

address situations in which local resources do not permit more advanced diagnostic testing. Finally, there is limited agreement regarding the long-term medical management of patients with MINOCA.

The purpose of this statement is to provide a formal and updated definition for the broadly labelled term MINOCA (incorporating the definition of AMI from the newly released “Fourth Universal Definition of Myocardial Infarction”¹²) and to provide a clinically useful framework and algorithms pertaining to the diagnostic evaluation and management of these patients.

EPIDEMIOLOGY

Clinical studies have reported a prevalence of MINOCA of 5% to 6% of AMI cases,⁶ with a range between 5% and 15% depending on the population examined.^{3,6,14–16} Although MINOCA can present with or without ST-segment elevation on the ECG, patients with MINOCA are less likely to have electrocardiographic ST-segment deviations and have smaller degrees of troponin elevation than their AMI counterparts with obstructive CAD (AMI-CAD).^{14,15}

The demographic and clinical characteristics of MINOCA patients differ from other patients with AMI. MINOCA patients are usually younger^{6,14–16} than patients with AMI-CAD. In a large systematic review, the average age of patients with MINOCA was 58 years, compared with 61 years among those with AMI-CAD.⁶ Women are disproportionately represented among individuals with MINOCA^{3,6,14,15}; they make up close to 50% of the MINOCA population but only 25% of the population with AMI-CAD.⁶ Women presenting with AMI are more than twice as likely as men to have MINOCA, whereas men presenting with AMI are more likely than women to have AMI-CAD.^{3,6,14,15,17,18} MINOCA is also more likely to occur in patients of black, Maori, or Pacific race and Hispanic ethnicity.^{3,14,16}

The prevalence of traditional CAD risk factors and clinical features also varies among patients with MINOCA versus AMI-CAD. MINOCA patients have a lower prevalence of dyslipidemia than their counterparts with AMI-CAD.^{6,14,15,18} Other traditional CAD risk factors, such as hypertension, diabetes mellitus, tobacco abuse, and a family history of myocardial infarction, are less frequent in MINOCA patients,^{14,15,18} although this has not been consistently observed in all studies.⁶

DEFINITIONS

Key Issues in Defining MINOCA

The rationale for defining MINOCA as a distinct entity is based on key clinical observations and premises, including the following: (1) Patients with MINOCA generally have a better prognosis than patients with AMI-CAD;⁶

(2) multiple atherosclerotic and nonatherosclerotic causes with heterogeneous pathophysiological mechanisms can cause MINOCA^{12,16}; and (3) unlike AMI-CAD, there is a paucity of dedicated studies examining MINOCA and therefore a lack of evidence-based therapies in these patients.¹² Given the aforementioned suppositions, standardization of the definition of MINOCA is clinically pragmatic, has operational utility, and serves a key purpose in promoting clinical awareness and research into the condition.

The European Society of Cardiology¹² developed the first international position article on MINOCA and proposed the following MINOCA criteria: (1) AMI criteria as defined by the “Third Universal Definition of Myocardial Infarction”¹⁹; (2) nonobstructive coronary arteries as per angiographic guidelines,²⁰ with no lesions $\geq 50\%$ in a major epicardial vessel; and (3) no other clinically overt specific cause that can serve an alternative cause for the acute presentation. Fundamental to the definition of MINOCA is the diagnosis of AMI with an elevated cardiac biomarker, typically a cardiac troponin >99 th percentile of the upper reference level with a rise or fall in the level on serial assessment. Although elevated troponin levels are indicative of myocyte injury with release of this intracellular protein into the systemic circulation, the process is not disease specific and can result from either ischemic or nonischemic mechanisms. Given this limitation of the troponin bioassay, the “Fourth Universal Definition of Myocardial Infarction” (by the Joint European Society of Cardiology/American College of Cardiology/American Heart Association/World Heart Federation Task Force for the Universal Definition of Myocardial Infarction) recently redefined the concept of myocardial injury.¹² Similar to myocardial infarction, the hallmark of myocardial injury is an elevated troponin beyond the 99th percentile of the upper reference level. However, these entities differ conceptually, because myocardial injury is attributable to nonischemic mechanisms of myocyte injury (eg, myocarditis), whereas myocardial infarction arises from ischemic mechanisms (eg, plaque disruption or supply-demand mismatch). The clinical diagnostic challenge is to delineate these entities, because patients with myocardial injury can present with symptoms that mimic myocardial infarction at the initial presentation.

With this revised concept of AMI, the term MINOCA should be reserved for patients in whom there is an ischemic basis for their clinical presentation. Thus, in the evaluation of patients with a suspected AMI (based on cardiac biomarkers and corroborative clinical evidence), despite the absence of obstructive CAD, it is imperative to exclude (1) clinically overt causes for the elevated troponin (eg, sepsis, pulmonary embolism), (2) clinically overlooked obstructive disease (eg, complete occlusion of a small coronary artery subsegment resulting from plaque

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George A. Pankey



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