

Unstable Plaque: The Killer in Your Arteries – Kerry Bone

Arterial disease, leading to heart attacks and strokes, is a significant cause of death and disability in our communities. This is despite all the research advances and the billions of dollars spent by drug companies and universities. It is certainly an issue where prevention is better than cure. The famous Framingham Heart Study was the first to clearly define the risk factors for arterial disease, factors that could be targeted to help prevent it. As a result, there is currently a big focus in conventional medicine on drugs that lower cholesterol and blood pressure. But as we have read and seen, these issues are very complex and controversial.

Imagine the possibility of achieving a new clarity among this confusion by understanding the latest thinking about what usually triggers a heart attack and stroke. Consider that this becomes particularly powerful when combined with newly discovered properties of herbs and supplements, especially how they seem to work in harmony with the cell's basic metabolism, rather than interfering with it. What we will be covering in this article will give you a radical new framework for cardiovascular health.

You might be feeling this is a tall agenda, that the whole field is so confused with differing opinions and conflicting studies. Concerns over cholesterol are misplaced, or it's too hard to treat cardiovascular disease naturally. Or even that we are falling into the same trap as conventional medicine, with an ill-considered focus on risk factors, with the evidence never reaching anything conclusive.

Let's press on and examine new information about the extreme importance of the role of vulnerable plaque in hard cardiovascular events and what you can do about it. It's a radical new concept and a fertile field of discovery for natural medicine. Currently, it is the elephant in the room of cardiovascular risk assessment.

The elephant in the room

Our arteries are subject to a disease known as atherosclerosis, where a build-up of abnormal tissue (known as plaque) occurs in the blood vessel wall. Unstable or vulnerable plaque is an atherosclerotic plaque at a high short-term risk of rupture. When rupture does occur, it results in a massive aggregation of platelets (known as a thrombus) which can block the artery. Depending on where this blockage

occurs, a heart attack or stroke might follow. Here's the intriguing thing, only plaque with a very thin fibrous cap is at risk of rupture and even just a small area of such plaque is life threatening. These plaques are essentially unstable because of a deficiency of connective tissue. In a sense arterial plaque is a type of wound on the blood vessel wall, and vulnerable plaque can be seen as not being able to heal appropriately, somewhat like an ulcer or abscess. Even in the presence of widespread arterial disease, rarely more than a few plaques appear to be at risk of rupture at any given moment.¹

Plaque rupture was described as far back as 1844, but it was only in the late 1980s that its true relevance to heart attacks and strokes was recognised. ii In reality, we can now propose that there are in fact two sets of risk factors for heart attacks and strokes: one set of factors that predicts the risk for development of diseased arteries (arterial plaque), and a second set that predicts the risk for unstable plaque and it potential rupture. Surely, if someone already has arterial plaque, the best way to stop an acute event (heart attack or stroke) would be to focus on this second set of risk factors. The research is still ongoing, but the list of factors predicting likelihood of plaque rupture contains a few surprises, and also confirms some folk wisdom.

What causes plaque rupture?

One major review of the research found a strong link between acute coronary syndromes and acute respiratory infections. Both peak in winter, and acute infections could precede up to one third of heart attacks. Large and well-designed retrospective studies consistently find a 2- to 3-fold increase

in risk within 1-2 weeks after a respiratory infection, especially marked in the first few days (a 5-fold increase). Up to half of all deaths during influenza epidemics are attributable to cardiovascular causes. The above review described the detailed and complex mechanisms as to how an infection can trigger plaque rupture. So surprisingly, taking Echinacea root every day to prevent infections might actually lower your risk of a heart attack!

Other factors linked to plaque vulnerability and rupture include:

- Low circulating fibrocytes (maybe indicative of a poor healing capacity)^{iv}
- Elevated C-reactive protein (CRP) and other inflammatory markers^{v,vi}
- Lower serum eicosapentaenoic acid (EPA)/arachidonic acid (AA) ratio (not enough omega-3 fatty acids in your system)^{vii}
- Age^{viii}
- Lower plasma glutathione^{ix}
- Lower regulatory T cells and serum interleukin-10^x
- An elevated low-density lipoprotein to high-density lipoprotein cholesterol ratio^{xi}

Many of these point to increased inflammation, a key factor behind unstable plaque.

