CLINICAL RESEARCH

Evolution of acute coronary syndrome with normal coronary arteries and normal cardiac magnetic resonance imaging

Évolution des syndromes coronaires à coronaires angiographiquement normales et IRM cardiaque normale

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Received 15 March 2011; received in revised form 12 May 2011; accepted 17 May 2011
Available online 1 September 2011

KEYWORDS
Acute coronary syndrome; Tako-tsubo cardiomyopathy; Myocarditis; Cardiac magnetic resonance imaging; Evolution

Summary
Background. — Acute coronary syndrome (ACS) with normal coronary angiography is a frequent clinical situation with an uncertain prognosis. Cardiac magnetic resonance imaging (CMRI) is a powerful tool for differential diagnosis between myocardial infarction (MI), acute myocarditis and Tako-tsubo cardiomyopathy (TTC). Data are sparse regarding the evolution of patients presenting an ACS with normal coronary arteries and normal CMRI.

Aims. — To evaluate the evolution of patients presenting an ACS with normal coronary arteries and normal CMRI, with a 1-year follow-up.

Methods. — Eighty-seven consecutive patients (mean age, 53 years; 40.2% men) presenting an ACS with troponin elevation and normal coronary arteries by angiography were prospectively included. All patients underwent CMRI at 3-Tesla. Adverse events were recorded with 1-year follow-up.

Abbreviations: ACS, acute coronary syndrome; AMI, acute myocardial infarction; CMRI, cardiac magnetic resonance imaging; cTnI, cardiac troponin I; ECG, electrocardiogram; LGE, late gadolinium enhancement; LV, left ventricular; LVEDI, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; LVESI, left ventricular end-systolic volume index; PE, pulmonary embolism; TTC, Tako-tsubo cardiomyopathy; TTE, transthoracic echocardiography.

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Results. — A likely aetiology for the acute clinical presentation was established by CMRI in 63.2% of patients (22.7% MI, 26.4% acute myocarditis, 11.5% TTC). During follow-up, one patient in the MI group had a stroke (1.2%). In the myocarditis group, there was one initial cardiogenic shock, one episode of congestive heart failure (1.2%) and nine patients had recurrent chest pain without troponin elevation (10.3%). Two TTC group patients initially presented with cardiogenic shock (2.4%); there were no other adverse events in this group during follow-up. In the remaining 36.7% patients, no clear diagnosis could be identified by CMRI, and no adverse events occurred during follow-up.

Conclusion. — CMRI is a useful tool for the management of ACS presenting with normal coronary angiography, as it helps to ascertain the diagnosis and adapt treatment in a large proportion of cases. Nonetheless, patients with no abnormalities identified by CMRI have an excellent evolution.

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Introduction

Acute coronary syndromes (ACS) are one of the leading causes of death and morbidity in industrialized countries [1]. Typical presentation includes acute chest pain and cardiac troponin I (cTnI) elevation, possibly associated with electrocardiogram (ECG) abnormalities. Subsequent coronary angiography usually reveals flow-limiting epicardial stenoses [2]. However, coronary angiography can be normal in 1 to 12% of cases, depending on the definition of “normal” coronary arteries [3—5].

Differential diagnosis can thus be challenging and possible diagnoses include acute myocardial infarction (AMI), generally limited to the subendocardial territory [6], and two non-ischemic myocardial diseases, namely acute myocarditis [7] and transient apical ballooning syndrome, also known as Tako-tsubo cardiomyopathy (TTC) [8].

Cardiac magnetic resonance imaging (CMRI) is a highly sensitive noninvasive technique for detecting myocardial damage in ischemic [9] and non-ischemic cardiac disease, including acute myocarditis [10] and TTC [11].

Management strategy and follow-up can be thus adapted secondarily, further to the results of CMRI, with the initiation of secondary prevention treatment, including antiplatelet therapy, when CMRI confirms a diagnosis of AMI [2]. However, Assomull et al. demonstrated that CMRI can identify the basis for cTnI elevation in only 65% of patients presenting ACS symptoms and unobstructed coronary arteries [12]. Moreover, the evolution of this clinical situation, usually reported in the past as uniformly benign [4,13], might in fact be less favourable, with one recent study reporting a significantly high rate of death and major adverse events [5].

In this context, the aim of this study was to evaluate the evolution of patients presenting with ACS and normal
coronary arteries and normal CMRI, with a follow-up of 1 year. For CMRI evaluation, the latest technology available in clinical practice was used with a high field of Tesla.

