contraction/ventricular tachycardia's originating from mitral annulus are rarely reported (1).

PVCs arising from the mitral annulus frequently originate from anterolateral, posteroseptal and posterior sites (2). It has been reported that 2/3 of the PVCs arising from the mitral annulus originate from anterolateral site (2). Furthermore, small part of these arrhythmias originates from the anteroseptal site of the mitral annulus. Ablation of this site may be technically very challenging. Cases have been reported that successful catheter ablation of the premature ventricular contraction origin from the anteroseptal site of the mitral annulus can be performed either by a transseptal or transaortic approach in literature (3, 4). Anterolateral site of the mitral annulus is in close proximity to anterior of the right ventricle outflow tract, left ventricular epicardium near to the left sinus Valsalva and subvalvular region of the left ventricular outflow tract. Idiopathic PVC/VTs frequently originates from these sites that support this theory (5). In our case, early activation sites are not detected at the aortic root region and left ventricle outflow tract. By mapping of the left ventricle, at the time of PVCs, earliest ventricular activity is recorded in the anterolateral of the mitral annulus. In this site during the PVC, local ventricular activation preceded the QRS onset by 28 ms, when radiofrequency ablation applied to this site, PVCs immediately disappeared.

Adequate analysis of characteristics of ECG helps to determine the origin of mitral annulus sourced PVC/VT and may shorten the duration of the electrophysiological study. While the PVCs originating from anterolateral of mitral annulus has inferior axis, those originating from posterior annulus has superior axis. While QRS polarity in DI and aVL leads of PVCs originating from anterior annulus is negative, those originating from posterior annulus have positive QRS polarity in DI and aVL leads. Additionally it is shown that all the patients ECGs with mitral annulus originated PVC/VTs have s waves in lead V6 (2). In our case, ventricular premature contractions showed right bundle branch block pattern. Derivasyon lead (DI) showed rS pattern, V6 lead had an s wave and inferior axis. QRS notching in the inferior leads supported anterolateral origin. All these ECG findings showed that premature contractions were originating from anterolateral site of mitral annulus.

Conclusion

Premature ventricular contraction with right bundle branch block pattern can originates from mitral annulus. Medical therapy is the treatment of choice in these patients. Radiofrequency catheter ablation should be considered in patients' refractory to medical therapy.

Ömer Uz, Fethi Kılıçaslan, Mehmet Tezcan
Department of Cardiology, Gülhane Military Medical Academy, Haydarpaşa, İstanbul-*Turkey*

References

1. Jia-Feng L, Yue-Chun L, Jia L, Kang-Ting J, Jia-Xuan L, Ji-Fei T, et al. Successful epicardial ablation of idiopathic mitral annular ventricular tachycardia from the great cardiac vein. Pacing Clin Electrophysiol 2012; 5: 120-3. [CrossRef]
2. Tada H, Ito S, Naito S, Kurosaki K, Kubota S, Sugiyasu A, et al. Idiopathic ventricular arrhythmia arising from the mitral annulus: a distinct subgroup of idiopathic ventricular arrhythmias. J Am Coll Cardiol 2005; 15: 877-86. [CrossRef]
3. Yamada T, McElderry HT, Doppalapudi H, Epstein AE, Plumb VJ, Kay GN. Catheter ablation of premature ventricular contractions arising from the mitral annulus after mitral valvoplasty. Pacing Clin Electrophysiol 2009; 32: 825-7. [CrossRef]
4. Yamada T, McElderry HT, Allison JS, Doppalapudi H, Epstein AE, Plumb VJ, et al. Successful transseptal catheter ablation of premature ventricular contractions arising from the mitral annulus: a case with a pure annular origin. Pacing Clin Electrophysiol 2009; 32: 680-2. [CrossRef]
5. Kimber SK, Downar E, Harris L, Langer G, Mickleborough LL, Masse S, et al. Mechanisms of spontaneous shift of surface electrocardiographic configuration during ventricular tachycardia. J Am Coll Cardiol 1992; 15: 1397-404. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Ömer Uz
Gülhane Askeri Tıp Akademisi Haydarpasa, Kardiyoloji Kliniği, İstanbul-*Türkiye*
Phone: +90 216 542 34 65 Fax: +90 216 348 78 80
E-mail: homeruz@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 22.06.2012

©Telif Hakkı 2012 AVES Yayıncılık Ltd. Şti. - Makale metnine www.anakarder.com web sayfasından ulaşılabilir.
©Copyright 2012 by AVES Yayıncılık Ltd. - Available on-line at www.anakarder.com
doi:10.5152/akd.2012.162

Prolonged asystole during hypobaric chamber training

Alçak basınç ortamında oluşan hipokside uzamış asistoli

Introduction

An asystole, defined as the absence of myocardial electrical activity (1), is a state, which may occur due to acute hypobaric hypoxia. It can be seen even in completely healthy individuals (2) and may cause hazardous results compromising flight safety. Asystole is usually associated with an organic heart disease; coronary heart disease, myocardial infarction, myocarditis, congenital heart diseases, hypoxia, acidosis, hypo-hyperkalemia.

