



Superficial Dental Intervention Induced Myopericarditis: A Case Report

Yüzeyel Dental Girişimin Tetiklediği Miyoperikardit: Vaka Sunumu

Dental Intervention Induced Myopericarditis

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Öz

Dental girişim sonrasında gelişen miyoperikardit vakası literatürde şimdiye kadar raporlanmamıştır. Biz, yüzeyel dental girişim sonrasında plöritik tipte göğüs ağrısı ile hastaneye başvuran 26 yaşında bir erkek hasta sunduk. Hasta, labaratuvar, elektrokardiyografik ve ekokardiyografik bulguları ile değerlendirildiğinde miyoperikardit tanısı aldı. Miyoperikardit etyolojisinde yer alan diğer nedenler dışlandıktan sonra, dental girişim öyküsü etyolojik neden olarak suçlandı. Bakteriye miye sekonder gelişen kardiyak olaylar nadirdir ve sıklıkla infektif endokardit olarak prezente olur. Biz dental girişim sonrası miyoperikardit kliniği gelişen bir vaka sunduk. Her dental girişim bakteriyemiye neden olabilir ancak her bakteriyemi kardiyak komplikasyonlar ile sonuçlanmaz. Miyoperikardit patogenezinde bakteri invazyonu, toksini yada eksternal uyarılara karşı gelişen immünolojik reaksiyonlar yer alır. Bu bağlamda, her bakteriyemi, immünolojik mekanizmalar aracılığı ile kardiyak komplikasyonlara yol açabilir.

Anahtar Kelimeler

Miyoperikardit; Dental Girişim; Bakteriye mi

Abstract

A myopericarditis case following dental intervention has not yet been reported in the literature yet. We present the case of a 26-year-old man admitted with pleurotic type chest pain following superficial dental intervention. He was diagnosed with myopericarditis based on laboratory parameters and electrocardiographic and echocardiographic findings. Other known causes of myopericarditis were excluded and dental intervention was concluded to be the probable cause of myopericarditis. Cardiac manifestations secondary to bacteremia are rare complications and most present as infective endocarditis. This case reports the first patient presented with myopericarditis following superficial dental intervention. All dental interventions can cause bacteremia; however, each case of bacteremia does not result in cardiac complications. Bacterial invasion, toxins or immune responses to external stimulations act on pathogenesis of myopericarditis. In this regard, it is possible that bacteremia may lead to cardiac complications via immune mechanisms.

Keywords

Myopericarditis; Dental Intervention; Bacteremia

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Introduction

Myopericarditis is an inflammatory process affecting the heart muscle and pericardium. Acute pericarditis is responsible for up to 5% of admissions to the emergency department and may result from drugs, infections, or autoimmune or idiopathic disorders [1]. There are four known mechanisms of myopericarditis etiopathogenesis: 1) direct damage to cardiac myocytes by the microorganism; 2) damage to infected myocyte via immunoresponse to bacteria; 3) cytotoxicity by viral antigen specific immunoreaction; and 4) cytotoxicity via a circulating toxin [2]. Cardiac manifestation secondary to bacteremia is very rare complication following superficial dental interventions. No cardiac complication has been reported in patients following a dental thinning procedure before. In this paper, we report a case of myopericarditis probably having occurred by an immunoresponse to bacteremia which develop following a dental thinning procedure. We evaluate the clinical and laboratory findings of myopericarditis and discuss them with respect to the available literature.

Case Report

We report the case of a 26-year-old man admitted with pleurotic type chest pain. He had no medical history of chronic diseases such as coronary artery disease, valvular heart disease, diabetes mellitus, or hypertension and had no familial history of cardiovascular disease. He didn't have a history of drug intake continuously or recently. He was not a smoker and did not have history of drug addiction. He had no viral or bacterial actual infection symptoms. He had been suffering from pleurotic type chest pain for five days and palpitation for three days before admission to our clinic. His physical examination did not show any relevant findings including fever, lymphadenopathies, rash, arthritis or heart murmurs. His abdominal examination was unremarkable. Laboratory finding were white blood cells count of 8.77 U/L (4.0-10.0 10^3 u/L), lymphocyte count of 1.08 U/L (0.6-3.4 10^3) and hemoglobin levels of 8.63 g/dl (13.2-17.3g/dL). There was an elevation in cardiac enzymes. Creatine-kinase (CK) was 1413 (< 190 U/L), CK-MB was 36 (0.6-6.3ng/dL), lactate dehydrogenase was 405 (< 190 U/L), and troponin I (TnI) was 1.65 (< 0.09 ng/mL). Erythrocyte sedimentation rate was 5 mm/h (<15 mmm/h) and C-reactive protein was 1.0 mg/L (<10). Autoimmune (antinuclear antibody test with IFA was negative) and microbiological studies (EBV VCA IgM, CMV IgM, HSV type I-II IgM with ELISA were negative, influenza IgA PCR was negative, blood cultures were negative) failed to show an etiology for his disease. Electrocardiogram indicated sinus tachycardia and nonspecific changes on inferior derivations (Figure1). Echocardiography showed echogenic focus on inferior segment of myocardium without any pericardial effusion (Figures 2-3). These findings indicated a diagnosis of myopericarditis and the patient was given nonsteroidal-anti-inflammatory-drugs. The patient was discharged after clinical symptoms and laboratory findings were resolved. We learned of a history of a dental thinning procedure two days before the initiation of chest pain according to the patient's detailed anamnesis. After excluding the other causes, we concluded that the etiology of myopericarditis was like related to the dental thinning procedure.

