

was firstly described by Joseph Osborn in 1953 (1). These waves are a deflection with hump configuration occurring at the J point on the ECG. Mostly seen in hypothermia but they may be associated with sepsis, hypokalaemia, hypercalcemia, hypoglycemia, diabetic ketoacidosis, neuroleptic drug abuse, Brugada syndrome, damage to brain and ischemic heart disease (2-4). Recent findings suggest that hypothermia has conduction delay effects but the Osborn waves are directly associated with high CO₂ and low pH levels under hypothermic conditions. Some animal studies demonstrates that, although hypothermia is corrected, Osborn waves still maintain because of low pH levels (5).

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Increased level of cardiac troponin-I due to rheumatoid factor positivity in a healthy patient with normal coronary arteries

Normal koroner damarlara sahip sağlıklı bir hastada serum romatoid faktör pozitifliğine bağlı artmış kardiyak troponin değerleri

Dear Editor,

Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit together with evidence of myocardial ischemia are required for the diagnosis of myocardial infarction (1).

A 56-year-old man was admitted to emergency department with dyspepsia and epigastric pain. Physical examination was normal except a mild tenderness in the epigastrium. He did not have an allergy.

Electrocardiography was normal. Laboratory test were normal except increased troponin-I which was 0.5ng/mL (reference level, <0.01 ng/mL; the diagnostic cut-off for major myocardial injury is 0.4 ng/mL) with normal creatine kinase and creatine kinase-MB fraction and showed no serial increase in subsequent blood tests. Echocardiography showed normal cardiac functions. Coronary angiography demonstrated normal coronary arteries (Fig. 1). The serologic tests for viral etiology were negative for hepatitis B and C virus, human immunodeficiency virus, Coxsackie virus-B, adenovirus and parvovirus B19, Cytomegalovirus and Epstein-Barr virus. His rheumatoid factor status was positive. At one month follow-up visit cardiac troponin-I level was 0.6ng/mL and so interference studies were carried out which showed antibody interference in the troponin assay.

The presence of heterophilic antibodies in high titers may lead to analytical errors in two-site immunoassays (2). Our patient had no recent history of animal exposure, vaccination and antiserum therapy. Rheumatoid factor is a heterogeneous group of auto antibodies that are directed against immunoglobulin-G and presented in the sera of many patients with rheumatoid arthritis, other immune diseases and healthy individuals. It has been reported that 5% of healthy patients might have circulating rheumatoid factor, and approximately 1% of patients with elevated cardiac troponin-I levels may have this elevation solely because of the rheumatoid factor (3). Our patient had rheumatoid factor positivity but we did not have specific agent to remove in our laboratory. So we performed polyethylene glycol which is used to precipitate large immune complexes in the serum as a simple, inexpensive and easy method (4). Treating with polyethylene glycol made the test negative. Although highly specific and sensitive to acute myocardial injury, pulmonary embolism, congestive heart failure, cardiac trauma, cardiopulmonary resuscitation, electrical cardioversion, sepsis, end stage renal disease, arrhythmias, epileptic seizures, stroke and cardiac interventions may also increase cardiac troponins. High sensitive cardiac troponins are novel markers of cardiac injury having high sensitivity and specificity besides providing accurate early diagnosis, risk stratification and screening in compared to standard troponin assays (5). In this patient we exclude all cardiac and non-cardiac reasons of true positivity of troponin levels. Only logical explanation remaining was false positivity of cardiac troponin-I level so we have performed polyethylene glycol for definitive diagnosis, which confirmed our hypothesis.

In conclusion, whenever unexpected cardiac troponin-I results are encountered in a inconsistent clinical picture it is wise to ask to the laboratory for their assistance for the probability of false positive results as immunoassays are widely used nowadays and results from antibody interference cause troublesome consequences, misdiagnosis, unnecessary and expensive procedures to investigate this unexpected laboratory abnormality.

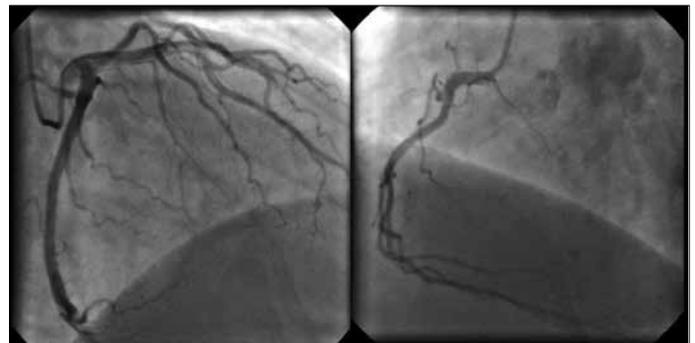


Figure 1. Coronary angiography view of normal coronary arteries

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Multilateral coronary fistulas terminating in the lung parenchyma

