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Review / Derleme

Cardiac myxoma: a rare cause of acute myocardial infarction

Kardiyak miksoma: Akut miyokard enfarktüsünün nadir bir nedeni

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ABSTRACT

Background: This study aims to investigate the clinical features of acute myocardial infarction secondary to cardiac myxoma.

Methods: Data source of the study was based on a comprehensive retrieval of the English literature between the dates January 1st 1990 and August 31st 2014. A total of 48 patients (mean age 45.2±15.3 years; range 9 to 70 years) with acute myocardial infarction secondary to cardiac myxoma were included in the study.

Results: Most patients had an acute onset with chest pain being the prevailing onset symptom accounting for 75.6%. Majority of the patients had elevated serum myocardial enzyme levels. Inferior myocardial infarction was the most frequent followed by anterior myocardial infarction. A normal coronary artery was found in 20 patients (48.8%), coronary occlusive lesion in 21 patients (51.2%), while coronary conditions were not given in seven patients. Cardiac myxoma was diagnosed solely by transthoracic echocardiography in 83.7% of the patients and 93.3% of the cardiac myxomas were in the left atrium. Conservative management including thrombolytic and/or anticoagulant therapies was described in 17 patients (35.4%). Two patients had sudden death without an opportunity for surgical treatment. A surgical resection of the myxoma was performed in 44 patients (95.7%). Percutaneous coronary intervention failed in two patients (40%). As a result, there were 38 (88.4%) event-free survivals, three (7.0%) survivals with disabilities, and two (4.7%) deaths.

Conclusion: In young patients with acute myocardial infarction without apparent risk factors of coronary artery disease, a myxoma-related acute myocardial infarction should be considered and an echocardiographic screening is necessary for a differential diagnosis with conventional coronary artery disease. Coronary angiogram is crucial in identifying the status of coronary emboli and for decision-making for further management. Associated peripheral embolic events frequently warrant an embolectomy.

Keywords: Acute coronary syndrome; cardiac surgical procedures; myxoma.

ÖZ

Amaç: Bu çalışmada kardiyak miksomaya bağlı akut miyokard enfarktüsünün klinik özellikleri araştırıldı.

Çalışma planı: Çalışmanın veri kaynağı 01 Ocak 1990 ve 31 Ağustos 2014 tarihleri arasındaki İngilizce literatürden kapsamlı bir veri edinimine dayandırıldı. Kardiyak miksomaya bağlı akut miyokard enfarktüsü olan toplam 48 hasta (ort. yaş 45.2±15.3 yıl; dağılım 9-70 yıl) çalışmaya dahil edildi.

Bulgular: En yaygın başlangıç semptomu, hastaların %75.6'sında görülen göğüs ağrısı ile akut başlangıç idi. Hastaların çoğunda serum miyokard enzimlerinin düzeyi yüksekti. Anterior miyokard enfarktüsünü takiben inferior miyokard enfarktüs en yaygın olandı. Yirmi hastada (%48.8) normal bir koroner arter, 21 hastada (%51.2) koroner tıkayıcı lezyon bulunurken yedi hastada koroner koşullar belirtilmemişti. Kardiyak miksoma tanısı hastaların %83.7'sinde sadece transtorasik ekokardiyografi ile konuldu ve kardiyak miksomaların %93.3'ü sol atriyumda idi. On yedi hastada (%35.4) trombolitik veya antikoagülan tedavileri içeren konservatif tedavi tanımlandı. İki hastada cerrahi tedavi fırsatı olmaksızın ani ölüm gelişti. Kırk dört hastada (%95.7) miksoma için cerrahi rezeksiyon uygulandı. Perkütan koroner girişim iki hastada (%40) başarısız oldu. Sonuç olarak, 38 (%88.4) olaysız sağkalım, üç (%7.0) engellilikli sağkalım ve iki (%4.7) ölüm vardı.

Sonuç: Koroner arter hastalığın belirgin risk faktörleri olmayan genç akut miyokard enfarktüslü hastalarda miksoma ilişkili akut miyokard enfarktüsü dikkate alınmalıdır ve konvansiyonel koroner arter hastalığın ayırıcı tanısı için ekokardiyografik görüntüleme gereklidir. Koroner embolinin durumunu tanımlamak ve ileri tedaviye karar vermek için koroner anjiyogram çok önemlidir. İlişkili periferik embolik olaylar sıklıkla embolektomi gerektirir.

