A PROSPECTIVE STUDY OF KOUNIS SYNDROME: CLINICAL EXPERIENCE AND CARDIAC MAGNETIC RESONANCE IMAGING FINDINGS FOR 21 PATIENTS

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ABSTRACT

Background: *Thus far, there are only case reports about allergic angina (Kounis Syndrome, KS). Neither incidence nor imaging findings have been reported.*

Aim*: To determine the incidence of KS and evaluate the role of cardiac magnetic resonance imaging (CMRI) in detecting KS among allergy patients in the emergency department (ED).*

Material and Methods: The study population included patients over 18-year-old suffering from KS. Detection of pathologies on at least one of the electrocardiogram (ECG), cardiac enzyme, or CMRI tests was identified as the criterion for KS. As part of the CMRI procedure, T2-weighted imaging, early gadolinium enhancement, and late gadolinium enhancement were performed.

Results*: Incidences of KS in all admissions and among allergy patients were 19.4 per 100 000 and 3.4%, respectively. The most common etiology was the use of medications (81%). Of the patients, 47.6% described palpitations, 71.4% chest pressure, 52.4% shortness of breath, and 14.3% syncope. Examinations showed that 76.2% of the patients had hives, 47.6% oedema of the uvula, 24% angioneurotic oedema, and 19% hypotension. CMRI showed early-stage subendocardial contrast involvement and oedema in T2-weighted images in all patients, pericardial effusion in 28.6%, and hypokinesis in the contrast defect site in 38.1%. The ECG results for 4 patients (19.1%) were normal, and only 4 patients had a high troponin level.*

Conclusion*: These patients almost always present with reversible changes in the CMRI. The left ventricular lateral wall and the septal region of the heart are particularly sensitive to KS according to the CMRI findings*

Key words*: Kounis Syndrome; angioneurotic oedema; acute coronary syndrome; cardiac magnetic resonance imaging; hives; urticeria.*

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Introduction

The clinical picture of myocardial ischemia accompanying allergic reactions was first recognized in 1991[(1)] and is defined as Kounis syndrome (KS) or allergic angina[(2)]. KS is rare, and the exact incidence is unknown as emergency doctors are unaware of the diagnosis and no prospective trial has been performed[(2)]. The mechanism of an allergic acute coronary syndrome is thought to be due to coronary spasm or mast cell activation in the heart tissue as a result of systemic or local increase in allergic mediators[(2)]. Respectively, these mediators trigger coronary spasm or induce plaque rupture and coronary or stent thrombosis[(3)].

Anamnesis, clinical findings, electrocardiography (ECG), and cardiac enzymes are important in the diagnosis of KS. Histamine, tryptase, and skin tests showing allergy are also used. Percutaneous coronary intervention (PCI) is commonly used in patients who are admitted to our Acute Coronary Syndrome (ACS) clinic, and coronary vessels reported to be normal in type 1 KS rendered frequent use of this invasive test in these patients questionable. Computerized tomography angiography is a non-invasive test. However, this procedure, similar to PCI, needs contrast material injection that may cause additional allergic reactions in atopic and sensitive patients; moreover, these procedures expose patients to radiation, and the coronary arteries are generally found to be normal even in the event of changes in ECG results[(4)]. The use of non-invasive and radiation-free cardiac magnetic resonance imaging (CMRI) offers a unique combination

of safety, clarity of anatomical visualization, and quantitative accuracy of functional and morphologic cardiac assessment[5,6]. CMRI allows the early detection of ischemic heart disease and differentiation of nonischemic disorders[6]. We have previously discussed the importance of CMRI in KS and reported that lesions could be shown on CMRI even if ECG and troponin were normal in type 1 KS[7].

This prospective study aims to determine the incidence of KS among allergy patients admitted to our hospital's emergency department (ED) and to identify findings associated with KS in cardiac MRI.

