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ORIGINAL ARTICLE

Extent of coronary artery disease in patients undergoing angiography for stable or acute coronary syndromes

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Abstract *Background:* We aimed to investigate whether the angiographic extent of coronary artery disease (CAD) differs in patients undergoing coronary angiography for stable CAD or acute coronary syndrome (ACS) and identify predictors of CAD extent in these patients.
Methods: We enrolled 584 consecutive patients (463 with stable CAD, 121 with ACS) with angiographically established CAD ($\geq$1 stenosis >25%). The Gensini score was used to assess the extent of coronary atherosclerosis.
Results: Stable CAD patients had greater Framingham risk score and greater prevalence of hypertension, hypercholesterolemia, and diabetes ($p<0.05$ for all). Fasting glucose and systolic and diastolic blood pressure were higher, while high-sensitivity C-reactive protein (hsCRP) levels were lower in patients with stable CAD than in those with ACS ($p<0.05$ for all). No difference in Gensini score was observed between the two groups ($p=0.118$), but patients with ACS were more likely to have at least one significant epicardial angiographic lesion (>50% stenosis) (OR 2.0, $p=0.022$). Higher Gensini score was independently associated with (i) higher

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hsCRP and glucose levels, hypercholesterolemia, and increased age in stable CAD patients (R^2 0.15, $p<0001$) and (ii) increased age and higher glucose and hsCRP levels in patients with ACS (R^2 0.17, $p<0001$).

Conclusions: Patients undergoing coronary angiography for ACS or stable CAD presented with a similar extent of angiographic CAD, although patients with ACS had a higher prevalence of significant lesions in the presence of a better cardiovascular risk profile and higher inflammation levels. The extent of angiographic CAD in both the groups shared common determinants such as hsCRP, age, and hyperglycemia, but these appeared to explain only a small part of the variation of coronary atherosclerosis.

1. Introduction

Coronary artery disease (CAD), as a result of the atherosclerotic process in coronary arteries, is the leading cause of mortality and morbidity worldwide.[1] Coronary atherosclerosis may be detected early in life, but it usually progresses from subclinical asymptomatic to clinical overt disease over several decades. The progressive obstruction of coronary arteries and the subsequent severe restriction of blood flow to the myocardium lead to the appearance of chronic ischemia and symptoms of stable CAD. Rupture of a vulnerable atherosclerotic plaque along with the superimposition of thrombus is the major pathophysiological mechanism leading to acute coronary syndromes (ACS; unstable angina and acute myocardial infarction).[2,3]

Differences in the severity and extent of coronary atherosclerosis between patients with stable and unstable CAD have been little investigated previously, with controversial results. Early studies had implicated mild to moderately severe coronary atherosclerotic plaques in the occurrence of ACS,[4–7] while later studies have shown that unstable CAD may occur more often at significant coronary lesions.[8–10] Furthermore, it has been reported that a greater extent of coronary atherosclerosis may be associated with the occurrence of unstable coronary syndromes compared to stable CAD.[11] Whether the differences in the severity and extent of atherosclerosis may be attributed to differences in cardiovascular risk factors observed in patients with stable vs. unstable CAD has not been previously studied. Moreover, it has not been clarified whether patients with stable and unstable CAD share the same determinants of coronary atherosclerosis burden.

The aims of this study were to investigate whether the angiographic extent of CAD is different in patients undergoing coronary angiography for stable CAD or ACS and identify predictors of the extent of CAD in these patients.

2. Methods

2.1. Study Population

In the present study, we prospectively enrolled 1,107 consecutive patients who underwent a diagnostic coronary angiography for ACS or elective coronary angiography for stable angina or suspected stable CAD and consented to participate in the study. Patients with a history of severe valvular disease, congenital heart disease, and cardiomyopathy and those on hemodialysis were excluded. Patients eligible for this study (n = 584) were those with angiographic confirmation of at least one coronary atherosclerotic lesion with >25% luminal stenosis (as needed for the calculation of Gensini score) without a previously established history of CAD. Patients with other evident causes of coronary pain such as significant myocardial bridging or diffuse coronary spasm during angiography were excluded. Myocardial infarction was defined according to standard criteria on the basis of clinical symptoms, electrocardiographic findings, and raised troponin.[12] Unstable angina was diagnosed using Braunwald's criteria,[13] i.e., the presence of new onset or accelerated effort angina or chest pain at rest within the preceding 48 h or within the past month with transient ST-T segment depression and/or T-wave inversion but without elevated troponin (<0.5 ng/ml). Patients with suspected stable CAD were referred for diagnostic angiography because of either stable angina (typical ischemic chest pain that did not change its pattern in the preceding 2 months) or suspicious atypical symptoms (most frequently chest pain or dyspnea) along with a positive stress test (abnormal nuclear imaging, stress echo, or exercise stress test).

In all subjects, a full medical history was acquired and a complete physical examination was performed. Blood samples were drawn early in the morning after an overnight fast and just before coronary angiography for stable coronary syndromes. In patients with unstable coronary syndromes, blood samples for the determination of lipid and fasting glucose levels were drawn either before coronary angiography or the next morning, depending on the presence of fasting state. The study protocol was approved by the local Ethics Committee. The study complied with the Declaration of Helsinki, and all participants provided written informed consent.

2.2. Cardiovascular risk factor evaluation

Weight and height were measured, and body mass index was calculated. Hypertension was defined as systolic blood pressure (SBP) >140 mmHg and/or diastolic blood pressure (DBP) >90 mmHg or administration of antihypertensive medications. Hypercholesterolemia was defined as low-density lipoprotein cholesterol >115 mg/dl or administration of anti-cholesterolemic medications (statins or

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