Anahtar sözcükler: Akut koroner sendrom; kardiyak cerrahi işlemler; miksoma.

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Although systemic embolization due to a cardiac myxoma is frequent, the involvement of the coronary artery leading to occlusion is extremely rare, and the myxoma rarely causes acute myocardial infarction (AMI).[1] Acute coronary embolization may be secondary to infective endocarditis, intracardiac thrombus, prosthetic valve emboli, paradoxical emboli, cardiac myxoma, or calcium deposits from the calcified valves during cardiac interventions or operations.[2] The nonspecific clinical manifestations of cardiac myxomas from one or more of the triad of embolism, intracardiac obstruction and constitutional symptoms[3] make an early diagnosis a challenge. It was reported that 41% of the myxomas had surface thrombus.[4] As the potential sequels of cardiac myxomas can sometimes be life-threatening, an early diagnosis is a key to improved prognosis. To our knowledge, a comprehensive study on cardiac myxoma related AMI is lacking. The present study aims to investigate the clinical features of AMI secondary to cardiac myxoma.

MATERIALS AND METHODS

Comprehensive retrieval of the English literature was performed in the PubMED, Highwire Press and Google search engine from January 1st 1990 to 31 August 2014. The search terms included "cardiac myxoma" and "acute myocardial infarction". There were totally 46 articles[1,2,5-48] involving 48 patients with 22 (46.8%) males and 25 (53.2%) females while gender was not given in one patient. They aged 45.2±15.3 (range, 9-70; median, 46) years (n=47). Only articles published in English language were retained. Articles describing patients with coronary artery disease associated with a cardiac myxoma were excluded from the statistical analysis.

Data were extracted from the texts, figures, or tables and included details of the study population, demographics, diagnosis of the coronary lesion and cardiac myxoma, location of the myocardial infarction and the hypo- and/or akinesis of the ventricular wall, management strategies, prognosis, and follow-up.

Statistical analysis

Data were expressed as mean ± standard deviation with range and median values and were compared by independent sample t-test. Categorical variables were compared by Fisher exact test. A p value of <0.05 was considered statistically significant.

RESULTS

Most of the patients had an acute onset of symptoms. The time interval from onset to presentation was reported in 21 patients: 17 patients (81.0%) presented within 24 hours and four patients (19.0%) presented two days to six months after onset. The mean interval was 254.7±944.7 (range, 0.5-4320; median, 3) hours (n=21). Chest pain was the prevailing onset symptom accounting for 75.6% (Table 1).

Heart murmur was reported in two patients.[29,36] Serum troponin was detected in 16 patients (33.3%) including troponin I in 11 (68.8%) and troponin T in four (25%), while troponin type was unknown in one patient (6.3%). The troponin I on admission was normal in five patients (45.5%) and elevated in six (54.5%) with a mean of 67.6±177.3 (range, 0.00189-570; median, 3.135) μg/L (n=10). Two of these patients had a remarkably elevated troponin I to 307.6 and 31.21 μg/L during the hospitalization. The troponin T values were positive in all four patients with a mean of 3.5±4.3 (range, 0.31-9.37; median, 2.14) μg/L (n=4). Elevated serum enzymes including lactate dehydrogenase 1063.9±754.4 (range, 225-6245; median, 978) U/L (n=8), creatinine phosphokinase 1314.5±1239.8 (range, 45-4000; median, 739.5) U/L (n=16), and creatine kinase MB (CK-MB) 133.3±155.8 (range, 4.5-500; median, 57) U/L (n=10) were also noted.

Three patients (6.3%) had a previous history of cerebral infarct and four patients (8.3%) presented with embolic events (cerebral and multiple peripheral in two, cerebral in one, and femoral embolic events in one patient).

Four patients (8.3%) deteriorated into cardiogenic shock (which required intraaortic balloon pump) with pneumonia, severe pulmonary edema, multiple organ failure or ventricular tachycardia in each (25%).

