Cardiac Computed Tomography for the Evaluation of the Acute Chest Pain Syndrome: State of the Art

Christopher L. Schlett, MD, MPH[a,b], Udo Hoffmann, MD, MPH[b], Tobias Geisler, MD[c], Konstantin Nikolaou, MD[d], Fabian Bamberg, MD, MPH[b,d,*]

KEYWORDS

• Acute chest pain syndrome • Acute coronary syndrome • Cardiac computed tomography • CCTA

KEY POINTS

- Coronary computed tomography angiography (CCTA) is considered appropriate for the triage of acute chest pain in patients with a low-to-intermediate likelihood for acute coronary syndrome.
- Absence of any coronary artery disease (CAD) confirmed by CCTA allows rapid emergency department discharge.
- This article reviews the current scientific evidence and controversies on CCTA-based triage as a modality that is as safe as traditional triage, reduces the hospital length of stay, and may provide cost-effective or even cost-saving care.

PATHOPHYSIOLOGY AND CLINICAL PRESENTATION OF ACUTE CORONARY SYNDROME

The acute coronary syndrome (ACS) is mainly caused by underlying coronary atherosclerosis. Other, but extremely rarer pathophysiologic mechanisms are microvascular dysfunction or coronary vasospasm.[1] Today coronary atherosclerosis is known as a chronic inflammatory process within the tunica intima, forming a vessel wall thickening, which is called plaque if it reaches specific morphologic characteristics.

High-Risk Plaques and Significant Stenosis

There are two main concepts of how coronary, arteriosclerotic plaque can lead to ACS. In the first concept, ACS occurs after development of a superficial thrombus and subsequent obstruction of blood flow to downstream coronary segments. The formation of a thrombus is typically due to

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[a] Department of Diagnostic and Interventional Radiology, University Hospital Heidelberg, Im Neuenheimer Feld 110, Heidelberg 69120, Germany; [b] Cardiac MR PET CT Program, Massachusetts General Hospital, Harvard Medical School, 165 Cambridge St, Suite 400, Boston, MA 02114, USA; [c] Department of Cardiology and Cardiovascular Medicine University Hospital of Tübingen, Hoppe-Seyler-Straβe 3, Tübingen 72076, Germany; [d] Department of Diagnostic and Interventional Radiology, University Hospital of Tübingen, Hoppe-Seyler-Straβe 3, Tübingen 72076, Germany
* Corresponding author. Department of Diagnostic and Interventional Radiology, University Hospital Tübingen, Hoppe-Seyler-Straße 3, Tübingen 72076, Germany.
E-mail address: fabian.bamberg@med.uni-tuebingen.de

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rupture (~60%) or erosion (~40%) of an underlying atherosclerotic plaque.[2,3] Particular morphologically features have been observed at the site of plaque rupture or erosion, including a large lipid/necrotic core, a thin fibrous cap, and small calcified embedded nodules. These form the concept of vulnerable plaque, which are at high risk for developing ACS (also called "high-risk plaque").[2] Also, a high inflammatory activity is discussed as a vulnerability factor following the observed increased density of macrophages in ruptured plaques.[2] Although factors such a large necrotic core favor that high-risk plaques are located in areas of high plaque burden, it is not necessarily associated with severe luminal stenosis. In approximately 5% to 20% of all ACS events, no significant stenosis is captured by standard invasive angiogram corresponding to the event location.[2,4]

The second concept is based on increasing luminal narrowing to due atherosclerotic plaque progression, in which a significant stenosis causes an imbalance between downstream blood-flow or oxygen supply and oxygen myocardial demand. Depending on the literature, a significant (possible flow-limiting) stenosis is considered equal to or greater than 50% luminal (diameter) narrowing and severe (probable flow-limiting) stenosis to be equal to or greater than 70% luminal (diameter) narrowing.[5] However, it is known from invasive fractional flow reserve measurements in combination with outcome trials that the decreased oxygen supply (and the survival of the patient) is not only influenced by the stenotic degree of a lesion.[6] These pathophysiologic concepts for developing ACS are not exclusively and often clinically not discriminable.

Unstable Angina Pectoris, Non–ST-Elevation Myocardial Infarction, and ST-Elevation Myocardial Infarction

Acute onset of chest pain with a burning sensation, pressure, or tightness is the typical symptom of ACS. However, chest pain can have other differential diagnoses, such as gastroesophageal reflux disease or musculoskeletal problems. On the other hand, many symptoms, including dyspnea or diaphoresis, may be clinical manifestations of ACS that complicate the diagnosis based on just clinical presentation. Furthermore, ACS consists of 3 subdiagnoses: ST-elevation myocardial infarction (STEMI), non-STEMI (NSTEMI), and unstable angina pectoris (UAP).[7,8] Whereas STEMI and NSTEMI are defined by the presence of myocardial necrosis (manifested by troponin elevation), UAP is defined as chest pain due to ischemia without the presence of myocardial necrosis.[8,9]

Traditional Triage of Patients with Acute Chest Pain

The traditional diagnostic workup of patients with acute chest pain for presence of ACS includes patient's history, physical examination, 12-lead electrocardiogram (ECG), and initial measurement of cardiac biomarkers.[8] Patients with high likelihood for ACS can be directly referred for further invasive diagnosis and treatment. Extreme low-risk patients can be readily discharged for outpatient follow-up. Patients with low-to-moderate likelihood for ACS after the initial evaluation remain for further observation and examination, including serial cardiac biomarker and ECG testing over the next 24 hours. These are frequently followed by a stress test for risk stratification, if subsequent ECG and biomarkers tests are inconclusive.

However, this traditional work-up of patients with acute chest pain, particularly in the triage in the emergency department (ED) has encountered several issues. Each year, more than 7 million patients are admitted to the ED in the United States with acute chest pain as their chief complaint, making it one of the most frequent causes of ED visits.[10] However, only 2% to 8% are diagnosed with ACS, most have chest pain of noncardiac origin.[11] Furthermore, a single troponin measurement is not sufficient to safely rule-out ACS because plasma troponin values have a delay function between the coronary event and the observed elevation at which serial cardiac biomarker measurements are recommended. With the introduction of high-sensitivity troponin (hsTn) measurements, this issue has been partially overcome, but a significant decrease in specificity was observed because hsTn is also associated with structural heart disease.[12] These issues lead to increased test burden and prolonged stay in the ED or chest pain unit. Despite that, about 2% to 3% of all patients suffering from ACS within 72 hours of ED presentation are erroneously discharged, contributing to missed ACS as the number-one cause for ED malpractice costs in the United States.[13,14] Accordingly, a great need for novel and improved triage strategies exists and coronary computed tomography angiography (CCTA) has been suggested as a safe, fast, and cost-effective modality to overcome these challenges.

EVIDENCE FOR USING COMPUTED TOMOGRAPHY ANGIOGRAPHY FOR THE EVALUATION FOR ACUTE CORONARY SYNDROME

Recently, several observational and interventional trials have been conducted to prove the accuracy,