Commentary

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Ischemia and low-back pain—is it time to include lumbar angina as a cardiovascular disease?

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Ischemia and low-back pain—is it time to include lumbar angina as a cardiovascular disease?

by Henrik Bøggild, PhD[1]

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Angina pectoris, abdominal or intestinal angina, and claudicatio intermittens are all well-known clinical pain syndromes related to ischemia as a result of regional atherosclerotic arteries. A decade ago Kauppila (1) hypothesized that low-back pain could be due to atherosclerosis of the small lumbar arterial vessels. The hypothesis was later supported by studies using magnetic resonance (MR) (2), which has shown occlusion in these arteries more often in patients with low-back pain than in controls, and, in postmortem studies, occluded or narrowed arteries were more often found in patients with low-back pain (3). Most of the studies have been cross-sectional or have at least collected information on low-back pain in retrospect, with the risk of information bias.

In this issue of the *Scandinavian Journal of Work Environment & Health*, Leino-Arjas and her co-workers report a cohort study (4) in which risk factors for cardiovascular disease (CVD) were related to the development of low-back pain 28 years later among industrial employees. The paper finds that, especially for the men, smoking, high body mass index (BMI), high cholesterol, high triglycerides, and high blood pressure, alone and in combination, predict low-back pain and thus support the atherosclerotic origin of low-back pain.

Is it then time to expand the list of CVD pain syndromes, currently consisting of angina pectoris, abdominal angina, and claudicatio intermittens to include also *lumbar angina*, pain in the area nourished by lumbar and sacral arteries?

The implications would be large. If low-back pain turns out to be a disease of the arteries, the inference for diagnostics, treatment, and prevention would be huge. Suddenly, the diagnosis of low-back pain should no longer be based on self-reported information alone, but could be distinguishable in arteriographia, scanning or other tests; in the future the management of low-back pain might include known anti-angina medicines like nitrates in addition to physical training; and back pain prevention would have to expand its focus also to include risk factors for atherosclerosis.

Taking the role of the Devil's advocate, I propose that a couple of issues should be taken into account before the hypothesis is assumed to be proved by this study. Although the cohort was followed for 28 years, and thus has a time perspective that is relevant in relation to the development of CVD, the cohort was—as often is the case—originally designed to address other issues, and we must carefully explore what it is that is used as proxy measures for CVD and low-back pain in this study.

Information on low-back pain was not clinically ascertained, but was gathered by summarizing information from five items on different symptoms and their frequency in relation to the lumbar area of the back and pain radiating to the left and right legs. In a comparison with the baseline information, an increase in the low-back pain score for a group of participants with more severe pain was identified. We do not know how the score compares with the results of a clinical examination, and we do not know whether changes in the score on this index 28 years apart are measuring anything relevant.

The exposures are all well-known risk factors for CVD. The authors use the risk factors both by themselves and in summing up values for trichotomized BMI, cholesterol, triglycerides, systolic blood pressure, diastolic blood pressure, smoking, and physical activity into an index, and the results are then again trichotomized.

But smoking and physical inactivity have repeatedly been shown to be associated with BMI, blood pressure, and lipids. Probably due to their place in different levels of a causal chain, smoking and physical inactivity causally lead to higher BMI, blood pressure, and

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lipids, and BMI causally leads to higher blood pressure. Summing up interdependent risk factors into one score or examining results in a logistic regression model without allowing for these factors leads to a risk of spurious results.

BMI has long been recognized also as an independent risk factor for low-back pain, and it has been postulated that this relationship is due to biomechanical changes in body posture due to a higher weight on the lumbar spine and its muscles. In clinical experience, weight loss often leads to a reduction in back pain. Likewise, a lack of physical activity is related to both the risk of having low-back pain and to a reduction in pain when exercise is intensified. Both weight loss and exercise rapidly lead to a reduction in low-back pain—more rapidly than should be expected if the pain were due to atherosclerosis.

A detailed examination of the Leino-Arjas et al's results (tables 2 and 3) (4) suggests that current smoking, triglycerides, and blood pressure are more predictive of radiating pain to the lower extremities than to local low-back pain. In this context, it could be possible, that, in some persons, *claudicatio intermittens* mimicks the radiating pain of low-back pain and, therefore, explains the association with CVD risk factors.

It is not stated whether the "increase in the low-back pain score" was measured from the total score or whether it allowed for comparing the regional pain and the radiating pain separately. It is also not clear whether the association between CVD risk factors 28 years ago and a successive rise in low-back pain was due to changes in the lumbar part or the radiating part of the index.

As both exposure and, especially, outcome were measured by composite scores, the aforementioned problems should lead to a very careful and preferably theory-driven examination of the associations.

The jury is still out. The verdict as to whether we should regard low-back pain as a clinical entity of CVD cannot be given on the evidence presented.

But before using all of the other population-based studies that have information on both CVD risk factors and questions relating to low-back pain for examining whether these studies give the same results, I would suggest that we use clinical, as well as epidemiologic and statistical, skill to focus study objectives and methods so that results are less prone to interpretation.

Information on both exposure and outcome should be examined carefully in order to ensure that it would be possible to separate the known biomechanical risk factors from those related to arterial function (eg, lipids, endothelial markers) and to measure information on low-back pain that would be both clinically relevant and measured changes over a long period of time. The statistical road would include careful modeling, including, for example, the sampling units, multilevel regression modeling, or merely taking advantage of the old tool of stratification to control for BMI and exercise.

In the end, examining whether well-known CVD-related interventions like blood pressure reduction or a reduction in cholesterol and triglycerides using diet or medication as fibrates leads to less back pain could settle the question more effectively than another "me too" study.

References

1. Kauppila LI. Can low-back pain be due to lumbar-artery disease? Lancet 1995;346: 888–9.
2. Kauppila LI, Mikkonen R, Mankinen P, Pelto-Vasenius K, Maenpaa I. MR aortography and serum cholesterol levels in patients with long-term nonspecific lower back pain. Spine 2004;29:2147–52.
3. Kauppila LI. Prevalence of stenotic changes in arteries supplying the lumbar spine: a post-mortem angiographic study on 140 subjects. Ann Rheum Dis 1997;56:591–5.
4. Leino-Arjas P, Solovieva S, Kirjonen J, Reunanen A, Riihimäki H. Cardiovascular risk factors and low back pain in a long-term follow-up of industrial employees. Scand J Work Environ Health. 2006;32(1):12–9.