



Review

Multimodality Imaging of Sudden Cardiac Death and Acute Complications in Acute Coronary Syndrome

Giuseppe Muscogiuri ^{1,2,*}, Andrea Igores Guaricci ^{3,†}, Nicola Soldato ³, Riccardo Cau ⁴, Luca Saba ⁴, Paola Siena ³, Maria Grazia Tarsitano ⁵, Elisa Giannetta ⁶, Davide Sala ⁷, Paolo Sganzerla ⁷, Marco Gatti ⁸, Riccardo Faletti ⁸, Alberto Senatieri ², Gregorio Chierchia ², Gianluca Pontone ⁹, Paolo Marra ^{2,10}, Mark G. Rabbat ^{11,12} and Sandro Sironi ^{2,10}

- ¹ Department of Radiology, Istituto Auxologico Italiano IRCCS, San Luca Hospital, Piazzale Brescia 20, 20149 Milan, Italy
 - ² School of Medicine, University of Milano-Bicocca, 20126 Milan, Italy
 - ³ University Cardiology Unit, Department of Interdisciplinary Medicine, University of Bari, 70121 Bari, Italy
 - ⁴ Department of Radiology, Azienda Ospedaliero Universitaria (A.O.U.), di Cagliari-Polo di Monserrato, 09124 Cagliari, Italy
 - ⁵ Department of Medical and Surgical Science, University Magna Grecia, 88100 Catanzaro, Italy
 - ⁶ Department of Experimental Medicine, Sapienza University of Rome, Viale Regina Elena, 324, 00161 Rome, Italy
 - ⁷ Department of Cardiac, Neurological and Metabolic Sciences, San Luca Hospital, Istituto Auxologico Italiano IRCCS, 20149 Milan, Italy
 - ⁸ Radiology Unit, Department of Surgical Sciences, University of Turin, 10124 Turin, Italy
 - ⁹ Centro Cardiologico Monzino IRCCS, 20138 Milan, Italy
 - ¹⁰ Department of Radiology, ASST Papa Giovanni XXIII, 24127 Bergamo, Italy
 - ¹¹ Division of Cardiology, Loyola University of Chicago, Chicago, IL 60611, USA
 - ¹² Edward Hines Jr. VA Hospital, Hines, IL 60141, USA
- * Correspondence: giuseppe.muscogiuri@unimib.it
† These authors contributed equally to this work.



Citation: Muscogiuri, G.; Guaricci, A.I.; Soldato, N.; Cau, R.; Saba, L.; Siena, P.; Tarsitano, M.G.; Giannetta, E.; Sala, D.; Sganzerla, P.; et al.

Multimodality Imaging of Sudden Cardiac Death and Acute Complications in Acute Coronary Syndrome. *J. Clin. Med.* **2022**, *11*, 5663. <https://doi.org/10.3390/jcm11195663>

Academic Editors: Marco Guglielmo and Anna Giulia Pavan

Received: 13 August 2022

Accepted: 22 September 2022

Published: 26 September 2022

Publisher's Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.



Copyright: © 2022 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

Abstract: Sudden cardiac death (SCD) is a potentially fatal event usually caused by a cardiac arrhythmia, which is often the result of coronary artery disease (CAD). Up to 80% of patients suffering from SCD have concomitant CAD. Arrhythmic complications may occur in patients with acute coronary syndrome (ACS) before admission, during revascularization procedures, and in hospital intensive care monitoring. In addition, about 20% of patients who survive cardiac arrest develop a transmural myocardial infarction (MI). Prevention of ACS can be evaluated in selected patients using cardiac computed tomography angiography (CCTA), while diagnosis can be depicted using electrocardiography (ECG), and complications can be evaluated with cardiac magnetic resonance (CMR) and echocardiography. CCTA can evaluate plaque, burden of disease, stenosis, and adverse plaque characteristics, in patients with chest pain. ECG and echocardiography are the first-line tests for ACS and are affordable and useful for diagnosis. CMR can evaluate function and the presence of complications after ACS, such as development of ventricular thrombus and presence of myocardial tissue characterization abnormalities that can be the substrate of ventricular arrhythmias.

Keywords: ischemic cardiomyopathy; acute myocardial infarction; cardiac arrhythmias; myocardial edema; late gadolinium enhancement; ventricular thrombus

1. Introduction

Coronary artery disease (CAD) represents one of the most important causes of death, especially in low and middle-income countries, caused by acute coronary syndrome (ACS) or cardiac complication related to ACS [1–3]. Prevention of acute events or non-invasive evaluation after the development of ACS can be performed using a multimodality approach [4,5]. Theoretically, an approach based using different non-invasive technique

in ACS could result in an economical advantage, reduction of invasive procedures, and identification of patients that can have poor outcome after ACS [6]. Electrocardiogram (ECG) and echocardiography still represent the first line techniques in the presence of ACS. Cardiac magnetic resonance (CMR) is mainly confined to the evaluation of acute myocardial infarction in the early stage [5]; in particular, the evaluation of myocardial tissue abnormalities such as the presence of myocardial edema, microvascular obstruction (MVO), and the presence of left ventricular thrombus, can play a key role in terms of prognostic stratification in the early stage [5]. In the acute setting, coronary computed tomography angiography (CCTA) can be extremely important to rule out CAD [7], identify presence of high-risk coronary plaque [8], or severe stenosis, that may lead to acute myocardial infarction [9]. Furthermore, the application of CCTA can be extremely important for the identification of differential diagnosis causing chest pain and increased troponin, such as acute myocarditis [10].

The growing application of artificial intelligence (AI) in cardiac imaging is rapidly changing the diagnostic and therapeutic approach of patients with ischemic cardiomyopathy [11]. Potentially, the impact of AI will rapidly modify the management of patients with suspected ACS providing information regarding stenosis [12] and risk stratification [13].

In this review, we will demonstrate the application of non-invasive, multimodality imaging for evaluation of CAD, and possible sequelae that can lead to sudden cardiac death (SCD).

2. ECG Findings

ECG is an indispensable tool that help physicians to find potentially fatal situations after ACS occurrence. ECG is useful to identify cardiac arrhythmias. Indeed, the predominant causes of SCD are represented by tachyarrhythmias, such as ventricular fibrillation (VF) and sustained ventricular tachycardia (VT), or bradyarrhythmia-asystole events [14–17]. Ventricular arrhythmias (VA) are temporally distributed in two phases: early acute phase (from incident event up to 72 h), when ischemic dynamic changes and reperfusion occur; and intermediate-chronic phase (weeks-to-months later), when remodeling occurs.

Premature ventricular contractions (PVC) are common in the early phase of ACS. Both primary VF and VPC (particularly R-over-T phenomenon) occur during the early phase of ST elevation myocardial infarction (STEMI), when electrical instability is predominant. Although R-over-T beats can trigger VF, the sensitivity and specificity of these electrocardiographic findings during monitoring is too low to identify patients at increased risk for VT [18]. The pathophysiologic mechanism of fatal VA is based on the concept that an acute destabilizing event acts on a susceptible myocardial substrate. In this scenario, a SCA causes a myocardial scar, which generates a biochemical, electrolyte, and mechanical dysfunction, and creates the substrate for multiple arrhythmic re-entrant pathways [19].

Primary percutaneous coronary intervention (PCI), combined with the use of beta-blockers, have reduced the incidence of VT or VF during the first 48 h after a myocardial infarction [20–22]. Nevertheless, many patients present recurrent VA within the first 48 h and their response to pharmacological antiarrhythmic agents is variable [20]. Approximately 10% of post-MI survivors remain at high risk of dying in the first months or years following hospital discharge (mortality >25% at 2 years) [23,24]. Patients who develop sustained VF or VT after 48 h after their index MI have a predominantly higher rate of all-cause mortality [20]. However, the relationship between early (within 48 h of the index MI) VF/VT and mortality remains controversial [20]. The risk of recurrent cardiac arrest is usually in the range of 10–20%. Patients who experienced recurrent arrhythmias have a mortality rate of 50% [25].

On the other hand, ischemic lesions may cause a conduction block at any level of the atrioventricular or intraventricular system [26]. First-degree and second-degree type one atrioventricular (AV) blocks do not appear to affect survival and they are more often associated with occlusion of the right coronary artery and ischemia of the AV node [27]. Type II second-degree AV block is generally transient after an inferior-posterior infarction

and manifests itself with a narrow/junctional QRS escape rhythm. However, after infarction of the anterior-wall, the block usually located below the His-bundle could evolve into a complete block [28,29]. On ECG, an unstable escape rhythm with wide QRS complexes can occur with ventricular rate <40 bpm and ventricular asystole can suddenly occur [30]. However, SCD may also occur after mechanical complications of ACS [31,32]. As mentioned before, twelve-lead EKG is more useful to find arrhythmic complications with respect to mechanical complications of MI. On the other hand, ECG together with clinical and echocardiographic findings (see below, echocardiography section), may be useful to identify post-myocardial infarction acute mechanical complications that may lead to SCD [33,34]. For instance, a ventricular septal defect may lead to the signs of evolving myocardial infarction, Q wave in the respective coronary affected territory and ventricular arrhythmias. Clinically, the patient may present with a new murmur or signs of acute heart failure and circulatory collapse.

A free wall rupture, a life-threatening condition, typically occurs after ineffective reperfusion therapy or delayed presentation of MI [35,36]. ECG may show a new ST-segment elevation, because outflow of blood irritates the pericardium. Patients may present with new episodes of chest pain, nausea, hemodynamic instability, or collapse.

Lastly, ECG may detect electrolyte imbalances, a condition typically observed in ACS patients, which represents a pro-arrhythmic burden [37–39]. For instance, hyperkalemia may be suspected in patients who present with increased voltage of the T-waves, with pointed, narrow and symmetrical morphology, widening of the QRS-duration (right bundle branch-block-like), reduction of the amplitude of the P-wave, and ST-segment elevation in the right precordial leads [40]. Similarly, hypokalemia is also common in this setting of patients, and it can cause VA [41]. It may cause reduction of the amplitude of the T wave until its disappearance, QT interval prolongation, atrioventricular blocks, and ST-segment depression [42].

The role of ECG in patient with ACS can be definitely extremely important for the assessment of complications due to AMI and identifications of patients that could potentially develop arrhythmogenic SCD.

A representative case is shown in Figure 1.

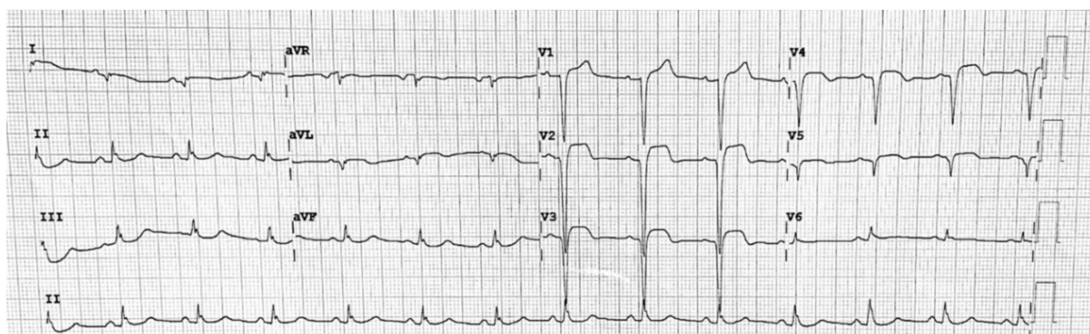


Figure 1. Twelve-lead EKG from a 60 years old patient five days after admission in our referral hospital for antero-lateral ST-segment elevation myocardial infarction (STEMI). Patient underwent primary revascularization of left descending coronary artery. Five days after reperfusion, patient experienced hypotension and new chest pain episode. EKG showed a new ST-segment elevation in the anterior precordial leads. Prompt bedside echocardiography showed an acute mild-to-moderate pericardial tamponade. The origin site of this tamponade was not observed.

3. Echocardiography

Echocardiography is the first imaging tool used both in suspected or established ACS [43]. This is due both to its feasibility as a bedside diagnostic technique and to the relatively recent application to assess aspects of myocardial mechanics and advanced systolic and diastolic function [44–47]. The first aim of the echocardiographic examination in cases of ACS is to assess the presence and extension of kinesis abnormalities, which represent

markers of ischemia (hypo-dys-akinesia) [44,48–50]. In addition, echocardiography helps physicians to assess overall ventricular volumes and left ventricle ejection fraction (LVEF), possible extension of ischemia to the right ventricle, subsequent pericardial disease, and possible mechanical complications [51,52]. In the ACS diagnostic pathway, echocardiography and secondary imaging exams, such as CCTA or CMR, could identify other potentially deadly pathologies as differential causes of symptoms: aortic dissection; right-sided overload from acute pulmonary embolism; hypertrophic cardiomyopathy; aortic stenosis; and pericardial pathology [53].

In the era of primary coronary reperfusion therapy for ACS, both mortality and incidence of mechanical complications (0.27%), such as intraventricular septal rupture, free wall rupture with or without tamponade, or acute mitral regurgitation (MR) have decreased [54–58]. Nevertheless, mechanical complications can lead to mortality and significant morbidity, especially in older patients, so they require early diagnosis and therapeutic management. Patients with mechanical complications tend to be older, female, have a history of heart failure, chronic kidney disease, and are often presenting with their first ACS [58–60]. In this scenario, prompt use of transthoracic echocardiography is essential [57,61–63].

Several studies showed that free wall rupture (FWR) is the most frequent mechanical complication post-MI presenting as a mild pericardial effusion, chest pain and nausea, and rapid development of cardiac tamponade, or as SCD due to electromechanical dissociation [64]. Echocardiography is useful to confirm the diagnosis and promptly guide surgical repair. Nevertheless, in-hospital mortality for FWR, even if treated surgically, still remain > 35% [65]. Although uncommon, aneurysms and pseudoaneurysms (PAN), which derive from an incomplete rupture of the LV free wall, may occur after ACS and have a high mortality rate up to 20% [66]. Aneurysms are located in the left ventricle apex in 80% of cases, while PANs are typically located in the anterior or lateral wall of the LV [67]. Echocardiographic features are the following: a cavity which communicates with the LV chamber through a large (aneurysm) or tight (PANs) neck; a thrombus into either aneurysms or PANs may be found; doppler and color flow imaging may show bidirectional blood flow via the site of rupture [68]. Although echocardiography is useful to promptly find aneurysms or PANs, sometimes-detected characteristics are less typical. In this case, cardiac magnetic resonance (CMR) may be useful [69].