Methods

Study population

The patients consented to the use of their imaging data in this study, and approval was obtained from the hospital's Ethics Committee (Reg. no: B.30.2.ATA.0.01.00/134). The study population included patients over 18-year-old, admitted to the ED between January 1, 2012, and December 31, 2012, with a suspected diagnosis of clinical KS (presence of chest pain presenting with allergy symptoms), who signed the informed consent document, and for whom contrast CMRI scans were performed within 24 hours. Detection of pathologies in at least one of the ECG, cardiac enzyme, or CMRI tests was identified as the criterion in addition to clinical examination for diagnosing patients with KS. This study intended to exclude patients with previously known coronary artery disease, previous PCI, or CABG history presenting with allergic angina, and patients with wall motion abnormality such as akinesia or aneurysm recorded on transthoracic echocardiography. However, no patients met these criteria.

A total of 138,911 patients were admitted to the ED during a period of 1 year, with 793 admitted with complaints of allergy. Twenty-nine of the 793 patients had chest pain. No CMRI scans were performed for 6 patients (due to claustrophobia or no desire for CMRI), and KS was not suspected in 2 of the remaining 23 patients as their ECG, cardiac enzymes, and CMRI were normal. The study finally included 21 patients. Of them, 2 patients had chronic renal failure, 2 patients had hypertension, 1 patient had allergic asthma, and 1 patient had depression in their medical history.

Definition

The presence of chest pain (or angina equivalent such as chest pressure) with allergic findings in the form of urticarial and/or angioedema was defined as clinical KS. Hemogram, biochemistry, CK-MB, and cardiac troponins were collected. Cardiac biomarkers and ECG follow-up were performed every 6 hours. Patients with a high troponin level (n=4, >0.60 μg/L at our own laboratory) were taken into the catheterization laboratory to eliminate the possibility of an ACS.

Cardiac MRI procedure: CMRI was performed for all patients within 24 h. MRI was performed on a 1.5 T scanner (Siemens AG, Magnetom Avanto, 2008- Germany) using a 16-channel cardiac coil. Patients were placed in a relaxed supine position, and after a rapid survey to determine the exact heart axis, cine loops in short-axis (SA) and 3 long-axis (LA) planes (4-, 3-, and 2-chamber view) were acquired for further planning. Short-axis cine SSFP images were acquired to cover the entire LV. The imaging parameters were as follows: image size, 256×256; contiguous sections, 10-14; section thickness, 8-10 mm; slice gap, 0 mm; and number of phases, 20. CMRI was started 10 min after 0.2 mmol/kg of gadopentetate dimeglumine (Gd-DTPA, Magnevist, Bayer Healthcare, Germany) was injected. Late gadolinium enhancement (LGE) imaging involves T1-weighted inversion-recovery imaging approximately 10 min after intravenous administration of gadolinium contrast. All CMRI images were evaluated by MK and YK, expert radiologists.

Statistical analysis

Normally distributed numerical variables are shown as mean±standard deviations, non-normally distributed numerical variables are shown as medians (minimum-maximum), and categorical variables are shown in percentages. The incidence of KS is described as the ratio of the number of patients admitted to the hospital's ED during a period of 1 year and clinically diagnosed with KS to the total number of admitted patients. The definitive statistical analysis of this study was made using SPSS version 20.0 for Windows (Chicago, Illinois) statistical software.

Results

A total of 138,911 patients were admitted to the hospital's emergency department in 2012. Of

these patients, 793 presented with complaints of allergy (769 with urticaria and 24 with angioneurotic oedema). The incidence of allergy admissions in 2012 was 5.7 per 1000 for the unit. The incidence of KS at the ED in 2012 among all admissions and allergy patients was 19.4 per 100 000 (27/138,911) and 3.4% (27/793), respectively.

Eleven (52.4%) of 21 patients included in this study were male. The patients' ages ranged between 18 and 60 (median: 37). The patients were most often admitted in February (4 patients, 19%) and November (4 patients, 19%). Autumn and winter were the seasons of highest admissions (8 patients, 40%, and 8 patients, 38.1%, respectively). Possible factors were taking pills in 17 (81%) patients (5 patients antibiotics + analgesics, analgesics in 5 patients, antibiotics in 4 patients, antibiotics + lansoprazol in 1 patient, lansoprazol in 1 patient, antidepressant in 1 patient), hair dye in 2 (10%) patients, bee sting in 1 (5%) patient, and food allergy in 1 (5%) patient.

