Apical Hypertrophic Cardiomyopathy and Arrhythmia in Military Pilots

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CASE REPORT

Apical hypertrophic cardiomyopathy (ApHCM), a subtype of hypertrophic cardiomyopathy, may be found incidentally in healthy young adults. Arrhythmias are poor prognostic signs, and are the most frequent cause of sudden cardiac death. We present two cases of military aviators with ApHCM. One was a high-performance jet weapon system operator, who had asymptomatic non-sustained ventricular tachycardia (NSVT) and subsequently a symptomatic episode of paroxysmal atrial fibrillation. The second was a helicopter pilot, who had asymptomatic NSVT.

Keywords: non-sustained ventricular tachycardia, atrial fibrillation.

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disease of the myocardium caused by a mutation in the genes encoding the sarcomeric proteins (12). Apical hypertrophic cardiomyopathy (ApHCM) is a subtype of HCM in which the hypertrophy of the myocardium predominantly involves the apex of the left ventricle. The main symptoms are angina, atypical chest pain, effort-induced dyspnea, palpitations, and weakness. Giant inverted T waves and tall peaked QRS waves are seen in the chest leads on electrocardiogram (ECG). Echocardiography demonstrates isolated hypertrophy of the apex or combined hypertrophies of the intra-ventricular septum and the apex. Hypercontractility of the hypertrophied apex commonly causes a systolic spade shape during left ventriculography (9).

The prognosis of patients with ApHCM is better than that of HCM. The reported annual cardiovascular mortality rate is 0.1% (2). This rate can be considered compatible with flying, with various restrictions, in selected cases. We present two military aviators with incidentally discovered ApHCM who had arrhythmic episodes during follow-up and nevertheless continued active aviation.

CASE REPORTS

Case 1

A 43-yr-old previously healthy asymptomatic weapon system operator (WSO) operating on an A-4 jet was routinely examined in our institution. Giant inverted T waves in the precordial leads were recorded on ECG. An intraventricular septum of 11 mm (normal ≤ 11 mm) and hypertrophy of the apex of the left ventricle were demonstrated on echocardiography. Cardiac stress test was inconclusive regarding ischemia due to resting ECG abnormalities. Features consistent with inferior wall ischemia were recorded on a thallium radionuclide scan. No evidence of atherosclerotic disease was found during cardiac catheterization and 24-h rhythm monitoring was normal, but a spade-shaped pattern was observed during ventriculography. The WSO continued active operational duties. During the next 7 yr, echocardiography and 24-h rhythm monitoring were repeated every 12 mo. A gradual widening of the intra-ventricular septum up to 13 mm was observed. Rhythm recordings were normal. In 2004, an asymptomatic 15-s long non-sustained ventricular tachycardia (NSVT) (rhythm of 120 bpm) was recorded. Echocardiography demonstrated a normal left ventricular function with no enlargement of the left atrium and ventricle. Repeated rhythm recordings were normal, with no abnormalities during stress testing. A few months later the aviator developed atrial fibrillation (AF) during physical exertion, during which he remained hemodynamically stable. He underwent successful pharmacological cardioversion (using propafenone), and aspirin and bisoprolol treatment were initiated. No abnormalities were seen on repeated rhythm monitoring and stress testing. The WSO was transferred to basic navigation instruction on a low-performance platform. To date, he remains asymptomatic, with no rhythm disturbances on repeated monitoring.

Case 2

A 31-yr-old UH-60 helicopter pilot, with recently diagnosed hypertension and dyslipidemia, was routinely examined at our institution. He was treated with an ACE inhibitor, beta blocker, thiazide diuretic, and statin...
tin. His BP and blood lipid levels were well controlled. Secondary hypertension was excluded. On ECG, ST changes and inversion of T waves in the precordial leads were noticed. Previous ECG recordings were normal. Echocardiography demonstrated apical hypertrophy, with intra-ventricular septum width of 12 mm (normal ≤ 11 mm) and left atrial enlargement (diameter 40 mm, normal ≤ 36 mm; area 22 cm², normal ≤ 20 cm²). Cardiac stress test was inconclusive due to resting ECG abnormalities. No evidence of ischemia was observed on thallium radionuclide imaging and 24-h rhythm recording was normal. During the subsequent 4 yr, during which he was regularly assessed by echocardiography and 24-h rhythm monitoring, a gradual widening of the intra-ventricular septum up to 14 mm was observed and an asymptomatic NSVT (140 bpm) lasting 4 s was recorded. Thallium scanning demonstrated a pattern of inferior-lateral wall ischemia. Cardiac catheterization demonstrated a non-significant lesion in a small diagonal branch. A spade shape of the left ventricle was demonstrated on ventriculography. Subsequent rhythm and stress testing were normal.

A waiver to return to his two-seated low-performance platform (helicopter) was granted. During the next 4 yr, no further widening of the intra-ventricular septum was demonstrated. Repeated 24-h rhythm recordings did not demonstrate rhythm disturbances. No evidence of ischemia was found on repeated exercise echocardiography studies. The aviator continued his medication and remained asymptomatic. He continued his active aviation duty.