The main echocardiographic findings of pericardial tamponade are represented by pericardial effusion, inferior vena cava (IVC) dilatation, diastolic right ventricular collapse, and systolic right atrial collapse [70]. IVC plethora consists of dilatation of the IVC > 20 mm without respiratory collapse (<50% of the basal diameter) and it is a very sensitive sign of cardiac tamponade (92%). Atrial collapse is usually observed before ventricular collapse during progressive cardiac tamponade. Right atrium collapse is usually observed in early systole, when intracavity pressure is lower. Furthermore, a prolonged duration of either atrial collapse (more than one-third of the cardiac cycle) and RV collapse have been described as highly sensitive and specific sign of clinical cardiac tamponade [71–73]. Ventricular septal defects (VSD) have an incidence of 0.3%, mainly due to early reperfusion therapy. Many studies show that VSD is generally caused by multivessel coronary disease [74,75] and generally occurs 3–5 days after an MI. It is characterized by a high mortality rate (85% of patients within 2 days) and evolution into cardiac failure and cardiogenic shock if not treated effectively [58,59]. VSD are more frequently located in the anterior than inferior/lateral wall (66% versus 34%). Although uncommon, inferior VSDs are more complex with multiple, irregular, and/or variable interventricular connections and they are associated with a higher mortality rate and SCD, probably due to contemporary right ventricle dysfunction [75]. Posterior VSDs are often associated with mitral regurgitation secondary to ischemic tethering. The rupture of the septum is shown as a partial opening during diastole and closure in systole. A left-to-right shunt via the VSD is usually found using color-doppler imaging. Patients with inferior/posterior VSD, early onset of

symptoms and signs of right ventricular failure represent a subgroup at higher risk of SCD (Figure 2) [76–78].

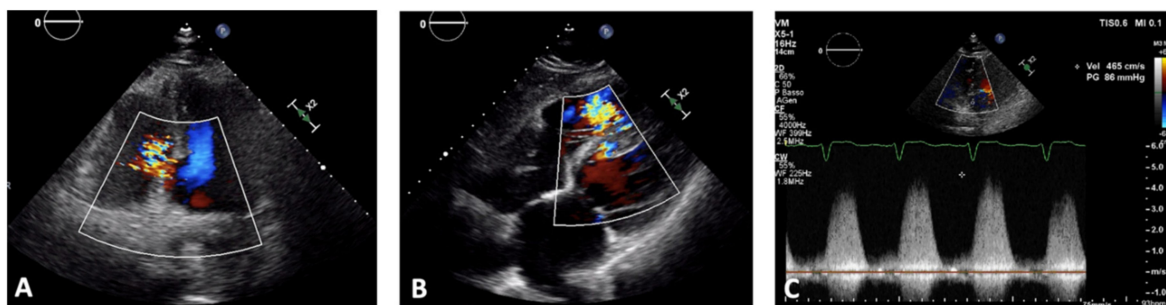


Figure 2. Ventricular septal defect (VSD) in a patient diagnosed with subacute myocardial infarction 3 days after reperfusion of right coronary artery. (A) Apical four-chamber view shows presence of a posterior VSD. Color Doppler mode demonstrated a high-velocity flow through the neck of the VSD, which corresponds to left-to-right shunt. (B) Subcostal view confirmed presence of a serpiginous posterior VSD with left-to-right shunt. (C) Continuous-wave Doppler imaging showed presence of a high velocity shunt, although underestimated because of non-completely parallel positioning of doppler-marker as respect to flow direction.

Acute ischemic MR is a rare complication (0.05%), which typically occurs 2–7 days after AMI [61,79,80]. It is generally caused by the rupture of a papillary muscle or chorda tendineae [81]. Acute MR generates a massive blood flow which is ejected into the left atrium during ventricular systole. This causes a reduction of the ejection fraction and consequently a cardiocirculatory collapse. In addition, massive regurgitation via the incompetent valve leads to a volume and pressure overload in the left atrium, reflecting in higher pulmonary pressure and congestion [82]. Echocardiography is mandatory to assess the presence and severity of MR, especially in cases of cardiogenic shock. The direction of the regurgitant jet on color flow doppler may help physician to identify the etiology of the MR. In papillary muscle rupture, echocardiography can show a mobile mass prolapsing into left atrium during ventricular systole. However, only a leaflet prolapse may be found [83].

Prediction of ventricular arrhythmias after MI is challenging. Echocardiographic evaluation of the global systolic function assessed by LVEF still remains the primary approach to stratify patients with ACS at high risk of SCD [49]. SCD is most frequently caused by ventricular arrhythmias resulting from electrical and mechanical changes in the affected myocardium and may be prevented by an implantable cardioverter-defibrillator (ICD) [84]. It is well established that patients with LVEF < 35% after ACS present an improvement in overall survival by receiving an ICD, due to a reduction in arrhythmic fatal events [85,86]. Similarly, patients who receive biventricular pacing appear to have a lower rate of mortality due to tachyarrhythmias [84,87,88]. Several studies demonstrate that LV hypertrophy with eccentric remodeling and dilatation represent risk factors for SCD in ACS patients [88–90]. However, many patients suffering from SCD after AMI have a LVEF > 35%. This reflects a poor sensitivity of LVEF as a unique mechanical risk-stratifying parameter for this subgroup of patients [91]. In this scenario, myocardial strain represents a novel and accurate tool able to quantify global and regional myocardial function and the timing of contraction. Particularly, several studies have found that global longitudinal strain (GLS) is more accurate than LVEF in assessing LV function post-MI and in predicting mortality and ventricular arrhythmias [92,93]. GLS is a more accurate marker to detect subtle changes in myocardial function, which may be important in arrhythmic risk stratification [94,95]. Furthermore, a novel strain echocardiography parameter is mechanical dispersion, which is defined as the time to peak negative strain from the 16 LV segments and represents a measure of myocardial contraction heterogeneity [96,97]. Mechanical dispersion appears to be a new tool in the risk assessment of SCD because it predicts arrhythmias in patients

after myocardial infarction independently by GLS and traditional parameters such as EF, volumes, and diastolic dysfunction [92].

Echocardiography still represents the first technique useful for the evaluation of AMI complications. In particular, it can be extremely helpful for the identification of patients that can develop structural complications and fatal arrhythmias with ACS.

4. Cardiac Magnetic Resonance

CMR can play a key role in patients with acute myocardial infarction but also with chronic ischemic cardiomyopathy [4,5,98–101]. In particular, the additional value of CMR over echocardiography is represented by the possibility to identify reversible or irreversible damaged myocardial areas, the latter of which could potentially be a trigger of arrhythmias [102,103]. The assessment of myocardial damage using CMR in ischemic cardiomyopathy can be differentiated considering the acute or chronic setting [4,104]. In the acute setting, it is possible to evaluate the presence of myocardial edema, MVO and late gadolinium enhancement (LGE) [5,105]; in patients with chronic ischemic cardiomyopathy, LGE can be useful for evaluation of viability as well as the presence of heterogeneous LGE, which is associated with increased incidence of arrhythmias [5].

Two cases of AMI evaluated with CMR are shown in Figures 3 and 4.

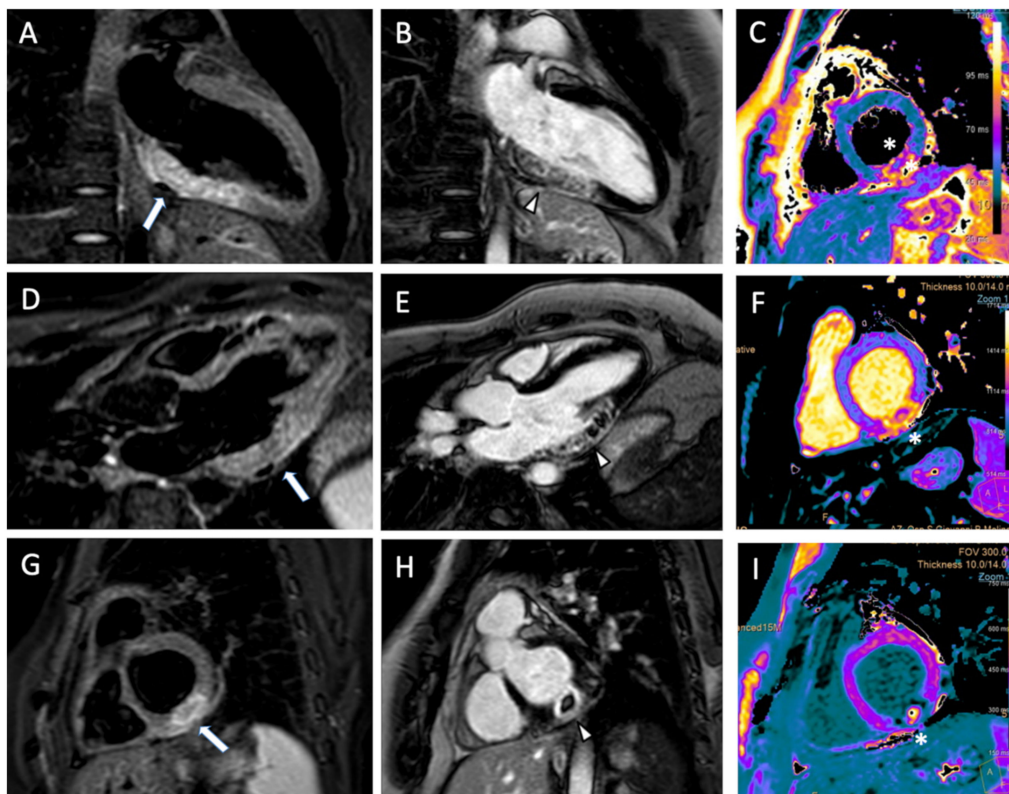


Figure 3. (A) 41 year old male patient underwent cardiac magnetic resonance for acute myocardial infarction. T2 black blood images show myocardial edema on inferolateral wall in two chamber (arrow, (A)), three chamber (arrow, (D)), and short axis (arrow, (G)). The myocardial infarcted area with presence of microvascular obstruction was observed on late gadolinium enhancement in two chamber (arrowhead, (B)), three chamber (arrowhead, (E)), and short axis (arrowhead, (H)). Microvascular obstruction was observed on short axis T2 mapping (asterisk, (C)), native T1 mapping (asterisk, (F)) and post-contrast native T1 (asterisk, (I)).

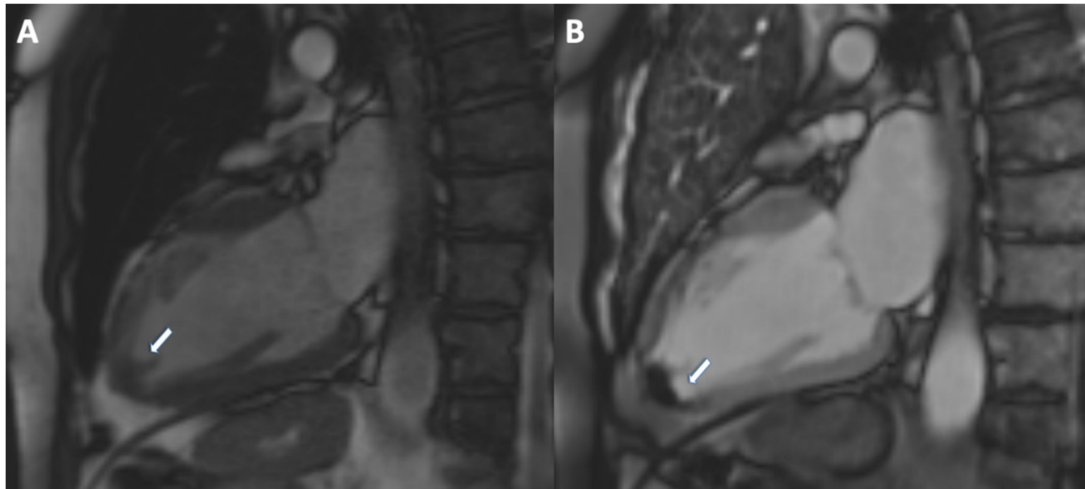


Figure 4. (A) 72 year old male patient with previous AMI on LAD territory. The patient was admitted to hospital due to chest pain. Images show possibility of a left ventricular thrombus on two chamber cine image ((B), arrow). Finding was confirmed on two chamber early gadolinium enhancement ((B), arrow).

5. Myocardial Edema and Area at Risk

In the past, the evaluation of myocardial edema in clinical practice has been performed using T2 black blood sequences [106]. When acquiring T2 black blood (T2BB) sequences, myocardial edema is hyperintense, normal myocardium has normal signal intensity, while blood pool shows black signal intensity [5]. The acquisition of T2BB is routinely performed in clinical practice using T2 short-tau inversion recovery (T2 STIR) and T2 spectral adiabatic inversion recovery (T2 SPAIR). The longtime of acquisition is one of the common disadvantages of both T2 STIR and T2 SPAIR; furthermore, T2 STIR can show artifacts due to misalignment of three radiofrequency pulses; T2 SPAIR can show artifacts related to presence of B_0 inhomogeneities [107].

In order to overcome the limitations due to T2 BB images, the approach with T1 and T2 mapping has been developed [108]. Native T1 mapping is sensitive to increased extracellular volume; however, it is not specific for edema [109], conversely, T2 mapping is very sensitive and specific for presence of edema [110].