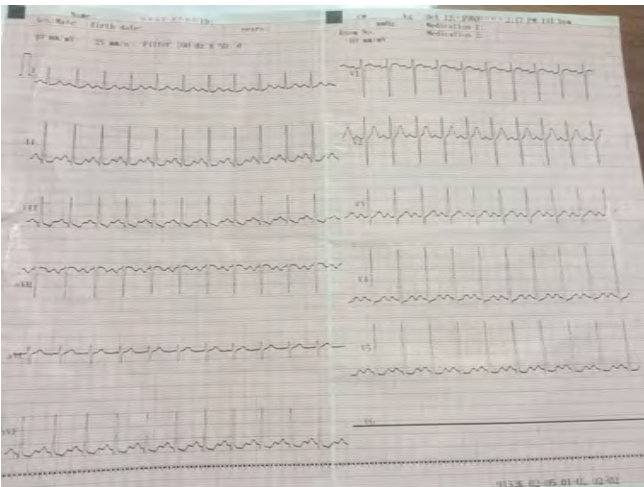


Figure 1. Electrocardiography. Electrocardiogram indicated the sinus tachycardia and non specific changes on inferior derivations.



Figure 2. Echocardiography-Parasternal short axis view. Echocardiography showed the echogenic focus on inferior segment of myocardium.

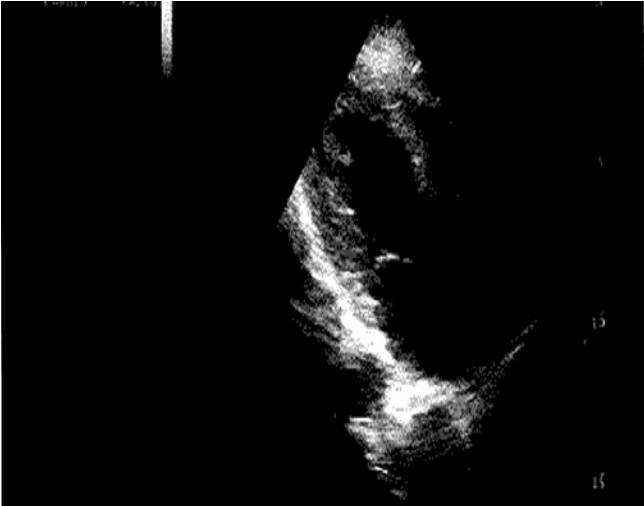


Figure 3. Echocardiography-Parasternal long axis view. Echocardiography showed the echogenic focus on inferior segment of myocardium.

Discussion

Myopericarditis is a myocardial injury added to the pericardial inflammation, on the basement. It can be clinically diagnosed by findings of acute pericarditis and elevated cardiac markers of injury (TnI or TnT, and CK-MB) without depressed left ventricular function by echocardiogram [3]. The pericardium may be affected by all categories of diseases, including infectious diseases such as viral, bacterial, fungal, parasitic, or

autoimmune diseases such as systemic lupus erythematosus, Sjögren's syndrome, scleroderma, Takayasu's arteritis, Behçet's syndrome, or rheumatoid arthritis. It can also be affected by neoplastic diseases such as pericardial mesothelioma, lung and breast carcinoma, lymphoma, or iatrogenic and traumatic conditions such as penetrating thoracic injury, esophageal perforation, radiation injury, or metabolic events such as uraemia and myxoedema. Classic myocarditis, an inflammation of the heart muscle, occurs as a result of the triggering of external antigens including viruses, bacteria, parasites or drugs (anthracyclines, cocaine, clozapine, sulfonamides, cephalosporins, penicillins and tricyclic antidepressants), or internal stimulants such as autoimmune activation against self-antigens [4]. In our case, we concluded that dental intervention was the probable cause of myopericarditis via immunostimulation against bacteria when other known causes had been eliminated.

Cardiac complication associated with dental intervention is mostly shown to be associated with infective endocarditis (IE) in the literature. Incidence of IE is approximately 10 per 100,000 of the population per year. The incidence of IE that is a result of dental treatment is as low as 4% [5]. The majority of patients who develop endocarditis were thought have a pre-existing cardiac defect. More recently, this perception has changed with the information that nearly half of the cases of endocarditis did not have known previous cardiac disease [6]. Guidelines had recommended that antibiotics be administered prior to invasive dental procedures to those patients who with a high risk of endocarditis. However, recent guidance by the National Institute for Health and Care Excellence (NICE) in England and Wales now recommends that antibiotics are not required. Many dental procedures cause bacteremia and this may lead to endocarditis in some people. Bacteria may enter the blood through a variety of portals but generally via mucosal surfaces. The gingiva and periodontal ligament which surrounds all teeth may cause a degree of inflammation and this lead to entry of bacteria to the bloodstream. Indeed, it has been demonstrated that daily activities such as toothbrushing can cause bacteremia [7,8]. In this regard, bacteremia have been not resulted with a cardiac complication mostly. However, underlying immunologic interactions could play a role in cardiac manifestations such as endocarditis or myopericarditis.

In our case we diagnosed myopericarditis by clinical presentation and elevated cardiac enzymes. After excluding the other possible causes including autoimmune disorders, viral and bacterial infections, and drugs used, we suspected superficial dental intervention as an etiologic reason. As a result, we report the first case of myopericarditis that was secondary to superficial dental intervention. All dental interventions could can bacteriemia; however, each case of bacteremia does not result in cardiac complications. In this regard, it is possible that bacteremia may lead to cardiac complications via immune mechanisms.

Conclusion

Dental interventions should be considered in the differential diagnosis of myopericarditis etiology.

Competing interests

The authors declare that they have no competing interests.

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