CASE REPORT

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THE CARDIOMYOPATHY THAT NO LONGER EXISTS: THE EVOLVING APPROACH TO LEFT VENTRICULAR NONCOMPACTION ILLUSTRATED BY THE CASE OF A FLIGHT TECHNICIAN

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Abstract: For decades, left ventricular hypertrabeculation was regarded as a rare congenital cardiomyopathy characterized by an excessively developed trabecular endocardial layer with deep intertrabecular recesses. It most commonly presented with heart failure, arrhythmias, and/or thromboembolic complications in young adults. Temporary or permanent suspension from flying duties was the usual response to a diagnosis of left ventricular noncompaction (LVNC) cardiomyopathy, given the potential risk of arrhythmia and sudden in-flight incapacitation. However, the 2023 European Society of Cardiology (ESC) guidelines have radically changed the approach to this imaging phenotype.

Hypertrabeculation is no longer classified as a separate cardiomyopathy but as a phenotypic trait that may occur in isolation or accompany other heart conditions. This fundamental change in classification calls for a reassessment of existing aeromedical certification procedures and may affect the ability of aircrew previously disqualified on these grounds to continue their careers.

The practical implications of this reclassification are illustrated by the case of a 41-year-old flight technician with left ventricular hypertrabeculation. In the era of "the cardiomyopathy that no longer exists," this case underscores the importance of individual risk assessment and the need to adapt certification procedures to current medical knowledge.

Keywords: left ventricular hypertrabeculation, aviation medicine, cardiac arrhythmias, pilots

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INTRODUCTION

The current 2023 ESC definition of hypertrabeculation

The latest European Society of Cardiology (ESC) guidelines on cardiomyopathies, published in 2023, introduced a major change in the approach to left ventricular hypertrabeculation (formerly referred to as left ventricular noncompaction [LVNC]) [9,33,34]. Hypertrabeculation is no longer regarded as a distinct disease entity or a separate cardiomyopathy. As the ESC emphasizes, this "phenotypic trait" may occur not only in healthy individuals but also in various cardiomyopathies (e.g., hypertrophic or dilated) and in certain physiological states (pregnancy, intense athletic training) [9,21,33,34]. The term "hypertrabeculation" is preferred over "left ventricular noncompaction" (LVNC), especially when the finding is transient or appears in adulthood [9,33].

Imaging criteria

Hypertrabeculation is diagnosed mainly by imaging: echocardiography and cardiac magnetic resonance imaging (MRI) [19,33]. The most commonly used quantitative criteria are a noncompacted-to-compacted layer thickness ratio of >2:1 at end-systole (echocardiography) or >2.3:1 at end-diastole (MRI) [19]. As the ESC emphasizes, however, the presence of hypertrabeculation is not equivalent to a diagnosis of cardiomyopathy if other signs of myocardial damage, such as systolic dysfunction, ventricular dilation, or clinical symptoms, are absent [9,19,33,34].

Clinical significance

Hypertrabeculation should be regarded as a phenotype that may accompany other cardiomyopathies or represent a normal variant [9,21,33,34]. In clinical practice, overdiagnosis of this trait as a disease should be avoided, particularly in asymptomatic individuals with no other abnormalities on imaging.

Evolution of the concept of left ventricular noncompaction

Left ventricular noncompaction (LVNC) cardiomyopathy was long classified as a distinct disease entity within the group of primary cardiomyopathies. It was first described by Grant in 1926 but was not characterized in detail until the 1990s. The historical definition of LVNC was that of a congenital cardiomyopathy in which an excessively developed trabecular endocardial layer contained deep intertrabecular recesses [18] communicating with the left ventricular cavity. The condition is thought to result from impaired myocardial compaction during fetal development.

In 2006, the American Heart Association (AHA) recognized LVNC as a genetic cardiomyopathy, whereas the European Society of Cardiology (ESC) regarded it as an unclassified cardiomyopathy [18,20].

The clinical manifestations of LVNC are nonspecific and include progressive dyspnea, reduced exercise tolerance, chronic fatigue, and signs and symptoms of heart failure such as peripheral edema and nocturia. Arrhythmias, including ventricular tachycardia or atrial fibrillation, may be the initial presentation in some patients and can lead to presyncope, syncope, or even sudden cardiac death. In addition, some patients develop intracardiac thrombi, which increase the risk of systemic embolism, including stroke.

LVNC is diagnosed primarily by transthoracic or transesophageal echocardiography, which shows a bilayered left ventricular myocardium with a compacted outer layer and a noncompacted (trabecular) inner layer, with a noncompacted-to-compacted thickness ratio of >2:1 in systole. A more detailed structural assessment and exclusion of other forms of cardiomyopathy can be achieved with cardiac MRI. The electrocardiogram (ECG) often reveals nonspecific changes such as signs of left ventricular hypertrophy, conduction blocks, or ventricular arrhythmias. The workup also includes 24-hour Holter ECG monitoring and assessment of the left ventricular ejection fraction. Genetic counseling is indicated when a familial form is suspected.