Evaluation of edema in acute myocardial infarction can be performed using qualitative or semiquantitative approaches [5] and in the acute setting allows the ability to identify edema and area at risk (AAR); the latter represents the mismatch between the edematous myocardium and myocardial area of LGE [5] and potentially the myocardium that can be salvaged by timely revascularization [5].

Comparing the T2 BB versus the T1 and T2 mapping approach, Bulluck et al., showed that the approach using mapping allows a minor variability in terms of inter and intrareader agreement for the evaluation of AAR [111]. Although AAR can be extremely important in the acute setting, it is extremely important to consider the “bimodal wave” of myocardial edema development in patients with acute myocardial infarction [112]. Indeed, the development of edema follows a dynamic process beginning with the development due to reperfusion, followed by edema associated with inflammation and remodeling [112]. Furthermore, it is important to consider that development of myocardial edema can be variable considering the myocardial territory supplied, the coronary configuration and the culprit lesion [113].

Zorzi et al., evaluated the impact of edema depicted on CMR in a mixed population comprising sudden cardiac arrest due to ischemic and non-ischemic cardiomyopathy. The authors found that myocardial edema was associated with a significantly higher survival free from ICD interventions (long rank: 0.04) and ICD shock (long rank: 0.03) [114]. Therefore, the authors hypothesize that myocardial edema represents only a transient

arrhythmogenic substrate [114]. Similar results were observed in a smaller cohort of patients by Zorzi et al.; the authors showed that in patients who experienced out-of-hospital cardiac arrest, the presence of edema was associated with a favorable outcome [115].

Therefore, it appears that the presence of edema depicted on CMR represents a transient arrhythmogenic substrate for development of arrhythmias.

6. Microvascular Obstruction

The development of MVO during acute myocardial infarction can be related to different factors, such as ischemic injury, reperfusion injury, distal embolization, and individual predisposition [116,117]. The presence of MVO in the acute setting can be depicted using perfusion, early gadolinium (EGE), or LGE sequences, after the administration of a gadolinium-based contrast agent, demonstrated as a myocardial dark area with ischemic distribution in the damaged myocardium [5]. The presence of MVO decreased over time and amount during perfusion is usually greater compared to LGE [118]. Using a non-contrast approach, it is possible to depict MVO in native T1 mapping showing decreased values compared to surrounding myocardial tissue [119].

Usually, MVO is associated with left ventricle remodeling, increased left ventricle volume, and impairment of left ventricle ejection fraction, that lead to poor outcome of patients with acute myocardial infarction [120,121]. Despite the evaluation that was observed with myocardial contrast echocardiography, it seems that in the early phase the presence of no reflow areas is not associated with an increased development of arrhythmias [122], while in a short follow-up the presence of MVO and expression of irreversibly damaged myocardium, can lead to development of arrhythmias [123].

The development of MVO causes the development of intramyocardial hemorrhage (IMH), the expression of blood cell deposition, vascular endothelial damage, and iron deposition [124].

The depiction of IMH can be evaluated using T2* sequences demonstrating iron deposition within the damaged myocardium [125]; while native T1 mapping can be used for the depiction of MVO in the infarcted myocardium, differentiating it from salvageable and remote myocardium that shows higher values of native T1 [119].

The intramyocardial deposition of iron is associated with an arrhythmogenic trigger [126,127].

Cokic et al., in an animal model, compared the ECG findings between two subgroups showing absence and presence of iron deposition after ACS [127]. Animals showing iron deposition demonstrated an elevated QT interval and QT corrected ($p < 0.05$) during the day, night and 24 h, compared to animals negative for iron deposition [127].

Mather et al., analyzed the impact of IMH demonstrating that patients with the presence of IMH showed a significantly ($p = 0.04$) longer filtered QRS (125 ms) compared to patients without IMH (109 ms) [126]. Furthermore, the authors showed that the size of MVO was not an independent predictor of prolonged filtered QRS [126].

These manuscripts demonstrate that arrhythmias can be developed following the iron deposition within myocardium.

7. Ventricular Thrombus

The development of thrombus after ACS is caused by the presence of Virchow's triad after an acute event comprising myocardial wall motion abnormalities, subendocardial inflammation, and systemic hypercoagulability due to ACS [128].

The presence of ventricular thrombus can be identified as a hypointense area inside the ventricular cavity using EGE sequences, in particular EGE should be acquired within two minutes after administration of the contrast agent [129]. LGE can be useful for the assessment of left ventricular thrombus showing a specificity of 99% and a sensitivity of 88% [130]. An approach without contrast can be feasible using native T1 mapping, which shows lower values compared to patients with blood pool [131]. Despite the incidence of thrombus after acute myocardial infarction is more frequent in the anterior or apical

myocardial infarction, in patients with ACS the incidence of left ventricular thrombus detected using CMR ranges between 6.3% and 12.2%, respectively [128,132]. In the era of thrombolytic therapy, the presence of left ventricular thrombus causing stroke is around 2–3%, while limited data is still available for patients treated with percutaneous coronary intervention [128]. The thrombus that can lead to the development of emboli are mainly represented by a protruding shape and/or cavitory motion [133]. A CMR protocol dedicated for the evaluation of LV thrombus should be mandatory for the evaluation of LV thrombus.

8. Late Gadolinium Enhancement

To date, late gadolinium enhancement sequences are the most studied in the field of magnetic resonance, and its prognostic power has been demonstrated in numerous contexts of the cardiovascular field [134–136]. The evaluation of LGE using CMR in the acute setting could be important in terms of risk stratification of acute events, but also as sequelae of myocardial infarction [137,138]. The evaluation of LGE in acute myocardial infarction is performed 10–15 min after the administration of a gadolinium-based contrast agent [139–141]. The wash-out of gadolinium in the infarcted myocardial areas is decreased, therefore it is possible to obtain images with remote myocardium with a null signal, bright blood pool, and hyperintense signal intensity of the infarcted myocardium showing an ischemic pattern [4]. Black-blood LGE (BB-LGE) is another approach developed for the identification of the infarcted area [99,101,142]. Using BB-LGE it is possible to obtain hyperintense signal intensity of infarcted myocardium, black blood pool, and grey signal intensity of remote myocardium [101].

The main advantage of BB-LGE over the standard sequences used for the evaluation of LGE is represented by the possibility to better evaluate subendocardial infarction and infarcted papillary muscles [99,101].

In terms of arrhythmogenic stratification, the assessment of LGE in patients with ACS should be carefully evaluated considering that the amount of LGE can decrease over the time [5].

The presence of myocardial infarction heterogeneity and the development of ventricular arrhythmias have been explored by Robbers et al. [138]. The authors demonstrated that a large proportion of penumbra within the myocardial enhanced area can increase the risk of VT ($p = 0.003$); the latter was associated with an increased prevalence of sudden death [138]. Furthermore, the presence of ventricular fibrillation, prior PCI, and the proportional amount of penumbra within the enhanced area in a multivariate analysis are independently associated to VT [138].

During CMR, it is important to evaluate the presence of infarcted papillary muscles, which can trigger an arrhythmia [143,144]. Although the study was performed using echocardiography, the authors found that patients with infarcted papillary muscles showed a greater QRS (150 ± 15 ms) compared to patients with fascicular arrhythmias (127 ± 11 ms) ($p = 0.001$). Echocardiographic data were confirmed using CMR by Bogun et al. [137] who showed that heterogenous LGE was more frequent in arrhythmogenic papillary muscles compared with papillary muscles not involved in ventricular arrhythmias ($p = 0.01$) [137].

9. Cardiac Computed Tomography Angiography

According to the ESC and AHA guidelines, CCTA is considered one of the best non-invasive techniques to rule out the presence of CAD [145–151]. Despite the low positive predictive value in terms of ischemic coronary lesions [7], CCTA represents a unique tool for the noninvasive evaluation of plaque characteristics. The association between specific plaque features and adverse plaque characteristics has been linked with worse outcomes (Figure 5) [8,152,153]. Furthermore, CCTA it is a useful tool for the evaluation of anomalous origin or course of coronaries that can be associated with a worse outcome [154].

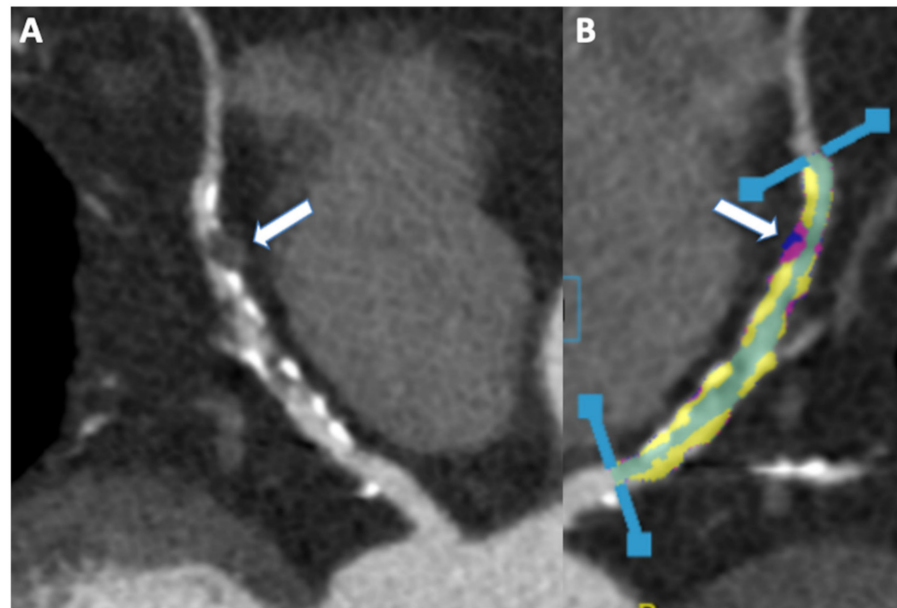


Figure 5. (A) 55 year old male patient showing diffuse coronary artery disease on left anterior descending artery (panel (A)). The predominant plaques were calcified, a fibro-fatty plaque with positive remodeling was observed on mid-LAD (arrow, (A)). Plaque analysis confirm huge coronary calcification (yellow, (B)), while fibrofatty and remodeled plaque (arrow, (B)) show fibrofatty plaque (purple) associates with necrotic core (blue).

Tissue characterization is another interesting tool that can be extremely important for assessment of patients with chest pain and increased troponin (Figure 6) [155].

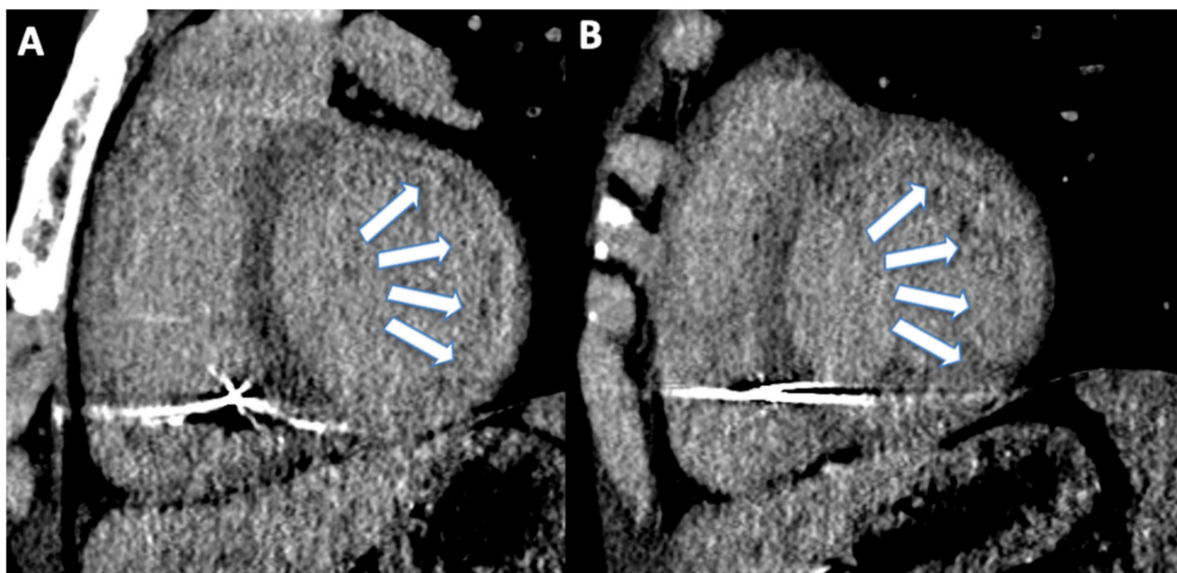


Figure 6. (A) 23 year old male patient with history of cardiac arrest. After the implantation of implantable cardioverter defibrillator, the patient acquired cardiac computed tomography angiography for the assessment of late iodine enhancement. Late iodine enhancement reconstructed on short axis showed intramyocardial late iodine enhancement on anterior, lateral, and inferior wall of basal (arrows, (A)) and mid-ventricular (arrows, (B)) left ventricle.

In summary, CCTA can be easily identify patients with characteristic of coronary plaque and stenosis or myocardial tissue abnormalities that can lead to development of SCD.

10. Coronary Stenosis and Plaque

The assessment of coronary plaque using CCTA may be indicative of patients that can develop sudden cardiac death [156–159].

Min et al., demonstrated that patients with stenosis < 50% have a higher survival rate (survival 99.7%) compared to patients with left main disease \geq 50% (survival 85%) [160]. Beyond stenosis, Min et al., demonstrated that the location of disease was important in terms of survival [160]. The presence of significant proximal stenosis was associated with a worse outcome if the stenosis was evident in the left anterior descending artery [160].

Beyond the location of coronary stenosis, CCTA can play a key role in the non-invasive evaluation of plaque burden and adverse plaque characteristics that has been associated with worse outcomes [8].

Chang et al., demonstrated that more than 65% of patients developing acute coronary syndrome have non-obstructive CAD and 52% of cases have high risk plaque (HRP) [158]. In particular, the adjusted hazard ratio was 1.010 for percentage of diameter stenosis, while 1.593 for plaque showing necrotic core [158].

