Address for Correspondence/Yazışma Adresi: Dr. Turgay Işık
Osmangazi Mah. Erkaraduman sitesi A blok No:22 Palandöken, Erzurum-*Türkiye*
Phone: +90 442 232 58 80 Fax: +90 442 232 50 38
E-mail: isikturgay@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 13.03.2012

©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.2012.077

Author's Reply

Dear Editor,

We would like to thank the author of the letter for kind interest to our article and for sharing their study results. In our case, myocardial infarction (MI) after carbon monoxide (CO) intoxication developed in a patient with underlying coronary artery disease (previous stent implantation) (1). However, in their case MI developed in a young subject with no history of coronary artery disease. They reported that, although successful reperfusion was achieved in infarct related artery, complete recovery of myocardial function cannot be achieved. The causes of these were discussed. We congratulate the authors for this very interesting and important report.

Whatever the status of the patient, immediate reperfusion (preferably primary percutaneous intervention if possible) treatment is mandatory as recommended in guidelines in these patients. Major issue is that, reperfusion treatment may not be enough in CO intoxication induced MI. Oxygen administration or hyperbaric oxygen therapy should be considered in patients with CO poisoning and cardiac toxicity (MI). Because, as reported in the case, successful TIMI-3 flow restoration may not completely salvage myocardium. The reason of is that CO has a more generalized toxic effect on myocardium apart from a limited toxic effect localized at infarct related area.

CO intoxication may cause acute MI in those with or without preexisting CAD, through various complex mechanisms. CO attaches to the hemoglobin (Hb) and blocks the capacity to carry oxygen. Carboxyhemoglobin may cause MI by severe generalized tissue hypoxia (2). Secondly, CO also has direct toxic effect on myocardial mitochondria (3). Thirdly, CO can trigger thrombus formation due to increased platelet aggregability and polycythemia (2). Consequently, CO intoxication may cause acute MI in those with or without preexisting CAD.

We recommend strict electrocardiographic and enzymatic monitoring of all patients in the first hours after CO exposure.

Ercan Varol
Department of Cardiology, Faculty of Medicine, Süleyman Demirel University, Isparta-*Turkey*

References

1. Varol E Özaydın M, Aslan SM, Doğan A, Altınbaş A. A rare cause of myocardial infarction: acute carbon monoxide poisoning. Anadolu Kardiyol Derg 2007; 7: 322-3.
2. Marius-Nunez AL. Myocardial infarction with normal coronary arteries after acute exposure to carbon monoxide. Chest 1990; 97: 491-4. [CrossRef]
3. Weaver LK, Hopkins RO, Elliott G. Carbon monoxide poisoning. N Engl J Med 1999; 340: 1290-2. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Ercan Varol
Süleyman Demirel Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Isparta-*Türkiye*
Phone: +90 246 232 45 10
E-mail: drercanvarol@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 13.03.2012

Diphtheria myocarditis in Turkey after years

Yıllar sonra Türkiye'de difteri miyokarditi

A 34-years-old female without any chronic disease history applied to otorhinolaryngology department with fever, throat pain, dysphagia and unwellness for 3 days. White membranes were seen on uvula and soft palate. The patient was diagnosed as cryptic tonsillitis and peritonsillar abscess and was hospitalized. Throat culture was taken and 1 gr of sulbactam-ampicillin three times per day and 2.5 mg of metamizole sodium four times per day were administered. Because of ongoing fever and unwellness, the patient was consulted to infectious disease department. The result of throat culture was normal so nasopharyngeal swab was taken for microscopic inspection and tularemia, Coxsackie virus A-B and adenovirus IgM and IgG antibodies and diphtheria toxin were prospected. The diphtheria toxin was found positive and 40.000 unit diphtheria antitoxin was given intravenously. Despite of these medications, urine output was decreased, serum creatinine level was elevated up to 3.6 mg/dl and the patient began to experience exertional dyspnea and orthopnea so that cardiology consultation was asked. Blood pressure was 119/79 mm Hg, pulse rate was 108/min and bilateral crepitant rales were detected. Cardiovascular examination was normal except rhythmic tachycardia. ECG revealed ST segment depression in DI-II, aVL and V2-6 leads and ST segment elevation in DIII, aVR and V1 leads (Fig. 1). Echocardiography was performed immediately and global hypokinesia was detected with an ejection fraction of 25%. Cardiac enzymes were examined and creatinine phosphokinase was 1945 unit/L, creatine phosphokinase MB isoenzyme was 213 unit/L and Troponin I level was 49 ng/ml. The patient was transferred to cardiology intensive care unit with a diagnosis of diphtheria myocarditis with permission of infectious disease department. Ventricular tachycardia developed on the second day of intensive care unit and electrical cardioversion was performed because of hemodynamic instability. Later on ventricular tachycardia developed over and over again. Therefore magnesium and amiodarone were administered intravenously and plasma electrolyte levels were checked. In spite of all, her general medical condition was deteriorated increasingly. Cardiac arrest was developed due to intractable ventricular arrhythmia despite of all anti-arrhythmic medications. The patient was resuscitated for two hours but she passed away.

