Requiem for the ‘vulnerable plaque’

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myocardial infarction (STEMI), the survivors may develop more
heart failure in the long term.

We contend that we currently find ourselves amidst a shift in dis-
ease manifestations of atherosclerosis due to altered demographics,
attendant changes in risk factor profiles, efforts to control tobacco
abuse, and the increased prevalence of statin treatment. Myocardial
infarction has ‘gone global’, not only affecting predominantly middle-
aged Caucasian males in higher socioeconomic strata, and no longer
associates mainly with cigarette smoking, hypertension, and high
low-density lipoprotein (LDL). Women, non-Caucasians, younger
individuals, and those with obesity, insulin resistance or frank dia-
betes, and high triglycerides and low high-density lipoprotein
(HDL) represent an increasing proportion of our patients with ACS.

We also now find ourselves the midst of a transition in the pres-
entation of ACS, with STEMI on the wane and non-ST segment

Table 1  Challenges to the ‘vulnerable plaque’ concept

- Thin-capped, lipid-rich atheromata are not solitary, rather often
  multiple, and affect several arterial beds in the same individual.
- Thin-capped, lipid-rich atheromata most often persist for years
  without causing a clinical event.
- The risk profile and demographics of acute coronary syndrome
  patients are shifting worldwide (global burden, younger patients,
  more women, more insulin resistance/diabetes, more
  hypertriglyceridaemia, and less low-density lipoprotein excess).
- Statin treatment and other preventive measures have begun to
  modify atherosclerotic disease.
- ST segment elevation myocardial infarction wanes as non-ST
  segment elevation myocardial infarction waxes.
- Plaque rupture declines as a cause of acute coronary syndromes
  while superficial erosion appears on the rise.
- Plaques underlying cerebrovascular events reveal more stable
  fibrous characteristics compared with that 10 years ago.

Plaque rupture declines as a cause of acute coronary syndromes
while superficial erosion appears on the rise.

Figure 1  Contrasts between superficial erosion and fibrous cap rupture as causes of arterial thrombosis. LDL, low-density lipoprotein.