The impact of plaque characteristic has also been evaluated in the SCOT-HEART trial, showing that patients with adverse plaque showed death or myocardial infarction three times more frequent ($p < 0.01$ and HR: 3.01) and two times more frequent in patients with obstructive coronary artery disease ($p = 0.02$ and HR: 1.99) [159]. The combination of obstructive CAD and adverse remodeling demonstrated a 10-fold increase of death or myocardial infarction compared to patients with normal coronary arteries ($p < 0.01$ and HR: 11.50) [159].

Considering the impact of plaque characteristics and coronary artery stenosis in terms of prognosis, the reporting system of CCTA has been modified, taking into account that both metrics can be useful in terms of diagnosis and prognosis [161–163]; especially considering that patients after CCTA, beyond invasive coronary angiography, could be potentially treated with aggressive medical therapy [161].

11. CCTA in Emergency

CCTA in an emergency can [164] play a key role to rule out CAD in select cases [155], especially in patients with acute chest pain, increase of troponin and inconclusive diagnosis (discrepancy with marker, symptoms, ECG and echocardiography) [10].

The ROMICAT trial described the impact of CCTA in patients with acute chest pain [165]. Hoffmann et al., demonstrated that in patients with acute chest pain, CCTA was able to rule out 50% of patients with low to intermediate likelihood of ACS with a negative predictive value of 100% [165]. The ROMICAT II trial highlighted the importance of plaque characteristics in patients who underwent CCTA in an emergency setting [164]. Patients with ACS showed HRP more frequently ($p: 0.006$ with an OR: 8.9) [164].

Another useful approach of CCTA can be represented by tissue characterization in the acute setting [10].

Esposito et al., demonstrated that the application of CCTA in the emergency department can provide information regarding myocarditis or non-ischemic cardiomyopathies [155]. A study with a larger cohort of patients showed that using CCTA was possible to overcome the concept of “triple rule out” entering in the new era of “quadruple rule out” improving diagnosis [10].

12. Application of Artificial Intelligence to Non-Invasive Multimodality Imaging

The application of artificial intelligence (AI) in non-invasive cardiovascular imaging is rapidly growing [11]. In particular, the application of artificial intelligence can modify the approach to CAD in clinical practice speeding up the time of acquisition, reporting, and building models for risk stratification [12,100,166–168].

Focusing on echocardiography, the manuscript of Narang et al., demonstrated the possibility to evaluate left ventricle and left atrium volume in 35 ± 17 s [169]. Furthermore, algorithms trained for the depiction of regional wall motion abnormalities can provide

results similar to experienced sonographer (AUC 0.99 for deep learning model vs. 0.98 for sonographers) [170].

In terms of CMR, the application of AI ranges from image quality to image analysis [100,171,172].

In particular, Moccia et al., demonstrated that it was possible to segment the myocardial scar using a fully convolutional neural network with a sensitivity and Dice score of 88.07% and 71.25%, compared to manual segmentation [172].

The application of AI in CCTA aimed to provide information regarding the stenosis, plaque characteristics, association with ischemia, and risk stratification [12,167,173].

Evaluation of coronary stenosis has been described by several authors founding a good agreement for the depiction of coronary artery stenosis [12,174,175]. Furthermore, AI algorithms can be used for risk stratification as demonstrated by Motwani et al. [176]. In particular, the authors demonstrated that combining the data of machine learning (ML) with clinical data was possible to predict better the risk of all-cause mortality, compared to clinical score in a follow up of 5 years [176]. The impact of ML has been demonstrated also by Van Rosendaal et al. [177]. The authors demonstrated that an algorithm based on ML is able to stratify better patients compared to current CCTA integrated risk score [177].

In summary, the application of AI can definitely speed up the time of reporting of imaging modalities and it could potentially provide data regarding prognosis.

13. Future Perspective

A multimodality approach in the application of cardiovascular disease plays a key role for the prevention of sudden cardiac death due to coronary artery disease.

Therefore, it is not surprising that a multimodality imaging approach combined with artificial intelligence algorithms may change the management of patients with CAD and reduce the risk of fatal arrhythmias.

Author Contributions: G.M., A.I.G.: Conceptualization and writing; N.S., S.S., P.S. (Paolo Sganzerla), R.C., L.S., P.S. (Paola Sienaand), P.M.: writing; M.G.T., E.G., D.S., G.P., M.G., R.F., A.S., G.C.: Data Curation and writing; M.G.R., G.P., S.S.: Supervision. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: The patients provided informed consent to publish the images.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Vedanthan, R.; Seligman, B.; Fuster, V. Global perspective on acute coronary syndrome: A burden on the young and poor. *Circ. Res.* **2014**, *114*, 1959–1975. [[CrossRef](#)] [[PubMed](#)]
2. Zipes, D.P.; Wellens, H.J. Sudden cardiac death. *Circulation* **1998**, *98*, 2334–2351. [[CrossRef](#)] [[PubMed](#)]
3. Hess, P.L.; Wojdyla, D.M.; Al-Khatib, S.M.; Lokhnygina, Y.; Wallentin, L.; Armstrong, P.W.; Roe, M.T.; Ohman, E.M.; Harrington, R.A.; Alexander, J.H.; et al. Sudden Cardiac Death After Non-ST-Segment Elevation Acute Coronary Syndrome. *JAMA Cardiol.* **2016**, *1*, 73–79. [[CrossRef](#)] [[PubMed](#)]
4. Muscogiuri, G.; Guglielmo, M.; Serra, A.; Gatti, M.; Volpato, V.; Schoepf, U.J.; Saba, L.; Cau, R.; Faletti, R.; McGill, L.J.; et al. Multimodality Imaging in Ischemic Chronic Cardiomyopathy. *J. Imaging* **2022**, *8*, 35. [[CrossRef](#)] [[PubMed](#)]
5. Gudenkauf, B.; Hays, A.G.; Tamis-Holland, J.; Trost, J.; Ambinder, D.I.; Wu, K.C.; Arbab-Zadeh, A.; Blumenthal, R.S.; Sharma, G. Role of multimodality imaging in the assessment of myocardial infarction with nonobstructive coronary arteries: Beyond conventional coronary angiography. *J. Am. Heart Assoc.* **2022**, *11*, e022787. [[CrossRef](#)]
6. Wang, Y.; Wang, Z.; Tian, J.; Lu, M. Editorial: Multimodality Imaging in Acute Coronary Syndrome. *Front. Cardiovasc. Med.* **2022**, *9*, 939428. [[CrossRef](#)]
7. Pontone, G.; Guaricci, A.I.; Palmer, S.C.; Andreini, D.; Verdecchia, M.; Fusini, L.; Lorenzoni, V.; Guglielmo, M.; Muscogiuri, G.; Baggiano, A.; et al. Diagnostic performance of non-invasive imaging for stable coronary artery disease: A meta-analysis. *Int. J. Cardiol.* **2020**, *300*, 276–281. [[CrossRef](#)]

8. Conte, E.; Annoni, A.D.; Pontone, G.; Mushtaq, S.; Guglielmo, M.; Baggiano, A.; Volpato, V.; Agalbato, C.; Bonomi, A.; Veglia, F.; et al. Evaluation of coronary plaque characteristics with coronary computed tomography angiography in patients with non-obstructive coronary artery disease: A long-term follow-up study. *Eur. Heart J.-Cardiovasc. Imaging* **2017**, *18*, 1170–1178. [[CrossRef](#)]
9. Budoff, M.J.; Lakshmanan, S.; Toth, P.P.; Hecht, H.S.; Shaw, L.J.; Maron, D.J.; Michos, E.D.; Williams, K.A.; Nasir, K.; Choi, A.D.; et al. Cardiac CT angiography in current practice: An American society for preventive cardiology clinical practice statement. *Am. J. Prev. Cardiol.* **2022**, *9*, 100318. [[CrossRef](#)]
10. Palmisano, A.; Vignale, D.; Tadic, M.; Moroni, F.; De Stefano, D.; Gatti, M.; Boccia, E.; Faletti, R.; Oppizzi, M.; Peretto, G.; et al. Myocardial Late Contrast Enhancement CT in Troponin-Positive Acute Chest Pain Syndrome. *Radiology* **2022**, *302*, 545–553. [[CrossRef](#)]
11. van Assen, M.; Muscogiuri, G.; Caruso, D.; Lee, S.J.; Laghi, A.; De Cecco, C.N. Artificial intelligence in cardiac radiology. *Radiol. Med.* **2020**, *125*, 1186–1199. [[CrossRef](#)] [[PubMed](#)]
12. Muscogiuri, G.; Chiesa, M.; Trotta, M.; Gatti, M.; Palmisano, V.; Dell’Aversana, S.; Baessato, F.; Cavaliere, A.; Cicala, G.; Loffreno, A.; et al. Performance of a deep learning algorithm for the evaluation of CAD-RADS classification with CCTA. *Atherosclerosis* **2020**, *294*, 25–32. [[CrossRef](#)] [[PubMed](#)]
13. Nicol, E.D.; Weir-McCall, J.R.; Shaw, L.J.; Williamson, E. Great debates in cardiac computed tomography: OPINION: “Artificial intelligence and the future of cardiovascular CT—Managing expectation and challenging hype”. *J. Cardiovasc. Comput. Tomogr.* **2022**. [[CrossRef](#)]
14. Huikuri, H.V.; Castellanos, A.; Myerburg, R.J. Sudden Death Due to Cardiac Arrhythmias. *N. Engl. J. Med.* **2001**, *345*, 1473–1482. [[CrossRef](#)] [[PubMed](#)]
15. Cobb, L.A.; Fahrenbruch, C.E.; Olsufka, M.; Copass, M.K. Changing Incidence of Out-of-Hospital Ventricular Fibrillation, 1980–2000. *JAMA* **2002**, *288*, 3008–3013. [[CrossRef](#)] [[PubMed](#)]
16. Nichol, G.; Thomas, E.; Callaway, C.W. Regional variation in out-of-hospital cardiac arrest incidence and outcome. *JAMA* **2008**, *300*, 1423–1431. [[CrossRef](#)]
17. Weisfeldt, M.L.; Everson-Stewart, S.; Sitlani, C.; Rea, T.; Aufderheide, T.P.; Atkins, D.L.; Bigham, B.; Brooks, S.C.; Foerster, C.; Gray, R.; et al. Ventricular Tachyarrhythmias after Cardiac Arrest in Public versus at Home. *N. Engl. J. Med.* **2011**, *364*, 313–321. [[CrossRef](#)]
18. Bhar-Amato, J.; Davies, W.; Agarwal, S. Ventricular Arrhythmia after Acute Myocardial Infarction: ‘The Perfect Storm’. *Arrhythmia Electrophysiol. Rev.* **2017**, *6*, 134–139. [[CrossRef](#)]
19. Wit, A.L. Basic Electrophysiologic Mechanisms of Sudden Cardiac Death Caused by Acute Myocardial Ischemia and Infarction. *Card. Electrophysiol. Clin.* **2017**, *9*, 525–536. [[CrossRef](#)]
20. Demidova, M.M.; Smith, J.G.; Höijer, C.-J.; Holmqvist, F.; Erlinge, D.; Platonov, P. Prognostic impact of early ventricular fibrillation in patients with ST-elevation myocardial infarction treated with primary PCI. *Eur. Heart J. Acute Cardiovasc. Care* **2012**, *1*, 302–311. [[CrossRef](#)]
21. Khairy, P.; Thibault, B.; Talajic, M.; Dubuc, M.; Roy, D.; Guerra, P.G.; Nattel, S. Prognostic significance of ventricular arrhythmias post-myocardial infarction. *Can. J. Cardiol.* **2003**, *19*, 1393–1404. [[PubMed](#)]
22. Fan, X.; Hua, W.; Xu, Y.; Ding, L.; Niu, H.; Chen, K.; Xu, B.; Zhang, S. Incidence and predictors of sudden cardiac death in patients with reduced left ventricular ejection fraction after myocardial infarction in an era of revascularisation. *Heart* **2014**, *100*, 1242–1249. [[CrossRef](#)] [[PubMed](#)]
23. Zheng, Z.-J.; Croft, J.B.; Giles, W.H.; Mensah, G. Sudden Cardiac Death in the United States, 1989 to 1998. *Circulation* **2001**, *104*, 2158–2163. [[CrossRef](#)] [[PubMed](#)]
24. Ruberman, W.; Weinblatt, E.; Goldberg, J.D.; Frank, C.W.; Chaudhary, B.S.; Shapiro, S. Ventricular premature complexes and sudden death after myocardial infarction. *Circulation* **1981**, *64*, 297–305. [[CrossRef](#)]
25. Uretsky, B.F.; Sheahan, R.G. Primary prevention of sudden cardiac death in heart failure: Will the solution be shocking? *J. Am. Coll. Cardiol.* **1997**, *30*, 1589–1597. [[CrossRef](#)]
26. Atkins, J.M.; Leshin, S.J.; Blomqvist, G.; Mullins, C.B. Ventricular Conduction Blocks and Sudden Death in Acute Myocardial Infarction. Potential indications for pacing. *N. Engl. J. Med.* **1973**, *288*, 281–284. [[CrossRef](#)]
27. Kosmidou, I.; Redfors, B.; Dordi, R.; Dizon, J.M.; McAndrew, T.; Mehran, R.; Ben-Yehuda, O.; Mintz, G.S.; Stone, G.W. Incidence, Predictors, and Outcomes of High-Grade Atrioventricular Block in Patients with ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention (from the HORIZONS-AMI Trial). *Am. J. Cardiol.* **2017**, *119*, 1295–1301. [[CrossRef](#)]
28. Kosmidou, I.; Redfors, B.; McAndrew, T. Worsening atrioventricular conduction after hospital discharge in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention: The HORIZONS-AMI trial. *Coron. Artery Dis.* **2017**, *28*, 550–556. [[CrossRef](#)]
29. Gang, U.J.O.; Hvelplund, A.; Pedersen, S.; Iversen, A.; Jons, C.; Abildstrøm, S.Z.; Haarbo, J.; Jensen, J.S.; Thomsen, P.E.B. High-degree atrioventricular block complicating ST-segment elevation myocardial infarction in the era of primary percutaneous coronary intervention. *Europace* **2012**, *14*, 1639–1645. [[CrossRef](#)]