The take-home message from this case is the possibility of reoccurrence of serious diphtheria infections in Turkey after years and diphtheria infections should also come to mind in patients with high fever, sore throat and un-wellness. Multidisciplinary approach may enable early diagnosis and early diagnosis could be life-saving.

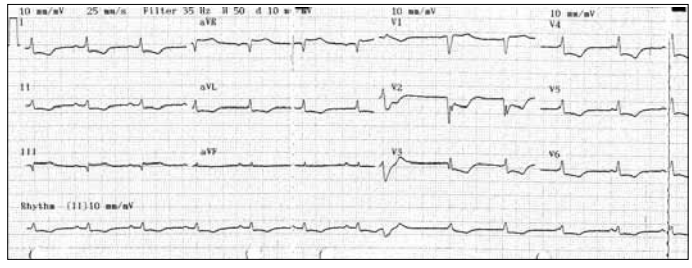

Figure 1. Electrocardiogram recording of ST segment depression in DI-II, aVL and V2-6 leads and ST segment elevation in DIII, aVR and V1 leads

Ender Örnek, Çağın Mustafa Üreyen[1], Alparslan Kurtul, Fatih Öksüz
Clinic of Cardiology, Etlik Training, Education and Research Hospital, Ankara
[1]Clinic of Cardiology, Atatürk State Hospital, Antalya-*Turkey*

Address for Correspondence/Yazışma Adresi: Dr. Çağın Mustafa Üreyen
Uncalı Mah. 23. Cadde 55A Demirel Sitesi Kat:5 No:9 07070 Antalya-*Türkiye*
Phone: +90 242 229 08 86 Fax: +90 242 334 33 73
E-mail: drcaginureyen@gmail.com
Available Online Date/Çevrimiçi Yayın Tarihi: 13.03.2012

©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.2012.078

Mitral valve leaflet strain imaging with echocardiography opens new windows to mitral valve study

Ekokardiyografi `strain` görüntüleme ile mitral kapak yaprakçık gerginliği, mitral kapak çalışmasına yeni pencereler açar

The mitral valve (MV) is a complex anatomical structure consisting of two leaflets, an annulus, chordae tendinae, and papillary muscles. Elucidation of the role of each component and their interactions is critical to improving our understanding of MV function, and to form the developmental basis of new surgical strategies/techniques, so it needs a complete understanding of normal MV dynamics. In addition, repair of the posterior leaflet has been highly successful, while repair of the anterior leaflet has not demonstrated the same level of success due to anatomical complexities of the anterior leaflet (1). It may show some understanding importance of biomechanical behaviors of native MV tissues. The quantification of the mechanical properties of native MV tissues can be used to develop constitutive models, which can be applied to numerical simulations and optimization strategies for repair techniques.

Multiple studies in animal mitral valve tissue showed mitral valve leaflets are stretchable tissue: anterior leaflet experienced large, anisotropic and stretched during closure. Once the valve is closed, further leaflet deformation ceases. These studies suggest that the anterior leaflet does not function as a simple coapting membrane structure; but rather deforms in a complex, finely tuned manner. This information will be useful in furthering our understanding of MV function for both surgical repair and functional tissue engineering (2, 3).

In a recent human study, three- dimensional transesophageal echocardiography was used for evaluation of mitral valve leaflet strain. Serial images of normal human MVs were used to construct models at end diastole and end-isovolumic contraction to detect any deformation during isovolumic contraction. Results showed minimal mitral valve leaflets deformation during isovolumic contraction against other animal study (4). However, in a new study in sheep on cardiopulmonary bypass and with biplane fluoroscopy significant strain of anterior mitral valve was demonstrated (5). It seems this difference can be due to software used in later study.

