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EDITOR'S PAGE

Ischemic Cardiomyopathy: An Oxymoron?

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I was on rounds recently as the Attending Physician on the Cardiology Service when I was confronted with one of the concepts in medicine to which I most object. The medical resident nonchalantly announced that the patient being seen had an "ischemic cardiomyopathy." My thoughts immediately reverted to my early cardiology training when it was drilled into my head that cardiomyopathy was a disease of the myocardium independent of disease in any other cardiovascular structure. Thus, cardiomyopathy was a primary myocardial disease not caused by disease of the heart valves, pericardium, great vessels (systemic or pulmonary hypertension), congenital anomalies, or especially, coronary atherosclerosis. It would therefore seem to be an oxymoron to say that someone had both ischemic heart disease and cardiomyopathy. Nevertheless this term has been repeated over and over on hospital wards and medical records, at medical meetings, and even in medical journals. It has also engendered the antithetical category of non-ischemic cardiomyopathy, dividing left ventricular dysfunction and heart failure into a term that is either a non sequitur or one that is redundant.

The confusion surrounding ischemic cardiomyopathy should not be surprising given the multiple definitions and classifications of cardiomyopathy that have existed through the years. It was initially described as uncommon, non-coronary heart muscle disease, and subsequently as disease of unknown etiology associated with cardiac dysfunction.[1] Given his expertise and stature in the field, the cardiovascular community was greatly influenced by a seminal publication by John Goodwin in 1972.[2] In that paper he first introduced the concept of primary myocardial disease that was categorized as dilated, hypertrophic, or restrictive. Goodwin's definitions were largely adopted and formalized by the World Health Organization in 1980 and have persisted to some degree or another even to the present time. Based upon research advances and greater understanding of the condition, particularly in regard to the role of genetic abnormalities, altered classifications of cardiomyopathy were subsequently promulgated by the American Heart Association[3] and European Society of Cardiology[4] in 2006 and 2008. The most recent classification endorsed by the World Federation of Cardiology[5] classified cardiomyopathy according to detailed morphofunctional, organ involvement, genetic, and etiological criteria, with an additional designation of stage [MOGE(S)]. Obviously, any condition with more than three definitions and classifications can be very confusing. Nevertheless, throughout all of these varying categorizations, a constant theme has been that these are primary diseases of heart muscle independent of abnormalities of any other cardiovascular structure.

Clinically, varying degrees of increased LV volume, mass, systolic dysfunction and heart failure are often the final manifestation of many types of cardiovascular disease. Given the prevalence of coronary atherosclerosis, CAD is the etiology for many of the patients exhibiting end-stage heart disease. Since dilated, poorly contractile ventricles with failure are the most common presentation of cardiomyopathy, it was perhaps predictable that someone would describe end-stage ischemic ventricular dysfunction as ischemic cardiomyopathy. The first recorded use of the term was by George Burch in 1970,[6] but it was used only infrequently. However, as the treatment of coronary disease has progressed, and more and more patients are surviving events only to develop LV dysfunction and heart failure, the prevalence of the term in medical parlance has dramatically increased. The phrase has been warmly embraced by the heart failure/transplant community as it tends to place these patients under their purview.

Although the origination of the name ischemic cardiomyopathy is very understandable, it is still wrong. Not only do ischemic heart disease and primary myocardial disease differ in etiology, pathophysiology, and prognosis, but they also differ markedly in treatment except for the most end-stage patients. The therapeutic potential of revascularization has no role in cardiomyopathy. The attribution of cardiac disease to coronary atherosclerosis may lead to a failure to diagnose the many systemic diseases for which cardiomyopathy is the initial or most prominent finding. In addition, it seems to me to be intellectually sloppy to conflate a primary disorder of the myocardium with fundamental disease of the coronary arteries.

The confusion regarding ischemic cardiomyopathy is further amplified by the possibility of having both abnormalities present. Patients with severe coronary artery disease who have had prior myocardial infarction(s) and present with a dilated, dyskinetic left ventricle can be confidently diagnosed as having severe ischemic LV dysfunction or heart failure. However, it is not uncommon for patients with high grade, disseminated multivessel coronary atherosclerosis to present with normal contractile function and ejection fraction.

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Therefore, the presence of CAD without evidence of prior infarction cannot establish an ischemic etiology for advanced LV dysfunction with certainty. More problematic is the patient with one or two vessel CAD without, or with only a small prior infarction, but with severe, generalized ventricular dyskinesis and dilation. In such cases the myocardial abnormality is disproportionate to the coronary lesions, and it is likely that a primary myocardial process is also present and etiologic. As a former professor of mine often said, "just because you have fleas doesn't mean that you can't have lice too." Such patients should be characterized as having both ischemic disease and cardiomyopathy, whereas using the current vernacular they would be said to have both ischemic and non-ischemic cardiomyopathy.

The difficulty in assessing the possible presence of both ischemic and primary myocardial heart disease is exhibited well by an examination of heart failure patients published by Felker and co-authors.[7] They demonstrated that, as a group, patients with an ischemic etiology had a worse prognosis than those with other causes of heart failure. However, analysis revealed that a subgroup of patients with single vessel disease not involving the left main or proximal LAD without prior infarct had the same prognosis as those without an ischemic etiology despite the same degree of heart failure/dysfunction. More in-depth assessment likely would have revealed that there exist intermediate groups between single vessel and diffuse three-vessel disease in whom the natural history more closely resembled those without CAD. Labeling such groups as non-ischemic cardiomyopathy shrouds the myocardial component and the characteristic etiologies, prognosis, and therapy.

As has been my custom for many years, whenever I encounter the term ischemic cardiomyopathy, whether in presentations, rounds, or publications, I point out the inconsistency. In fact, the positions of the American Heart Association, European Society of Cardiology, and World Heart Federation are all supportive of my position. However, it appears that I am losing ground, as the phraseology is becoming more and more ubiquitous. I feel a bit like Don Quixote, tilting at windmills. Nevertheless, I plan to persist in this effort, since I believe that ischemic cardiomyopathy is more than just a name, but represents a concept of a disorder that is just wrong.

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