HYPERTENSIVE HEART DISEASE

Pages with reference to book, From 43 To 44

Nuzhat Rafique (PMRC Research Centre, Jinnah Postgraduate Medical Centre, Karachi.)

Hypertensive heart disease can be defined as response of the heart to the after load imposed on the left ventricle by the progressively increasing arterial pressure and peripheral vascular resistance. Other than left ventricular hypertrophy other cardiac manifestations of hypertension include congestive heart failure, cardiac dysrhythmias and ischaemic heart disease. Although risk of atherosclerotic coronary heart disease is related to the systolic and diastolic pressure, most clinical trials have not found a relation between antihypertensive therapy and reduced incidence of myocardial infarction. This observation is explained by multi factorial nature of both hypertensive and coronary heart disease, age of patient, experimental design and metabolic effects of antihypertensive agents[1]. Some have suggested the possibility of harmful effect if diastolic pressure is reduced too much[2]. Relation between the height of arterial pressure and left ventricular hypertrophy has been repoted[3-6]. Consequences of hypertensive heart disease and obstructive coronary disease are difficult to distinguish, former involves increased myocardial oxygen demand associated with comparatively reduced coronary blood for hypertrophied left ventricle and associated micro vascular disease, later results in reduced myocardial blood supply due to occlusive atherosclerotic epicardial arterial disease. Atherosclerotic arterial disease has its own natural progression, but hypertension facilitates this process. The heart maintains its chamber size in proportion to its workload and to body weight, growth and maturation[7]. The type of cardiac overload determines the pattern of hypertrophy. Volume overload produces increased ventrical cavity volume on proportion to mass called “eccentric hypertrophy"8, whereas pressure overload produces increased left ventricular mass out of proportion to volume known as “concentric hypertrophy” [8]. Physiologic hypertrophy refers to increased left ventricular mass in athletes, but very few studies have assessed whether exercise induced hypertrophy is physiologic or pathologic[9]. Hypertension has been reported as the most common precursor of the coronary heart failure[10,11]. Although concentric left ventricular hypertrophy maintains systolic function at a near normal level, left ventricular relaxation is impaired with long standing pressure overload reflecting reduced distensibility of the left ventricle. The clinical recognition of cardiac involvement in hypertension depends upon non-invasive methods that detect hypertrophy or abnormal left ventricular function. The most widely used clinical classification of the severity of hypertension is based on the level of systolic and diastolic pressure[27], which are risk factors for left ventricular hypertrophy. On physical examination forceful and sustained apical impulse with a fourth heart sound suggests hypertrophy, an unsustained apical impulse with a faster heart rate indicates hyperdynamic circulation, lateral displacement of apical impulse shows left ventricular dialatation and presence of third heart sound along with it suggest congestive heart failure[15-17]. ECG the standard method of recognizing left ventricular hypertrophy and staging hypertensive heart, is recommended for every hypertensive patient. Although ECG detects only a minority of instances of left ventricular hypertrophy identified by echocardiography but despite the relative insensitivity of this method the development or reversal of ECG changes of left ventricular hypertrophy has been a strong predictor of patients outcome[28,29]. Definite electrocardiographic evidence of hypertrophy provides proof of target organ demage-amendate for blood pressure control - portends a poorer prognosis. Echocardiography provides good information about the structural changes in hypertensive heart disease[30-32] like posterior wall and inter ventricular septal thickness and size of left ventricular chamber. Abnormality of shape and wall motion can be detected by two-dimensional echocardiography[33]. Exercise electrocardiography and newer imaging methods are being used to work out true deficits in myocardial perfusion due to

hypertensive arteriolar disease[34,35]. Positive exercise tests are common in hypertensive patients without obstructive epicardial coronary artery disease. Nuclear magnatic resonance imaging or CT scan with proper cardiac-cycle gating, provides high quality representation of cardiac size, geometry and function[36-38], but these techniques are currently too expensive as compared with echocardiography, so can only be used in very selected cases. Complications of hypertensive heart includes cardiac hypertrophy, which is an adaptive benefit but confers substantial risk on the patient due to myocardial ischaemia and increased minimal vascular resistance[5,6,11]. Greater susceptibility to leathal dysrhythmias and ectopic ventricular activity is also a risk factor for sudden death[39]. Development of cardiac failure, in which diastolic dysfunction precedes systolic dysfunction during long procession of hypertensive heart disease to cardiac failure. Main therapeutic concerning coversing left ventricular hypertrophy is the need to prevent its development in the first place. This requires a commitment to the early treatment of hypertension before hypertrophy develops. Almost all antihypertensive agents, when used for long enough periods will reduce left ventricular mass, but not all agents in some class of drugs have the same effect on cardiac mass[40]. Only certain drugs reduce mass within a period of weeks. Short term therapy with diuretics and direct acting smooth muscle vasodilators such as hydralazine will not reduce left ventricular mass, whereas short term therapy for four to eight weeks with beta adrenergic receptor blockers, centrally acting adrenolytic drugs such as methyldopa, angiotensive converting enzyme inhibitors and calcium channel blockers will reduce left ventricular mass rapidly. These findings suggest that the reversal of left ventricular hypertrophy after short term therapy is unclear, but they may include inhibition of adrenergic or renin-angiotensin system, changes in intracellular calcium and induction or inhibition of humoral substances, growth factors or proto oncogenes[20,23,24,41,42]. It is still too early to identify which forms of therapy are best. In this regard it will be important to learn whether the reversal of left ventricular hypertrophy with specific antihypertensive agents increases survival or not?

