The small plaques

Nowadays, the conventional view assumes that the majority of heart attacks originate from moderate, apparently harmless „non-critical“ alterations in the vessels. It is thought that about ¾ of all heart attacks occur due to acute blockage of the coronary arteries as a result of torn “soft, unstable plaques”. In medical language, “plaques” are arteriosclerotic lumps in the vessel walls. When these plaques have a soft, fatty core containing cholesterol they have a tendency to tear and cause a heart attack due to thrombosis. This is why they are called “soft, unstable plaques”.

Assuming this, most people in the industrialized world should therefore be at constant risk of having a heart attack. The statistics quoted in the chapter “Doubt” strikingly show the high frequency of severe arteriosclerosis in industrial countries. If moderate alterations in the wall of the vessels are now also a risk, even a great danger, then we are all more or less affected. It is perhaps not a coincidence that this idea established itself when “cholesterol inhibitors” (“statins”) came onto the market. If this assumption is correct, then it is only natural that such medication should be put to extensive use.

Such soft unstable plaques are only a danger when they are still small and have not yet caused severe stenoses in the coronary arteries. Severe stenoses are safeguarded by collaterals. Moderate stenoses that do not yet obstruct the blood flow do not induce the development of collaterals. **An acute blockage that develops within a few hours from an apparently small stenosis leads to a heart attack; there can be no doubt about this.**

Nowadays, it is assumed that almost ¾ of all heart attacks are caused by “soft plaques”. This idea is based on studies that compare the findings of a coronary angiogram performed after a heart attack with an angiogram carried out before the heart attack. The diameter of the stenosis blocked during the heart attack was compared with its diameter beforehand. It was repeatedly found that a large proportion, in some studies up to ¾ of all stenoses found in the “angio” before the attack, were only moderate in size (29,30,31). In all these studies, however, there was a relatively large time span between catheterizations, which ranged on average from many months to several years. Arteriosclerosis is recognized to be a chronic progressive process. One cannot, therefore, draw the decisive conclusion from any of these studies that a moderate “non-critical” stenosis was the origin of the acute blockage of the vessel. This originally moderate stenosis could have developed into a severe stenosis in the meantime.

How often does this actually occur? – This is the deciding issue.

This dilemma can only be solved by a pathologist. Pathologists’ statements on this subject are unambiguous: **autopsy findings almost always show high-grade “critical stenoses” to be the origin of thromboses (32).** Baroldi reports that 93% of all thromboses found in heart attacks develop from severe vessel stenoses (16).

About 20 years ago, there was an era during which emergeny treatment was given to dissolve a blood clot in the case of acute heart attack. When an angiogram was performed after this treatment it was possible to judge the degree of stenosis at the place the blood clot had

become attached. An extrapolation from various scientific publications resulted in a quota of 84% of cases in which a thrombosis originated from severe “critical” stenoses (16).

As a consequence, it can be assumed that in the majority of cases, acute blockage of a coronary artery originates from an existing stenosis, which is already sufficiently surrounded by collaterals, without appreciable consequences, as shown in the example of the highway. **There remain, however, at the most 16% of cases in which smaller stenoses are involved and “soft plaques“ can be assumed to have caused the heart attack. This number is greater than zero, but it is not ¾ of all heart attacks.** An occluding thrombosis originating in small soft plaques as a cause of heart attack is a rather rare event. Soft plaques and, in general, coronary vessel factors should be credited with a more minor role in the development of heart attacks.

Another important argument: Pieces of blood clots that originate from small soft plaques would repeatedly break away in the pulsating blood stream and drift to the periphery of the vessels, where they may cause an acute blockage and thus a heart attack. Such coagulations or embolies that originate as thromboses in the carotid artery and pass into the brain are an important cause of stroke. Heart attacks are never (!), however, caused by embolies that originate from the coronary arteries (16), except after interventions such as catheterization or bypass surgery.

All things considered, small “soft plaques” that are not yet protected by collaterals **are <u>one</u> of the causes of heart attacks.** In this respect, the coronary arteries can cause damage and are dangerous. **But they are rare events. Coronary vessel factors play a rather modest role in causing heart attacks.**