Locations of AMI were evident by electrocardiogram in 37 patients, where the most frequent site of AMI was the inferior wall followed by the anterior (Figure 1). Echocardiography was inspected before

Table 1. Onset symptoms

Onset symptoms	n	%
Chest pain (concurrent dyspnea, syncope, dizziness, mental status alteration, and dizziness plus mental status alteration in one each)	31	75.6
Chest discomfort	3	7.3
Dyspnea	2	4.9
Epigastralgia	2	4.9
Dyspnea, aphasia, and weakness	1	2.4
Hemiparesis	1	2.4
Palpitation	1	2.4

coronary angiogram in 31 (70.5%) (coronary angiogram was not performed in one of them), coronary angiogram first in 12 (27.3%), and coronary computed tomographic angiogram first without coronary angiogram in one (2.3%) patient. Three (25%) of the patients with coronary angiogram first had a delayed diagnosis of cardiac myxoma, and one (33.3%) of them deteriorated rapidly. In two patients, embolectomy was performed for the peripheral embolic events and pathologically myxomatous nature of the excavated samples led to the suspicion of cardiac myxoma.

Coronary lesion was confirmed by autopsy in two patients and by coronary angiogram in 39 patients. A normal coronary artery was found in 20 patients (48.8%) and coronary occlusive lesion in 21 patients (51.2%). The culprit coronary arteries included eight circumflex arteries (38.1%), six right coronary arteries (28.6%), five left anterior descending coronary arteries (23.8%), one left anterior descending coronary artery and right coronary artery (4.8%), and one not stated culprit coronary artery (4.8%). Neovascularization of the cardiac myxomas was noted in eight patients (66.7%) who underwent a coronary angiogram with a supplying artery of the right coronary artery in (62.5%) patients, circumflex artery in two (25%) patients, and in one (12.5%) patient whose supplying artery was unknown.

Of the 20 patients with a normal coronary angiogram, seven were males and 13 were females with no sex predominance (χ^2=3.6, p=0.113). The patients with a normal coronary angiogram was younger than those with a myxoma thrombosed coronary artery with no statistical significance (42.9±15.6 years vs. 48.6±17.4 years, p=0.324).

Cardiac myxoma was diagnosed by transthoracic echocardiography in 36 (83.7%), transthoracic and transesophageal echocardiography in four (9.3%) (inconsistent results of both transthoracic and transesophageal echocardiogarphy were noted in one patient), and computed tomography aortogram, transthoracic and transesophageal echocardiography and magnetic resonance imaging and intraoperative transesophageal echocardiography in one (2.3%) each. Hypo- and/or akinesis of the ventricular wall was observed by echocardiography in 28 patients (45.9%), the inferior wall being the most frequently involved (Figure 2). Moreover, five patients (10.4%) had urgent coronary angiography[2,13,18,21,40] and one patient (2.1%) had urgent cardiac computed tomography[28] done for the diagnostic purpose of the coronary lesions.

Of the 45 cardiac myxomas with a location reported, 42 (93.3%) were in the left atrium (one of them was on the mitral valve) and one (2.2%) each in the left ventricle, both left atrium and right atrium, and on the aortic valve. Nineteen (44.2%, 19/43) of the atrial myxomas prolapsed into the left ventricle during diastole. At least 13 (27.1%) myxomas were pedunculated. Fourteen (82.4%) myxomas were regular and three (17.6%) irregular on gross appearance. Dimension of the myxomas was 44.7±19.5 (range, 10-90; median; 46) mm (n=31). Three patients (6.3%) had a hemorrhagic myxoma.

Conservative management strategies were described in 17 patients (35.4%). Ten (58.8%) of them were performed thrombolysis with recombinant tissue plasminogen activator (n=3), streptokinase (n=2), tenecteplase (n=2), urokinase (n=1), and agent unknown (n=2). In one patient, thrombolytic therapy failed in recanalization of the coronary artery and heparin was used instead. In the patient who was administered bisoprolol, acetylsalicylic acid, perindopril, fragmin, and atorvastatin were used thereafter. In the seven patients without receiving a thrombolytic therapy,

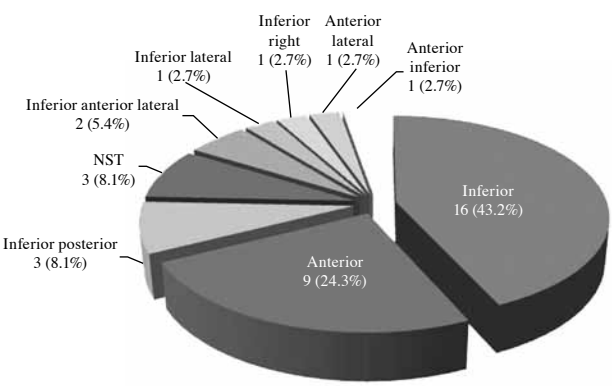

Figure 1. Distribution of myocardial infarction sites. NST: Non-ST segment elevation myocardial infarction.

