Therapeutical Approach of Arterial Hypertension

Sur Genel1*, Sur Lucia1, Bulata Bogdan2, Sur Maria1, Kudor-Szabadi Liana1, Sur Daniel1 and Samasca Gabriel2*

1University of Medicine and Pharmacy Iuliu Hatieganu, Cluj-Napoca, Romania
2Emergency Clinical Hospital for Children, Cluj-Napoca, Romania

Background

Arterial hypertension treatment is based upon all available evidence about the benefits of antihypertensive therapy and of different classes of therapeutic agents [1].

Applying antihypertensive treatment induces a significant decrease of cardiovascular morbidity and mortality. Treatment is associated with a major decrease of 30-40% in the risk of stroke and also, with a decrease of coronary events of 20% and an important decrease in heart failure [2-5].

Content

When starting arterial hypertension therapy one should always consider blood pressure values and cardiovascular risk. All patients with arterial hypertension stage 2 or 3 must undergo treatment. There is still a cautious attitude over the benefits of treatment applied for hypertension stage. First measure for hypertension of all stages (1 to 3) should be lifestyle modification and based on arterial pressure values and cardiovascular risk, pharmacotherapy should follow. The main goal of treatment is to reduce long term cardiovascular risk. These peculiar effects have lower benefits than the protective dominant effect induced by arterial pressure decrease [6-9].

Several meta-analyses revealed that drug classes have different effects on heart failure occurrence. Diuretics and beta adrenergic antagonists, angiotensin converting enzyme inhibitors and angiotensin receptors blockers have a better action in preventing heart failure than calcium channel blockers, independently of blood pressure values [10-12].

Randomized trials that studied target organ damage have an increased value for medical practice. Studies on the effect of different drugs on ventricular hypertrophy showed that a decrease in blood pressure values generates a slower rate for the augmentation of ventricular mass, no matter the type of pharmacologic agents used. There was noticed the same efficiency for the angiotensin converting enzyme inhibitors, angiotensin receptors blockers and calcium channel blockers [13-17]. On concern to diuretics, studies ascertained indapamide’s efficiency [18].

The best option for first line antihypertensive therapy seems to be: beta adrenergic antagonists, diuretics, calcium channel blockers, angiotensin converting enzyme inhibitors and angiotensin receptors blockers. These drugs may be used as monotherapy or as a combination. Each of the drug classes mentioned above may have specific characteristics, advantages and limits and they have to be adjusted to patient necessities. Beta-blockers offer protection against undesired coronary events and mortality, but are less efficient for protection against stroke-they are indicated in cases of: angina pectoris, heart failure and recent myocardial infarction. As side effects must be mentioned: potential weight gain, side effects on lipids metabolism and increase of diabetes type 2 prevalence. In conclusion beta-blockers should not be administrated to hypertensive patients with multiple metabolic risk factors as metabolic syndrome with abdominal obesity, altered glycemia values, altered glucose tolerance. Thiazides diuretics also have a dismetabolic and diabetogenic effects. In exchange, vasodilator beta-blockers as carvedilol and nebivolol have a decreased or absent dismetabolic and diabetogenic action [19-23].

Antihypertensive drug classes have a different action on target organs damage. Angiotensin converting enzyme inhibitors and angiotensin receptors blockers have a high efficiency in reducing left ventricular hypertrophy; they also have good results in reducing microalbuminuria, proteinuria, in preserving renal function and preventing chronic renal disease [24-27]. Calcium channel blockers are efficient in reducing left ventricular hypertrophy and in slowing down coronary atherosclerosis progression. Studies revealed that, generally, therapy using a combination of drugs have a higher efficiency [28,29].

Drug classes have different types and frequency of side effects, and patients have a different behavior regarding side effects. Antihypertensive medication may influence distinguishly risk factors, target organs damage, specific cardiovascular events and provide specific protective action for different groups of patients. For this reason selection of drug combination should be based on certain circumstances. When choosing or avoiding a medication one should always take in consideration a series of factors: the experience (positive or negative) that a patient has with that medicine including the decrease of blood pressure values and side effects, the action of medication on cardiovascular risk factors (adjusted to each patient), target organs damage (cardiovascular disease, renal disease or diabetes mellitus should benefit of drugs that already proved to be efficient for this pathology), presence of some affections may limit the use of some drugs, avoiding interaction with other treatment that the patient follows and the cost-benefit rate [4,6,8,30].

Other principles that may be useful when starting antihypertensive therapy are: cost-benefit considerations should not be decisive for medication choice, long time action drugs should be preferred (they effectively decrease arterial pressure values), a simple therapy may increase patients compliance, but we must take in consideration how we may obtain a 24 hour efficient control.

One must always pay attention on avoiding or limiting side effects: for calcium channel antagonists , thiazides diuretics and beta-blockers is the dose dependence.