We measured anterior mitral valve leaflet strain in 12 normal subjects by My-lab 60 equipment echocardiography in apical four-chamber view by velocity vector imaging. In presystolic time, five-point place on anterior mitral leaflet, one point in junction of anterior mitral leaflet and septum and five-point in left atrium as shown in Figure 1. Our results showed maximal mitral valve leaflets deformation during mid-systolic period in some of them with identifiable curve (Fig. 1). We purpose to develop software with ability to measure mitral valve leaflet strain with

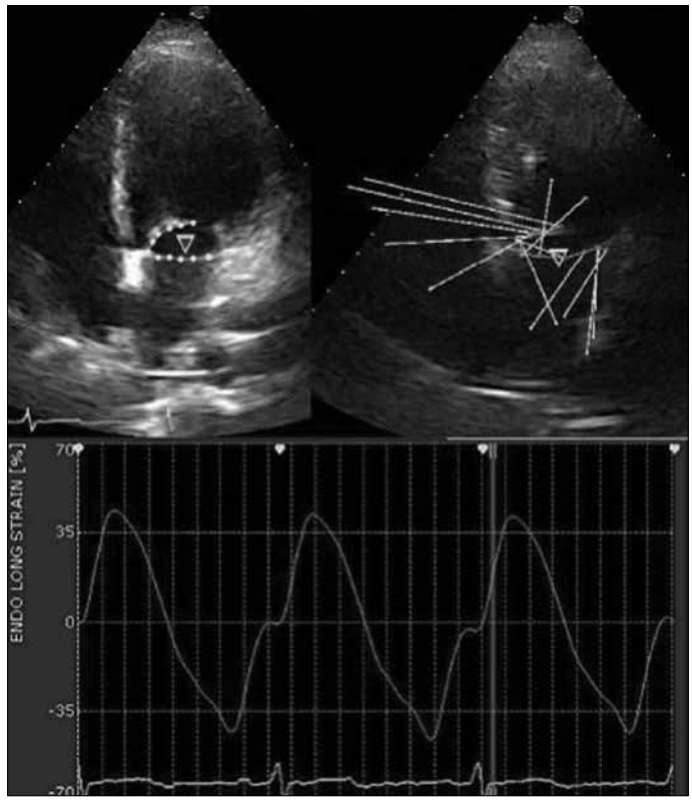

Figure 1. Anterior mitral leaflet strain measurement by velocity vector imaging

echocardiography open new windows to mitral valve mechanic study and mitral valve repair.

Ali Hossein Sabet, Azin Alizadehasl
Department of Cardiology, Madani Heart Hospital, Tabriz University of Medical Sciences, Tabriz-*Iran*

References

1. Hearn TC, Mazumdar J , Hubbard R , Eyster G. Temporal and heart-size effects in first-heart-sound spectra. Med Biol Eng Comput 1979; 17: 563-8. [CrossRef]
2. Grashow JS, Yoganathan AP, Sacks MS. Biaixal stress-stretch behavior of the mitral valve anterior leaflet at physiologic strain rates. Ann Biomed Eng 2006; 34: 315-25. [CrossRef]
3. He Z, Sacks MS, Baijens L, Wanant S, Shah P, Yoganathan AP. Effects of papillary muscle position on in-vitro dynamic strain on the porcine mitral valve. J Heart Valve Dis 2003; 12: 488-94.
4. Xu C, Brinster CJ, Jassar AS, Vergnat M, Eperjesi TJ, Gorman RC, et al. A novel approach to in vivo mitral valve stress analysis. Am J Physiol Heart Circ Physiol 2010; 299: H1790-4. [CrossRef]
5. Rausch MK, Bothe W, Kvitting JP, Göktepe S, Miller DC, Kuhl E. In vivo dynamic strains of the ovine anterior mitral valve leaflet. J Biomech 201; 44: 1149-57.

Address for Correspondence/Yazışma Adresi: Azin Alizadehasl, MD
Department of Cardiology, Tabriz University of Medical Sciences,
Madani Heart Hospital, Daneshgah st., Tabriz-*Iran*
Phone: 8 411 3363880
E-mail: alizadeasl@yahoo.com
Available Online Date/Çevrimiçi Yayın Tarihi: 13.03.2012

©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.2012.079