ESC calls for research into vulnerable plaques

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The European Society of Cardiology (ESC) Working Group of Atherosclerosis and Vascular Biology has published a position paper to raise the profile of vulnerable plaques and the need for greater use of therapies to promote plaque stabilisation. The position paper, published online today in *Thrombosis and Haemostasis*, is also calling for more research into the causes of plaque rupture, and for the development of better diagnostics and treatments.

"We want more medical professionals to understand the concept that stabilising vulnerable plaques offers a fundamental approach to preventing cardiovascular events," said Seppo Ylä-Herttuala, chairman of the position paper task force.
Indeed, he added, several statin trials for secondary prevention have reported a reduction in cardiovascular events, and furthermore anti platelet therapies have been shown to have a beneficial effect.

"Introducing stabilisation of vulnerable plaques as part of secondary prevention would offer the opportunity to wipe out half of coronary events," said Ylä-Herttuala, from University of Eastern Finland (Kuopio, Finland).

"Wide spread stabilization of vulnerable plaques would also have important socio economic implications dramatically reducing the need for invasive treatments," said Christian Weber, also a member of the working group.

The idea of vulnerable plaques is that not all plaques (the fatty deposits in arterial walls) are equal and that some are particularly prone to rupture and causing cardiovascular events . These plaques are not necessarily the same as those that cause symptoms such as angina. Explaining the concept of vulnerable plaques, Weber, from Ludwig-Maximilians-University (Munich, Germany) said that it is thought that inflammatory cells resulting from ongoing inflammation destabilise the structure of the plaque. "It is believed that they degrade the fibres that make the plaque stable, leading to a greater potential for the plaque to rupture," he said.

The concept of plaque stabilisation was introduced to explain how acute coronary events could be reduced by lipid lowering therapy without accompanying regression of coronary atherosclerosis seen on angiography.

Part of the motivation for producing the working paper, said Ylä-Herttuala, was to provide general clinicians with greater guidance. "The whole field can be really confusing. After patients have been treated with statins for two or three years family doctors can be really concerned that they see no changes on angiograms. In such cases there's a danger that they may decide to stop life saving treatment."

The position paper reviewed the current state of knowledge around unstable plaques exploring the role of inflammation, chemokines, growth factors, platelets, angiogenesis and smoking. Evidence for therapies such as statins, antiplatelet therapies, and antihypertensive treatments were outlined, in addition to reviewing new approaches ,such as the development of drugs targeting the fibrous cap. Detection of unstable plaques through genetic testing, biomarkers and imaging was also explored.

"The single most important advance that would help us to tackle vulnerable plaques would be to have a non invasive imaging tool that

would allow us to identify at risk patients before they suffer an event," said Ylä-Herttuala.

The position paper is also calling for more translational research into imaging, biomarkers and the development of new treatments. "There is a real need to develop treatments specifically for the purpose of stabilising vulnerable plaques. At the moment we only have treatments that were discovered to have a beneficial effect through serendipity," said Weber.

Provided by European Society of Cardiology