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The influence of type A personality in cardiovascular disease

Abstract

The correlations among the psychic repercussions of acute and chronic stress on the atherosclerotic process due to psychosomatic disorders were described more consistently since the middle of the 20th century. The revival of the attentions to them has been revitalized by the necessity to understand, diagnose and treat the effects of the Covid-19 pandemics in the cardiac patients. A pivotal exemplary work demonstrated the relationship of stress and atherosclerosis was experimentally compared when rabbits were placed on a lipid-rich diet in which atherosclerosis was developed (about 40%); however, if in addition to the diet, the animals were submitted to a continuous stress or the administration of benzedrine, about 90% of them presented generalized atheromas The increased risk of coronary heart disease in stress patients is well known. However, prisoners in concentration and war camps, under a strong state of stress, did not present the risk of coronary heart disease, but those who suffered an impact from a hurricane, earthquake or deprivation or even ignominy are deeply affected, bringing harm to the body. Attention must be payed to the importance of the peculiarities of each individually; responses to the stressful factor may depend on their genetic predisposition and specific unconscious fantasies, own and characteristics of each person. Unconscious conflicts would prepare their responses to stressful factors. Studies presented from different perspectives to link them the environmental stress as is presently the Covid-19 occurrence.

Keywords: psychosomatic medicine, stress, Covid-19, coronary, atherosclerosis, cardiac symptoms, anxiety, depression

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Introduction

The year of 2020, the Covid-19 year, is being a threat not only to infectious diseases specialists but to all the clinical and basic sciences researchers, due to the multiple array of manifestations that can occur during the pandemic. The resulting stress manifestations, mainly anxiety and depression, cause alterations that present psychosomatic symptoms. Attention has been brought to the occurrence of its repercussions in the emotional, psychological, psychosomatic and psychiatric deleterious effects. The focus of this communications is aimed at the primordial relations of the cardiometabolic and somatic reactions of the heart related to the coronary heart disease and the atherosclerotic progression.[1]

It has to be noted that its understanding has its roots in earlier works that serve as a basis for its comprehension.

Psychosomatic Medicine since its early days of the modern era until today devotes enormous attention to emotional problems related to cardiovascular disorders, beginning with the famous works of Selye.[2–10] As described in previous work,[11] the main manifestations occur in Type A personality and can be summarized as follows: marked tendency to achieve usually undefined goals, marked drive to compete, persistent desire for recognition and progress, continuously involved in multiple functions, not having time to finish them, getting hurt and distressing at the last moment to perform them, propensity to accelerate the pace of physical and mental activities, extraordinarily concerned, physically and mentally.

Another anxious characteristic of these individuals is their inability to relax, that is, these individuals are never satisfied with what they accomplish, because their degree of ambition is always above what they get. Retrospective studies on profile A and coronary arteriopathies indicate a real and evident link between these two pathological situations. One of the epidemiological studies on this correlation was conducted by the Western Collaborative Group Study (WCGS) involving 3000 normal men for 8 and a half years revealing twice the risk of acquiring coronary atherosclerosis to those with profile A, confirmed by the Framingham Studies of Coronary Risk, whose research also shows women with profile A reaching the same incidence as men. Byrne, Rosenmann think steam-type A patients behave unintentionally, increasing their occupational and social stress.[11]

The relationship of stress and atherosclerosis was experimentally compared when rabbits were placed on a lipid-rich diet in which atherosclerosis was developed however, if in addition to the diet, the animals were submitted to a continuous stress or the administration of benzedrine, about 90% of them presented generalized atheromas.[12]

In the case of chicks placed in an isolated compartment, they have higher intensity of atherosclerosis (including death) compared to those reared in gregarious life. Isolation represents, therefore, a "stress" for these birds, mainly because it is a "critical period" of growth. However, the main experiment performed by Myasnikov[12] was that in which the excitation of the ventromedian nuclei and tuber produces increased atheroma depot in the aorta and coronary artery, and can be avoided by prophylactic action of the psychotropic. These experiences highlight the importance of emotions at the origin of this disease. Grundy, Griffin[13] revealed that in medical students during the testing period, the cholesterolemia rate increased by 23% to 27% compared to the levels found during regular periods of classes.

Russek[14] found that emotional factors mobilize lipids from tissue to blood, including the sudden triggering of coronary occlusion. Myasnikov[12] in hypertensive patients with atherosclerosis found that caffeine and benzedrine increased cholesterolemia, and, on the contrary, phenobarbital, hydrated chloral and heparin lowered it. Brogdonoff, Estes[15] found rapid and significant increase in free fatty acids during periods of emotional stress. Sloane et al.[16] putting

individuals on a low-fat diet for 8 days, divided them into 2 groups called "high" (cholesterolemia greater than 165.5mg%) and "low" (less than 107 mg%); they found that the group of "highs" was composed of aggressive, ambitious and dictatorial individuals and, although not distressed, were always dissatisfied with themselves.