30. Hamm, W.; Rizas, K.D.; von Stülpnagel, L.; Vdovin, N.; Massberg, S.; Kääb, S.; Bauer, A. Implantable cardiac monitors in high-risk post-infarction patients with cardiac autonomic dysfunction and moderately reduced left ventricular ejection fraction: Design and rationale of the SMART-MI trial. *Am. Heart J.* **2017**, *190*, 34–39. [[CrossRef](#)]
31. Maeda, H.; Michiue, T.; Zhu, B.-L.; Ishikawa, T.; Quan, L.; Bessho, Y.; Okazaki, S.; Kamikodai, Y.; Tsuda, K.; Komatsu, A.; et al. Potential risk factors for sudden cardiac death: An analysis of medicolegal autopsy cases. *Leg. Med.* **2009**, *11* (Suppl. S1), S263–S265. [[CrossRef](#)] [[PubMed](#)]
32. Risum, N.; Valeur, N.; Søgaard, P.; Hassager, C.; Køber, L.; Ersbøll, M. Right ventricular function assessed by 2D strain analysis predicts ventricular arrhythmias and sudden cardiac death in patients after acute myocardial infarction. *Eur. Heart J.-Cardiovasc. Imaging* **2017**, *19*, 800–807. [[CrossRef](#)] [[PubMed](#)]
33. Connolly, N.P.; Hennessey, B.; Mylotte, D. Non-ST-segment elevation myocardial infarction with evidence of transmural infarction complicated by left ventricular rupture during percutaneous coronary intervention. *BMJ Case Rep.* **2020**, *13*, e235459. [[CrossRef](#)] [[PubMed](#)]
34. Olsovsky, M.R.; Topaz, O.; DiSciascio, G.; Vetrovec, G.W. Acute traumatic ventricular septal rupture. *Am. Heart J.* **1996**, *131*, 1039–1041. [[CrossRef](#)]
35. Damluji, A.A.; van Diepen, S.; Katz, J.N.; Menon, V.; Tamis-Holland, J.E.; Bakitas, M.; Cohen, M.G.; Balsam, L.B.; Chikwe, J. Mechanical Complications of Acute Myocardial Infarction: A Scientific Statement from the American Heart Association. *Circulation* **2021**, *144*, e16–e35. [[CrossRef](#)]
36. Reddy, Y.; Al-Hijji, M.; Best, P.J.; Sinak, L.J.; Suri, R.M.; Ijioma, N.N.; Aberle, S.J.; Goyal, D.G.; Singh, M. Diagnosis of Free-Wall Rupture by Left Ventricular Angiogram After Inferior ST-Segment–Elevation Myocardial Infarction. *Circulation* **2015**, *132*, e31–e33. [[CrossRef](#)]
37. Thu Kyaw, M.; Maung, Z.M. Hypokalemia-Induced Arrhythmia: A Case Series and Literature Review. *Cureus* **2022**, *14*, e22940. [[CrossRef](#)]
38. El-Sherif, N.; Turitto, G. Electrolyte disorders and arrhythmogenesis. *Cardiol. J.* **2011**, *18*, 233–245.
39. Diercks, D.B.; Shumaik, G.M.; Harrigan, R.A.; Brady, W.J.; Chan, T.C. Electrocardiographic manifestations: Electrolyte abnormalities. *J. Emerg. Med.* **2004**, *27*, 153–160. [[CrossRef](#)]
40. Littmann, L.; Gibbs, M.A. Electrocardiographic manifestations of severe hyperkalemia. *J. Electrocardiol.* **2018**, *51*, 814–817. [[CrossRef](#)]
41. Ashurst, J.; Sergeant, S.R.; Sergeant, B.R. Evidence-Based Management of Potassium Disorders in the Emergency Department. *Emerg. Med. Pract.* **2016**, *18*, 1–24. [[PubMed](#)]
42. Olshansky, B.; Bhattacharya, S.K. Electrolytes and the ECG Intervals: Big Data and Little Insight. *J. Am. Coll. Cardiol.* **2019**, *73*, 3132–3134. [[CrossRef](#)] [[PubMed](#)]
43. Roffi, M.; Patrono, C.; Collet, J.P. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). *Eur. Heart J.* **2016**, *37*, 267–315. [[PubMed](#)]
44. Lang, R.M.; Badano, L.P.; Mor-Avi, V.; Afilalo, J.; Armstrong, A.; Ernande, L.; Flachskampf, F.A.; Foster, E.; Goldstein, S.A.; Kuznetsova, T.; et al. Recommendations for Cardiac Chamber Quantification by Echocardiography in Adults: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J. Am. Soc. Echocardiogr.* **2015**, *28*, 1–39. [[CrossRef](#)]
45. Cameli, M.; Sciacaluga, C.; Loiacono, F.; Simova, I.; Miglioranza, M.H.; Nistor, D.O.; Bandera, F.; Emdin, M.; Giannoni, A.; Ciccone, M.M.; et al. The analysis of left atrial function predicts the severity of functional impairment in chronic heart failure: The FLASH multicenter study. *Int. J. Cardiol.* **2019**, *286*, 87–91. [[CrossRef](#)] [[PubMed](#)]
46. Merlo, M.; Porcari, A.; Pagura, L.; Cameli, M.; Vergaro, G.; Musumeci, B.; Biagini, E.; Canepa, M.; Crotti, L.; Imazio, M.; et al. A national survey on prevalence of possible echocardiographic red flags of amyloid cardiomyopathy in consecutive patients undergoing routine echocardiography: Study design and patients characterization—The first insight from the AC-TIVE Study. *Eur. J. Prev. Cardiol.* **2021**, *29*, e173–e177. [[CrossRef](#)] [[PubMed](#)]
47. Pastore, M.C.; Mandoli, G.E.; Giannoni, A.; Benfari, G.; Dini, F.L.; Pugliese, N.R.; Taddei, C.; Correale, M.; Brunetti, N.D.; Carluccio, E.; et al. Sacubitril/valsartan reduces indications for arrhythmic primary prevention in heart failure with reduced ejection fraction: Insights from DISCOVER-ARNI, a multicenter Italian register. *Eur. Heart J. Open* **2022**, *2*. [[CrossRef](#)]
48. Prastaro, M.; Pirozzi, E.; Gaibazzi, N.; Paolillo, S.; Santoro, C.; Savarese, G.; Losi, M.A.; Esposito, G.; Filardi, P.P.; Trimarco, B.; et al. Expert Review on the Prognostic Role of Echocardiography after Acute Myocardial Infarction. *J. Am. Soc. Echocardiogr.* **2017**, *30*, 431–443. [[CrossRef](#)]
49. Guaricci, A.I.; Chiarello, G.; Gherbesi, E.; Fusini, L.; Soldato, N.; Siena, P.; Ursi, R.; Ruggieri, R.; Guglielmo, M.; Muscogiuri, G.; et al. Coronary-specific quantification of myocardial deformation by strain echocardiography may disclose the culprit vessel in patients with non-ST-segment elevation acute coronary syndrome. *Eur. Heart J. Open* **2022**, *2*. [[CrossRef](#)]
50. Takeuchi, M.; Nishikage, T.; Nakai, H.; Kokumai, M.; Otani, S.; Lang, R.M. The Assessment of Left Ventricular Twist in Anterior Wall Myocardial Infarction Using Two-dimensional Speckle Tracking Imaging. *J. Am. Soc. Echocardiogr.* **2007**, *20*, 36–44. [[CrossRef](#)]
51. Gaibazzi, N.; Porter, T.; Lorenzoni, V.; Pontone, G.; De Santis, D.; De Rosa, A.; Guaricci, A.I. Effect of Coronary Revascularization on the Prognostic Value of Stress Myocardial Contrast Wall Motion and Perfusion Imaging. *J. Am. Heart Assoc.* **2017**, *6*, e006202. [[CrossRef](#)] [[PubMed](#)]

52. Gaibazzi, N.; Davies, J.; Tuttolomondo, D.; Pontone, G.; Guaricci, A.I.; Lorenzoni, V.; Benatti, G.; Siniscalchi, C.; Pastorini, G. Association of coronary artery Doppler-echocardiography diastolic-systolic velocity ratio at rest with obstructive coronary artery stenosis on the left main or left anterior descending coronary artery. *Int. J. Cardiol.* **2019**, *281*, 1–7. [[CrossRef](#)] [[PubMed](#)]
53. Neumar, R.W.; Shuster, M.; Callaway, C.W. Part 1, Executive Summary: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation* **2015**, *132*, S315–S367. [[CrossRef](#)] [[PubMed](#)]
54. Peterson, E.D.; Shah, B.R.; Parsons, L.; Pollack, C.V.; French, W.J.; Canto, J.G.; Gibson, C.M.; Rogers, W.J. Trends in quality of care for patients with acute myocardial infarction in the National Registry of Myocardial Infarction from 1990 to 2006. *Am. Heart J.* **2008**, *156*, 1045–1055. [[CrossRef](#)]
55. Damluji, A.A.; Forman, D.E.; van Diepen, S.; Alexander, K.P.; Page, R.L.; Hummel, S.L.; Menon, V.; Katz, J.N.; Albert, N.M.; Afilalo, J.; et al. Older Adults in the Cardiac Intensive Care Unit: Factoring Geriatric Syndromes in the Management, Prognosis, and Process of Care: A Scientific Statement from the American Heart Association. *Circulation* **2020**, *141*, e6–e32. [[CrossRef](#)]
56. O’gara, P.T.; Kushner, F.G.; Ascheim, D.D.; Casey, D.E.; Chung, M.K.; De Lemos, J.A.; Ettinger, S.M.; Fang, J.C.; Fesmire, F.M.; Franklin, B.A.; et al. 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: Executive Summary: A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Am. Coll. Cardiol.* **2013**, *61*, e78–e140. [[CrossRef](#)]
57. Ibáñez, B.; James, S.; Agewall, S.; Antunes, M.J.; Bucciarelli-Ducci, C.; Bueno, H.; Caforio, A.L.; Crea, F.; Goudevenos, J.A.; Halvorsen, S. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. *Rev. Esp. Cardiol.* **2017**, *70*, 1082. [[CrossRef](#)]
58. Rogers, W.J.; Frederick, P.; Stoehr, E.; Canto, J.G.; Ornato, J.P.; Gibson, C.M.; Pollack, C.V.; Gore, J.M.; Chandra-Strobos, N.; Peterson, E.D.; et al. Trends in presenting characteristics and hospital mortality among patients with ST elevation and non-ST elevation myocardial infarction in the National Registry of Myocardial Infarction from 1990 to 2006. *Am. Heart J.* **2008**, *156*, 1026–1034. [[CrossRef](#)]
59. French, J.K.; Hellkamp, A.S.; Armstrong, P.; Cohen, E.; Kleiman, N.S.; O’Connor, C.M.; Holmes, D.R.; Hochman, J.; Granger, C.B.; Mahaffey, K.W. Mechanical Complications After Percutaneous Coronary Intervention in ST-Elevation Myocardial Infarction (from APEX-AMI). *Am. J. Cardiol.* **2010**, *105*, 59–63. [[CrossRef](#)]
60. Moreyra, A.E.; Huang, M.S.; Wilson, A.C.; Deng, Y.; Cosgrove, N.M.; Kostis, J.B.; MIDAS Study Group (MIDAS 13). Trends in Incidence and Mortality Rates of Ventricular Septal Rupture During Acute Myocardial Infarction. *Am. J. Cardiol.* **2010**, *106*, 1095–1100. [[CrossRef](#)]
61. Elbadawi, A.; Elgendy, I.Y.; Mahmoud, K.; Barakat, A.F.; Mentias, A.; Mohamed, A.H.; Ogunbayo, G.O.; Megaly, M.; Saad, M.; Omer, M.A.; et al. Temporal Trends and Outcomes of Mechanical Complications in Patients with Acute Myocardial Infarction. *JACC Cardiovasc. Interv.* **2019**, *12*, 1825–1836. [[CrossRef](#)] [[PubMed](#)]
62. Honda, S.; Asami, Y.; Yamane, T.; Nagai, T.; Miyagi, T.; Noguchi, T.; Anzai, T.; Goto, Y.; Ishihara, M.; Nishimura, K.; et al. Trends in the Clinical and Pathological Characteristics of Cardiac Rupture in Patients with Acute Myocardial Infarction Over 35 Years. *J. Am. Heart Assoc.* **2014**, *3*, e000984. [[CrossRef](#)] [[PubMed](#)]
63. Sulzgruber, P.; El-Hamid, F.; Koller, L.; Forster, S.; Goliash, G.; Wojta, J.; Niessner, A. Long-term outcome and risk prediction in patients suffering acute myocardial infarction complicated by post-infarction cardiac rupture. *Int. J. Cardiol.* **2017**, *227*, 399–403. [[CrossRef](#)]
64. Matteucci, M.; Fina, D.; Jiritano, F.; Meani, P.; Blankesteyn, W.M.; Raffa, G.M.; Kowaleski, M.; Heuts, S.; Beghi, C.; Maessen, J.; et al. Treatment strategies for post-infarction left ventricular free-wall rupture. *Eur. Hear. J. Acute Cardiovasc. Care* **2019**, *8*, 379–387. [[CrossRef](#)]
65. Formica, F.; Mariani, S.; Singh, G.; D’Alessandro, S.; Messina, L.A.; Jones, N.; Bamodu, O.A.; Sangalli, F.; Paolini, G. Postinfarction left ventricular free wall rupture: A 17-year single-centre experience. *Eur. J. Cardio-Thoracic Surg.* **2018**, *53*, 150–156. [[CrossRef](#)] [[PubMed](#)]
66. Hung, M.-J.; Wang, C.-H.; Cherng, W.-J. Unruptured left ventricular pseudoaneurysm following myocardial infarction. *Heart* **1998**, *80*, 94–97. [[CrossRef](#)] [[PubMed](#)]
67. Goraya, M.H.N.; Kalsoom, S.; Almas, T.; Amin, M.K.; Hussain, N.; Awan, J.R.; Ehtesham, M.; Niaz, M.A.; Virk, H.U.H.; Filby, S.J. Simultaneous Left Ventricular Aneurysm and Ventricular Septal Rupture Complicating Delayed STEMI Presentation: A Case-Based Review of Post-MI Mechanical Complications Amid the COVID-19 Pandemic. *J. Investig. Med. High Impact Case Rep.* **2021**, *9*. [[CrossRef](#)] [[PubMed](#)]
68. El Ouazzani, J.; Jandou, I. Aneurysm and pseudoaneurysm of the left ventricle. *Ann. Med. Surg.* **2022**, *75*, 103405. [[CrossRef](#)]
69. Caldeira, A.; Albuquerque, D.; Coelho, M.; Côte-Real, H. Left Ventricular Pseudoaneurysm: Imagiologic and Intraoperative Images. *Circ. Cardiovasc. Imaging* **2019**, *12*, e009500. [[CrossRef](#)]
70. Alerhand, S.; Carter, J.M. What echocardiographic findings suggest a pericardial effusion is causing tamponade? *Am. J. Emerg. Med.* **2019**, *37*, 321–326. [[CrossRef](#)]
71. Singh, S.; Wann, L.; Klopfenstein, H.; Hartz, A.; Brooks, H.L. Usefulness of right ventricular diastolic collapse in diagnosing cardiac tamponade and comparison to pulsus paradoxus. *Am. J. Cardiol.* **1986**, *57*, 652–656. [[CrossRef](#)]
72. Gillam, L.D.; Guyer, D.E.; Gibson, T.C.; King, M.E.; Marshall, J.E.; Weyman, A.E. Hydrodynamic compression of the right atrium: A new echocardiographic sign of cardiac tamponade. *Circulation* **1983**, *68*, 294–301. [[CrossRef](#)] [[PubMed](#)]

