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 un-wellness for 3 days. White membranes were seen on uvula and soft palate. The patient was diagnosed as cryptic tonsillitis and periostal abscess and was hospitalized. Throat culture was taken and 1 gr of sulbactam-ampicillin three times per day and 2.5 mg of metazime sodium four times per day were administered. Because of ongoing fever and un-wellness, 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 crepitation 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 electric 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.

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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

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

The mitral valve (MV) is a complex anatomical structure consisting of two leaflets, an annulus, chordae tendineae, 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 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 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 of the anterior leaflet (4). It may show some understanding 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.

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

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