Methods

Study population

Patients with clinical suspicion of ACS (ST-segment elevation myocardial infarction or non-ST-segment elevation ACS) and with strictly normal coronary arteries on coronary angiography were prospectively recruited in this observational study. Inclusion criteria were defined as: the association of new onset chest pain present at rest, lasting for longer than 30 minutes; elevated cTnI; and normal coronary angiography, performed no later than 72 hours after admission and reviewed by two experienced observers. Other initial investigations and searches for alternative causes of cTnI elevation, systematically including transthoracic echocardiography (TTE), had proved diagnostically inconclusive. Intracoronary methylergonovine testing was also systematically performed during angiography to exclude coronary vasospasm. Exclusion criteria included prior history of cardiovascular disease, previous coronary intervention, high Framingham risk score (10-year risk greater than 10%) and standard CMRI contraindications. Patients presenting with cardiac rhythm disorders, such as atrial fibrillation, which could explain cTnI elevation, were also excluded.

Data collected included baseline characteristics, medication at admission and ECG abnormalities using the Minnesota code [14]. Daily blood samples were obtained to assess peak cTnI, lipid profile, inflammatory markers including C-reactive protein, B-type natriuretic peptide, creatinine concentration to evaluate glomerular filtration rate. The threshold used in the core laboratory to define a positive cTnI was 0.15 ng/mL and cTnI assessment was performed every 6 hours until peak concentrations were reached. Framingham [15] and Global Registry of Acute Coronary Events (GRACE) [16] risk scores were calculated for each patient to evaluate long- and short-term risk of death. Myocardial tissue biopsy was not envisaged to establish a diagnosis.

All patients initially received standard medical therapy for ACS, including antiplatelet therapy, statins, angiotensin-converting enzyme inhibitors and beta-blockers, according to international guidelines [2,17]. Treatment strategy was subsequently adjusted according to the results of CMRI. In case of myocarditis, anti-inflammatory therapy was initiated if chest pain indicative of associated pericardial reaction persisted. For TTC, treatment was initiated at the physician’s discretion, combining an inhibitor of the renin-angiotensin system with beta-blocker therapy. If CMRI was normal, all therapy was stopped.

Cardiac magnetic resonance imaging

CMRI scans were performed during hospital stay or within 3 weeks of initial presentation and repeated at 3 months in case of suspected TTC to evaluate improvement in left ventricular (LV) function. CMRI studies were conducted at 3.0 field strength (General Electric Healthcare Company, Signa HD, Milwaukee, WI, USA) with a standard 40mT/m gradient, using an eight-element phased array surface coil. Left ventricular function was assessed by ECG-gated cine steady-state free precession (SSFP) breath-hold sequences in the two-chamber and four-chamber views as well as in the short cardiac axis from base to apex (30 phases per cardiac cycle; repetition time, 3.5 ms; echo time, 1.2 ms; flip angle, 45°; typical voxel size, 1.92 × 1.25 × 8.0 mm) [18]. T2-weighted images (triple inversion recovery; TE, 60 ms; TR, 2 × R-R interval; TI, 170 ms; slice thickness, 7 mm; flip angle, 180°; pixel size, 2.3 × 1.3 mm) were acquired in the short-axis plane. First-pass perfusion imaging was performed using a T1-weighted fast gradient echo sequence (FGRE TR/TE = 3.5 ms/1.5 ms) with a notched saturation pulse, after the injection of a bolus of gadolinium (Dota-Gd; Guerbet, Roissy, France) in a brachial vein at a single dose of 0.2 mL/kg (0.1 mmol/kg). The field of view was 400 mm and the matrix size was 256 × 224 interpolated to 256 × 256. The heart was imaged in the short-axis plane with four to six slices, 8 mm thick with a gap of 1 mm. One image per slice was acquired every two cardiac cycles, leading to a temporal resolution of 2 R-R per imaging plane. Forty frames were obtained from each image plane within 80 R-R. For late gadolinium enhancement (LGE) imaging at 3 and 15 minutes, a breath-hold ECG-gated T1-weighted sequence was used (TE=MinFull; field of view, 440 mm; TI optimised to obtain optimal myocardial nulling; matrix size, 256 × 224 interpolated to 256 × 256; slice thickness, 8 mm; gap, 1 mm) [19]. The number and position of the slices were the same as used for the perfusion first-pass imaging.