Dilated cardiomyopathy, myocarditis, and valvular heart disease should be considered in the differential diagnosis, along with other conditions featuring myocardial hypertrophy or trabeculation, including athlete's heart, sarcoidosis, and storage diseases.

Diagnostic and clinical significance of hypertrabeculation

Population-based studies indicate that the left ventricular hypertrabeculation phenotype may be present in up to 5% of the general population, often without a significant effect on clinical prognosis [20].

However, according to recent data from the Copenhagen General Population Study, this phenotype is associated with a significantly higher risk of adverse cardiovascular events, including heart failure (hazard ratio [HR], 2.63) and all-cause mortality (HR, 2.08) [23]. For aircrew, whose certification may be affected even by subclinical

abnormalities, these data are particularly important to medical assessment.

In aviation medicine, both structural and functional myocardial abnormalities, including ventricular and supraventricular arrhythmias associated with hypertrabeculation, pose a significant diagnostic and certification challenge. Because of the risk of sudden decompensation or arrhythmic episodes in flight, particular caution is required when assessing fitness for flying duties.

CASE PRESENTATION

History and initial evaluation

In 2015, a 41-year-old career soldier serving as a flight technician was referred to the Regional Military Aeromedical Board (RWKL-L) for an aeromedical fitness examination ahead of a planned change of assignment. The patient was asymptomatic, with no prior treatment for internal medical conditions and no history of chronic or cardiac disease.

Records from previous periodic examinations, conducted since 2008, noted abnormal T waves and nonspecific ST-T changes on the resting ECG. Isolated premature ventricular complexes were recorded during an exercise ECG test performed as part of screening for coronary artery disease after the age of 35.

Sinus rhythm at 72/min and frequent premature ventricular complexes were recorded on the resting ECG obtained at this assessment. Transthoracic echocardiography showed normal global left ventricular systolic function without regional wall motion abnormalities. Prominent ventricular trabeculation and visible intertrabecular recesses were noted, raising suspicion of left ventricular noncompaction.

Consequently, the soldier was referred to the Department of Internal Disease at the Military Institute of Aviation Medicine (WIML) for further diagnostic workup. No significant abnormalities were found on physical examination at admission, and the patient's general condition was good.

Laboratory tests showed mildly elevated total cholesterol, low-density lipoprotein (LDL) cholesterol, and total bilirubin levels.

Diagnostic workup: imaging and electrophysiology

During hospitalization, 24-hour Holter ECG monitoring recorded a total of 24,092 monomorphic premature ventricular complexes (26% of all beats), including very frequent couplets (1,437), episodes of bigeminy and trigeminy, and 9 runs of nonsustained ventricular tachycardia (nsVT); the patient was unaware of the arrhythmia.

A bilayered structure of the left ventricular myocardium, with deep intertrabecular recesses predominantly in the apical segments, was visualized on cardiac MRI. At end-diastole, the maximum ratio of noncompacted (NC) to compacted (C) layer thickness was 6.6 (NC = 20 mm, C = 3 mm), which met the previously used diagnostic criteria for left ventricular noncompaction cardiomyopathy [9]. No late gadolinium enhancement (LGE) was found, which is a favorable prognostic factor.

On the basis of the history, imaging findings, and cardiac rhythm monitoring, the patient was diagnosed with ventricular arrhythmia and left ventricular noncompaction cardiomyopathy.

Treatment and certification decisions

Bisoprolol, a beta-blocker, was initiated at 2.5 mg daily, and the patient was selected for percutaneous radiofrequency (RF) catheter ablation of the ectopic focus responsible for the ventricular arrhythmia. Because the ventricular arrhythmia was potentially malignant, the flight technician was temporarily removed from duty until the ablation had been performed and its effectiveness assessed.

Follow-up 24-hour Holter ECG monitoring after the ablation showed a substantial reduction in ventricular ectopy: 10,178 premature ventricular complexes (11% of all beats) and a single 3-beat run of ventricular tachycardia. Only 674 multiform premature ventricular complexes (0.82% of all beats) were recorded at the next assessment 4 months later; no complex ventricular arrhythmias were detected.

After 6 months off duty, another Holter ECG recording showed no ventricular arrhythmia. An exercise test also showed no arrhythmias, with good exercise capacity (10.9 metabolic equivalents [METs]).

On the basis of these findings, the flight technician was cleared to return to flying duties.

