Comment on “Clinical characteristics of spontaneous coronary artery dissection in young female patients with acute myocardial infarction in Korea”

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I immediately read with great attention and interest the significant and well-presented article recently published in an issue of this journal by Kim et al. [1] entitled “Clinical characteristics of spontaneous coronary artery dissection in young female patients with acute myocardial infarction in Korea.” They evaluated the prevalence, characteristics, and clinical outcomes of spontaneous coronary artery dissection (SCAD) in young female patients with acute myocardial infarction in Korea.

SCAD is increasingly recognized as an important cause of acute myocardial infarction (AMI) in young female patients, accounting for almost 35% of acute coronary syndrome (ACS) in women aged 50 years old or less [2,3]. The underlying pathophysiological mechanisms of SCAD are not well understood. Conventional risk factors for coronary artery disease, such as smoking and hypertension, only have a weak association with SCAD. Female sex hormones are thought to play a role in SCAD in young female patients with AMI [4,5]. The correlation of female sex hormones with SCAD in female is not fully understood. However, the changes of hormones may lead to alterations in the architecture of the coronary arterial wall. Degeneration of collagen induced by female sex hormones, fragmentation of reticulin fibres along with decreased mucopolysaccharide content of the arterial media, which weaken the artery wall, and haemodynamic changes are thought to play a crucial role in young female patients with SCAD [4,5]. Some hypotheses suggest that the female sex hormones receptors present in the coronary arteries may mediate these changes [5].

Although various potential risk factors for SCAD have been evaluated in the study of Kim et al. [1], the influence of hormone has not. However, clinical data have demonstrated that patients under either oral contraceptive use or hormone replacement therapy have an increased risk for developing SCAD. Though hormone is now recognized to be associated with SCAD, it is still an uncommon occurrence, and this may have led to the delay in both presentation and diagnosis. ACS in an otherwise healthy woman without traditional risk factors for coronary heart disease should raise the possibility of a SCAD especially if the patient is using oral contraceptives or having hormone replacement therapy.

If the prior medical history of oral contraceptive use or hormone replacement therapy has been evaluated in the study presented by Kim et al. [1], it
would be of utmost interest to consider this factor in the study to evaluate its potential contribution to SCAD. Such knowledge would not only reinforce the clinical relevance of hormone effects on SCAD but also enable preventive recommendations.

Conflict of interest
No potential conflict of interest relevant to this article was reported.

REFERENCES