REFERENCES

1. Samuelsson, 0., Wilhelmsen, L, Anderason, OK, Pennert, K., Bergiund, C. Cardiovascular morbidity in relation to change in blood preesure and serum cholesterol levels in treated hypertenaion: results from the Primary Prevention Trial in Goteborg. Sweden. JAMA., 1987;258:1768-76.
2. Cruickshankç J.M. coronary flow reserve and the 3 curve relation between distolic blood pressureand myoeardial infarction. BMJ.. 1988;297: 1221-30.
3. Sokotow, M., Perloff, D.The prognosis of essential hypertension trested conservatively. circulation, 1961;23:697-713.
4. Breziin, D.J., Gifford, R.W. Jr., Fairbairn, J.F. II. Essential hypertension: a twenty-year follow-up study. Circulation, 1966;33:87-97.
5. Kannel, W.B.. Gordon, T., Offutt, D. Left ventricular hypertrophy by eleetro-cardiogram: prevalence, incidence and mortality in the Framingham Study. Ann.lntern. Med., 1969;71:89-105.
6. Levy, D., Anderson. KM., Savage, D.D., Kannel, W.B., Christiansen, J.C., Castelli, W.P. Echocardiographically detected left ventricular hypertrophy: prevalence and risk factors: the Framinghsm Hesrt Study. Ann. intern. Med., 1988;108:7-13.
7. Holt, J.P., Rhode, LA, Kines, H. ventricular volumes and body weight in mammala. Am.J.Physiol., 1968;215:704-15.
8. Maron, B.J., Roberts, W.C., McAllister, HA., Easing, DR. Epstein, SE. Sudden dsath in youngathletes. circulation, 1980;62:218-29.
9. Granger, CB., Karimeddini, M.K., Smith, V.E Shapiro, Hit, Katz, AM., Ribs, AL Rspid ventricular filling in left ventricular hypertrophy. I. Physiologic hypertrophy. 3. Am. CoIl. CardioL 1988;5:862-8.

10. Kannel, W.B., Castelli, W.P., McNamara, P.M., McKee, P.A, Feinleib, M. Roleof blood pressure in the development of congestive heart failure: the Frangham Study. N. Engl. 3. Med., 1972:287:781-7.
11. Kannel, W.B., Dannenberg, AL, Levy, D. Population implications of electrocardiographic leftventricular hypertrophy. Am.J., CsrdioL, 1987;60:851-931.
12. Smith, V.E, Schulman, P., Karimmeddini, M.K., White, W.B., Meeran, M.K., Kata, AM. Rapidventriculsr fillingin leftventricular hypertrophy. II. Pathologic hypertrophy. J.Am.Coll.Cardiol., 1985;5:869-74.
13. lnouye, I., Massie, B., Loge, D. et al. Abnormal left ventricularfillinv an early finding in mild to moderate systemichypertension. Am.J. CsrdioL, 1984;53:120-6.
14. Topol, E.J., Traill,T.A., Fortuin, N.J. Hypertensive hypertrophic cardiorayopathy of the elderly. N.Engl.J.Med., 1985;312:277-83.
15. Tarazi, R.C., Miller, A, Frohlich, ED., Dustan, H.P. Electrocardiographic changes reflecting left atrial abnormality in hypertension. Circulation, 1966;34:818-22.
16. Tarazi, R.C., Frohlich, D.E., Duatan, H.P. Left atrial abnormality and ventricular pre-ejection period in hypertension. Dis. Chest,, 1969;55:214-18.
17. Frohlich, ED., Tarazi, R.C., Dustan, H.P. Clinical physiological correlations in the development of hypertensive heart disease, Circulation, 1971;44:448-55.
18. Drcslinski, O.R, Frohlich, ED., Dunn, F.G., Messerli, F.H., Suarez, D.H., Reisin, 8. Echocardiographic diastolicventrieular abnormality in hypertensive heartdisesse: atrial emptying index Am.J.Cardiol., 1981;47:1087-90.
19. Devereux, RB., Pickering T.G., Harshfield, G.A et al. Left ventricular bypertrophyin patients with hypertension: importance of blood pressure responae to regularly recurring stress Circulation, 1983;68:470-76.
20. Drayer, 3.1., Weber, M.A DeYoung, J.L BP ass determinant ofcardiac leftvcntricular muscle mass. Arcls Intern. Med., 1983;143:90-92.
21. ken, J.F., Hakki, AU., Kotler, M.N., Iskandrian, AS. Exercise systolic blood pressure: a powerful determinant of increased left ventricular mass in patientswith hypertension. J.Am.Coll.Cardiol., 1985;5:1224-31.
22. Frohlich, ED., Tarazi, RC. is arterial pressure the sole factor responsible for hypertensive cardiac hypertrophy? Am.J. Cardiol., 1979;44:959-63.
23. Frohlich, ED. Uemodynamics and other determinants in the development of left ventricular hypertrophy. Fed. Proc., 1983;42:2709-15.
24. ldem. The first Irvine H. Page lecture: the mosaic of hypertension: past, present and future.). Hypertens. SuppL, 1988;6:SuppL4:S-2-S-11.
25. Walford, G.D. Spurgeon, H.A, Lakatta, E.G. Diminished cardiac hypertrophy and muscle performance in older compared with younger adult rats with chronic atrioventricular block. Circ. Res., 1988;63:502-11.
26. Isoyama, S., Wein, J.Y., lzumo, S., Fort. P., Schocn, F.)., Grossmsn, W. Effectof age on the development cardiac hypertrophy produced by aortic constriction in the rat. Circ.Res., 1987;61:337-45.
27. 1988 joint National Committee. The 1988 report of the Joint National Committee on Detection: evaluation and treatment of high blood pressure. Arch. Intern. Mcd., 1988:148:1023-38.
28. Kannel, W.B., Abbott, R.D. A prognostic comparison of asymptomatie leftventricular hypertrophy and unrecognized myocardial infarction; the Framingham Study. Am.Hesrt 3., 1986;111:391-97.
29. ClassIc, P.N., Devereux, RB., Alonso, DR., Campo, B., Kligfield, P. Improved sex-specific criteria of left ventricular hypertrophy for clinical and compulor interpretation of electrocardiograms: validation with autopsy Findings. Circulation, 1987;75:565-72.
30. Dunn, P.C., Chandrarstns, P., deCarvalho, J.G., Basta, LL., Frohlich, ED. Pathophysiologicaasessmentof hypertensive hesrtdiseaaewith echocardiography. Am.J.Cardiol,, 1977;39:789-95.