DISCUSSION

Hypertrabeculation and arrhythmias

The risk factors for cardiac arrhythmias include left ventricular hypertrabeculation. The literature indicates a significant association between left ventricular hypertrabeculation and cardiac arrhythmias. Ventricular arrhythmias are estimated to occur in approximately 47% of patients with hypertrabeculation, and atrial fibrillation in more than 25%. Complex ventricular arrhythmias, including episodes of nonsustained ventricular tachycardia, dominated the clinical picture in

this patient and provided the main indication for antiarrhythmic treatment.

Notably, the prognosis of patients with hypertrabeculation may vary. A meta-analysis published in 2020 [2] showed that individuals diagnosed with this phenotype have a risk of cardiovascular mortality, all-cause mortality, thromboembolic events, and ventricular arrhythmias comparable to that of patients with dilated cardiomyopathy. Reduced left ventricular ejection fraction remains the key predictor of poor prognosis in patients with hypertrabeculation.

An increased risk of cardiovascular complications has not been clearly linked to left ventricular hypertrabeculation in isolation, without coexisting systolic dysfunction or other abnormalities, according to a growing body of evidence. Large population-based studies, such as MESA (Multi-Ethnic Study of Atherosclerosis), have shown that in asymptomatic individuals, hypertrabeculation is not associated with adverse cardiac remodeling or with an increased risk of myocardial fibrosis [19]. Similarly, analyses comparing individuals with LVNC and the general population have shown that patients with a normal ejection fraction and isolated apical trabeculation have survival similar to that of individuals without this trait [19,27].

Coexisting abnormalities, especially left ventricular systolic dysfunction and fibrosis (LGE on MRI), are the main determinants of cardiovascular complication risk in individuals with hypertrabeculation [4,6,19]. Meta-analyses and cohort studies have shown that rates of mortality, arrhythmias, and thromboembolic complications are similar to those observed in dilated cardiomyopathy rather than higher than in the general population [2,4,16]. The key prognostic factor is the left ventricular ejection fraction, not the extent of trabeculation itself [2,4,19,27].

The literature confirms an association between LVNC and an increased incidence of ventricular and supraventricular arrhythmias, including atrial fibrillation, ventricular tachycardia, and conduction blocks [18,19,20]. However, patients with reduced ejection fraction, left ventricular dilation, or fibrosis on MRI face the highest risk [3,6,13,17,26]. MRI studies have shown that late gadolinium enhancement correlates with a higher risk of arrhythmia and a worse prognosis, regardless of the extent of trabeculation itself [3,6,19].

The 2023 ESC guidelines and recent literature reviews emphasize that hypertrabeculation is a phenotypic trait rather than a distinct disease entity. Standard cardiology follow-up is sufficient for asymptomatic individuals without systolic dysfunction: no increased risk of complications has been identified, and neither aggressive treatment nor additional surveillance is recommended [4,9,19].

In asymptomatic individuals with normal systolic function and no fibrosis on MRI, periodic cardiology follow-up is sufficient: ECG, 24-hour Holter ECG monitoring, and echocardiography every 1 to 2 years, with MRI if there is diagnostic uncertainty or clinical deterioration [9].

If arrhythmia, systolic dysfunction, or fibrosis is found, management should follow the standard approach used in other cardiomyopathies, including consideration of pharmacotherapy, ablation, and, in selected cases, implantable cardioverter-defibrillator (ICD) implantation [3,4,6,9].

Decisions should be made on an individual basis, preferably by a multidisciplinary team, and treatment should be guided by symptoms and the clinical phenotype rather than by the trabeculation pattern alone [4,6,9].

The aviation medicine perspective: stressors of the flight environment

Military pilots are exposed to adverse factors at work including acceleration, vibration, abrupt changes in atmospheric pressure and temperature, information-related stress, chronic fatigue with circadian rhythm disruption, and altitude hypoxia. Each of these factors can modulate myocardial automaticity, conduction, and repolarization, increasing the likelihood of arrhythmias. Therefore, the key criterion for certification and clearance to continue flying duties is the risk of sudden in-flight incapacitation. The mission profile, availability of immediate intervention, and potential consequences of transient or permanent incapacitation in flight must inform the operational assessment of even asymptomatic arrhythmias, which are often benign in the general population [16,35,36].

Altitude hypoxia activates peripheral chemoreceptors, shifting the autonomic balance toward sympathetic activation and relative parasympathetic withdrawal. This results in tachycardia and increased cardiac output as compensatory mechanisms to maintain oxygen delivery. This profile promotes arrhythmias by increasing automaticity, facilitating triggered activity (early and delayed afterdepolarizations [EADs/DADs]), and modulating the conditions for reentry (e.g., by altering refractoriness and the heterogeneity of repolarization). Ventricular ectopy and brief, self-limited episodes of tachyarrhythmia may occur even in individuals without diagnosed cardiovascular disease, according to high-altitude studies [5,10,14,22,24,30].