Offline image analysis was performed on a dedicated workstation (General Electric Healthcare Company, Milwaukee, WI, USA). The endocardial border was drawn manually on each dynamic image. Left ventricular ejection fraction (LVEF), end-diastolic and end-systolic volume indexes (LVEDI and LVESI, respectively) and LV mass index were calculated from the short-axis view [20]. Qualitative interpretation of CMRI scans was performed by two reviewers by consensus. Readers were blinded to the clinical situation. The portion of the heart exhibiting late enhancement and/or T2 high signal intensity was classified according to the segmentation established by the American Heart Association, which divides the left ventricle into 17 segments [21]. LGE images and corresponding T2-weighted images were assessed for subendocardial signal abnormalities in the distribution of a coronary artery, compatible with AMI [9]. Diagnosis of acute myocarditis was made according to criteria previously described by Friederich et al. with detection of oedema in T2-weighted images, hyperaemia and capillary leakage in myocardial early (3 minutes) gadolinium enhancement sequences and necrosis or fibrosis in LGE images detected in the midwall/subepicardial regions [10]. TTC was suspected on initial CMRI if there were no signs of delayed enhancement, T2 signal intensity and LV dysfunction, especially with mid/apical ballooning; TTC was confirmed by complete normalization of LVEF on the follow-up CMRI at 3 months. Scans with volumes and function within the normal range, with no LGE and T2 signal intensity abnormalities were considered normal by CMRI.
Outcomes

The subsequent progress of patients was assessed by telephone contact at 1, 3, 6 and 12 months. In case of clinical events, these were confirmed by hospital records or contact with the general practitioner. Clinical endpoints evaluated were death, recurrent ACS, congestive heart failure, stroke and recurrence of chest pain without cTnI elevation.

Statistical analysis

Continuous data are expressed as mean±standard deviation. Baseline characteristics were compared between the diagnostic CMRI group and the group in which CMRI was judged to be normal, using an independent sample t test and the Chi² test for categorical variables, as appropriate. All tests were two sided. A P value of less 0.05 was considered statistically significant. All statistical analyses were performed using MedCalc© (Mariakerke, Ghent, Belgium).

Results

Baseline characteristics

Among a total of 1687 patients admitted to our institution for ACS between January 2008 and September 2009, 87 (5.1%) had a normal coronary angiogram and met the inclusion criteria. Baseline characteristics are summarized in Table 1. The mean age was 53±18.5 years and 59.8% were women (n=52). No patient had pre-existing renal impairment, prior exposure to cardiotoxic drugs or prior chemotherapy. At the time of cTnI evaluation, no patient had symptoms suggestive of alternative cause of cTnI elevation, such as septicaemia, stroke, renal insufficiency, pulmonary embolism (PE) or aortic dissection. Eight patients (9.2%) described psychological stress before clinical presentation. The prevalence of cardiovascular risk factors and primary prevention therapy was moderate. No patient underwent thrombolytic therapy. Sixty-nine (75.8%) patients had an abnormal ECG on presentation. ST-segment depression was the most commonly detected abnormality (59.4%), followed by T wave changes (26.5%). One patient presented left bundle branch block (1.1%) and none had supraventricular or ventricular tachycardia. No patient with ST-segment elevation was eligible for inclusion. No criteria of right ventricle overload indicative of PE with cTnI elevation were found in the initial TTE and 32 patients (36.8%) underwent further investigation by CT pulmonary angiography (n=22) and ventilation perfusion isotope scans, which did not show evidence of embolism in any patient.

Cardiac magnetic resonance imaging

The median time from presentation of ACS to CMRI was 10.3 days (range 5–22 days). One scan was repeated because the quality was suboptimal. Thus, complete CMRI images were available for all patients. CMRI dimensions were 86.7±24.7 mL/m² for LVEDI and 41.7±14.5 mL/m² for LVESI. LV mass index was 86.4±18.6 g/m² and LVEF was 65.1±13.6%. The diagnoses determined by CMRI are summarized in Table 2. CMRI provided a diagnosis in 55 patients (63.2%).

