

HOW TO TREAT HYPERTENSION IN BLACKS: REVIEW OF THE EVIDENCE

Key words: African-Americans, antihypertensive treatment, arterial hypertension, hypertensive blacks, hypertensive complications, sub-Saharan Africans

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ABSTRACT

Presentation, response to therapy, and clinical outcome differ according to race for patients with hypertension.

Black patients have a higher prevalence and earlier onset of hypertension than other ethnic groups, with poorer prognosis than white patients. Blacks are more likely to be salt-sensitive, and to have a low plasma renin activity than are whites. They are at much greater risk of developing cardiovascular and renal complications. Despite many advances in the understanding and treatment of cardiovascular diseases, black patients continue to have increased morbidity and mortality from the end-organ complications of hypertension. The explanations for these observations remain incompletely understood, but genetic differences,

added to socio-economic and environmental factors, have been proposed to explain this disparity.

The first therapeutic approach is to decrease salt and increase potassium intakes. Diuretics (thiazides and potassium-sparing agents) and calcium channel blockers constitute the first antihypertensive drug choices. The angiotensin-converting-enzyme inhibitors, the angiotensin II receptor blockers and beta-blockers appear to be less effective in blacks with regard to uncomplicated hypertension, especially in older people, but addition of a small dose of diuretic improves their efficacy. These combinations are preferred among patients with chronic kidney disease or heart failure.

The goal for blood pressure target is the same in blacks as it is in whites, being a blood pressure of less than 140/90 mmHg in uncomplicated hypertension and less than 130/80 mmHg in patients with diabetes mellitus or chronic kidney disease.

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INTRODUCTION

It has long been recognised that high blood pressure (BP) in people of African descent and of black race is more frequent, more severe, more complicated, and more difficult to manage than it is in white people (1,2).

The reasons for this are incompletely understood, and probably involve environmental and genetic factors (3) as well as socio-economic differences.

We have much information in the hypertensive field that originates from the United States (US) where there are over 30 million people of African origin. We

will summarize data concerning hypertensive US black people. Whenever it has been possible, we have cited results for other black populations especially from sub-Saharan Africa (SSA).

The goal of this review is to provide an update of the current knowledge on hypertension (HTN) in black people.

EPIDEMIOLOGIC CHARACTERISTICS OF HTN IN BLACK RACE

The prevalence of HTN in African-Americans is among the highest in the world at nearly 40 percent. Compared to the general population, this HTN has an earlier onset, and is accompanied by a 80-percent higher stroke mortality rate, a 50-percent higher heart disease mortality rate, and a 32-percent greater rate of HTN-related end-stage renal disease (ESRD) (4).

In SSA, the prevalence of HTN and cardiovascular disease is also increasing rapidly (5-7), particularly in urban societies (8). In these developing countries, urban populations have higher prevalence of HTN (20 to 30%) compared with their rural counterparts (8 to 10%) in almost all studies (9).

In addition to this high prevalence of HTN, black people have also a lower awareness of their condition than do white people (10).

PATHOPHYSIOLOGIC CHARACTERISTICS

This greater prevalence and greater rate of end-organ damage has prompted investigations to explain this disparity. Several factors have been identified (11).

Sodium Sensitivity and Sodium Retention

The relationship between dietary salt intake and the development of HTN has been well studied, especially in view of the increased rate of cardiovascular disease in African-Americans (12). Indeed, the latter have consistently been shown to have a greater frequency of BP sensitivity to salt loading than do whites (13). Yet, the dietary intake of salt does not differ among African-Americans when compared with other ethnic groups in the USA (14). This suggests that the increased prevalence of salt-sensitive HTN in African-Americans is secondary to an impaired ability to excrete salt, and that the higher BP is a physiologic response to decreased natriuresis. This is the conclusion of Luft et al. (15) who

demonstrated slower salt excretion in hypertensive African-Americans after a dietary salt load.

This could be due perhaps to upward resetting of the set point for tubuloglomerular feedback (16).

Black individuals were also reported to have higher vasopressin levels than whites (17). There is a possible contribution of vasopressin to high BP by its antidiuretic effects, mediated by the renal V2 receptors, located in the thick ascending limb of Henle's loops and the collecting ducts. Moreover, the V1a receptor, expressed in the macula densa could influence the renin secretion (18).