*Corresponding author: Gabriel Samaşca, Department of Immunology, Croitorilor Street, 19-21 No, Iuliu Hatieganu University of Medicine and Pharmacy Cluj-Napoca, Romania, E-mail: Gabriel.Samasca@umfcluj.ro

Received November 01, 2011; Accepted November 05, 2011; Published November 07, 2011

Citation: Genel S, Lucia S, Bogdan B, Maria S, Liana KS, et al. (2011) Therapeutical Approach of Arterial Hypertension. Pharm Anal Acta 2:108e. doi:10.4172/2153-2435.1000108e

Copyright: © 2011 Genel S, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.
When starting antihypertensive treatment: first it is indicated to use a single pharmacologic agent that must be administered in a low dose; if arterial pressure is not controlled we must increase the dose or change the drug class. A combination of 2 or more drugs is used in most studies; when combined the first drug as well as second one can be administered in lower doses, so that side effects are avoided and, in the same time, a good control of hypertension is achieved.

Indication of using different classes of antihypertensive therapy

Angiotensin converting enzyme inhibitors: heart failure, left ventricular dysfunction, after myocardial infarction, diabetic nephropathy, left ventricular hypertrophy, carotids atherosclerosis, microalbuminuria, metabolic syndrome, atrial fibrillation, angina pectoris, left ventricular hypertrophy, coronary atherosclerosis, pregnancy, supraventricular tachycardia.

β-blockers: Angina pectoris, after myocardial infarction, heart failure, tachyarrhythmia, glaucoma, pregnancy.

Thiazides diuretics: isolated systolic hypertension, heart failure.

Loop diuretics: heart failure, terminal renal/kidney disease.

A combination of drugs may be used: if drugs have different and complementary mechanism of action, there is efficiency on lowering blood pressure values, tolerance profile is good.

Possible combinations:

- Thiazides diuretic + angiotensin converting enzyme inhibitor
- Thiazides diuretic + angiotensin receptors blocker
- Calcium channel antagonist + angiotensin converting enzyme inhibitor
- Calcium channel antagonist + angiotensin receptors blocker
- Calcium channel antagonist + thiazides diuretic
- β-blocker + calcium channel antagonist [31-36].

References

1. Flack JM (1999) Optimal blood pressure on antihypertensive medication. Curr Hypertens Rep 1: 381-386.
2. Goldstein LB, Bushnell CD, Adams RA, Appel LJ, Braun LT, et al. (2011) Guidelines for the Primary Prevention of Stroke. A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke 42: 517-584.
3. Paker M, Cohn JN (1999) Consensus recommendations for the management of chronic heart failure. Am J Cardiology 83: 1-20.
4. Mancia G (2006) Role of outcome trials in providing information on antihypertensive treatment: importance and limitations. Am J Hypertens 19: 1-7.
5. Conroy RM, Pyorala K, Fitzgerald AP, Sans S, Menotti A, et al. (2003) Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. Eur Heart J 24: 987-100.
6. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, et al. (2003) The seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure: the JNC 7 report. JAMA 289: 2560-2572.
7. De Backer G, Ambrosioni E, Borch-Johnsen K, Brotons C, Cifkova R, et al. (2003) European guidelines on cardiovascular disease prevention in clinical practice. Third Joint Task Force of European and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. Eur Heart J 24: 1601-1610.
8. Hajjar I, Kotchen TA (2003) Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988-2000. JAMA 290: 199-206.
9. European Society of Hypertension – European Society of Cardiology (2003) Guidelines for the management of arterial hypertension. J Hypertens 21: 1011-1053.
10. Hansson L, Lindholm LH, Niskanen L (1999) Effect of angiotensin converting enzyme inhibition compared with conventional therapy on cardiovascular morbidity and mortality in hypertension, Captopril project. Lancet 353: 611-616.
11. Ramhi TM (2000) Beta-blocker therapy for chronic heart failure. Am Fam Physician 62: 2267-2274.
12. Lee DS, Vasan RS (2006) Goals and guidelines for treating hypertension in a patient with heart failure. Curr Treat Options Cardiovasc Med 8: 334-344.
13. Hansson L, Hedner T, Lund-Johansen P, Kjeldsen SE, Lindholm LH, et al. (2000) Randomised trial of effects of calcium antagonists compared with diuretics and beta-blockers on cardiovascular morbidity and mortality in hypertension: the Nordic Diltiazem (NORDIL) study. Lancet 356: 359-365.
14. Verdecchia P, Reboldi G, Angeli F, Gattobigio R, Bertiovoglio M, et al. (2005) Angiotensin-converting enzyme inhibitors and calcium channel blockers for coronary heart disease and stroke prevention. Hypertension 46: 386-392.
15. Neal B, MacMahon S, Chapman N, Blood Pressure Lowering Treatment Trialists' Collaboration (2000) Effects of ACE inhibitors, calcium antagonists, and other blood-pressure-lowering drugs: results of prospectively designed overviews of randomised trials. Blood Pressure Lowering Treatment Trialists' Collaboration. Lancet 356: 1995-1964.
16. Morgan T, Lauri J, Bertram D, Anderson A (2004) Effect of different antihypertensive drug classes on central aortic pressure. Am J Hypertens 17: 118-123.
17. Muijesan ML, Salvetti M, Montedoro C, Bonzi B, Paini A, et al. (2004) Left ventricular concentric geometry during treatment adversely affects cardiovascular prognosis in hypertensive patients. Hypertension 43: 731-738.
18. Goss P, Sheridan DJ, Zannad F, Dubourg O, Gueret P, et al. (2000) Regression of left ventricular hypertrophy in hypertensive patients treated with indapamide SR 1.5 mg versus enalapril 20 mg: the LIVE study. J Hypertens: 1485-1475.
19. Messerli F, Bangalore S, Ruschitzka F (2009) Angiotensin receptor blockers: baseline therapy in hypertension? Eur Heart J 30: 2427-2430.
20. Kostis JB, Packer M, Black HR, Schmieder R, Henry D, et al. (2004) Omapatrilat and enalapril in patients with hypertension: the Omapatrilat Cardiovascular Treatment vs. Enalapril (OCTAVE) trial. Am J Hypertens 17: 103-111.
21. Yusuf S, Sleight P, Pogue J, Bosch J, Davies R, et al. (2000) Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. N Engl J Med 342: 145-153.
22. Wong ND, Plo JR, Franklin SS, L' Italien GJ, Kamath TV, et al. (2003) Preventing coronary events by optimal control of blood pressure and lipids in patients with the metabolic syndrome. Am J Cardio 91: 1421-1426.
23. Narkiewicz K (2006) Diagnosis and management of hypertension in obesity. Obes Rev 7: 155-162.
24. Olsen MH, Wachtell K, Ibsen H, Lindholm LH, Dahlöf B, et al. (2006) Reduction in albuminuria and in electrocardiographic left ventricular hypertrophy independently improve prognosis in hypertension: the LIFE study. J Hypertens 24: 775-781.
25. Bianchi S, Bigazzi R, Campese VM (1999) Microalbuminuria in essential hypertension: significance, pathophysiology, and therapeutic implications. Am J Kidney Dis 34: 973-995.
26. Casas JP, Chua W, Loukogeorgakis S, Vallance P, Smeeth L, et al. (2005) Effect of inhibitors of the renin-angiotensin system and other antihypertensive drugs on renal outcomes: systematic review and meta-analysis. Lancet 366: 2026-2033.