CMRI revealed subendocardial or transmural T2 high signal area and corresponding LGE in the distribution of coronary artery compatible with AMI in 22 patients (22.7%) (Fig. 1). CMRI suggestive of AMI was located in the anterior wall in two cases (9.1%), in the lateral wall in eight cases (36.6%) and in the inferior wall in 12 cases (54.4%). LGE was transmural for four patients. A diagnosis of myocarditis was established in 23 patients (26.4%) by gadolinium enhancement and T2-weighted sequences (Fig. 1). The most common midwall/epicardial high signal intensity areas were found in the lateral wall (15 patients; 65.2%). All patients had evidence of acute inflammation with increased midwall/epicardial T2 signal intensity.

A diagnosis of TTC was confirmed in ten patients (11.5%) with normalization of LV systolic function at 3 months (Fig. 2). In three patients (3.8%), LVEF at 3 months was estimated by TTE as CMRI was not available. We observed high signal intensity areas with T2-weighted imaging being suggestive of myocardial oedema in four patients.

In the 32 remaining patients (36.7%), there was no detectable myocardial necrosis or inflammation and left ventricular function was normal and no new diagnosis was made.

<table>
<thead>
<tr>
<th>Table 1 Baseline characteristics.</th>
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<tbody>
<tr>
<td>Characteristics</td>
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<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Men</td>
</tr>
<tr>
<td>History of smoking</td>
</tr>
<tr>
<td>Hypertension</td>
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<tr>
<td>Dyslipidaemia</td>
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<tr>
<td>Diabetes mellitus</td>
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<tr>
<td>Obesity (body mass index &gt; 30)</td>
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<tr>
<td>Family history of ischemic heart disease</td>
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<tr>
<td>Peak cardiac troponin I</td>
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<td>Peak C-reactive protein (mg/L)</td>
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<td>Leukocytes (g/L)</td>
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<td>Fibrinogen (g/L)</td>
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<td>Glomerular filtration rate (mL/minute)</td>
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<td>Glycaemia (g/L)</td>
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<td>Framingham risk score</td>
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<td>GRACE risk score</td>
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<tr>
<td>Medications on admission</td>
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<tr>
<td>Antiplatelet therapy</td>
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<tr>
<td>Statins</td>
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<tr>
<td>Angiotensin-converting enzyme inhibitor/angiotensin II blocker</td>
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<tr>
<td>Beta-blocker</td>
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<tr>
<td>Patient thrombolysed</td>
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</tbody>
</table>

Data are mean±standard deviation or number (%).
Table 2  Cardiac magnetic resonance imaging diagnosis.

<table>
<thead>
<tr>
<th>CMRI diagnosis</th>
<th>Assomull et al. [12] n (%)</th>
<th>Current study n (%)</th>
<th>1-year follow-up adverse events, current study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial infarction</td>
<td>7 (11.6) 30 (50.0)</td>
<td>22 (22.7) 23 (26.4)</td>
<td>One stroke (1.2%) One cardiogenic shock (1.2%); one congestive heart failure (1.2%); nine recurrences of chest pains (10.3%)</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>1 (1.7) 21 (35.0)</td>
<td>10 (11.5) 32 (36.7)</td>
<td>Two cardiogenic shocks (2.4%) None</td>
</tr>
</tbody>
</table>

CMRI: cardiac magnetic resonance imaging; TTC: Tako-tsubo cardiomyopathy.

Normal versus abnormal cardiac magnetic resonance imaging

There was no significant difference in baseline characteristics such as age, sex, cardiovascular risk factors or risk scores between the groups with and without CMRI abnormalities. As regards biological markers, only cTnI elevation was significantly lower in the normal CMRI group (3.48 ± 4.3 vs 11.86 ± 11.99 µg/L, P = 0.0028). ECG findings were also similar in both groups. CMRI LV dimensions and function as well as time to CMRI did not differ between groups (Table 3).

Figure 1.  Cardiac magnetic resonance with late gadolinium enhancement imaging in acute myocarditis (A and B) and in acute myocardial infarction (C and D). Typical pattern of acute myocarditis with patchy mid-wall delayed enhancements localized in the lateral wall (arrows); short-axis (A) and four-chamber (B) views. Transmural delayed enhancement in the inferior wall suggestive of myocardial infarction (arrows); black regions within the white areas of late enhancement corresponding to persistent microvascular obstruction (arrows); short-axis (C) and four-chamber (D) views.
Figure 2. Serial late gadolinium enhancement cardiac magnetic resonance imaging in Tako-tsubo cardiomyopathy. Images acquired at baseline scanning demonstrate apical hypokinesia (A and B) in the absence of late gadolinium enhancement (C). A repeat scan 3 months later shows complete normalization of left ventricular function (D and E) with no late gadolinium enhancement detectable (F).

