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Table 1 Clinical Characteristics and Pharmacologic Treatments According to ABI Ranging From 0.91 to 1.39 and ≥1.40

	ABI 0.91–1.39 (n = 1,381)	p Value	ABI ≥1.40 (n = 204)
Age 65–74 yrs	347 (25)	NS	41 (20)
Age ≥75 yrs	603 (44)		94 (46)
Female	642 (47)	<0.002	71 (35)
Paroxysmal NVAF	600 (44)	NS	87 (42)
Persistent NVAF	196 (14)		26 (13)
Permanent NVAF	585 (42)		91 (45)
AH	1,115 (81)	<0.04	177 (87)
Diabetes	265 (19)	<0.03	53 (26)
Smoking habit	194 (14)	NS	19 (9)
Previous TIA/stroke	136 (10)	NS	21 (10)
Vascular disease	215 (16)	NS	26 (13)
CHA_2DS_2-VASc*			
0	66 (5)	NS	7 (4)
1	181 (13)		31 (15)
≥ 2	1,134 (82)		166 (81)
Antithrombotic		NS	
None	217 (16)		30 (15)
OAC	836 (60)		138 (67)
APs	270 (20)		28 (14)
OAC + APs	58 (4)		8 (4)
Statins	496 (36)	NS	62 (31)
Oral hypoglycemic agents	166 (12)	NS	35 (17)

Values are n (%). *Vascular disease includes previous myocardial infarction, peripheral arterial disease, or aortic plaque.

ABI = ankle-brachial index; AH = arterial hypertension; APs = antiplatelet drugs; NVAF = nonvalvular atrial fibrillation; OAC = oral anticoagulants; TIA = transient ischemic attack; CHA_2DS_2-VASc = congestive heart failure [or left ventricular systolic dysfunction]; hypertension [blood pressure consistently >140/90 mm Hg or on hypertension medication]; age ≥75 years; diabetes mellitus; previous stroke, transient ischemic attack, or thromboembolism; vascular disease [e.g., peripheral artery disease, myocardial infarction, aortic plaque]; age 65 to 74 years; sex category [i.e., female]).

fibrillation patients with clear delineation of each step. In addition, the follow-up study required a repeat measurement of ABI every 12 months.

We agree with Dr. Aboyans and colleagues that our findings are particularly relevant in patients classified as CHA_2DS_2-VASc score 0 and 1 as the inclusion of low ABI may upgrade the risk and eventually change the therapeutic approach in patients considered at low-to-moderate risk. Inclusion of ABI ≤0.90 in the CHA_2DS_2-VASc might have important therapeutic implications only if incorporation of ABI into the CHA_2DS_2-VASc score is prospectively tested. Thus, the results of our ongoing prospective study will validate this and clarify its incremental value.

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New Insights on Plaque Erosion and Calcified Nodules

"Seeing Is Believing"

Plaque erosion (PE) and calcified nodules (CN) have been classically described in pathological studies as causes of acute coronary syndromes (ACS) (1). However, until very recently, the diagnosis of these entities in the clinical setting has remained largely elusive (1). In this regard, the study of Jia et al. (2), using optical coherence tomography (OCT) for the diagnosis of PE and CN, is of major clinical interest and raises several important issues. First, the investigators considered that OCT might provide a "definitive" diagnosis of PE when fibrous cap disruption is excluded and a thrombus overlying an "intact" plaque is visualized. However, considering that OCT lacks the resolution required to visualize mild superficial endothelial erosions and that coronary thrombi may displace along the vessel (either spontaneously or following instrumentation) and actually originate from a remote source (3), we believe that these OCT findings should be considered as diagnostic of "probable" PE. Likewise, lumen surface irregularities without associated thrombus and plaque attenuation by red thrombus (2) are probably better classified as "possible" PE. In our experience, in some patients, a repeated OCT study after several days of intense antithrombotic therapy may unravel the true characteristics of the underlying plaque, once the overlying thrombus has disappeared or drastically reduced in size (3). Indeed, in some of these patients, a previously hidden small plaque rupture—rather than an intact plaque—may be eventually visualized. As this strategy may help to better identify patients with PE, it will be of interest to know if similar "evolving" findings were found in some patients in this study. Second, until now, the value of OCT to detect CN has not been established. Nevertheless, anecdotal patients presenting with large "superficial" calcified plates and associated intracoronary thrombosis have been recently reported (4,5). However, we believe that visualization of a "rupture" should not be required for the diagnosis of CN (2), although this finding might be critical in the diagnosis of complicated CN. Notably, previous studies using virtual histology have demonstrated that silent, uncomplicated, protruding CN may be detected in nonculprit vessels of ACS patients and also that the prognosis of these plaques is rather benign (6). Furthermore,

according to classical OCT diagnostic criteria, the visualization of intense dorsal shadowing must be interpreted as the presence of red thrombus. However, we strongly believe that, in some patients, "bony" CN may also generate intense posterior shadowing (5). This pattern might mimic concomitant intracoronary thrombosis. Therefore, it would be of major interest to know whether in the study of Jia et al. (2) some images of dorsal shadowing were considered likely a result of "bony" CN rather than associated luminal red thrombi. Finally, we fully agree that in ACS patients, OCT is of major value to rule out other rare alternative diagnoses to plaque rupture, such as spontaneous coronary artery dissection (7).

We should be most grateful to these investigators for their unique and systematic diagnostic effort (2). The novel findings from this large OCT registry clearly advance our knowledge by unraveling the elusive multiple faces of PE and CN in patients with ACS.

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