COMMENTRY ARTICLE

Preventive cardiology as a subspecialty of cardiovascular medicine

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ABSTRACT

Despite considerable progress in reducing the worldwide burden of cardiovascular disease, efforts have mostly concentrated on the of evident disease rather than the prevention of occurrences. There is a huge chance to shift the focus away from intervention and toward prevention of cardiovascular disease. Long-established services such as cholesterol, diabetes, hypertension, and general cardiology clinics are giving way to the burgeoning specialty of "preventive cardiology." Because previous advances are endangered by the rising tide of obesity and diabetes, it is incumbent on the cardiology community to engage in cardiovascular prevention. Now is the time to create a specialized preventive cardiology specialist to educate the next generation of professionals. The goal of this American College of Cardiology. Council Perspective is to clarify what it means to be a cardiologist. We have seen tremendous decreases in morbidity and mortality from Cardiovascular Disease (CVD) during the last four decades. Despite this, cardiovascular disease continues to be the top cause of death in both men and women around the world. What is less well known is that at least half of the progress made in improving CVD outcomes has been due to increased access to procedural interventions and technological advancements that allow patients to live with and cope with Advanced Atherosclerotic Cardiovascular Disease (ASCVD) and heart failure rather than preventing the disease in the first place. Despite the fact that avoiding earlier needs for the cardiac patients in Namibia.

Key Words: *Cardiac rehabilitation; Core components; Guidelines; Heart valve surgery; Heart valve replacement*

INTRODUCTION

The Framingham Heart Study, launched in 1948, established the principle of CV risk, with its compounding predictive factors. Early work from Framingham opened up the whole field of preventive cardiology by identifying modifiable risk factors for heart disease. Next, clinical trials extended the epidemiological findings to demonstrate that managing hypercholesterolemia, hypertension, cigarette smoking, and diabetes reduced the risk for ASCVD across age and sex. Indeed, since the landmark publication of 4S study (Scandinavian Simvastatin Survival Study) in 1994, an uninterrupted stream of randomized controlled trials validated the effectiveness of statin drugs in virtually all clinically relevant patient group. _Due to the success of several early studies, trial design progressed from placebo-controlled to statin-controlled (high-intensity vs. low- or moderate-intensity) studies [1]. The clinical outcomes from these studies suggested that there was no Low-Density Lipoprotein Cholesterol (LDL-C) level below which patients did not receive further benefit, thus setting the stage for the current standard of statin recommendation for all individuals above a certain risk threshold [2]. The improved CVD outcomes observed across the statin mega-trials were impressive and strikingly consistent. Moreover, the results of these studies transformed the way clinicians perceived hypercholesterolemia and combined dyslipidemia, both in terms of risk assessment and treatment.

As the understanding that LDL-C lowering is safe, easy to achieve, and effective at mitigating atherosclerotic risk in broad populations became widely established, several national and international guidelines adopted LDL-C lowering as a top priority for ASCVD risk management. At the same time, proper risk assessment became a key driver of treatments that are long-term and occasionally associated

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with side effects. Specialty lipid clinics emerged to optimize implementation of evidence-based guidelines for lipid-lowering therapies, to provide management for patients with high-risk genetic lipid disorders, and for consultative guidance in the setting of treatment-associated adverse effects. However, it has become increasingly recognized that cholesterol management is most often required for individuals whose ASCVD risk. is driven by a multiplicity of comorbidities and risk exposures [3]. Thus, lipid management is component of a comprehensive intervention addressing multiple important ASCVD risk factors, often including a combination of therapeutic lifestyle changes and medical therapies. This concept should be extended to the management of hypertension, diabetes, obesity, and suboptimal lifestyle habits, and so on-all of which can be addressed under the auspices of a dedicated preventive cardiology program. As the need to identify more comprehensive preventive opportunities in larger populations emerged, the value of the limited specialty clinic model diminished and the roots of preventive cardiology took hold. It became increasingly apparent that to prevent ASCVD most effectively, multiple risk factor interventions are required[4].

Global risk assessment tools have limitations, with prior algorithms underestimating and more modern risk scores at times overestimating risk. Moreover, risk assessment tools for ASCVD, although helpful, continue to exhibit gaps, such as not accounting for the risk of heart failure, and also require frequent updating to incorporate contemporary data. Current work moves from population-level data and risk factors, such as cholesterol, to a new generation of precision medicine and tailored therapeutics. Within the realm of preventive cardiology, refinement in risk estimation and therapeutic decision making is being facilitated by atherosclerosis imaging (e.g., ultrasound for carotid intima-media thickness and carotid plaque, computed tomography for coronary artery calcium score), new biomarkers, advances in genetic testing (identification of causal monogenic mutations and development of actionable polygenic risk scores), and a plethora of pipeline and market pharmaceuticals with promise or proof of benefits. Given the proliferation of new data, approaches, and pharmacotherapy, preventive cardiology needs a structure to deliver specialized training and produce dedicated clinician-scientists and practitioners to serve this ever-growing medical need [5]. This new direction will be important to help us define risks early, initiate appropriate treatments, and identify new drugs/approaches for treatment and prevention delineates common indications for referral to preventive cardiology services.