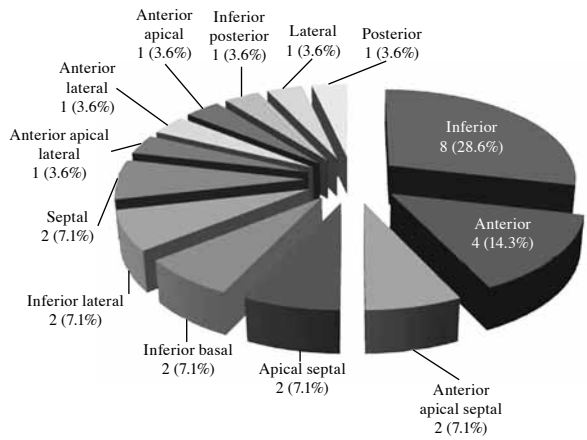

Figure 2. Distribution of hypo- and/or akinesis of the ventricular wall observed by echocardiography.

a coagulate therapy by acetylsalicylic acid, heparin, acetylsalicylic acid, clopidogrel, fragmin, or fibrinolytic agents was given.

Treatment was not described in two patients. Of the remaining 46 patients, two patients (4.2%) died suddenly and without an opportunity of surgical treatment. A surgical resection of the myxoma was performed in 44 patients (95.7%): a sole resection of the myxoma in 24 (54.5%), with concurrent coronary artery bypass grafting in six (13.6%), with concurrent percutaneous coronary intervention in five (11.4%), with concurrent coronary artery bypass grafting and atrial septal defect closure in one (2.3%), coronary artery bypass grafting and staged surgical embolectomy in one (2.3%), and staged surgical embolectomy in seven (15.9%) patients. An urgent cardiac surgery was performed in two patients (4.2%).[15,42] A delayed myxoma resection was performed in at least 10 patients (22.7%) for 19.6±10.8 (5-35; 19.5) days. Totally, eight patients had a staged surgical embolectomy, for the embolized femoral artery in two (25%), and for the emboli of the aorta, left anterior descending coronary artery, circumflex artery, right coronary artery, extremity artery and multiple sites in one (12.5%) each. In addition, there were three amputations, two fasciotomies, and one left ventricular aneurysmectomy. Percutaneous coronary intervention failed in two patients.

Five patients were under postoperative follow-up with a mean of 21.0±11.6 (range, 7-36; median, 24) months (n=5). Prognoses of 43 patients were presented, with 38 (88.4%) event-free survivals, three (7.0%) survivals with disabilities, and two (4.7%) deaths.

DISCUSSION

Cardiac myxomas are categorized into two types according to their gross appearances: type 1, with an irregular or villous surface and a soft consistency; and type 2, with a smooth surface and a compact consistency.[49] Type 1 myxoma seemed to be slightly more common (59.5%) than type 2 (41.5%).[50] Papillary or villous myxomas are more fragile and embolize more often than myxomas with a smooth surface.[10] The incidence of systemic embolism was higher with the polypoid type compared with round type (58% vs. 0%, $p<0.05$).[11] About two-thirds of the myxomas are smooth surfaced and globoid, and they seldom embolize. In minority of these, thrombus may form on the surface of the cardiac myxomas by repeated impingements against the atrial wall or the valve leaflets, and therefore embolic events frequently occur. Type 2 myxoma accounting for about one-third of myxomas is extremely fragile and predisposes to embolic events frequently.[51] After all, the type of the myxoma with embolic predilections remains speculative.[50] Mobility and friability rather than tumor size are the greatest risk factors for cardiogenic emboli from a cardiac myxoma.[52]