Follow-up

Follow-up information was available for all patients. No new or alternative diagnosis was made for any patient during follow-up. One-year follow-up adverse events are summarized in Table 2. No death or recurrent ACS was observed during follow-up. Three patients (3.5%) had cardiogenic shock at admission. For one patient, CMRI provided the diagnosis of acute myocarditis and for the two others, the diagnosis of TTC was made. Two (2.4%) patients were rehospitalized during follow-up: one for stroke (1.2%), in whom AMI had been diagnosed on initial CMRI, and one for congestive heart failure at 3 months, after an initial diagnosis of myocarditis with alteration of LV systolic function. Nine patients (10.3%) from the myocarditis group presented recurrent chest pain during follow-up, without

| Table 3 | Cardiac magnetic resonance imaging variables in normal and abnormal groups. |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| CMRI variable   | Normal(n = 32)  | Abnormal(n = 55) | P               |
| LVEDI (mL/m²)   | 85.5 ± 16.3     | 89.6 ± 18.4     | 0.35            |
| LVESI (mL/m²)   | 39.5 ± 14.5     | 44.6 ± 12.5     | 0.22            |
| LV mass index (g/m²) | 79 ± 23.7     | 84.7 ± 14.7     | 0.19            |
| LVEF (%)        | 65.7 ± 10.7     | 58.4 ± 9.2      | 0.16            |
| Interval to scan (days) | 7 (1–18)       | 9 (4–22)        | 0.84            |

Data are mean ± standard deviation or median (range). CMRI: cardiac magnetic resonance imaging; LV: left ventricular; LVEDI: left ventricular end-diastolic volume index; LVEF: left ventricular ejection fraction; LVESI: left ventricular end-systolic volume index.
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Discussion

Elevated cTnI concentration reflects with good sensitivity and specificity the existence of myocardial necrosis [22]. Nevertheless, there are a number of other causes of elevated cTnI in the absence of coronary artery disease, as recently summarized by Jeremias et al. [23], including cardiac arrhythmia and non-cardiac causes such as PE, sepsis or systemic inflammatory response syndrome, renal failure and neurological disorders. In our study, we excluded patients with any non-cardiac diseases that could interfere with cTnI concentration. In particular, no patient presented criteria of right ventricular overload evocative of PE, with cTnI elevation at initial TTE. Indeed, we performed further investigations in 36.8% of patients to exclude a diagnosis of PE. However, there is always the possibility that an episode of rapid cardiac rhythm disorder, undetected at admission or during follow-up, may have caused the elevation of cTnI. Furthermore, we retained only three possible aetiologies that could explain a clinical presentation associating acute chest pain, elevated cTnI and normal coronary angiography, namely AMI [24], acute myocarditis [7] and TTC [8].

AMI with normal coronary angiography is a common clinical situation and several different pathophysiological mechanisms have been proposed. Firstly, coronary angiography suffers limitations as a diagnostic technique due to its spatial resolution, which is limited to 200 μm, and the fact that only arterial patency can be judged, without providing much information on the arterial wall. However, most ACSs derive from atheroma causing less than 50% diameter stenosis, which occurs more frequently than tighter stenoses [25]. Vulnerable plaques that give rise to ACS present a positive remodelling effect, with expansion towards the outer layer of the vessel rather than towards the lumen [26]. In AMI with normal coronary arteries, rupture or erosion of a vulnerable plaque can occur, causing transitory occlusion that resolves spontaneously, without leaving any residual visible intracoronary lesion [27]. The simultaneous use of coronary angiography and invasive catheter-based techniques, such as intravascular ultrasound or optical coherence tomography, could make it possible to establish an ischemic cause of ACS with normal coronary arteries, by identifying, for example, thrombus or vulnerable plaque with rupture of a thin-cap fibroatheroma [28,29]. However, these imaging techniques do not investigate distal vessels or small-calibre side branches. Furthermore, other mechanisms can also, albeit more rarely, give rise either separately or in combination, to distal embolisation [30], coronary vasospasm [31], inflammation [32] or coronary dissection [33]. It is now well established that acute myocarditis can mimic ACS [7], with elevation of cTnI in up to 49% of cases [34]. The most common aetiology of myocardial inflammation is viral infection, mainly involving adenoviruses and enteroviruses [35]. The clinical presentation of TTC is also practically indistinguishable from that of ACS, but with normal coronary angiography and initial transient LV dysfunction. The role of catecholamine-induced myocardial stunning or multivessel epicardial or microvascular spasm have been previously been suggested as possible causes of TTC [36].