27. Volpe M, Mancia G, Trimarco B. (2006) Angiotensin receptor blockers and myocardial infarction: the importance of dosage. J Hypertens 24: 1681-1682.

28. Lubsen J, Wagener G, Kirwan BA, de Brouwer S, Poole-Wilson PA, et al. (2005) Effect of long-acting nifedipine on mortality and cardiovascular morbidity in patients with symptomatic stable angina and hypertension: the ACTION trial. J Hypertens 23: 641-648.

29. The ALLHAT Officers, Coordinators for the ALLHAT Collaborative Research Group (2002) Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker vs diuretic: The Antihypertensive and Lipid-Lowering treatment to prevent Heart Attack Trial (ALLHAT). JAMA 288: 2981-2987.

30. Sur G, Sur M, Kudor-Szabadi L (2011) Difficulties in Achieving Arterial Hypertension control. Maedica 6: 114-119.

31. Goldstein S, Fagerberg B, Hjalmarson A, Kjekshus J, Waagstein F, et al. (2001) Metoprolol controlled release in patients with severe heart failure. J Am College of Cardiology 38: 932-938.

32. Pitt B, Zannard F, Remme WJ, Cody R, Castaigne A, et al. (1999) The effect of spironolactone on morbidity and mortality in patients with severe heart failure. New England J Med 341: 709-717.

33. Prichard BJ (2001) Beta-blockers, when are they really indicated? Clin Bas Cardiol 4: 3-25.

34. Dhakam Z, McEniery CM, Yasmin, Cockcroft JR, Brown MJ, et al. (2006) Atenolol and eprosartan: differential effects on central blood pressure and aortic pulse wave velocity. Am J Hypertens 19: 214-219.

35. Dahlof B, Devereux RB, Kjeldsen SE, Julius S, Bevers G, et al. (2002) Cardiovascular morbidity and mortality in the Losartan Intervention for Endpoint reduction in hypertension study (LIFE): a randomised trial against atenolol. Lancet 359: 995-1003.

36. Black HR, Elliott WJ, Grandits G, Grambsch P, Lucente T, et al. (2003) Principal results of the Controlled Onset Verapamil Investigation of Cardiovascular Endpoints (CONVINCE) trial. JAMA 289: 2073-2082.