Case Report

A 36-year-old, male helicopter pilot was taken to hypobaric chamber training. His electrocardiography, chest X-Ray and biochemical parameters revealed to be completely normal. He had no history of syncope or presyncope. He was exposed to hypobaric environment for about one hour including 5 minutes staying at a simulated altitude of 30.000 feet. On the 47th minute from the training onset, the pilot had nausea, vomiting, excessive sweating, and loss of positional awareness symptoms and finally lost his consciousness on the 46th second after the mask off. The training was stopped and the pilot was assessed for emergency treatment by the internal observer. He was taken the oxygen mask on and regained his consciousness while being placed in Trendelenburg position. After the training the subject was re-examined by cardiologist and his vital values, electrocardiography, echocardiography and head-up tilt tests were normal. During this event an ambulatory blood pressure monitoring (ABP) and 12-lead rhythm monitoring were being performed for a planned study, although it was not a routine assessment. In his Holter recordings there were no signs of arrhythmia, however asystole lasting 16 second followed by a sinus bradycardia lasting 10 second were seen on the monitor (Fig. 1) (Video 1. See corresponding video/movie images at www.anakarder.com). The heart rate, ABP and heart rate variability (HRV) parameters of the subject were recorded (Table 1, 2). Due to the absence of any complaint of performing the daily activities further researches including electrophysiology study were not conducted. Two months after discharge,

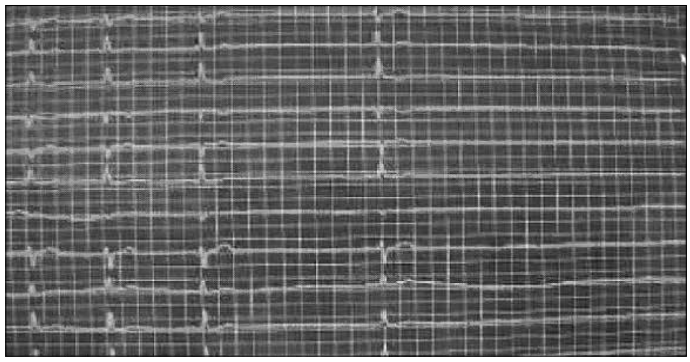

Figure 1. ECG recording during maximum hypoxia exposure onset showing asystole (paper speed- 25 mm per second and amplitude - 10 mm per mV)
ECG - electrocardiogram

Table 1. The Heart rate, ECG and ABP parameters of the subject

Parameters	Pre-hypoxia	Maximum hypoxia	Post-hypoxia
ABP, mmHg	146/95	140/95	147/86
Mean BP, mmHg	104	105	111
HR, bpm	97	108	70
Max QT, msn	360	320	400
Min QT, msn	288	200	288
QTd, msn	72	120	112
Max P, msn	128	112	112
Min P, msn	80	80	88
Pd, msn	48	32	24

ABP - arterial blood pressure, BP - blood pressure, ECG - electrocardiogram, HR - heart rate, Pd-P - wave dispersion, QTd - QT interval dispersion

Table 2. Heart rate variability parameters

SDNN 24 hour	77
SDANN index, ms	61
SDNN index, ms	58
rMSSD	24
pNN50, %	3
Spectral power-24 hour,	4633.5
Min spectral power/ Hour, ms^2	1870.9
Max spectral power /Hour, ms^2	5638.1

pNN50 - proportion of adjacent normal-to-normal (NN) intervals differing by >50 ms, rMSSD - root mean square of successive R - R interval differences, SDANN - standard deviation of the average NN intervals calculated over 5 - minute periods throughout the recording, SDNN - standard deviation of all, R - R intervals, SDNN index-mean of the standard deviation of the 5 - minute NN intervals over the entire recording

transthoracic echocardiography revealed no abnormality. Ejection fraction was within normal limits. A symptom-limited exercise electrocardiography (ECG) performed up to Bruce stage V, showed no evidence of myocardial ischemia and hyperventilation test was also normal. After this episode, he was allowed for full flight duties.