All patients described chest pains, with 15 (71.4%) describing a feeling of pressure on the chest, 11 (52.4%) shortness of breath, 10 (47.6%) palpitations, 10 (47.6%) uvula oedema, and 3 (14.3%) syncope. Sixteen (76.2%) patients had hives, 5 (24%) patients had angioneurotic oedema, and 4 (19%) patients had hypotension. Leucocytosis was identified in 67% of the patients. The patients stayed at the hospital for a median 24 h (min 7 h, max 96 h). Sixteen (76.2%) of the patients were followed in the ED, 4 (19.1%) were hospitalized in the cardiology unit, and 2 patients (9.5%) with chronic renal failure were hospitalized in the nephrology clinic. Cardiac catheterization findings for the 4 patients hospitalized in the cardiology unit were normal. All patients were discharged in good condition.

Although 9 (43%) patients presented with ST elevation in ECG, 8 (38.1%) patients had non-specific ST-T changes, 7 (33.3%) patients had tachycardia, 2 (10%) patients had bradycardia, 1 (4.8%) patient had paroxysmal AF, and 4 (19.1%) patients had normal ECG findings. A more detailed ECG analysis is given in Table 1. During observation, the ST segment elevations resolved in all patients.

CMRI showed early phase subendocardial contrast defect and oedema in T2-weighted images in all patients. Subendocardial contrast defect was in the (Fig. 1) septum in 11 (52.4%) patients, the left ventricular free wall in 6 (29%) patients, and the left ventricular apex in 4 (19.1%) patients. In addition to these findings, 8 (38.1%) patients had hypokinesia in the contrast defect site, and 6 (28.6%) patients had pericardial effusion (Table 1).

Age, Gender	İnitial ECG	Location of defect in the early phase of contrast in CMRI	Pericardial effusion	Hypokinesia	Initial Trop I
20, M	ST elevations (2mm) in D2, 3, avF, V4, 5, 6 ST depressions in V1, 2	Proximal septum	+	-	7.97
51, F	Sinus tachycardia, Poor R wave progression in anterior leads	Interventricular and apical septum	+	-	.10
48, F	Paroxysmal AF; ST elevations (1mm) in V1, 2, 3, 4, 5	Midventricular septum	-	+	.14
45, F	ST elevations (1mm) in V2, 3	Interventricular and apical septum	+	-	.85
35, F	ST elevations in V2, 3, 4 (1mm) and V5, 6 (0.5 mm)	Midventricular septum	-	-	.11
37, F	Sinus tachycardia, normal ECG	Apical septum	-	+	.11
27, F	Poor R wave progression in anterior leads	Apex of LV and Interventricular septum	-	+	.22
18, M	Sinus bradycardia, T wave negativity in V3, 4, 5, 6 P wave negativity in D2, 3, avF.	Midventricular septum	-	-	.10
26, F	T wave negativity in V1, 2, 3 voltage drop in extremity derivations	Interventricular septum	-	+	.21
54, F	Normal ECG	Interventricular septum, subendokardial	-	-	.10
27, M	ST elevations in V3, 4, 5, 6 (1mm)	LV free wall	+	-	.18
37, M	Sinus tachycardia, ST elevations in V1, 2, 3 ,4, 5 (1mm)	LV apex	-	-	.10
46, M	ST elevations in V2, 3 ,4, 5, 6 (1mm)	LV apical septum	-	+	.10
53, M	Normal ECG	LV apex and apicoposterior septum	+	+	.12
19, M	Sinus bradycardia, ST elevations in D2, 3, aVF (2mm) and V2, 3 (2mm)	Proximal part of LV free wall	+	-	3.95
60, M	Non-specific ST-T changes	Proximal part of LV free wall	-	-	.10
57, F	Non-specific ST-T changes	Mid ve apikal part of Interventricular septum	-	-	.10
18, M	Sinus tachycardia, T wave negativity in D3, ST elevations in D1, 2 (1 mm) and V2, 3, 4, 5 (2 mm), sharp T in V2 (13 mm)	LV free wall close to apex	-	+	21.09
49, F	Sinus tachycardia, Poor R wave progression in anterior derivations	LV free wall close to apex	-	-	.00
19, M	Sinus tachycardia, early repolarization in V3, 4	LV free wall close to apex	-	-	.01
55, M	Sinus tachycardia, Normal ECG	Midventricular septum and apex	-	+	.00

