Pathology of the Heart and Blood Vessels: Emerging Concepts

**Editorial**

“True knowledge exists in knowing that you know nothing” Socrates.

In the past century, evidence indicated that cardiovascular disease was a world plague for living individuals with regard to either epidemiological results or the clinical-therapeutic approach. However, Gould [1] literally wrote that “Cardiac pathology is sharing in the exciting and imaginative development of modern medicine, and the pathologist has become a vital member of the cardiologic team” in the Preface of his Textbook with a title almost similar to this editorial. As can be seen, not withstanding, no significant progress in the development of the characteristics of the cardiovascular disease, nevertheless an enthusiastic approach to the study of the heart and blood vessels existed.

In the 1960-70’ years, the support of the cardiologists led to the study of valvular heart disease [2], primarily mitral valve stenosis related to rheumatic fever [3] and semilunar aortic valve alterations, whose etiology recognized syphilis or atherosclerotic disease [4,5]. In addition, autopsy findings contributed to the knowledge and classification of the pathological characteristics of the congenital heart disease [6]. Hypertension was recognized as a potential cardiovascular alteration with no dramatic effects as documented today and, in addition, discontinuously treated by a scarce number of drugs [7] like alpha-methyl-dopa, some diuretics, and reserpine.

Following this period, however, some newer and stimulating concepts began to characterize the pathology of ischemic heart disease, primarily acute myocardial infarction because of the observations of some eminent pathologists [8-10], who analyzed the composition and development of coronary thrombi. They hypothesized, at that time, a coronary thrombus secondary to the ischemic disease and a consequence of the rheological changes in the area of the infarct, which impeded the laminar blood flow in the coronary artery related to the altered myocardium. A dramatic change of this hypothesis occurred in the 1990’ys when the obstructive atherosclerotic plaque and its fissuring (vulnerable plaque) was demonstrated to be in the coronary arteries of individuals affected by acute myocardial infarction [11,12].

These findings deeply modified the concepts related to the pathogenetic mechanisms of coronary heart disease and, at the same time, favored the development of newer diagnostic and therapeutic approaches to the management of both medical and surgical type [13]. Similarly, a dramatic change in the treatment of elevated blood pressure as well as the duration of the therapy was systematically assessed in the hypertensive individuals because of a large number of antihypertensive drugs to be used [14].

Findings related to cardiomyopathies, primarily hypertrophic and dilated cardiomyopathy followed by heart failure as a cardiac end-event showed a strong impulse [15]. The first emerging concept to be taken into account is the change of class that characterized both clinical and pathological events related to cardiovascular findings consisted of the enormous progress in pharmacological management of cardiovascular disease associated with a careful epidemiological survey of related pathology. The alterations observed demonstrated to be in considerable increase of the rate. Thus, ischemic heart disease and hypertension with its complications were the focus of the findings on the heart and blood vessels in the current century.

With regard to ischemic heart disease, a basic concept emerged. Different pathogenic mechanisms, not always due to obstructive coronary pathology, characterized the patterns of the various constituents of the ischemic disease. Thus, typical patterns of myocardial infarction and effort angina related to coronary atherosclerosis could be seen, but also vasospam (unstable angina) and sudden cardiac death, mainly due to sympathetic and adrenergic stimulation, can accompany the ischemic events. Both medical and/or surgical therapy, according to the characteristics of the pathology observed was and is continuously updated with very promising results, which are also a consequence of a major control of the coronary risk factors.

On the contrary, with regard to hypertension an established concept has been stably acquired: a lowering of blood pressure as high as possible in the hypertensive subjects, anyway obtained, is necessary to reduce the rate and/or protect against vascular complications, primarily stroke. At present, a satisfactory control of blood pressure can be achieved in view of the large number of antihypertensive drugs which can be safely used.

A very promising result, which then progresses in the cardiovascular finding provide, is a wider knowledge of the role of cardiovascular risk factors in the rate and appearance of any cardiovascular disease [16]. Hypertension, cigarette smoking, metabolic disorders of blood lipids and glucose as well as obesity
have been recognized to adversely influence the physiology and normal function of the heart and blood vessels with regard to either the rate or complicating events. However, the number of risk factors known is continuously in progress and characterized by implications sometimes worthy of discussion. It is worth noting that among the cardiovascular risk factors, there are some, like cigarette smoking, that could be absolutely avoided, some others, primarily metabolic disorders corrected by appropriate lifestyle changes and preventive measures, and, finally a group, primarily family history, age, and sex, which, although unmodifiable, could benefit of the protective measures of control conducted to prevent the effects of the other risk factors.