73. Leimgruber, P.P.; Klopfenstein, H.S.; Wann, L.S.; Brooks, H.L. The hemodynamic derangement associated with right ventricular diastolic collapse in cardiac tamponade: An experimental echocardiographic study. *Circulation* **1983**, *68*, 612–620. [[CrossRef](#)]
74. Jones, B.M.; Kapadia, S.R.; Smedira, N.G.; Robich, M.; Tuzcu, E.M.; Menon, V.; Krishnaswamy, A. Ventricular septal rupture complicating acute myocardial infarction: A contemporary review. *Eur. Heart J.* **2014**, *35*, 2060–2068. [[CrossRef](#)] [[PubMed](#)]
75. Crenshaw, B.S.; Granger, C.B.; Birnbaum, Y. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. *Circulation* **2000**, *101*, 27–32. [[CrossRef](#)]
76. Cohle, S.D.; Balraj, E.; Bell, M. Sudden death due to ventricular septal defect. *Pediatr. Dev. Pathol.* **1999**, *2*, 327–332. [[CrossRef](#)]
77. Di Summa, M.; Actis Dato, G.M.; Centofanti, P.; Fortunato, G.; Patanè, F.; Di Rosa, E.; Forsennati, P.G.; La Torre, M. Ventricular septal rupture after a myocardial infarction: Clinical features and long term survival. *J. Cardiovasc. Surg.* **1997**, *38*, 589–593.
78. Topaz, O.; Taylor, A.L. Interventricular septal rupture complicating acute myocardial infarction: From pathophysiologic features to the role of invasive and noninvasive diagnostic modalities in current management. *Am. J. Med.* **1992**, *93*, 683–688. [[CrossRef](#)]
79. Metzler, B.; Siostrzonek, P.; Binder, R.K.; Bauer, A.; Reinstadler, S.J. Decline of acute coronary syndrome admissions in Austria since the outbreak of COVID-19, the pandemic response causes cardiac collateral damage. *Eur. Heart J.* **2020**, *41*, 1852–1853. [[CrossRef](#)]
80. Birnbaum, Y.; Chamoun, A.J.; Conti, V.R.; Uretsky, B.F. Mitral regurgitation following acute myocardial infarction. *Coron. Artery Dis.* **2002**, *13*, 337–344. [[CrossRef](#)]
81. Durko, A.P.; Budde, R.P.J.; Geleijnse, M.L.; Kappetein, A.P. Recognition, assessment and management of the mechanical complications of acute myocardial infarction. *Heart* **2018**, *104*, 1216–1223. [[CrossRef](#)] [[PubMed](#)]
82. Bursi, F.; Enriquez-Sarano, M.; Nkomo, V.T. Heart failure and death after myocardial infarction in the community: The emerging role of mitral regurgitation. *Circulation* **2005**, *111*, 295–301. [[CrossRef](#)] [[PubMed](#)]
83. Watanabe, N. Acute mitral regurgitation. *Heart* **2019**, *105*, 671–677. [[CrossRef](#)] [[PubMed](#)]
84. Moss, A.J.; Zareba, W.; Hall, W.J.; Klein, H.; Wilber, D.J.; Cannom, D.S.; Daubert, J.P.; Higgins, S.L.; Brown, M.W.; Andrews, M.L. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N. Engl. J. Med.* **2002**, *346*, 877–883. [[CrossRef](#)]
85. Underwood, R.D.; Sra, J.; Akhtar, M. Evaluation and treatment strategies in patients at high risk of sudden death post myocardial infarction. *Clin. Cardiol.* **1997**, *20*, 753–758. [[CrossRef](#)] [[PubMed](#)]
86. Goldenberg, I.; Gillespie, J.; Moss, A.J. Long-term benefit of primary prevention with an implantable cardioverter-defibrillator: An extended 8-year follow-up study of the Multicenter Automatic Defibrillator Implantation Trial II. *Circulation* **2010**, *122*, 1265–1271. [[CrossRef](#)]
87. Bardy, G.H.; Lee, K.L.; Mark, D.B.; Poole, J.E.; Packer, D.L.; Boineau, R.; Domanski, M.; Troutman, C.; Anderson, J.; Johnson, G.; et al. Amiodarone or an Implantable Cardioverter-Defibrillator for Congestive Heart Failure. *N. Engl. J. Med.* **2005**, *352*, 225–237. [[CrossRef](#)]
88. Ruwald, M.H.; Solomon, S.D.; Foster, E. Left ventricular ejection fraction normalization in cardiac resynchronization therapy and risk of ventricular arrhythmias and clinical outcomes: Results from the Multicenter Automatic Defibrillator Implantation Trial with Cardiac Resynchronization Therapy (MADIT-CRT) trial. *Circulation* **2014**, *130*, 2278–2286.
89. Stevens, S.M.; Reinier, K.; Chugh, S.S. Increased left ventricular mass as a predictor of sudden cardiac death: Is it time to put it to the test? *Circ. Arrhythm. Electrophysiol.* **2013**, *6*, 212–217. [[CrossRef](#)]
90. Laukkanen, J.A.; Khan, H.; Kurl, S.; Willeit, P.; Karppi, J.; Ronkainen, K.; Di Angelantonio, E. Left Ventricular Mass and the Risk of Sudden Cardiac Death: A Population-Based Study. *J. Am. Heart Assoc.* **2014**, *3*, e001285. [[CrossRef](#)]
91. Buxton, A.E.; Ellison, K.E.; Lorvidhaya, P.; Ziv, O. Left ventricular ejection fraction for sudden death risk stratification and guiding implantable cardioverter-defibrillators implantation. *J. Cardiovasc. Pharmacol.* **2010**, *55*. [[CrossRef](#)]
92. Haugaa, K.H.; Grenne, B.L.; Eek, C.H. Strain echocardiography improves risk prediction of ventricular arrhythmias after myocardial infarction. *JACC Cardiovasc. Imaging* **2013**, *6*, 841–850. [[CrossRef](#)] [[PubMed](#)]
93. Edvardsen, T.; Gerber, B.L.; Garot, J.; Bluemke, D.A.; Lima, J.A.; Smiseth, O.A. Quantitative assessment of intrinsic regional myocardial deformation by Doppler strain rate echocardiography in humans: Validation against three-dimensional tagged magnetic resonance imaging. *Circulation* **2002**, *106*, 50–56. [[CrossRef](#)]
94. Eek, C.; Grenne, B.; Brunvand, H.; Aakhus, S.; Endresen, K.; Smiseth, O.A.; Edvardsen, T.; Skulstad, H. Postsystolic shortening is a strong predictor of recovery of systolic function in patients with non-ST-elevation myocardial infarction. *Eur. J. Echocardiogr.* **2011**, *12*, 483–489. [[CrossRef](#)]
95. Grenne, B.; Eek, C.; Sjøli, B.; Dahlslett, T.; Hol, P.K.; Ørn, S.; Skulstad, H.; Smiseth, O.A.; Edvardsen, T.; Brunvand, H. Mean Strain Throughout the Heart Cycle by Longitudinal Two-Dimensional Speckle-Tracking Echocardiography Enables Early Prediction of Infarct Size. *J. Am. Soc. Echocardiogr.* **2011**, *24*, 1118–1125. [[CrossRef](#)]
96. Haugaa, K.H.; Smedsrud, M.K.; Steen, T.; Kongsgaard, E.; Loennechen, J.P.; Skjaerpe, T.; Voigt, J.-U.; Willems, R.; Smith, G.; Smiseth, O.A.; et al. Mechanical Dispersion Assessed by Myocardial Strain in Patients After Myocardial Infarction for Risk Prediction of Ventricular Arrhythmia. *JACC Cardiovasc. Imaging* **2010**, *3*, 247–256. [[CrossRef](#)]
97. Dagues, N.; Hindricks, G. Risk stratification after myocardial infarction: Is left ventricular ejection fraction enough to prevent sudden cardiac death? *Eur. Heart J.* **2013**, *34*, 1964–1971. [[CrossRef](#)]