Table 1: ECG, cardiac magnetic resonance imaging and initial troponin results of the patients.
LV: left ventricle; CMRI: cardiac magnetic resonance imaging

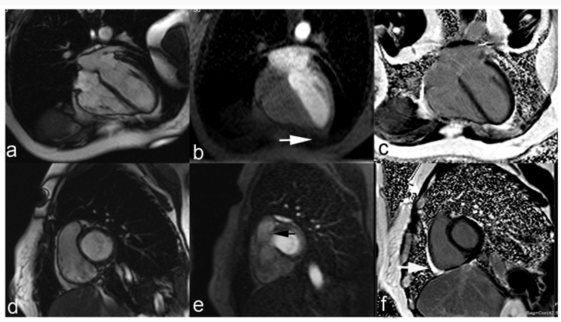

Fig. 1: a) T2-weighted, b) early gadolinium enhancement, and c) late gadolinium enhancement CMRI findings of a 27-year-old man presenting with chest pain due to drug allergy. In the early gadolinium enhancement view, the contrast defect in the apex is highlighted. d) T2-weighted, e) early gadolinium enhancement, and f) late gadolinium enhancement CMRI findings of a 45-year-old woman presenting with chest pain due to hair dye allergy. In the early gadolinium enhancement view, the contrast defect in the septum is highlighted. This contrast defect disappeared in the late series. Also noted is the pericardial effusion in the late images.

Discussion

To the extent of our knowledge, this is the first prospective study investigating the clinical demographics, electrocardiographic, and CMRI properties of patients with KS. This study aimed to determine the incidence of KS for the first time and presents CMRI findings for KS.

Today, anaphylaxis and KS diagnoses are two challenges for allergy patients[2]. Cardiovascular symptoms are important in the current definition of anaphylaxis, since reduced blood pressure is the only clinical finding that alone allows diagnosis of anaphylaxis after exposure to a known allergen[9]. Even if cutaneous, mucosal, or respiratory symptoms are often needed to establish a firm diagnosis of anaphylaxis[9], a study reported that these symptoms are not present in a large part of a group of 25 patients who died of anaphylaxis[10]. Our study found no skin findings (hives or angioneurotic oedema or mucosal symptoms) and/or hypotension in 3 patients (14.3%). A diagnosis of KS was considered following start of chest pain in 2 of these patients (aged 20 and 18, respectively) due to drug intake, high troponin value of 7 and 21, respectively, ST elevation on the ECG, and normal angiographic findings. The third patient (age 48) had a history of allergic asthma. The ECG for the patient who described angina and dyspnoea after a bee sting showed ST elevation in anterior derivations and paroxysmal AF, with normal cardiac enzymes. That is, skin and mucosal findings are not a requirement in all patients considered to have KS in the ED examination. Suspicion secondary to anamnesis is important in detecting these patients. However, PCI is inevitable in this patient group particularly if the troponin level is high.

On ECG, ST elevations in anterior and inferior leads have been the most reported findings in patients diagnosed with KS, although the ECG may be normal, or merely show nonspecific ST-T wave changes[11]. Our study findings showed normal or nonspecific ST-T changes in the ECG in 12 (57.1%) of our patients, and that the ST elevations were not high in inferior derivations as mentioned above in 3 (14.3%) patients. Our study found ST elevation in diffuse anterior derivations in 5 (24%) patients, in inferior + anterior derivations in 3 (14.3%) patients, and in septal derivations in 3 (14.3%) patients (Table 1). Interestingly, ST elevation in anterior derivations did not occur in 29% of the >2 mm amplitude patients in the AMI diagnosis criteria. In a previous case report, we hypothesized that, unlike ACS, ST elevation might not correlate with troponin I levels in KS[7]. Now, we have more evidence for this statement. Additionally, there is no need to calculate or try to achieve significance considering ST elevation is over 2 mm in anterior derivations, and over 1 mm in other derivations in patients considered having KS, as in AMI diagnosis criteria. In addition, based on the CMRI findings, ST is elevated on ECGs for patients with type 1 KS due to the formation of subendocardial effect rather than a transmural lesion as in STEMI.