**DISCUSSION**

The major risk factors for sudden death in HCM are a previous event of sudden death, previous sustained ventricular tachycardia, and a family history of sudden death. Minor risk factors are two or more episodes of syncope within 1 yr, left ventricular wall thickness of more than 30 mm, a failure to increase BP by more than 25 mmHg from baseline or a decrease of more than 10 mmHg from the maximal BP during exercise in an upright position, NSVT, a resting obstruction of the left ventricular outlet with gradient > 30 mmHg, microvascular obstruction, and high-risk genetic defects (13). The major risk factors for cardiac morbidity in ApHCM are age < 41 yr at diagnosis, left atrial enlargement, and New York Heart Association class II (2). The total annual cardiovascular mortality for ApHCM patients is 0.1%, which is much lower than the annual mortality of 1.4–4% for the entire HCM spectrum (10).

Cardiomyopathy disqualifies entry into civilian and military aviation training. In the United States Air Force and Navy, a finding of HCM disqualifies permanently for aviation duties (15,16). The Joint Aviation Authorities permit pilots with cardiomyopathy to continue operating on multicrew platforms. They are required to be asymptomatic, to have normal stress tests without excessive elevation of BP during exercise, normal cardiac rhythm recordings, normal left ventricular function, and a septal width < 25 mm (6). The Federal Aviation Authority restricts persons with hypertrophy or dilatations of the heart or with arrhythmias caused by organic heart disease from flying duties (3). Recently, an asymptomatic commercial pilot with HCM was granted a waiver for unrestricted flying by the National Transport Safety Board (5), although the FAA formally restricted him from flying (8). To our knowledge, only one case of a military aviator with ApHCM has been reported (11). He was asymptomatic during 15 yr of observation, and continued his active aviation duties on a low-performance platform.

Ventricular tachycardia with anatomic heart disease permanently disqualifies aviators in most civilian and military aviation authorities (6,15). High-performance platforms expose the aviator to high acceleration forces. These forces, especially +Gz, are arrhythmogenic. Most of the rhythm disturbances occur during acceleration and deceleration. Atrial and ventricular premature beats commonly occur and are benign during the acceleration phase, while supra-ventricular tachycardia and ventricular tachycardia are considered more dangerous. During deceleration, sinus bradycardia and atrial-ventricular block of various types are observed (7).

The prevalence of pre-syncope, syncope, or sudden death in aviators who suffered from NSVT and had a normal cardiac structure was 0.33% per year. Mitral valve prolapse increased the risk of a significant hemodynamic event to 2.3% per year (4). Non-sustained ventricular tachycardia was recorded in 23% of patients with ApHCM during long-term follow-up (2). Although NSVT increases the relative risk for sudden death by 1.9 times, the risk remains < 1% per year (1). Atrial fibrillation occurred in 12% of 125 ApHCM patients during a long-term follow-up study (2). Left atrial enlargement on the baseline echo was identified as the sole predictor of AF (2). In HCM patients, AF was associated with substantial functional deterioration in 84% of patients (14). There was an overall increase in mortality, mostly due to heart failure and stroke, and not due to sudden cardiac death. Onset of AF in those younger than 50 yr was associated with less favorable outcomes. Development of chronic AF was associated with higher mortality rates than paroxysmal AF. Ischemic stroke was eight times more prevalent, usually occurring more than 3.5 yr after AF onset. The prevalence of stroke was independent of whether AF was paroxysmal or chronic and of the number of AF paroxysms (14).

The Israeli Air Force Aeromedical Center uses the 1% threshold for asserting flying fitness. The 1% includes all events that may lead to sudden incapacitation; in this case sudden cardiac death, syncope, or stroke. Although symptoms of angina, atypical chest pain, dyspnea, and palpitations may be the presenting signs or develop in ApHCM patients, the two aviators discussed here were free of these symptoms, were New York Heart Association class I, and had no cardiac deterioration. Thorough follow-up, including yearly ECG, rhythm monitoring (Holter), echocardiography, stress tests or cardiac radio-nucleotide imaging, flight surgeon examination, and cardiac consultation was performed to exclude significant deterioration. Therapeutic measures were taken in both cases in order to modify the clinical finding. Case 1 was granted a waiver for
multicrew low-performance aviation. Although we observed gradual disease progression, the annual risk for sudden incapacitation due to cardiac death was considered by the authors to be less than 1%. Limiting the aviator to a low-performance platform abolished exposure to the arrhythmogenic +Gz force. Although the appearance of AF is an ominous sign in the aviator, flight safety would not be compromised by the sudden incapacitation of a navigation instructor on a multiple crew platform. Since Case 2 was not exposed to G forces, he continued his aviation duties without any restrictions in a dual-seated helicopter. Our two cases demonstrate that consideration should be given to permitting aviators with ApHCM to continue their aviation duties on condition that exposure to arrhythmogenic G force is eliminated and that thorough and regular cardiac assessment is done. As long as these two conditions are met, the aviator may continue flight duties in spite of isolated episodes of NSVT and even AF.

REFERENCES