Akciğer parenkiminde sonlanan multilateral koroner fistüller 

Dear Editor,

Coronary artery fistulas are rare angiographic findings with a 0.1%-0.2% incidence of all coronary angiograms (1). Most of them remain asymptomatic, however, they can lead to anginal symptoms, acute coronary syndromes, heart failure, arrhythmias as well as sudden cardiac death (1, 2). Majority of the fistulas are unilateral, usually originate from right coronary artery (RCA) and terminate in the low-pressure chambers, frequently in the right ventricle. Multilateral fistulas constitute only 2% of all cases (2, 3). Coronary fistula draining to lung parenchyma was reported with a few case reports and multilateral fistulas terminating in the lung parenchyma was not reported (4, 5).

Herein, we presented a case of multilateral coronary fistulas originating from proximal left anterior descending artery (LAD), circumflex artery (CX) and RCA and terminating in the lung parenchyma with multiple tracts. We also discussed the treatment alternatives for the patient.

A 62-year-old male patient presented with chest pain and shortness of breath lasting a year. He was a heavy smoker and under medication for hypertension. His physical examination was unremarkable and no

murmur heard on auscultation. Electrocardiography showed left axis deviation with normal rhythm. Echocardiography was normal besides left ventricular diastolic dysfunction and exercise stress test revealed inferolateral ST depression in the beginning of stage 3. In the coronary angiography, besides non-significant LAD ostial stenosis and 70% mid RCA stenosis (Fig. 1, 2), multiple coronary fistulas from all three main coronary vessels draining to lung parenchyma was obtained. Fistulas from proximal LAD were originating from two small side-branches and with a further ramification, which terminate in the lung parenchyma as a five tracts (Fig. 1, Video 1, 2. See corresponding video/movie images at www.anakarder.com). Fistula from proximal CX was originating from small side branch and fistula from proximal RCA was originating after sinus node artery and terminating in the lung parenchyma as two tracts (Fig. 2 and Video 3. See corresponding video/movie images at www.anakarder.com). After that, treatment alternatives were proposed either surgical ligation, graft stent deployment or percutaneous micro-coil embolization. Fistulas originating from ostial and proximal LAD were out of the classical surgical borders due to their origin and multiple ramifications. Thus, it might be difficult to identify exact side branches for surgical ligation. Graft stent deployment was seemed to be logical approach because of multiple fistulas. However, graft stent deployment to LAD ostium could yield to residual CX ostial stenosis and also, graft stent to proximal RCA could close the sinus node artery. Thus, percutaneous micro-coil embolization was the only available treatment approach. Percutaneous approach was proposed to patient for both RCA stenosis and RCA fistulas. Patient did not accept the percutaneous treatment and he was discharged with medical treatment.

In conclusion, coronary fistulas terminating in the lung parenchyma are rare clinical entity. It usually presents with anginal symptoms and dyspnea. Hemoptysis is another infrequent but pathognomonic symptom which was absent in our patient. Treatment strategies vary from surgical ligation to percutaneous embolization according to extent and anatomical features of the fistula. Small asymptomatic fistulas could be followed up with medical treatment.

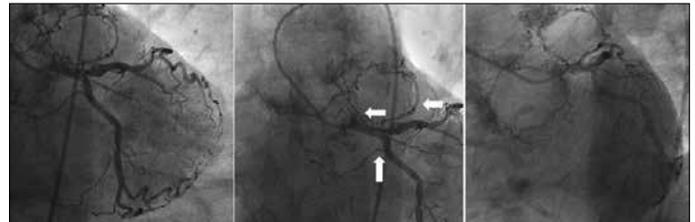


Figure 1. Left coronary system views from spider and anterior caudal projections (white arrows indicate origin of the fistulas in the proximal LAD and CX)
CX - circumflex artery, LAD - left anterior descending artery

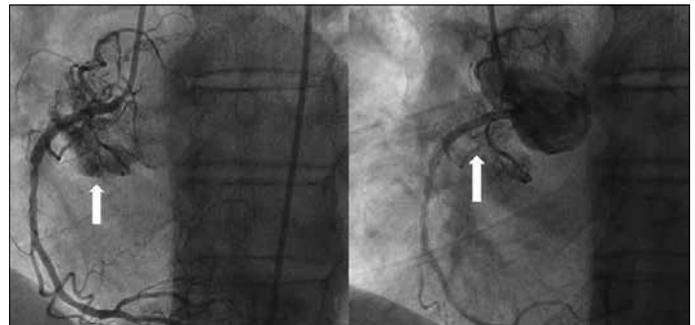


Figure 2. Right coronary artery views from left anterior oblique projection (white arrows indicate origin of the fistula and termination area in the lung parenchyma)