Thus, establishing an accurate diagnosis in the presence of ACS with normal coronary arteries is of paramount important, particularly so that management can be appropriately adjusted. Patients with confirmed AMI need long-term follow-up and secondary prevention treatment [2]. CMRI is increasingly used to diagnose ischemic heart disease and can provide non-invasive evaluation of myocardial wall motion, global function, perfusion and viability [9]. T2-weighted and LGE sequences are currently considered as the gold standard for in vivo detection of scarring associated with myocardial infarction [37], as well as for identifying other non-ischemic conditions, including myocarditis [10]. In case of TTC, CMRI can identify transient LV dysfunction on cine sequences, without any visible myocardial injury after injection of gadolinium [38].

CMRI is thus a useful noninvasive imaging tool that makes it possible to identify aetiology of ACS with normal coronary arteries in up to 65% of cases, as reported by Assomull et al. [12]. In our study, a diagnosis was established in 63.2% of cases, which is very similar to the rate observed in previous reports, as shown in Table 2. In our study, CMRI brought to light mainly myocarditis (26.4%), AMI (22.7%) and, to a lesser extent, TTC (11.7%). Most importantly, CMRI was normal and led to no definitive diagnosis being established in almost one third of patients, as in the study by Assomull et al., even with the use of high field 3-Tesla CMRI, which theoretically increases spatial resolution [39]. To the best of our knowledge, the current study was the first to assess myocardial injury in case of ACS with normal coronary angiography by using this new technology, but it does not appear to improve diagnostic accuracy. In our study, as reported elsewhere, the cTnI concentration was significantly lower in the group of patients with normal CMRI compared with those with pathological CMRI (3.48 ± 4.3 μg/L vs 11.86 ± 11.99 μg/L, P = 0.0028 in the current study). We hypothesize that in the group with normal CMRI, the myocardial injury, known to be correlated with cTnI [40], was probably too limited to be detected by CMRI at 1.5- or 3-Tesla, due to the limited spatial resolution with partial volume effects. It is noteworthy that a delayed contrast-enhanced cardiac computed tomography scan is also able to provide detailed information on myocardial tissue characteristics associated with myocardial infarction and myocarditis, with less contrast resolution than CMRI [41], and could be used in the clinical situation of ACS with normal coronary arteries.

Until recently, the prognosis of patients with ACS and normal coronary arteries was considered to be excellent, on the basis of several small studies showing a low recurrence rate of ACS and a high 10-year survival rate [4,5]. However, a more recent study that included 9796 patients with ACS undergoing coronary angiography showed a less favourable outcome [5]. In this study, patients with strictly normal angiograms (n = 273) were found to have a mortality of 1.8% at 30 days and of 4.0% at 1 year. However, to date, no study has used CMRI to distinguish between the possible aetiologies of ACS in this population or to evaluate prognosis. In general, patients with myocarditis seem to have favourable evolution and in most cases, early normalization of echocardiography and ECG variables, laboratory values and functional status is observed, although up to 13%
of patients reported atypical non-limited chest discomfort [42]. In TTC, the evolution is, again, generally favourable, with a 4-year survival that was not different from that of an age- and sex-matched population in one angiographic study [43]. Eitel et al. also failed to observe any major adverse events after a median follow-up of 3.3 months in patients with a diagnosis of TTC established by MRI [44]. The greatest risk exists during the initial phase, when complications such as ventricular arrhythmia or cardiogenic shock can be life threatening [43]. In our study, we observed 5.7% of major adverse cardiac events, with one stroke in the AMI group, one episode of congestive heart failure and three initial cardiogenic shocks but no death during the 1 year of follow-up. Furthermore, there was no adverse event in the group of patients who had normal coronary angiography and normal CMRI. Thus, it would appear that evolution in this population could be excellent and therapeutic strategy and follow-up could be secondarily adapted. A further study with a larger sample size and longer follow-up is necessary to provide confirmation of the positive tendency observed in our study.

Conclusion

CMRI is a useful tool for the management of ACS presenting with normal coronary angiography, as it helps to ascertain diagnosis and adapt treatment in a large proportion of cases. Nonetheless, patients with no abnormalities identified by CMRI have an excellent evolution.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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