98. Muscogiuri, G.; Gatti, M.; Dell'Aversana, S.; Andreini, D.; Guaricci, A.I.; Guglielmo, M.; Baggiano, A.; Mushtaq, S.; Conte, E.; Annoni, A.D.; et al. Diagnostic Accuracy of Single-shot 2-Dimensional Multisegment Late Gadolinium Enhancement in Ischemic and Nonischemic Cardiomyopathy. *J. Thorac. Imaging* **2020**, *35*, 56–63. [[CrossRef](#)]
99. Muscogiuri, G.; Gatti, M.; Dell'Aversana, S.; Guaricci, A.I.; Guglielmo, M.; Baggiano, A.; Andreini, D.; Mushtaq, S.; Conte, E.; Annoni, A.; et al. Image Quality and Reliability of a Novel Dark-Blood Late Gadolinium Enhancement Sequence in Ischemic Cardiomyopathy. *J. Thorac. Imaging* **2019**, *35*, 326–333. [[CrossRef](#)]
100. Muscogiuri, G.; Martini, C.; Gatti, M.; Dell'Aversana, S.; Ricci, F.; Guglielmo, M.; Baggiano, A.; Fusini, L.; Bracciani, A.; Scafuri, S.; et al. Feasibility of late gadolinium enhancement (LGE) in ischemic cardiomyopathy using 2D-multisegment LGE combined with artificial intelligence reconstruction deep learning noise reduction algorithm. *Int. J. Cardiol.* **2021**, *343*, 164–170. [[CrossRef](#)]
101. Muscogiuri, G.; Rehwald, W.G.; Schoepf, U.J.; Suranyi, P.; Litwin, S.E.; De Cecco, C.N.; Wichmann, J.L.; Mangold, S.; Caruso, D.; Fuller, S.R.; et al. T(Rho) and magnetization transfer and INvERSION recovery (TRAMINER)-prepared imaging: A novel contrast-enhanced flow-independent dark-blood technique for the evaluation of myocardial late gadolinium enhancement in patients with myocardial infarction. *J. Magn. Reson. Imaging* **2017**, *45*, 1429–1437. [[CrossRef](#)]
102. Roes, S.D.; Borleffs, C.J.W.; van der Geest, R.J.; Westenberg, J.J.; Marsan, N.A.; Kaandorp, T.A.; Reiber, J.H.; Zeppenfeld, K.; Lamb, H.J.; de Roos, A.; et al. Infarct Tissue Heterogeneity Assessed with Contrast-Enhanced MRI Predicts Spontaneous Ventricular Arrhythmia in Patients with Ischemic Cardiomyopathy and Implantable Cardioverter-Defibrillator. *Circ. Cardiovasc. Imaging* **2009**, *2*, 183–190. [[CrossRef](#)] [[PubMed](#)]
103. Pontone, G.; Andreini, D.; Bertella, E.; Loguercio, M.; Guglielmo, M.; Baggiano, A.; Aquaro, G.D.; Mushtaq, S.; Salerni, S.; Gripari, P.; et al. Prognostic value of dipyridamole stress cardiac magnetic resonance in patients with known or suspected coronary artery disease: A mid-term follow-up study. *Eur. Radiol.* **2016**, *26*, 2155–2165. [[CrossRef](#)] [[PubMed](#)]
104. Muscogiuri, G.; Ricci, F.; Scafuri, S.; Guglielmo, M.; Baggiano, A.; De Stasio, V.; Di Donna, C.; Spiritiglozzi, L.; Chiochi, M.; Lee, S.J.; et al. Cardiac Magnetic Resonance Tissue Characterization in Ischemic Cardiomyopathy. *J. Thorac. Imaging* **2022**, *37*, 2–16. [[CrossRef](#)] [[PubMed](#)]
105. Pontone, G.; Andreini, D.; Baggiano, A.; Bertella, E.; Mushtaq, S.; Conte, E.; Beltrama, V.; Guaricci, A.I.; Pepi, M. Functional Relevance of Coronary Artery Disease by Cardiac Magnetic Resonance and Cardiac Computed Tomography: Myocardial Perfusion and Fractional Flow Reserve. *BioMed Res. Int.* **2015**, *2015*, 297696. [[CrossRef](#)]
106. Kim, H.W.; Van Assche, L.; Jennings, R.B.; Wince, W.B.; Jensen, C.J.; Rehwald, W.G.; Wendell, D.C.; Bhatti, L.; Spatz, D.M.; Parker, M.A.; et al. Relationship of T2-Weighted MRI Myocardial Hyperintensity and the Ischemic Area-At-Risk. *Circ. Res.* **2015**, *117*, 254–265. [[CrossRef](#)]
107. Srichai, M.B.; Lim, R.P.; Lath, N.; Babb, J.; Axel, L.; Kim, D. Diagnostic Performance of Dark-Blood T2-Weighted CMR for Evaluation of Acute Myocardial Injury. *Investig. Radiol.* **2013**, *48*, 24–31. [[CrossRef](#)]
108. De Cecco, C.N.; Muscogiuri, G.; Varga-Szemes, A.; Schoepf, U.J. Cutting edge clinical applications in cardiovascular magnetic resonance. *World J. Radiol.* **2017**, *9*, 1–4. [[CrossRef](#)]
109. Muscogiuri, G.; Suranyi, P.; Schoepf, U.J. Cardiac Magnetic Resonance T1-Mapping of the Myocardium: Technical Background and Clinical Relevance. *J. Thorac. Imaging* **2018**, *33*, 71–80. [[CrossRef](#)]
110. Giri, S.; Chung, Y.-C.; Merchant, A.; Mihai, G.; Rajagopalan, S.; Raman, S.V.; Simonetti, O.P. T2 quantification for improved detection of myocardial edema. *J. Cardiovasc. Magn. Reson.* **2009**, *11*, 56. [[CrossRef](#)]
111. Bulluck, H.; White, S.K.; Rosmini, S.; Bhuvana, A.N.; Treibel, T.A.; Fontana, M.; Abdel-Gadir, A.; Herrey, A.S.; Manisty, C.H.; Wan, S.M.Y.; et al. T1 mapping and T2 mapping at 3T for quantifying the area-at-risk in reperfused STEMI patients. *J. Cardiovasc. Magn. Reson.* **2015**, *17*, 73. [[CrossRef](#)] [[PubMed](#)]
112. Ibanez, B.; Aletras, A.H.; Arai, A.E. Cardiac MRI Endpoints in Myocardial Infarction Experimental and Clinical Trials: JACC Scientific Expert Panel. *J. Am. Coll. Cardiol.* **2019**, *74*, 238–256. [[CrossRef](#)]
113. Fernández-Friera, L.; García-Ruiz, J.M.; García-Álvarez, A.; Fernández-Jiménez, R.; Sánchez-González, J.; Rossello, X.; Gómez-Talavera, S.; López-Martín, G.J.; Pizarro, G.; Fuster, V.; et al. Accuracy of Area at Risk Quantification by Cardiac Magnetic Resonance According to the Myocardial Infarction Territory. *Rev. Española Cardiol.* **2017**, *70*, 323–330. [[CrossRef](#)]
114. Zorzi, A.; Mattesi, G.; Baldi, E.; Toniolo, M.; Guerra, F.; Cauti, F.M.; Cipriani, A.; De Lazzari, M.; Muser, D.; Stronati, G.; et al. Prognostic Role of Myocardial Edema as Evidenced by Early Cardiac Magnetic Resonance in Survivors of Out-of-Hospital Cardiac Arrest: A Multicenter Study. *J. Am. Heart Assoc.* **2021**, *10*, e021861. [[CrossRef](#)] [[PubMed](#)]
115. Zorzi, A.; Susana, A.; De Lazzari, M.; Migliore, F.; Vescovo, G.; Scarpa, D.; Baritussio, A.; Tarantini, G.; Cacciavillani, L.; Giorgi, B.; et al. Diagnostic value and prognostic implications of early cardiac magnetic resonance in survivors of out-of-hospital cardiac arrest. *Heart Rhythm* **2018**, *15*, 1031–1041. [[CrossRef](#)] [[PubMed](#)]
116. Niccoli, G.; Scalone, G.; Lerman, A.; Crea, F. Coronary microvascular obstruction in acute myocardial infarction. *Eur. Heart J.* **2016**, *37*, 1024–1033. [[CrossRef](#)] [[PubMed](#)]
117. Pontone, G.; Andreini, D.; Guaricci, A.I. Association Between Haptoglobin Phenotype and Microvascular Obstruction in Patients With STEMI: A Cardiac Magnetic Resonance Study. *JACC Cardiovasc. Imaging* **2019**, *12*, 1007–1017. [[CrossRef](#)]
118. Abbas, A.; Matthews, G.H.; Brown, I.W.; Shambrook, J.; Peebles, C.R.; Harden, S.P. Cardiac MR assessment of microvascular obstruction. *Br. J. Radiol.* **2015**, *88*, 20140470. [[CrossRef](#)]

119. Chen, B.H.; An, D.A.; He, J.; Xu, J.R.; Wu, L.M.; Pu, J. Myocardial Extracellular Volume Fraction Allows Differentiation of Reversible Versus Irreversible Myocardial Damage and Prediction of Adverse Left Ventricular Remodeling of ST-Elevation Myocardial Infarction. *J. Magn. Reson. Imaging* **2020**, *52*, 476–487. [[CrossRef](#)]
120. Nijveldt, R.; Hofman, M.B.M.; Hirsch, A.; Beek, A.M.; Umans, V.A.W.M.; Algra, P.R.; Piek, J.J.; Van Rossum, A.C. Assessment of Microvascular Obstruction and Prediction of Short-term Remodeling after Acute Myocardial Infarction: Cardiac MR Imaging Study. *Radiology* **2009**, *250*, 363–370. [[CrossRef](#)]
121. Ito, H.; Tomooka, T.; Sakai, N.; Yu, H.; Higashino, Y.; Fujii, K.; Masuyama, T.; Kitabatake, A.; Minamino, T. Lack of myocardial perfusion immediately after successful thrombolysis. A predictor of poor recovery of left ventricular function in anterior myocardial infarction. *Circulation* **1992**, *85*, 1699–1705. [[CrossRef](#)] [[PubMed](#)]
122. Ito, H.; Maruyama, A.; Iwakura, K. Clinical implications of the ‘no reflow’ phenomenon. A predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction. *Circulation* **1996**, *93*, 223–228. [[CrossRef](#)] [[PubMed](#)]
123. De Waha, S.; Patel, M.R.; Granger, C.B.; Ohman, E.M.; Maehara, A.; Eitel, I.; Ben-Yehuda, O.; Jenkins, P.; Thiele, H.; Stone, G.W. Relationship between microvascular obstruction and adverse events following primary percutaneous coronary intervention for ST-segment elevation myocardial infarction: An individual patient data pooled analysis from seven randomized trials. *Eur. Heart J.* **2017**, *38*, 3502–3510. [[CrossRef](#)] [[PubMed](#)]
124. Hamirani, Y.S.; Wong, A.; Kramer, C.M.; Salerno, M. Effect of microvascular obstruction and intramyocardial hemorrhage by CMR on LV remodeling and outcomes after myocardial infarction: A systematic review and meta-analysis. *JACC Cardiovasc. Imaging* **2014**, *7*, 940–952. [[CrossRef](#)] [[PubMed](#)]
125. O’Regan, D.P.; Ariff, B.; Neuwirth, C.; Tan, Y.; Durighel, G.; Cook, S.A. Assessment of severe reperfusion injury with T2* cardiac MRI in patients with acute myocardial infarction. *Heart* **2010**, *96*, 1885–1891. [[CrossRef](#)]
126. Mather, A.N.; Fairbairn, T.A.; Ball, S.G.; Greenwood, J.P.; Plein, S. Reperfusion haemorrhage as determined by cardiovascular MRI is a predictor of adverse left ventricular remodelling and markers of late arrhythmic risk. *Heart* **2011**, *97*, 453–459. [[CrossRef](#)]
127. Cokic, I.; Kali, A.; Wang, X.; Yang, H.-J.; Tang, R.L.Q.; Thajudeen, A.; Shehata, M.; Amorn, A.M.; Liu, E.; Stewart, B.; et al. Iron Deposition following Chronic Myocardial Infarction as a Substrate for Cardiac Electrical Anomalies: Initial Findings in a Canine Model. *PLoS ONE* **2013**, *8*, e73193. [[CrossRef](#)]
128. Delewi, R.; Zijlstra, F.; Piek, J.J. Left ventricular thrombus formation after acute myocardial infarction. *Heart* **2012**, *98*, 1743–1749. [[CrossRef](#)]
129. Motwani, M.; Kidambi, A.; Herzog, B.A.; Uddin, A.; Greenwood, J.P.; Plein, S. MR Imaging of Cardiac Tumors and Masses: A Review of Methods and Clinical Applications. *Radiology* **2013**, *268*, 26–43. [[CrossRef](#)]
130. Roifman, I.; Connelly, K.A.; Wright, G.A.; Wijeyesundera, H.C. Echocardiography vs Cardiac Magnetic Resonance Imaging for the Diagnosis of Left Ventricular Thrombus: A Systematic Review. *Can. J. Cardiol.* **2015**, *31*, 785–791. [[CrossRef](#)]
131. Caspar, T.; El Ghannudi, S.; Ohana, M.; Labani, A.; Lawson, A.; Ohlmann, P.; Morel, O.; De Mathelin, M.; Roy, C.; Gangi, A.; et al. Magnetic resonance evaluation of cardiac thrombi and masses by T1 and T2 mapping: An observational study. *Int. J. Cardiovasc. Imaging* **2017**, *33*, 551–559. [[CrossRef](#)] [[PubMed](#)]
132. Bulluck, H.; Chan, M.H.H.; Paradies, V.; Yellon, R.L.; Ho, H.H.; Chan, M.Y.; Chin, C.W.L.; Tan, J.W.; Hausenloy, D.J. Incidence and predictors of left ventricular thrombus by cardiovascular magnetic resonance in acute ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention: A meta-analysis. *J. Cardiovasc. Magn. Reson.* **2018**, *20*, 72. [[CrossRef](#)] [[PubMed](#)]
133. Meltzer, R.S.; Visser, C.A.; Fuster, V. Intracardiac Thrombi and Systemic Embolization. *Ann. Intern. Med.* **1986**, *104*, 689. [[CrossRef](#)] [[PubMed](#)]
134. Guaricci, A.I.; Masci, P.G.; Muscogiuri, G. Cardiac magnetic Resonance for prophylactic Implantable-cardioverter defibrillator Therapy in Non-Ischaemic dilated Cardiomyopathy: An international Registry. *Europace* **2021**, *23*, 1072–1083. [[CrossRef](#)]
135. Al’Aref, S.J.; Altibi, A.M.; Malkawi, A.; Mansour, M.; Baskaran, L.; Masri, A.; Rahmouni, H.; Abete, R.; Andreini, D.; et al.; Writing Committee. Cardiac magnetic resonance for prophylactic implantable-cardioverter defibrillator therapy international study: Prognostic value of cardiac magnetic resonance-derived right ventricular parameters substudy. *Eur. Heart J.-Cardiovasc. Imaging* **2022**. [[CrossRef](#)]
136. Merlo, M.; Gagno, G.; Baritussio, A. Clinical application of CMR in cardiomyopathies: Evolving concepts and techniques: A position paper of myocardial and pericardial diseases and cardiac magnetic resonance working groups of Italian society of cardiology. *Heart Fail Rev.* **2022**. [[CrossRef](#)]
137. Bogun, F.; Desjardins, B.; Crawford, T.; Good, E.; Jongnarangsin, K.; Oral, H.; Chugh, A.; Pelosi, F.; Morady, F. Post-Infarction Ventricular Arrhythmias Originating in Papillary Muscles. *J. Am. Coll. Cardiol.* **2008**, *51*, 1794–1802. [[CrossRef](#)]
138. Robbers, L.F.H.J.; Delewi, R.; Nijveldt, R.; Hirsch, A.; Beek, A.M.; Kemme, M.; Van Beurden, Y.; Van Der Laan, A.M.; Van Der Vleuten, P.A.; Tio, R.A.; et al. Myocardial infarct heterogeneity assessment by late gadolinium enhancement cardiovascular magnetic resonance imaging shows predictive value for ventricular arrhythmia development after acute myocardial infarction. *Eur. Heart J.-Cardiovasc. Imaging* **2013**, *14*, 1150–1158. [[CrossRef](#)]
139. Kim, R.J.; Chen, E.-L.; Lima, J.A.; Judd, R.M. Myocardial Gd-DTPA Kinetics Determine MRI Contrast Enhancement and Reflect the Extent and Severity of Myocardial Injury After Acute Reperfused Infarction. *Circulation* **1996**, *94*, 3318–3326. [[CrossRef](#)]