Discussion

Asystole may occur in aviators during high altitude flights or simulated training conditions (3). A sinus arrest lasting longer than 3 seconds are generally considered abnormal and are suggestive of an underlying abnormality (1). The patient's ECG, echocardiography, treadmill, 24- hour rhythm Holter and head-up tilt tests were normal. Possible cause of the asystole is the imbalance of the sympathetic-parasympathetic systems in hypobaric environments. The inhibitory reflex called Bezold-Jarisch reflex is triggered when sensory receptors are simulated by mechanical stretch. Hypoxia enhances the normal vasodilator response to epinephrine and this enhancement contributes to the vascular collapse (3). The SA node, the primary pacemaker of the heart, is densely innervated by parasympathetic nerve fibers. It is observed in some studies that changes in choline acetyltransferase activity especially at high altitude are possible. The increase in choline acetyltransferase and the decrease in the amount of muscarinic receptor suggest that there is an increase in parasympathetic activity during hypobaric hypoxia (4). The inhibition of the epinephrine (sympathetic) and augmentation in vagal (parasympathetic) efferent discharge to the heart, bradycardia and dilatation of the peripheral blood vessels with resulting lowering of the blood pressure are presented. Asystole may be due to profound suppression of both atrial and ventricular activity, complete heart block and prolonged myocardial ischemia (1). The heart rate increases at altitude, a proportional increase occurred in the cardiac output (5). An acute ascent to 15.000 feet causes a decrease of 30% of the maximum oxygen uptake compared with the sea level. An increased coronary circulation in response to the metabolic requirements of the myocardium is required. Myocardial depression is a consequence due to the reduced arterial oxygen tension in parallel with decrease in cardiac functional reserve. Occasionally, in such circumstances, there is a severe compensatory vasoconstriction of the coronary vessels that swamps all other reflex responses and causes cardiac arrest (6).

A study reported a 34-year-old acclimatized patient with sleep apnea syndrome climbing mountain 7000 m above sea level and during sleep developed cyclic sinus arrhythmia with R-R intervals of up to 3.3 seconds. The subject had been known to have frequent premature atrial beats for thirteen years. Our case was not acclimatized, had only one episode of asystole lasting 16 second and had nor comorbid syndrome neither history of premature atrial beats. It seems most likely explanation for both cases is the vagal stimulation induced by hypoxia causing arrhythmia (7). Another research reported a 27 years-old pilot with neurovegetative dystonia who entered cardiac asystole for 35 seconds. Medical assistance was performed and cardiopulmonary resuscitation (CPR) was initiated. He developed convulsions for 3 seconds when he regained heart activity (8). Our case regained his consciousness while performing first step medical assistance so we did not perform CPR. Our case did not develop convulsions.

Conclusion

Although asystole is an expected event in the hypobaric chamber training. Asystole episode in our case did not require CPR and recovery was achieved with 100% oxygen administration while being placed in Trendelenburg position could be considered important. We also emphasize the importance of vagal stimulation induced by hypoxia causing asystole. It is recommended to be alert for asystole or a profound bradycardia in pilots undergoing hypobaric chamber training and to monitor the trainees in a careful manner.

Cengiz Öztürk, Tolga Çakmak[1], Süleyman Metin[1], Ahmet Akın[1], Ahmet Şen[1]
Clinic of Cardiology, Eskişehir Military Hospital, Eskişehir-*Turkey*
[1]Department of Aerospace Medicine, Gülhane Military Medical Academy, Eskişehir-*Turkey*

Video 1. ECG recording showing asystole lasting 16 second followed by a profound sinus bradycardia lasting 10 second at (paper speed- 25 mm per second and at amplitude - 10 mm per mV amplitude).
ECG - electrocardiogram

References

1. Bashian GG, Wazni O. Bradyarrhythmias, atrioventricular block, asystole, and pulseless electrical activity. In: Griffin BP, Topol EJ, editors. Manual of Cardiovascular Medicine. 3rd ed. Philadelphia: Lippincot Williams and Wilkins; 2009. p. 322-36.
2. Welch H. Physiological and clinical findings during latent hypoxia in the hypobaric chamber. In: Alonso D, Bardel M,. et al editors. NATO RTO Symposium 2001. Operational Medical RTO Meeting Proceedings 62 on ''Operational Medical Issues in Hypo-Hyperbaric Conditions'' 2000 Oct 16-19; Toronto, Canada. Ottawa: Canada; 2001. p. 368-71.
3. Westendorp RG, Blauw GJ, Frölich M, Simons R. Hypoxic syncope. Aviat Space Environ Med 1997; 68: 410-4.
4. Crockatt LH, Lund DD, Schmid PG, Roskoski R Jr. Hypoxia-induced changes in parasympathetic neurochemical markers in guinea pig heart. J Appl Physiol 1981; 50: 1017-21.
5. West JB, Schoene RB, Milledge JS, editors. High Altitude Medicine and Physiology, 4th edition London: Hodder Arnold; 2007. p. 89.
6. Gradwell DP. Hypoxia and hyperventilation. In: Rainford J, Gradwell DP, editors. Ernsting's Aviation Medicine, 4th Edition New York: Edward Arnold Ltd; 2006. p. 48.
7. Cummings P, Lysgaard M. Cardiac arrhythmia at high altitude. West J Med 1981; 135: 66-8.
8. Anghel M, Capanu I, Muresan M. Cardiac arrest during hypobaric chamber exposure in a young pilot. In: Vermeiren R, Horváth ZC, editors. ECAM 2008. European Conference on Aerospace Medicine; 2008 November 12–15; Budapest, Romania; 2008. p. 39.

