Replacing Cardiovascular Risk Factors with True AI and Absolute Quantifiable Measurement (FMTVDM) of Coronary Artery Disease

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In a relatively recent editorial Wilkins, et al [1] discussed the importance of critiquing the myriad of tests Cardiologists have been using to guestimate when someone might have heart disease - using what some people are calling artificial intelligence (AI). The amalgamation of multiple yes-no tests – Is the LDL cholesterol elevated?, Does the patient smoke?, et cetera – does not produce true AI. This approach is merely an accumulation of tests with sensitivity and specificity errors, the assembly of which does not produce AI – merely an algorithm (pseudo-AI) retaining the flaws of the initial tests.

All too often Cardiologists talk about risk factors and markers of inflammation. The Inflammation and Heart Disease Theory* (Figure 1) introduced almost two-decades ago [2] was never intended to result in people merely focusing on the surrogate blood markers themselves - factors associated with potential coronary artery disease - but rather to raise awareness that individuals have CAD for a variety of reasons. These surrogate markers of disease are found to varying extents in different people making it impossible to determine if someone has CAD merely upon the basis of these blood tests [2, 3].

Figure 1. Coronary artery disease is an inflammatory process precipitated by more than a dozen variables. Each variable contributes to inflammation within the blood vessels of the body, including the coronary arteries to varying degrees in different individuals [2].
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The use of yes-no testing, has resulted in further problems with both sensitivity and specificity, further confusing the diagnosis and treatment of individuals - placing the focus of medicine on blood tests and not the actual patient or the actual measurement of CAD itself [3-5]. The use of pseudo-AI and semi-quantitative tests, which we have discussed in the literature [4,5] pose an even greater potential threat to medical accuracy, as these tests mistakenly present clinicians with the presumption of greater accuracy when in fact they are only correlations of disease – correlations which do not match actual clinical measurement of CAD as shown in Figure 2 [6]. Correlations as we all know, do not represent cause and effect but merely a relationship, which can be very misleading [7].

![Blood Flow Image Change vs Blood Chemistry Changes](image)

**Figure 2.** The X-axis displays the composite blood profile including TC, fat, low HDL, IL-6, Lp, and Fib. The Y-axis displays changes in ischemia as measured by nuclear imaging. The standard regression analysis shows both the range of estimates (yellow) and the 95% confidence intervals (green). HDL, high-density lipoprotein; IL-6, interleukin-6; Lp-a, lipoprotein-a; Fib, fibrinogen; Tc, total cholesterol [6].

The true accurate diagnosis of Coronary Artery Disease in a given individual requires quantitative testing [4,5], which can accurately measure not merely presence or absence of disease (Figure 3), but the actual extent of inflammatory CAD present. While CAD is an inflammatory process [2], the measurement of that inflammatory process is not the measurement of surrogate blood markers of inflammation, but rather the actual measurement of the extent of the effect of the inflammatory process upon the coronary arteries themselves, with its associated physiologic consequence (Figure 3).
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Absolute quantification and subsequent treatment of CAD [2-6,8] with the removal of human qualitative interpretative errors - and semi-quantified measures such as SUV - is true AI. It is time to replace our qualitative imaging, semi-quantitative efforts and use of cardiovascular risk factors and pseudo-AI, with true AI and absolute quantifiable measurements (FMTVDM) of CAD.

ACKNOWLEDGMENTS: FMTVDM issued to first author. Figures expressly reproduced with permission of first author.

REFERENCES