140. Kim, R.J.; Fieno, D.S.; Parrish, T.; Harris, K.; Chen, E.-L.; Simonetti, O.; Bundy, J.; Finn, J.P.; Klocke, F.J.; Judd, R.M. Relationship of MRI Delayed Contrast Enhancement to Irreversible Injury, Infarct Age, and Contractile Function. *Circulation* **1999**, *100*, 1992–2002. [[CrossRef](#)]
141. Kim, R.J.; Shah, D.J.; Judd, R.M. How We Perform Delayed Enhancement Imaging. *J. Cardiovasc. Magn. Reson.* **2003**, *5*, 505–514. [[CrossRef](#)] [[PubMed](#)]
142. Holtackers, R.J.; Van De Heyning, C.M.; Nazir, M.S.; Rashid, I.; Ntalas, I.; Rahman, H.; Botnar, R.M.; Chiribiri, A. Clinical value of dark-blood late gadolinium enhancement cardiovascular magnetic resonance without additional magnetization preparation. *J. Cardiovasc. Magn. Reson.* **2019**, *21*, 1–11. [[CrossRef](#)] [[PubMed](#)]
143. Good, E.; Desjardins, B.; Jongnarangsin, K.; Oral, H.; Chugh, A.; Ebinger, M.; Pelosi, F.; Morady, F.; Bogun, F. Ventricular arrhythmias originating from a papillary muscle in patients without prior infarction: A comparison with fascicular arrhythmias. *Heart Rhythm* **2008**, *5*, 1530–1537. [[CrossRef](#)] [[PubMed](#)]
144. Guglielmo, M.; Fusini, L.; Muscogiuri, G.; Baessato, F.; Loffreno, A.; Cavaliere, A.; Rizzon, G.; Baggiano, A.; Rabbat, M.G.; Muratori, M.; et al. T1 mapping and cardiac magnetic resonance feature tracking in mitral valve prolapse. *Eur. Radiol.* **2020**, *31*, 1100–1109. [[CrossRef](#)]
145. Knuuti, J.; Wijns, W.; Saraste, A. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. *Eur. Heart J.* **2020**, *41*, 407–477. [[CrossRef](#)] [[PubMed](#)]
146. Writing Committee, M.; Gulati, M.; Levy, P.D. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J. Cardiovasc. Comput. Tomogr.* **2022**, *16*, 54–122.
147. Guaricci, A.I.; Maffei, E.; Brunetti, N.D.; Montrone, D.; Di Biase, L.; Tedeschi, C.; Gentile, G.; Macarini, L.; Midiri, M.; Cademartiri, F.; et al. Heart rate control with oral ivabradine in computed tomography coronary angiography: A randomized comparison of 7.5 mg vs. 5 mg regimen. *Int. J. Cardiol.* **2013**, *168*, 362–368. [[CrossRef](#)]
148. Maffei, E.; Seitun, S.; Martini, C.; Palumbo, A.; Tarantini, G.; Berti, E.; Grilli, R.; Tedeschi, C.; Messalli, G.; Guaricci, A.; et al. CT coronary angiography and exercise ECG in a population with chest pain and low-to-intermediate pre-test likelihood of coronary artery disease. *Heart* **2010**, *96*, 1973–1979. [[CrossRef](#)]
149. Maffei, E.; Seitun, S.; Martini, C.; Aldrovandi, A.; Cervellin, G.; Tedeschi, C.; Guaricci, A.I.; Messalli, G.; Catalano, O.; Cademartiri, F. Prognostic value of computed tomography coronary angiography in patients with chest pain of suspected cardiac origin. *La Radiol. Med.* **2011**, *116*, 690–705. [[CrossRef](#)]
150. Esposito, A.; Francone, M.; Andreini, D. SIRM-SIC appropriateness criteria for the use of Cardiac Computed Tomography. Part 1, Congenital heart diseases, primary prevention, risk assessment before surgery, suspected CAD in symptomatic patients, plaque and epicardial adipose tissue characterization, and functional assessment of stenosis. *Radiol. Med.* **2021**, *126*, 1236–1248.
151. Carrabba, N.; Pontone, G.; Andreini, D.; Buffa, V.; Cademartiri, F.; Carbone, I.; Clemente, A.; Guaricci, A.I.; Guglielmo, M.; Indolfi, C.; et al. Appropriateness criteria for the use of cardiac computed tomography, SIC-SIRM part 2, acute chest pain evaluation; stent and coronary artery bypass graft patency evaluation; planning of coronary revascularization and transcatheter valve procedures; cardiomyopathies, electrophysiological applications, cardiac masses, cardio-oncology and pericardial diseases evaluation. *J. Cardiovasc. Med.* **2022**, *23*, 290–303. [[CrossRef](#)]
152. Guaricci, A.I.; Arcadi, T.; Brunetti, N.D.; Maffei, E.; Montrone, D.; Martini, C.; De Luca, M.; De Rosa, F.; Cocco, D.; Midiri, M.; et al. Carotid intima media thickness and coronary atherosclerosis linkage in symptomatic intermediate risk patients evaluated by coronary computed tomography angiography. *Int. J. Cardiol.* **2014**, *176*, 988–993. [[CrossRef](#)] [[PubMed](#)]
153. Guaricci, A.I.; Pontone, G.; Fusini, L.; De Luca, M.; Cafarelli, F.P.; Guglielmo, M.; Baggiano, A.; Beltrama, V.; Muscogiuri, G.; Mushtaq, S.; et al. Additional value of inflammatory biomarkers and carotid artery disease in prediction of significant coronary artery disease as assessed by coronary computed tomography angiography. *Eur. Heart J.-Cardiovasc. Imaging* **2016**, *18*, 1049–1056. [[CrossRef](#)] [[PubMed](#)]
154. De Cecco, C.N.; Bastarrika, G.; Arraiza, M.; Enrici, M.M.; Pueyo, J.; Muscogiuri, G.; Fina, P.; Anselmi, A.; Di Girolamo, M.; David, V. Dual source CT: State of the art in the depiction of coronary arteries anatomy, anatomical variants and myocardial segments. *Minerva Cardioangiol.* **2012**, *60*, 133–146. [[PubMed](#)]
155. Esposito, A.; Palmisano, A.; Barbera, M. Cardiac Computed Tomography in Troponin-Positive Chest Pain: Sometimes the Answer Lies in the Late Iodine Enhancement or Extracellular Volume Fraction Map. *JACC Cardiovasc. Imaging* **2019**, *12*, 745–748. [[CrossRef](#)] [[PubMed](#)]
156. Van den Hoogen, I.J.; Gianni, U.; Al Hussein Alawamlh, O. What atherosclerosis findings can CT see in sudden coronary death: Plaque rupture versus plaque erosion. *J. Cardiovasc Comput. Tomogr.* **2020**, *14*, 214–218. [[CrossRef](#)]
157. Motoyama, S.; Ito, H.; Sarai, M.; Kondo, T.; Kawai, H.; Nagahara, Y.; Harigaya, H.; Kan, S.; Anno, H.; Takahashi, H.; et al. Plaque Characterization by Coronary Computed Tomography Angiography and the Likelihood of Acute Coronary Events in Mid-Term Follow-Up. *J. Am. Coll. Cardiol.* **2015**, *66*, 337–346. [[CrossRef](#)]
158. Chang, H.-J.; Lin, F.Y.; Lee, S.-E.; Andreini, D.; Bax, J.; Cademartiri, F.; Chinnaiyan, K.; Chow, B.J.; Conte, E.; Cury, R.C.; et al. Coronary Atherosclerotic Precursors of Acute Coronary Syndromes. *J. Am. Coll. Cardiol.* **2018**, *71*, 2511–2522. [[CrossRef](#)]
159. Williams, M.C.; Moss, A.J.; Dweck, M.; Adamson, P.D.; Alam, S.; Hunter, A.; Shah, A.S.; Pawade, T.; Weir-McCall, J.R.; Roditi, G.; et al. Coronary Artery Plaque Characteristics Associated with Adverse Outcomes in the SCOT-HEART Study. *J. Am. Coll. Cardiol.* **2019**, *73*, 291–301. [[CrossRef](#)]

160. Min, J.K.; Shaw, L.J.; Devereux, R.B.; Okin, P.M.; Weinsaft, J.W.; Russo, D.J.; Lippolis, N.J.; Berman, D.S.; Callister, T.Q. Prognostic Value of Multidetector Coronary Computed Tomographic Angiography for Prediction of All-Cause Mortality. *J. Am. Coll. Cardiol.* **2007**, *50*, 1161–1170. [[CrossRef](#)]
161. Cury, R.C.; Blankstein, R.; Leipsic, J. CAD-RADS 2.0—2022 Coronary Artery Disease—Reporting and Data System an expert consensus document of the Society of Cardiovascular Computed Tomography (SCCT), the American College of Cardiology (ACC), the American College of Radiology (ACR) and the North America society of cardiovascular imaging (NASCI). *J. Cardiovasc. Comput. Tomogr.* **2022**, in press.
162. Pontone, G.; Andreini, D.; Guaricci, A.I.; Guglielmo, M.; Baggiano, A.; Muscogiuri, G.; Fusini, L.; Soldi, M.; Fazzari, F.; Berzovini, C.; et al. Quantitative vs. qualitative evaluation of static stress computed tomography perfusion to detect haemodynamically significant coronary artery disease. *Eur. Heart J.-Cardiovasc. Imaging* **2018**, *19*, 1244–1252. [[CrossRef](#)] [[PubMed](#)]
163. Baggiano, A.; Fusini, L.; Del Torto, A. Sequential Strategy Including FFR(CT) Plus Stress-CTP Impacts on Management of Patients with Stable Chest Pain: The Stress-CTP RIPCARD Study. *J. Clin. Med.* **2020**, *9*, 2147. [[CrossRef](#)] [[PubMed](#)]
164. Puchner, S.B.; Liu, T.; Mayrhofer, T. High-risk plaque detected on coronary CT angiography predicts acute coronary syndromes independent of significant stenosis in acute chest pain: Results from the ROMICAT-II trial. *J. Am. Coll. Cardiol.* **2014**, *64*, 684–692. [[CrossRef](#)]
165. Hoffmann, U.; Bamberg, F.; Chae, C.U.; Nichols, J.H.; Rogers, I.S.; Seneviratne, S.K.; Truong, Q.A.; Cury, R.C.; Abbara, S.; Shapiro, M.D.; et al. Coronary Computed Tomography Angiography for Early Triage of Patients with Acute Chest Pain: The ROMICAT (Rule Out Myocardial Infarction using Computer Assisted Tomography) Trial. *J. Am. Coll. Cardiol.* **2009**, *53*, 1642–1650. [[CrossRef](#)]
166. Gautam, N.; Saluja, P.; Malkawi, A.; Rabbat, M.G.; Al-Mallah, M.H.; Pontone, G.; Zhang, Y.; Lee, B.C.; Al'Aref, S.J. Current and Future Applications of Artificial Intelligence in Coronary Artery Disease. *Healthcare* **2022**, *10*, 232. [[CrossRef](#)]
167. Muscogiuri, G.; Chiesa, M.; Baggiano, A.; Spadafora, P.; De Santis, R.; Guglielmo, M.; Scafuri, S.; Fusini, L.; Mushtaq, S.; Conte, E.; et al. Diagnostic performance of deep learning algorithm for analysis of computed tomography myocardial perfusion. *Eur. J. Pediatr.* **2022**, *49*, 3119–3128. [[CrossRef](#)]
168. Tat, E.; Bhatt, D.L.; Rabbat, M.G. Addressing bias: Artificial intelligence in cardiovascular medicine. *Lancet Digit. Health* **2020**, *2*, e635–e636. [[CrossRef](#)]
169. Narang, A.; Mor-Avi, V.; Prado, A.; Volpato, V.; Prater, D.; Tamborini, G.; Fusini, L.; Pepi, M.; Goyal, N.; Addetia, K.; et al. Machine learning based automated dynamic quantification of left heart chamber volumes. *Eur. Heart J.-Cardiovasc. Imaging* **2018**, *20*, 541–549. [[CrossRef](#)]
170. Kusunose, K.; Haga, A.; Abe, T.; Sata, M. Utilization of Artificial Intelligence in Echocardiography. *Circ. J.* **2019**, *83*, 1623–1629. [[CrossRef](#)]
171. Penso, M.; Pepi, M.; Mantegazza, V.; Cefalù, C.; Muratori, M.; Fusini, L.; Gripari, P.; Ali, S.G.; Caiani, E.G.; Tamborini, G. Machine Learning Prediction Models for Mitral Valve Repairability and Mitral Regurgitation Recurrence in Patients Undergoing Surgical Mitral Valve Repair. *Bioengineering* **2021**, *8*, 117. [[CrossRef](#)] [[PubMed](#)]
172. Moccia, S.; Banali, R.; Martini, C.; Muscogiuri, G.; Pontone, G.; Pepi, M.; Caiani, E.G. Development and testing of a deep learning-based strategy for scar segmentation on CMR-LGE images. *Magn. Reson. Mater. Physics, Biol. Med.* **2018**, *32*, 187–195. [[CrossRef](#)] [[PubMed](#)]
173. Muscogiuri, G.; Van Assen, M.; Tesche, C.; De Cecco, C.N.; Chiesa, M.; Scafuri, S.; Guglielmo, M.; Baggiano, A.; Fusini, L.; Guaricci, A.I.; et al. Artificial Intelligence in Coronary Computed Tomography Angiography: From Anatomy to Prognosis. *BioMed Res. Int.* **2020**, *2020*, 6649410. [[CrossRef](#)] [[PubMed](#)]
174. Choi, A.D.; Marques, H.; Kumar, V. CT Evaluation by Artificial Intelligence for Atherosclerosis, Stenosis and Vascular Morphology (CLARIFY): A Multi-center, international study. *J. Cardiovasc Comput Tomogr* **2021**, *15*, 470–476. [[CrossRef](#)]
175. Griffin, W.F.; Choi, A.D.; Riess, J.S. AI Evaluation of Stenosis on Coronary CT Angiography, Comparison with Quantitative Coronary Angiography and Fractional Flow Reserve: A CREDENCE Trial Substudy. *JACC Cardiovasc. Imaging* **2022**, in press. [[CrossRef](#)]
176. Motwani, M.; Dey, D.; Berman, D.S.; Germano, G.; Achenbach, S.; Al-Mallah, M.; Andreini, D.; Budoff, M.J.; Cademartiri, F.; Callister, T.Q.; et al. Machine learning for prediction of all-cause mortality in patients with suspected coronary artery disease: A 5-year multicentre prospective registry analysis. *Eur. Heart J.* **2016**, *38*, 500–507. [[CrossRef](#)]
177. Van Rosendael, A.R.; Maliakal, G.; Kolli, K.K.; Beecy, A.; Al'Aref, S.J.; Dwivedi, A.; Singh, G.; Panday, M.; Kumar, A.; Ma, X. Maximization of the usage of coronary CTA derived plaque information using a machine learning based algorithm to improve risk stratification; insights from the CONFIRM registry. *J. Cardiovasc. Comput. Tomogr.* **2018**, *12*, 204–209. [[CrossRef](#)]