Address for Correspondence/Yazışma Adresi: Dr. Cengiz Öztürk
Eskişehir Askeri Hastanesi, Kardiyoloji Kliniği 26035, Eskişehir-*Türkiye*
Phone: +90 222 220 45 30 Fax: +90 222 220 34 33
E-mail: drcengizozturk@yahoo.com.tr
Available Online Date/Çevrimiçi Yayın Tarihi: 22.06.2012

©Telif Hakkı 2012 AVES Yayıncılık Ltd. Şti. - Makale metnine www.anakarder.com web sayfasından ulaşılabilir.
©Copyright 2012 by AVES Yayıncılık Ltd. - Available on-line at www.anakarder.com
doi:10.5152/akd.2012.163

Conventional and computed tomography angiography views of a rare type of single coronary artery anomaly: right coronary artery arising from distal left circumflex artery

Tek koroner arter anomalisinin nadir bir tipinin konvansiyonel ve çok kesitli bilgisayarlı tomografi anjiyografi görüntüleri: Distal circumfleks arterden çıkan sağ koroner arter

Introduction

Single coronary artery anomaly (SCA) is defined as the coronary artery arising from a single coronary ostium, supplying the entire heart. Although the incidence of coronary artery anomalies ranges from 0.6% to 1.3% in angiography series, the prevalence of SCA was only found to be 0.02% in the population (1). SCA anomalies are usually benign and asymptomatic; however, serious complications such as sudden cardiac death and myocardial infarction resulting from these anomalies were also reported in the literature.

Right coronary artery (RCA) originating from left coronary sinus or proximal portions of left coronary arteries or left coronary system originating from right coronary sinus constitute the major proportion of SCA anomalies.

Herein, we report a case in which the RCA originates from the distal portions of left circumflex artery as a continuum of it. In addition to conventional angiography images; multi-detector computed tomography (MDCT) was used to confirm the diagnosis and determine the course of the anomalous coronary arteries in this case report.

Case Report

A 52-year old woman with hypertension and dyslipidemia was admitted to our clinics with class II exertional chest pain according to Canadian Cardiovascular Society classification. After 2 mm horizontal ST depression in the lateral leads with a Duke score of -10 was revealed on stress electrocardiography, coronary angiography was performed. Single coronary artery ostium was detected in which RCA was arising as a continuum of the left circumflex coronary artery (Fig. 1). To confirm this diagnosis and search for a possible cardiac anomaly, which may explain the patient's symptoms, 64-slice MDCT (Aquilion; Toshiba Medical Systems, Tokyo; Japan) was performed thereafter (Fig. 2). With the help of this method, we confirmed the SCA originating from solitary coronary ostium without an additional cardiac anomaly. The RCA was found to be

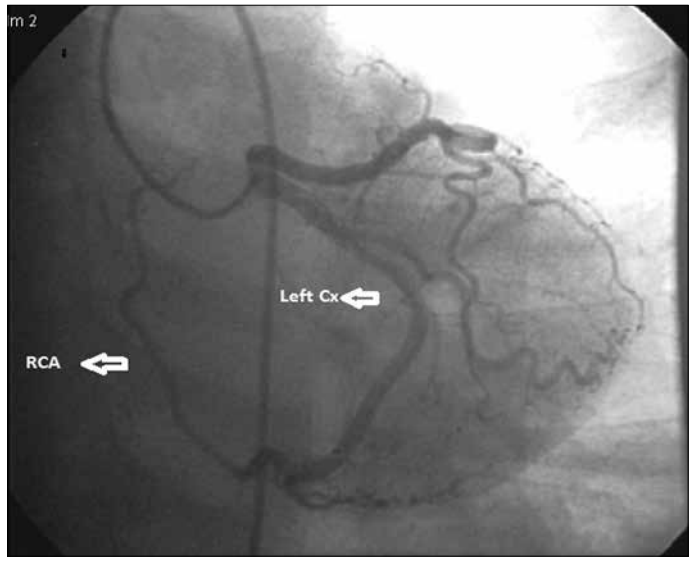

Figure 1. Conventional angiography image of the single coronary artery
Cx - circumflex artery, RCA - right coronary artery

Figure 2. Multidetector computed tomography (64-slice) views of single coronary artery
Cx - circumflex artery, LAD - left anterior descending artery RCA - right coronary artery