognizing this spectrum is essential for clinicians managing physically active and operationally stressed populations, in whom delayed or incomplete diagnostic evaluation may result in catastrophic outcomes.

Conclusions

1. This case series underscores the critical clinical significance of non-ischemic interventricular septal involvement as an important structural substrate for malignant arrhythmias and early heart failure in young, physically active individuals, particularly in military populations exposed to inflammation, thoracic trauma, and extreme physical exertion. Persistent physical activity during acute or subacute inflammatory states may contribute to focal myocardial injury and septal fibrosis, increasing the risk of life-threatening arrhythmias and sudden cardiac death.

2. Timely recognition of subclinical myocarditis and post-inflammatory myocardial injury is essential, as clinically significant cardiac involvement may occur despite normal biomarkers and baseline electrocardiography. Symptoms such as exertional dyspnea, dizziness, fatigue, or presyncope after infection or trauma should prompt comprehensive cardiovascular assessment.

3. Cardiac magnetic resonance imaging remains the most sensitive non-invasive modality for detecting focal septal fibrosis and subtle myocardial injury, often before abnormalities become apparent on conventional echocardiography. Early diagnosis enables timely activity restriction, monitoring, and targeted interventions, including permanent pacemaker implantation or medical therapy, thereby reducing the risk of fatal cardiac events.

4. High-grade atrioventricular block and early systolic dysfunction in young military personnel should not be considered benign. An assertive diagnostic approach incorporating advanced cardiac imaging is crucial for accurate risk stratification, prevention of sudden cardiac death, and preservation of operational readiness in this high-risk population.

Ethical approval
The research program was reviewed and approved by the Bioethics Commission of Zaporizhzhia State Medical and Pharmaceutical University (Protocol No. 6 dated April 16, 2026). The study complies with all applicable requirements for research involving human participants as established by international regulations and current Ukrainian legislation. All participants provided written informed consent.

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