Conclusion

In conclusion, a significantly different approach to the knowledge of the cardiovascular pathology has been achieved in this century by the development of more current findings of the disease of the heart and blood vessels, mainly due to the epidemiology and pharmacological studies. Newer concepts and updated observations have been identified as basis of interpreting the main cardiovascular alterations and their prevention associated with mass-media efforts to diffuse these occurrences among living individuals.

However, the most common and useful concept that should emerge is the cardiovascular pathology is always the same at any time, but the progresses in the knowledge of the responsible mechanisms continuously are changing according to more careful studies in the field of the disease of the heart and blood vessels. Thus, achieving the own knowledge, knowing to not do it is a fact to be always taken into account as well as the best emerging concept

References

1. Gould SE (1968) Preface. In Pathology of the Heart and Blood Vessels, SE Gould. (3rd edn), Charles C. Thomas Publisher, Springfield, Illinois, USA.
2. Pomerance A (1972) Pathology and valvular heart disease. Br Heart J 34(5): 437-443.
3. Selber A, Cohn KE (1972) Natural History of Mitral Stenosis: A Review. Circulation 45(4): 878-890.
4. Grabau W, Emanuel R, Ross D, Parker J, Hegde M (1976) Syphilitic aortic regurgitation. An appraisal of surgical treatment. Br J Vener Dis 52(6): 366-373.
5. Harris CN, Kaplan MA, Parker DP, Dunne EF, Cowell HS, et al. (1975) Aortic stenosis, angina, and coronary artery disease. Interrelations. Br Heart J 37(6): 656-661.
6. Lev M, Paul MH, Miller RA (1962) A classification of congenital heart disease based on the pathologic complex. Am J Cardiol 10(5): 733-737.
7. Gibb WE, Malpae JS, Turner P, White RJ (1970) Comparison of bethanidine, alpha-methyldopa, and reserpine in essential hypertension. Lancet 2(7667): 275-277.
8. Elliot RS, Baroldi G, Leone A (1974) Necropsy studies in myocardial infarction with minimal or no coronary luminal reduction due to atherosclerosis Circulation 49(6): 1127-1131.
9. Baroldi G, Radice F, Schmid G, Leone A (1974) Morphology of acute myocardial infarction in relation to coronary thrombosis. Am Heart J 87(1): 65-75.
10. Chandler AB, Chapman I, Erhardt LR, Roberts WC, Schwartz CJ, et al. (1974) Coronary thrombosis in myocardial infarction. Report of a workshop on the role of coronary thrombosis in the pathogenesis of acute myocardial infarction. Am J Cardiol 34(7): 823-833.
11. Falk E (1992) Why do plaque rupture? Circulation 86 (6 Suppl): III30-42.
12. Falk E, Shah PK, Fuster V (1995) Coronary plaque disruption. Circulation 92(3): 657-671.
13. Alexander KP, Peterson ED (2001) Medical and surgical management of coronary artery disease in women. Am J Manag Care 7(10): 951-956.
14. Leone A, Landini L, Leone A (2011) Epidemiology and costs of hypertension-related disorders. Curr Pharm Des 17(28): 2955-2972.
15. Yu CM, Fung WH, Lin H, Zhang Q, Sanderson JE, et al. (2003) Predictors of left ventricular reverse remodeling after cardiac resynchronization therapy for heart failure secondary to idiopathic dilated or ischemic cardiomyopathy. Am J Cardiol 91(6): 684-688.
16. Leone A (2016) Markers of atherosclerotic disease: What do they mean? Current opinion and future trends. Curr Pharm Des 22(1): 7-17.

Citation: Leone A (2016) Pathology of the Heart and Blood Vessels: Emerging Concepts. J Cardiol Curr Res 5(4): 00169. DOI: 10.15406/jccr.2016.05.00169