31. Devereux, RB., Casale, P.N., Kligfield, P. et aL Performance of primacy and derived M-mode echocardiographic messurements fordetection of leftventricular hypertrophy in necropsied subjects and in patients with systemic hypertension, mitral regurgitation and dilated cardiomyopsthy. AmA. Cardiol., 1986;57:1388-93.
32. Devereux, R.B. Reichek, N. Echocardiogrsphic determinationof leftventriculsr massin man: snatomicvalidation of the method. Circulation, 1977:55:613-18.
33. Stainm, R.B., Carabello, B.A, Mayers, DL, Martin, R.P. Two- dimensional echocardiographicmeasuremcntofleftventricularcjection fraction:prospectivesnalysisofwhat constitutes an adequate determination, Am.Heart 3., 1982; 104: 136-44.
34. Klcpzig, M., Eisentohr, H., Steindl, 3., Schmiebusch, H., Strauer, B.E. Media hypertrophy in hypertensive coronary resistance vessels. 3. Cardiovssc. Pharmacol., 1987; 10:Suppl. 6:S97 S102,
35. Brush, i.E., Jr., Cannon, R.O. Ill, Schcnke, W.H. et al. Angina due to coronary microvascular disease in hypertensive patients without left ventricular hypertrophy. N.Engl.J.Med., 1988;319:1302-7.
36. Keller, AM., Peshock, R.M., Malloy. CR. etaL In vivo measurement ofmyocardial mats using nuclearmagnetic resonance imaging, J.Am.Coll.Cardiol., i986;8: 113-17.
37. Feiring, A)., Rumberger, J.A Reiter, S.J. etai Determination ofleftventricutar mass in dogs with rapid-acquisition cardiac computer tomographie scanning. Circulation, 1985;72:1355-64.
38. Reiter, Si., Rumberger, 3.A, Feiring, A.J., Stanford, W., Marcus, M.I. Precision of measurements of right and left ventricular volume by cine computed tomography. Circulation, 1986;74:890-900.
40. Frohlich, E.D., Horinaka, S. Cardiacand sorticeffects ofangiotensin convertingenzyme inhibitors. Hypertension, 1991;18:Suppl. 11:11-2-11-17.
41. Khairallah, P.A., Robertson, AL, Davila, D. Effects of angiotenain II on DNA, RNA and protein synthesis. ln:Geness 3. KoiwE. eds. Hypertension, 1972, New York: Springer-Veriag. 1972:212-20.
42. Starksen, N.F,, Simpson, P.C., Bishopric, N. et aL Cardiac myocyte hypertrophy is associated with c-mye protoooncogene expression. Proc Natl. Acad. Sci, USA, 1986;83:8348-50.