Positive head-to-foot acceleration (+Gz) causes rapid redistribution of blood to the lower body and reduced venous return, leading to a transient decrease in cardiac output and activation of baroreceptor and mechanoreceptor reflexes. In response, heart rate increases and adrenergic activation intensifies [28,29].

Exposure to +Gz is rarely the only factor acting in operational settings. Anti-G straining maneuvers (AGSMs) increase intrathoracic pressure, modulate preload and afterload, and intensify cardiopulmonary mechanoreceptor stimulation. ECG recordings during human centrifuge testing or training have shown frequent premature ventricular and supraventricular complexes, paroxysmal supraventricular tachyarrhythmias, and, in some individuals, brief episodes of ventricular tachycardia, which usually resolved once exposure ended [29,32].

Whole-body vibration (WBV) should be regarded as an additional environmental stressor, particularly in transport aircraft and helicopters. Autonomic control may be modified by WBV, with accompanying changes in heart rate variability (HRV) parameters, according to research findings. The effect may depend on vibration frequency, exposure duration, and concurrent cognitive load. Evidence directly linking WBV to clinically significant arrhythmias in aircrew is limited, but WBV may be regarded as a factor that "amplifies" autonomic reactivity, particularly in combination with hypoxia and +Gz [15,25,31].

Operational stress and cognitive load generate strong adrenergic stimulation. Through ion channel mechanisms and increased automaticity, excessive sympathetic activation with reduced parasympathetic influence favors both supraventricular and ventricular arrhythmias. From an aeromedical perspective, risk should therefore be viewed as the cumulative effect of stimuli that, even if individually "subthreshold," may together narrow the safety margin [12,22].

In the context of left ventricular hypertrabeculation (LV hypertrabeculation/LVNC phenotype), the key distinction is between an isolated phenotype and one coexisting with arrhythmia, systolic dysfunction, or fibrosis on MRI. In the isolated form, the risk of adverse events may be low, whereas a high burden of premature ventricular complexes and episodes of nsVT, as in the case presented, are of particular importance regardless of the extent of trabeculation itself, because they relate directly to operational risk [1,6,8,11,27].

In summary, military aviation calls for an assessment of arrhythmia risk in terms of the combined effects of overlapping environmental stimuli. This combination of adverse factors does not necessarily result in arrhythmias in healthy people, but it may lower the arrhythmia threshold and narrow the safety margin in predisposed individuals, which is particularly relevant to aeromedical certification focused on the risk of sudden in-flight incapacitation.

In certification practice, multidimensional risk stratification is recommended, covering: (1) left ventricular function, (2) the presence and nature of structural abnormalities, (3) the arrhythmia profile on ECG monitoring (number and complexity of premature ventricular complexes, presence of nsVT), (4) exercise tolerance and the absence of symptoms suggestive of presyncope or syncope, and (5) the operational context (aircraft type, flight profile, exposure to hypoxia and +Gz, oxygen availability, stress, and fatigue). For aircrew exposed to hypoxia and/or +Gz, confirming a stable rhythm during exercise is especially important. In selected situations, assessment during environmental exposure matching the duty profile is also considered under local procedures [16,35–38].

The course of the disease described here, and in particular the very good outcome of the ablation, with ventricular ectopy reduced to trace levels, no complex forms of arrhythmia, and a good exercise test result, supported the possibility of a return to flying duties with periodic follow-up. This is consistent with the principle of aeromedical certification that fitness to fly is determined primarily by the clinical phenotype and documented operational risk rather than by an isolated morphological finding [7,16].

CONCLUSIONS

The history of "the cardiomyopathy that no longer exists" exemplifies the rapid evolution of medicine, with classifications and definitions changing as clinical knowledge advances. The reclassification of left ventricular hypertrabeculation from a disease to a phenotypic trait creates opportunities for more precise diagnosis and certification adapted to the clinical context, particularly in demanding settings such as aviation medicine [39].

AUTHORS' DECLARATION

Study Design: Daria Owsiak, Katarzyna Barwińska, Ewelina Zawadzka-Bartczak. **Statistical analysis:** Daria Owsiak, Katarzyna Barwińska, Ewelina Zawadzka-Bartczak. **Data Collection:** Daria Owsiak, Katarzyna Barwińska, Ewelina Zawadzka-Bartczak.

Manuscript Preparation: Daria Owsiak, Katarzyna Barwińska, Ewelina Zawadzka-Bartczak. The authors declare that there is no conflict of interest.

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