Sloane et al.[16] they find that these individuals sublimate this dissatisfaction by the strong need to be socially accepted, they have great aspirations and are extremely competitive, not accepting the rejection or loss of an object without externalizing themselves, because they do not know how to repress their aggressive feelings, which is why they channel us in professional activities. They're afraid of being alienated by others. The psychiatric study of these 2 groups[17] was able to predict more individuals with a statistically significant higher cholesterolemia rate (r = 0.58). Interestingly, students with "high cholesterolemia" said (consciously) that they had no ambition.

Animal experiments show that sudden death due to arrhythmia may occur, depending on environmental stress, as demonstrated by Lown et al.[18] and Verrier Lown,[19] whose biological mechanisms have been well studied. We can consider atherogenesis as an inflammatory response to injury of the endothelium and smooth muscle layer of the vessel, secondary to genetic, metabolic and hemodynamic influences, promoting the formation of a fibrofatty or fibrous plaque as a repair response of the arterial wall.[20,21]

The adhesion glycoproteins VCAM-1 and ELAM-1 (Endothelial Leukocyte Adhesion Molecule 1), generated by the endothelium, are related, respectively, to the adhesion of monocytes and polymorphonuclear leukocytes to the vessel wall.[22] The expression of the adhering molecules may also be induced by mediators such as gamma-interferon, IL-4 (Interleukin 4), IL-1 (Interleukin 1) beta and TNF-alpha (Tumor Necrosis Factor alpha), suggesting that certain pro-inflammatory cytokines may regulate the expression of the acceding molecules and be related to plaque development. After adhering to glycoproteins, monocytes migrate into the subendothelial space through intercellular junctions, under the influence of thygens, oxidized lipoproteins and mainly a specific protein called MCP-1 (Monocyte Chemotatic Protein -1), produced by endothelial cells and smooth muscles of the vessel wall.[23] In the subendothelial space, monocytes undergo a process of activation and differentiation, resulting in their phenotypic conversion to macrophages. Macrophages and, later, cells of the smooth muscle layer that migrate to the subendothelial space where they differ, accumulate lipids, giving rise to foamy cells. These, accompanied by T lymphocytes, will constitute the fatty stria, which is the earliest recognizable atherosclerotic lesion.[24]

The increased risk of coronary heart disease in stress patients is well known. However, prisoners in concentration and war camps, under a strong state of stress, did not present the risk of coronary heart disease,[25,26] but those who suffered an impact from a hurricane, earthquake or deprivation[27,28] or even ignominy are deeply affected, bringing harm to the body.[29] These apparently incomprehensible contrasts are due to stress due to the most varied causes, as some produce acute hormonal changes and other stresses cause chronic changes, both of which can damage the body in different ways.

Holmes Rahe,[30] through the Tests invented by them (Social Readjustment Rating Scale) found that certain individuals who accumulated high rates, had more coronary risks than the general population. Lazarus[31] argues that certain life events are not inherently stressful; they would depend on personal structure. Miller de Paiva[32] shows the importance of stym (haeceitas) that is, the peculiarity of the individual; responses to the stressful factor may depend on their genetic predisposition and specific unconscious fantasies, own and characteristics of each person. Unconscious conflicts would prepare their responses to stressful factors. Studies presented from different perspectives to link them between environmental stress and illness need to be viewed with caution due to various contamination factors, whether psychic (unconscious), organic and external. Connoly[33] was able to demonstrate that significant stresses preceded infarction crises.

Conclusion

In conclusion, the presence of stress in patients can be an agent of worsening the risk for coronary heart disease not only by its adrenergic effects but also for the detrimental lifestyle repercussions in unhealthy lifestyle habits that interfere with the major risk factors.