LIPID RELATED DANGERS

Since 1913, when Nikolai Ansitschkow a Russian pathologist, administered pure cholesterol to rabbits to develop aortic atherosclerosis, scientists and doctors from many backgrounds have been intrigued to the link between cholesterol and ASCVD. The Framingham Heart Study pioneered the notion of ASCVD risk factors, which is still relevant today [6]. Years later, physicist John Goffman identified plasma lipoproteins using analytical ultracentrifugation, demonstrating the direct and inverse correlations between LDL-C and high-density lipoprotein cholesterol levels and myocardial infarction rates, respectively. After identifying the intricate biochemical mechanism of cholesterol synthesis, Konrad Boch and Feodor Lynen were awarded the Nobel Prize ten years later. Michael Brown and Joe Goldstein produced their ground-breaking discovery about the invertebrates in 1973. The Framingham Heart Study pioneered the notion of ASCVD risk factors, which is still relevant today. Years later, physicist John Goffman identified plasma lipoproteins using analytical ultracentrifugation, demonstrating the direct and inverse correlations between LDL-C and high-density lipoprotein cholesterol levels and myocardial infarction rates, respectively [7]. After identifying the intricate biochemical mechanism of cholesterol synthesis, Konrad Bloch and Feodor Lynen were awarded the Nobel Prize ten years later. Michael Brown and Joe Goldstein produced important discoveries on the LDL receptor in 1973, with a young kid who suffered a heart attack due to homozygous familial hypercholesterolemia as the original inspiration for their work.

CVD RISK REDUCTION RESEARCH

Our understanding of lipid metabolism has been changed by the identification of Proprotein Convertase/Subtilisin Kexin Type 9 (PCSK9) as a master regulator of plasma LDL-C 19, 20. The US Food and Drug Administration (FDA) approved two completely human monoclonal antibodies that block PCSK9 function within 12 years after the protein's discovery, and landmark trials have shown modest improvements in CV outcomes when these treatments are coupled with statins. Alternative PCSK9 inhibitors are in the early stages of development. Beyond LDL-C, there is growing interest and impetus in targeting other atherogenic lipoproteins. Triglycerides and lipoprotein(a) lp(a) levels are anticipated to become targets of medication in the future to improve CV outcomes, according to both observational and genetic epidemiology. Targeting triglycerides with omega-3 polyunsaturated fatty acids Eicosapentaenoic Acid (EPA) only or a combination of EPA and docosahexaenoic acid) and a novel selective peroxisome proliferator-activated receptor alpha modulator, pemafibrate is assessing CV outcomes in randomized controlled studies. In fact, the REDUCE-IT (Reduction of Cardiovascular Events with Icosapent Ethyl–Intervention Trial), which compared the addition of 4 g/day of EPA to statin therapy in patients with established ASCVD and/or diabetes and at least one additional risk factor to placebo in patients with established ASCVD and/or diabetes and at least one additional risk factor, found significant improvements in CV outcomes [8]. Volanesorsen, an antisense oligonucleotide that inhibits the development of apoC III has shown to have a significant triglyceride-lowering effect while also appearing to be safe. Akcea, ApoCIII-Lrx, the second-generation (N-acetylgalactosamine form) of this medicine, is currently being tested in a Phase IIB clinical trial. Furthermore, the development of a particular antisense oligonucleotide that inhibits Apo lipoprotein(a) formation and hence substantially Lowers Plasma Lp(a) concentrations by roughly 80% 29, 30 has given Lp(a) a new lease on life. A large, randomized controlled trial is being planned to see if lowering Lp(a) is linked to better CV outcomes. As previously said, lipid science and its application to patient treatment have evolved and will continue to evolve, necessitating specialist knowledge to enhance the evaluation and management of patients at risk. Even among patients who are receiving rigorous treatment with established treatments, the rate of ASCVD events is unacceptably high. After

LDL-C has been reduced to its lowest level, the idea of residual CV risk suggests that other CV risk factors and comorbidities might be targeted to enhance outcomes. In the FOURIER (Further Cardiovascular Outcomes Research with PCSK9 Inhibition in Subjects with Elevated Risk) trial of evolocumab, despite achieving a median LDL-C of 30 mg/dl, there was a modest absolute risk reduction (1.5 percent) of the primary composite major adverse CV outcome. As a result, there has been a lot of interest in looking into additional options for improving CV outcomes. The Canakinumab Anti-Inflammatory Thrombosis Outcomes Study (CANTOS) [9]. randomized 10,061 subjects with established ASCVD and evidence of subclinical inflammation (based on elevated high-sensitivity C-reactive protein levels) to optimal medical therapy plus either the interleukin-1 antagonist canakinumab or placebo. Those who were given canakinumab had a statistically significant lower risk of recurrent episodes. As a result, inflammation might be included to the list of residual risk drivers. To that goal, a number of new anti-inflammatory drugs are now being investigated or assessed for their potential to reduce ASCVD occurrences. The path to regulatory approval and clinical adoption of powerful anti-inflammatory medicines for secondary prevention will almost certainly be winding and difficult. In truth, Novartis is a pharmaceutical company. The CIRT (Cardiovascular Inflammation Reduction Trial), testing low-dose methotrexate versus placebo in high-risk patients, failed to demonstrate an improvement in CV outcomes, suggesting that targeting specific inflammatory pathways will be necessary to garner therapeutic benefits. Indeed, there are ongoing studies testing the inflammatory hypothesis of ASCVD by suppressing inflammation with alternative approaches, including trials evaluating the role of low-dose colchicine for secondary prevention of CVD. The preventive cardiologist of the future will need to be well versed in assessing inflammatory risk and potentially modulating this risk with specific anti-inflammatory therapies.

TREATMENT FOR DIABETES

Type 2 diabetes puts you at a high risk of getting cardiovascular disease over the course of your life. This link is exacerbated by the fact that other metabolic risk factors for ASCVD and Heart Failure (HF) are frequently observed in diabetic patients. Indeed, ASCVD and heart failure (both with reduced, and increasingly with maintained, left ventricular ejection fraction, which can occur even in the absence of clinically evident ASCVD) are the greatest medical risks among diabetics. Despite the fact that glycemic management plays an important role in the prevention of some microvascular consequences, targeting plasma glucose as a strategy for reducing ASCVD and HF risks has yet to show considerable benefit.

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