Apart from the embolic potentials from the myxomatous or thrombus materials detached from the myxoma, another plausible hypothesis for AMI in association with cardiac myxoma is a highly vascularized mass in the cardiac chamber referring to a coronary steal phenomenon.[1] The potential predilection of inferior AMI caused by a cardiac myxoma is not known.[10] The rarity of coronary embolization could be ascribed to the natural protection offered by normal anatomy of the ostia and the coronary circulation.[10] The underlying anatomy of the right-angled junction of the ostium of the coronary arteries was thought to play a role in the low incidence of coronary embolism.[53] The caudally situated right coronary artery may be responsible for the predilection of cardiac myxoma-related coronary emboli.[9]

As a rare cause of AMI, cardiac myxoma is elusive and establishing the diagnosis can be challenging. In patients with peripheral embolism, the diagnosis of a cardiac myxoma source of embolus is often missed until pathology of aspirated or removed materials reveals myxomatous tissues.[24] Therefore, the check-out of the myxoma-embolic sources by echocardiography plays an important role in reaching an early diagnosis.[10]

The electrocardiogram changes are often non-specific; up to 33% may have left atrial hypertrophy. Chest X-ray may show left atrial enlargement or the presence of calcification in the tumor mass. The investigation of choice is transesophageal echo with a 100% sensitivity or transthoracic echocardiography that is 95% sensitive.[15] In patients with a suspicion of atrial myxoma, echocardiography is a reliable way for diagnosis.[12] The diagnosis of a cardiac myxoma-related AMI relies on coronary angiogram. A complete occlusion of the coronary artery without evidence of atherosclerotic obstruction,[54] a tumor blush as a consequence of marked tumor neovascularization,[10] or a filling defect [38] can be visualized during coronary angiogram. Neovascularization of the tumor mass may give a clue to its presence during cardiac catheterization.[8]

A differential diagnosis has to be made with coronary artery disease associated with a cardiac myxoma. As indicated previously, cardiac myxoma-related AMI often shows a sudden onset of chest

pain with elevated cardiac enzymes including cardiac troponins. The patients may lack a sign of coronary atherosclerosis on coronary angiogram but show a tumor brush or a filling defect. In such patients, a coronary surgery or intervention is only necessary in half of the patients.[10] However, patients with coronary artery disease associated with a cardiac myxoma can be even rarer.[55-58] They may have a history of coronary artery disease for years before the diagnosis of a cardiac myxoma is established.[57] Coronary atherosclerotic changes are evident on coronary angiogram,[55,58] whereas tumor brush or filling defect sign is absent. The patients are indicated for coronary artery bypass grafting with concurrent tumor resection.[55]

The present study revealed that 48.8% of this patient setting showed a normal coronary artery by coronary angiogram, significantly higher than the general population with AMI.[59] The underlying causes of the normal coronary angiogram in patients with cardiac myxoma-related AMI remain uncertain. However, it has been suggested that higher spontaneous recanalization rates of the myxomatous embolization should be a possible explanation.[7] The rate of spontaneous recanalization was fast and suggested that the breakdown of the myxomatous materials was rapid.[11]

Al Zahrani et al.[7] also hypothesized that the patients with a normal coronary angiogram were younger than those with myxoma-thrombosed coronary arteries. The present study revealed a 5.7-year age difference between the two groups with no statistical significance.

Intravenous or intracoronary thrombolysis is a treatment of choice for myxoma-related AMI, but failure to recanalize the culprit coronary lesion was once reported.[40] Otherwise, administration with heparin or alternative anticoagulants might be an option in those who are not indicated for or failing in thrombolytic therapy. A finding of acute coronary occlusion by coronary catheterization may be followed by an intracoronary aspiration of the thrombus, thereby avoiding the need of coronary artery bypass grafting.

Cardiac myxomas are indicated for an early removal in order to avoid further embolic events.[11] Cardiac myxomas produce constitutional symptoms by releasing interleukin-6 and depressing heart function. Without surgery, there is little opportunity to counter the effect of any tumor-mediated cardiac depressant effects. Reversal of cardiac dysfunction after surgical removal of myxoma suggests a myocardial depressant effect of myxoma not related to coronary occlusion.[54]

In conclusion, a myxoma-related acute myocardial infarction should be considered in young patients with acute myocardial infarction without apparent risk factors of coronary artery disease, and a screening echocardiography is necessary for a differential diagnosis with conventional coronary artery disease. Coronary angiogram is crucial in identifying the status of coronary emboli and for decision-making for further management. Associated peripheral embolic events frequently warrant an embolectomy.

Declaration of conflicting interests

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