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Conflicts of interest

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References

1. Murthy VL, Yu B, Wang W, et al. Molecular signature of multisystem cardiometabolic stress and its association with prognosis. *JAMA Cardiol*. 2020;5(10):1–10.
2. Selye H. Annual Report on Stress. Montreal: Acta Ed; 1951.
3. Selye H. Stress in health and disease. 1st edn. Sydiney: Butterworth–Heinemann; 1976.
4. Friedman HS, Booth–Kewley S. Personality, Type A behavior, and coronary heart disease: The role of emotional expression. *J Pers Soc Psychol*. 1987;53(4):783–792.
5. Schwalbe FC. Relationship between Type A personality and coronary heart disease. Analysis of five cohort studies. *J Fla Med Assoc*. 1990;77(9):803–805.
6. Riska E. The rise and fall of Type A man. *Soc Sci Med*. 2000;51(11):1665–1674.
7. Strike PC, Steptoe A. Psychosocial factors in the development of coronary artery disease. *Prog Cardiovasc Dis*. 2004;46(4):337–347.
8. Mitaishvili N, Danelia M. Personality type and coronary heart disease. *Georgian Med News*. 2006;(134):58–60.
9. Hawkins MAW, Callahan CM, Stump TE, et al. Depressive symptom clusters as predictors of incident coronary artery disease: a 15–year prospective study. *Psychosom Med*. 2014;76(1):38–43.
10. Bagheri B, Meshkini F, Dinarvand K, et al. Life psychosocial stresses and coronary artery disease. *Int J Prev Med*. 2016;7:106.
11. Silva AMAPN, Saldanha ALR, Margeotto APP, et al. Hypothetizing concomitant types A and D personalities in chronic kidney disease. *Int J Fam Commun Med*. 2020;4(6):158–160.
12. Myasnikov AL. Influence of some factors on development of experimental cholesterol atherosclerosis. *Circulation*. 1958;17: 99–113.
13. Grundy SM, Griffin AC. Relationship of periodic mental stress to serum lipoprotein and cholesterol levels. *J Am Med Assoc*. 1959;171:1794–1796.
14. Russek HI. Role of heredity, diet, and emotional stress in coronary heart disease. *JAMA*. 1959;171(5):503–508.

15. Brogdonoff MD, Estes H. Energy dynamics and acute states of arousal in man. *Psychosom Med*. 1961;23(1):23–32.

16. Sloane RB, Habib A, Eveson MB, et al. Some behavioural and other correlates of cholesterol metabolism. *J Psychosom Res*. 1961;5(3):183–190.

17. Sloane RB, Inglis J, Payne RW. Personal traits and maternal attitudes in relation to blood lipid levels. *Psychosom Med*. 1962;24:278–285.

18. Lown B, DeSilva RA, Reich P, et al. Psychophysiologic factors in sudden cardiac death. *Am J Psychiatry*. 1980;137(11):1325–1335.

19. Verrier RL, Lown B. Autonomic nervous system and malignant cardiac arrhythmias. *Res Publ Assoc Res Nerv Ment Dis*. 1981;59:273–291.

20. Steinberg D. Currents theories of the pathogenesis of atherosclerosis. In: Steinberg D, Olefsky JM, Editors. Hypercholesterolemia and Atherosclerosis. Pathogenesis and Prevention (CIEM). 1st edn. USA, New York: Churchill Livingstone; 1987.

21. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. *Nature*. 1993;362:801–809.

22. Weber C, Erl W, Pietsch A, et al. Antioxidants inhibit monocyte adhesion by suppressing nuclear factor–kappa B mobilization and induction of vascular cell adhesion molecule–1 in endothelial cells stimulated to generate radicals. *Arterioscler Thromb*. 1994;14(10):1665–1673.

23. Schwartz CJ, Valente AJ, Sprague EA. A modern view of atherogenesis. *Am J Cardiol*. 1993;71(6):9B–14B.

24. Chait A. Progression of atherosclerosis: the cell biology. *Eur Heart J*. 1987;8(suppl E):15–22.

25. Eitinger L, Strøm A. New investigations on the mortality and morbidity of Norwegian ex–concentration camp prisoners. Isr *J Psychiatry Relat Sci*. 1981;18(3):173–195.

26. Gill G. Study of mortality and autopsy findings amongst former prisoners of the Japanese. *J R Army Med Corps*. 1983;129(1):11–13.

27. Abrahams MJ, Price J, Whitlock FA, et al. The Brisbane floods, January 1974: their impact on health. *Med J Aust*. 1976;2(25–26):936–939.

28. Trichopoulos D, Katsouyanni K, Zavitsanos X, et al. Psychological stress and fatal heart attack: the Athens (1981) earthquake natural experiment. *Lancet*. 1983;1(8322):441–444.

29. Kosten TR, Jacobs S, Mason J, et al. Psychological correlates of growth hormone response to stress. *Psychosom Med*. 1984;46(1):49–58.

30. Holmes TH, Rahe RH. The social readjustment rating scale. *J Psychosom Res*. 1967;11(2):213–218.

31. Lazarus RS. Psychological stress and the coping process. New York: McGraw–Hill; 1966.

32. Miller de Paiva L. Psychoanalysis technique. "DIY" and Filigree. Brazil: Rio de Janeiro, Editora Imago; 1987. p. 364.

33. Connolly J. Life events before myocardial infarction. *J Human Stress*. 1976;2(4):3–17.

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