Cardiovascular events are triggered by stress

Remember the wife who pleaded with her husband not to get so stressed and angry, otherwise he might have a heart attack? She was right! Stress, by increasing the risk of plaque rupture, does indeed cause a heart attack. For example, anger and hostility were linked to increased incidence of heart attacks in previously healthy people, especially men. They are also are linked to poorer outcomes in people with existing heart disease. XII Another recent review found that a variety of mental stressors (anxiety, anger, bereavement, frustration, work stress, sexual activity), physical exertion, community-wide stresses (Monday, mornings, winter, blizzards, Christmas, heat waves, sports events, earthquakes, missile attacks, natural disasters) and other miscellaneous stresses (lack of sleep, over-eating, air pollution, infection) were all linked to an increased risk of a heart attack.xiii

Preventing plaque rupture

As indicated above, being more relaxed and less stressed will make a big difference in preventing plaque rupture. But there are also herbal measures that can help. Preventing infections as much as possible with immune herbs such as Echinacea root has already been mentioned. We can also speculate that herbs able to improve the stress response (adaptogens) such as Rhodiola, Withania and Eleutherococcus will be beneficial, together with regular use of calming herbs like valerian and kava.

Stabilising vulnerable plaque

If vulnerable plaque is a wound that has not healed properly, using herbs to promote

healing should help to stabilise it. By an amazing coincidence, we actually have clinical evidence that a key healing herb does exactly that (help plaque to heal). In two remarkable placebo-controlled clinical trials, gotu kola actives stabilised low-density carotidxiv and femoral plagues.^{xv} The dose used in both trials was 180 mg/day of gotu kola triterpenoids for 12 months. This clinical outcome was assessed by significant marked increases in echogenicity of the plaque compared to placebo. Low density plaque is not the same as unstable plaque (in fact we have no reliable way yet to detect unstable plaque), but these clinical trials do show that gotu kola can enhance the laying down of connective tissue in plaque, which is exactly what is needed.

As noted above, unstable plaque has been linked to low levels of omega-3, so eating plenty of fish and supplementing with oils rich in long-chain omega-3 fatty acids (EPA and DHA) is essential. Algal and fungal sources of EPA and DHA are also available.

Inflammation makes plaque more unstable, In particular, CRP and other inflammatory markers such as matrix metalloproteinase 9 (MMP-9) and interleukin-6 (IL-6) have been linked to vulnerable plaque. It is thought the ongoing inflammatory processes inside vulnerable plaque are key factors in its instability. A fascinating study assessing the clinical impact of Ginkgo on a number of novel cardiovascular risk factors, especially nanoplaque formation and lipoprotein(a) was published a few years ago. Now the authors have revisited blood samples taken from the trial participants and subjected them to further analysis. xvi As well as nanoplaque formation being reduced by 14.3% and nanoplaque size by 23.4%, oxidised LDL was reduced by 21%, lipoprotein(a) by 26.3%, interleukin-6 by 12.9%, MMP-9 by 32.9%,

white blood cell (WBC) count by 7.5% (mainly from monocytes and eosinophils) and highly-sensitive CRP (hs-CRP) by 39.3%.

CRP is usually measured to less than 5 mg/L. When it was realised that values less than this might also contribute to cardiovascular risk, a more sensitive technique was developed to measure these low numbers (hs-CRP). Interestingly, the Ginkgo reduced CRP regardless of the starting value. Hence, one patient with relatively low value of the 1.60 exhibited a reduction to 0.64, while in another a reading of 46.6 (indicating a high degree of inflammation) dropped to 29.3. The latter observation suggests Ginkgo might be a significant anti-inflammatory treatment in its own right. WBC count is emerging as a significant risk factor in patients with preexisting arterial disease. In particular, eosinophil activation has been linked to major adverse events in several clinical trials, especially in patients with stents. XVII Other anti-inflammatory herbs will also be highly valuable, particularly turmeric (especially as bioavailable forms of curcumin).

Unstable plaque has also been linked to lower plasma glutathione. All the Nrf2/ARE priming herbs such as turmeric, rosemary, green tea and Ginkgo will help boost plasma glutathione levels.

Summary and conclusions

If someone already has arterial plaque (and most of us do), there is probably only a modest benefit in reducing the formation of new plaque by addressing that first set of risk factors, the ones predicting the risk for plaque development. Stable plaque narrows arteries, and may eventually cause stable angina or

intermittent claudication, but other than that it has little adverse health consequences. Instead, a more risk-based approach will focus on unstable plaque, since in the majority of cases it is responsible for serious lifethreatening events (heart attacks and strokes).

In addressing the risk posed by unstable plaque, a two-pronged strategy is suggested: firstly stabilise the plaque, and secondly avoid those things known to trigger plaque rupture. As a consequence, a number of lifestyle, herbal and dietary measures are recommended such as avoiding stress (multiple herbs will help here) and taking Echinacea root (reducing acute infection risk), Ginkgo (reducing inflammation and increasing plasma glutathione), turmeric and bioavailable curcumin (reducing inflammation, priming Nrf2/ARE), gotu kola (healing plaque) and fish oil. If you need to lower your patients' cholesterol, then fine, do it. But ignoring the elephant of unstable plaque means you will be missing out on a huge extra benefit, one that might save their lives.

References

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