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The Similarities in Coronary Artery Disease and Cancer

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For decades, angina pectoris was thought to be due to a narrowing of a coronary artery producing decreased blood supply to an area of the heart. While it is true that a narrowed coronary artery may produce symptoms of angina, it is not true that this is the etiology of the symptoms perceived as the chest pain we call angina pectoris.

In reality, the symptom angina pectoris, was first scientifically proven and discussed at the 49th Annual American College of Cardiology (ACC) Conference in 2000 [1], and is the result of regional blood flow differences (RBFDs) between the different coronary artery vascular beds.

These differences are revealed, when sufficient demands are placed upon the arteries to increases their respective coronary blood flows, required to meet the metabolic requirements of the heart. This increased metabolic demand can be either the result of (A) naturally occurring increased cardiac work load (exercise/exertion) or (B) mimicked through pharmacologic manipulation triggering a subsequent regional change in coronary blood flow (e.g. inter alia dipyridamole, adenosine) resulting in vasodilation [2,3].

As scientifically proven, disease within the coronary arteries are first associated with a build up of inflammatory material within the walls of the arteries. This buildup in inflammatory material may or may not be associated with calcium. If calcium is present, coronary CT may be useful; however, the absence of calcium is no more exclusive of coronary artery disease (CAD) or RBFDs, than the absence of coronary lumen narrowing, which occurs later in the development of atherosclerotic plaque formation (Figure 1).

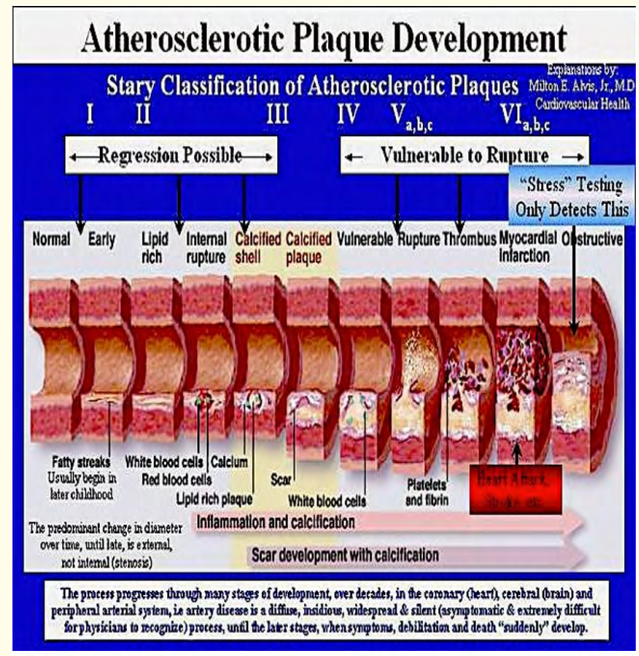

Figure 1: Sequential development of inflammatory coronary artery disease.

The introduction of inflammatory changes [1-4] within the walls of the arteries, is the true nidus for the development of regional blood flow differences (RBFDs), which may be expressed as coronary flow reserve (CFR), which ultimately will result in angina pectoris. While it is easy to understand why initial observations from more than half a century ago could lead to the misunderstanding that angina was the result of coronary artery lumen narrowing, we now know better [5].

RBFDs are not only the key to understanding, diagnosing and treating CAD, they are also the key to understanding, diagnosing and treating other diseases, such as cancer, hypertension, and diabetes.

To fully understand cancer, we need to comprehend that the development of cancer, is the result of transitional changes, which occur over time [6]. These changes in RBFDs are the result of changes in the metabolic activity of the cells in the region, including the native cells and the inflammatory cells recruited to the area in response to tissue changes already occurring.

These changes in RBFD and metabolism, can be directly measured using FMTVDM [7]. Like CAD, the enhancement of RBFDs using FMTVDM can be used to differentiate tissue changes on a transitional continuum, which can then be quantified to identify these changes. Just as FMTVDM can be used to find early transitional changes in CAD, long before coronary lumen narrowing has occurred, so too, is FMTVDM capable of measuring the transitional changes including the precancerous inflammatory process, which signals the beginning of tissue changes which may ultimately progress to and become what we have classically defined as cancer.

With our improved understanding and enhanced ability to understand, diagnose and treat CAD and cancer using FMTVDM, the similarities between CAD and cancer are now obvious. The changes that occur in response to tissue damage [6-8], which result in the development of CAD and cancer, are the direct result of measurable changes in metabolism and regional blood flow differences, that are a fundamental genetic response of our bodies to environmental insults.

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