One other issue of concern was whether the derivations in the pathologies observed on the ECG complied with the sites found to have pathologies on the MRI. CMRI explained the pathology found in 71.4% of the patients on the ECG imaging. In 19% of the patients, although the ECGs were normal, physiopathological changes were documented in CMRI.

CMRI not only shows myocardial perfusion to detect myocardial ischemia[12] but also defines the morphology of the myocardial tissue[13]. Thus, CMRI defines the type of myocardial injury whether ischemia or inflammation, acute or chronic, and reversible or irreversible[6]. There are three CMRI modalities: T2-weighted imaging, early gadolinium enhancement (first-pass perfusion imaging), and late gadolinium enhancement. T2-weighted imaging helps detect myocardial oedema, which is present in the acute stage of myocardial injury and represents the area compromised by ischemia[6]. We observed this oedema in the T2-weighted series of all patients with type 1 KS.

First-pass perfusion imaging assesses myocardial blood flow and ischemia. According to the wave front phenomenon[14], ischemic lesions mostly affect the subendocardial layer, while extension to the subepicardial portion is variable, and usually the distribution corresponds to the coronary territories. In contrast, nonischemic lesions-such as in inflammatory heart disease (myocarditis) or cardiomyopathies-are predominantly located in the subepicardial or middle portion of the myocardium with a more patchy distribution that is independent of the coronary territories[6,15]. Our study showed early-stage subendocardial contrast defect in all patients, which is an indicator of ischemia. Typical myocardial biopsy findings in KS also support this finding[8,16,17]. In our opinion, the cause of ischemia appears to be microvascular obstruction (MVO). MVO can be summarized as reperfusion injury and

is the consequence of clogging of small myocardial arterioles with embolic debris, acute inflammation, platelet aggregation, and vasospasm[18]. In our study, subendocardial contrast defect was in the septum in 52% of the patients, and in the left ventricular free wall or apex in 48% of the patients. Kounis-related studies conducted without using MRI and based on ECG data suggest that the right coronary artery is the vessel most often affected by vasospasm[8]. However, our study shows that the septum and the left ventricle were most often affected by ischemia. When we tried to theorize why the cardiac septum was sensitive to KS, we found that the formation theories of post-traumatic stroke disease, which is a very different injury and is seen in children less than 7 years of age, could create a similarity to KS. Cerebral infarction after minor head injury is characterized by infarction of the basal ganglia-internal capsule due to occlusion of the lateral lenticulostriate artery[19]. The lateral perforating branches of the middle cerebral artery follow a recurrent course to reach their points of penetration in the anterior perforating space; their angle of origin is very acute[20]. Stretching and distorting the angle of perforating branches lead to damage to the vessel, e.g., by "spasm," with a consequent decrease in local blood flow[21]. Capillaries in the intraventricular septum in the heart flow up at a vertical angle as in lenticulostriate artery branches. Vasospasm that occurs in these arterioles as a result of KS can explain septal ischemia. However, this mechanism cannot explain left ventricular involvement. Our study also found that hypokinesia was present in 38% of the patients in the contrast involvement site, and pericardial effusion in 29% of the patients.

Last, late gadolinium enhancement in the CMRG procedure identifies irreversible myocardial injury. Our study evaluated the late gadolinium enhancement as normal in all patients, and no infarct sites were observed even in patients with high troponin levels or hypokinesia. Our findings proved that there was no permanent heart damage in type 1 KS.

Limitations

As this study is a single-center report and as KS was clinically diagnosed, the incidence may be underestimated. Since the calculated incidence may be specific to our country and city, it may be difficult to generalize these findings. In addition, this study had a small sample size and did not include biopsy for confirmation. Skin prick testing or specific blood tests for allergy were not performed in the patients aimed at diagnosing the patients with allergy. Ours is just a preliminary study carried-out in emergency area and further study should be done to clarify this point.

Conclusion

Although KS is rare, there is a change on the ECG in an important part of the patients, and KS almost always presents with reversible changes on CMRI. In patients with Kounis syndrome, oedema is observed in T2-weighted series in CMRI, and subendocardial contrast defect in the early phase in cardiac sites consistent with ST elevation in the ECG. Based on the MRI findings, the left ventricular lateral wall and the septal region of the heart are particularly sensitive to KS. The sensitivity of the septal region may due to the arterioles flowing at a vertical angle.

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