Understanding and Treating Angina Postangioplasty

Editorial

Lead Article

Expert Answers to Three Key Questions

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Summaries of Ten Seminal Papers - *M. Marzilli*

Bibliography of One Hundred Key Papers

Editorial

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UNDERSTANDING AND TREATING ANGINA POSTANGIOPLASTY

Angina pectoris is commonly perceived as the clinical manifestation of coronary atherosclerosis. Based on this assumption, diagnostic and therapeutic algorithms have focused on identifying and removing "ischemia-causing" coronary atherosclerotic obstructions. Diagnosis and treatment of stable ischemic heart disease have been driven by coronary anatomy, particularly since the advent of percutaneous coronary interventions, to the point that the diagnosis of angina pectoris was denied in patients with typical symptoms and objective evidence of myocardial ischemia when "significant" stenosis was not identified at angiography ("false positive") and vice-versa.

Recently, evidence has strongly challenged this simplistic approach to ischemic heart disease. In this issue of *Dialogues in Cardiovascular Medicine,* Alda Huqi and Mario Marzilli discuss the new evidence and guidelines on understanding and treating angina postangioplasty. In patients undergoing provocative tests, those with an ischemic response to stress had a prevalence of coronary stenosis at invasive angiography that was similar to those with a negative response. In an international registry of patients undergoing noninvasive coronary angiography, those with typical angina had a prevalence of coronary stenosis similar to that of completely asymptomatic patients.

In patients with angina and "significant" coronary stenosis, the stenosis was removed by percutaneous coronary intervention in addition to optimal medical therapy. However, angina persistence occurred in 34% of patients at the 1-year follow-up despite removing the putative cause. The persistence of symptoms after the percutaneous or surgical removal of the presumed cause of angina has often been denied; therefore, the clinical relevance of persistent angina after "successful" coronary revascularization is underestimated. Recently, the negative prognostic impact of persistent angina has been documented. When diagnosed, angina occurring postangioplasty is attributed to incomplete coronary revascularization due to diffuse coronary atherosclerosis and/or

chronic total occlusions. However, similar rates of persistent angina and/or inducible myocardial ischemia have been reported in patients with no residual stenosis after a successful percutaneous coronary intervention.

In the meantime, the understanding of ischemic heart disease has evolved, and the European guidelines have included not only classic atherosclerotic obstructions, but also both microvascular dysfunction and coronary vasospasm among the possible causes of chronic angina pectoris. The guidelines have acknowledged that these multiple mechanisms may coexist in an individual patient. This new, multifactorial conception of chronic ischemic heart disease has several diagnostic and therapeutic implications, many of which are not fully understood and therefore not fully implemented. One of the immediate consequences of this new approach is the downgrading of the diagnostic role of coronary angiography. In fact, given that microvascular dysfunction and coronary vasospasm are not visible at routine angiography, invasive or noninvasive imaging of coronary anatomy will only be rarely necessary for the sole purpose of establishing or excluding the diagnosis of angina pectoris.

Along the same line of reasoning, the persistence of angina postangioplasty may not only be due to incomplete revascularization, but also to the coexistence of mechanisms that are not treated by a percutaneous coronary intervention, namely microvascular dysfunction and/or coronary vasospasm. Therefore, the persistence of angina after a percutaneous coronary intervention is nothing more than additional indirect evidence supporting the multifactorial nature of ischemic heart disease. If this interpretation is correct, given that angina persists in about one-third of percutaneous coronary intervention patients, an additional effort should be made to identify the cause(s) of angina before referring a patient for a potentially harmful revascularization procedure. This strategy could not only avoid inappropriate interventions, but it could also limit costs and complications, which would eventually improve the cost-benefit ratio of elective percutaneous coronary interventions that have so far been quite disappointing.

Three additional papers in this issue of *Dialogues in Cardiovascular Medicine* nicely expand this perspective. Carolyn M. Webb and Peter Collins summarize the diagnostic and therapeutic challenges of effort angina in patients without significant atherosclerotic obstructions, which was once diagnosed as Syndrome X and is now called microvascular angina. Ranil de Silva discusses the therapeutic options available for refractory angina. Finally, Christine Wright describes the role of a dedicated nurse specialist both in organizing the multidisciplinary treatment team and in generating a treatment plan for patients with refractory angina.