Another possible mechanism of salt sensitivity could be an abnormality at the chloride channel ClC-Kb level. This channel expressed in the basolateral cell membrane of the distal nephron participates in renal NaCl reabsorption. The prevalence of the mutant allele has been shown significantly higher in an African population from Ghana (22%) than in whites (12%) (19).

Renin-Angiotensin-Aldosterone System (RAAS)

This system has been well examined in the African-American population.

The first study to compare the RAAS in black and white hypertensive people was done in 1964. It reported that 30% of American blacks with HTN, had no detectable plasma renin activity (PRA). Renin may still be suppressed after the stimulus of a low salt diet and treatment with diuretics, even in normotensive black people (20-22).

Significant age-related differences in PRA have been demonstrated. Brunner and colleagues demonstrated that 70% to 80% of African-Americans aged 20 to 30 years have PRA levels within the normal range, whereas the majority of those aged 50 to 60 years have lower than normal PRA levels (23).

Moreover, Laragh et al. noted that younger patients responded better to beta-blockers, while older patients responded better to diuretics, and they linked this observation to the decline in plasma renin with age (24).

In a study that is consistent with a lesser activity of the renin-angiotensin system in blacks, Pratt and co-workers (25) reported that African-American children already secrete 40% less aldosterone than do caucasian children. Further, Fisher and colleagues (26) demonstrated that plasma aldosterone responses to stimulation were significantly lower in hypertensive African-Americans in comparison with hypertensive caucasians. Regardless of the mechanism, the lower degree of PRA is a marker in hypertensive African-

Americans. Low renin and angiotensin II levels have implications for the responsiveness to various classes of antihypertensive agents (Table 1).

Renin concentrations can also determine how far blood pressure falls on salt restriction (27).

Possible other hemodynamic and renal factors

Better treatment for HTN has led to a reduction in the occurrence of stroke and heart failure in both whites and blacks. Despite this clear cardiovascular benefit, there has been a continuing rise in the incidence of ESRD in older African-American patients disproportionately higher than among whites (28,29). This is presumably due to a renal consequence of HTN called the hypertensive nephrosclerosis (HN).

It remains uncertain why blacks might be particularly susceptible to renal disease. One possibility may relate to the autoregulation of renal blood flow. Parmer et al. (30) found that, when white and black patients with uncomplicated essential HTN were placed on a low salt diet and then changed to free salt intake (mean change in sodium excretion from 40 to 155 mEq/day), the high salt diet was associated with a rise in glomerular filtration rate of 17 ml/min in blacks while there was no change in whites. This glomerular hyperfiltration could probably be associated with intraglomerular HTN, a potential risk factor for progressive renal disease. One causal factor could be maternal malnutrition leading to low birth weight and a reduction in nephron number, in turn leading to obligatory hyperfiltration, i.e. higher single-nephron sodium load, in response to higher sodium intake.

SOCIO-ECONOMIC OR ENVIRONMENTAL STATUS?

The role of socio-economic factors in the racial differences in HTN has been studied (31) and shown to be relevant to HTN in African-American adults (32).

African-American youth living in economically depressed inner city areas have higher BP than do black youth living in more affluent areas (33).

Beside socio-economic status, per se, obesity could play a role.

Regarding body weight, there is a gradient of risk according to geography across the African diaspora with standardized prevalences of HTN of 14 percent in West Africa, 26 percent in the Caribbean, and 33 percent in the US (34). These differences in risk can be explained by differences in body mass index (35).

The problem of obesity is particularly common in black women. In the Hypertension Detection and Follow-up Program (HDFP), hypertensive African-Americans had a mean weight 20-25% above desired level (36). This pattern parallels the gradient in known risk factors, with obesity alone accounting for a third of the excess of HTN in the US compared to Africa.

COMPLICATIONS DUE TO HTN

Black patients with essential HTN are at much greater risk of developing cardiovascular and renal complications. One contributing factor to these risks is a lesser decline in nocturnal BP in blacks, which

Table 1 : Two types of HTN: the role of the RAAS

Type 1 HTN (vasoconstrictor, high renin)	Type 2 HTN (sodium dependent, low renin)
Renin secretion inappropriately high for blood pressure.	Renin secretion suppressed by kidney abnormalities.
Elimination of salt load exaggerated.	Role of macula densa in excessive sodium reabsorption.
Common in young white people.	Common in middle age or old or black people.
Better response to drugs which block the RAAS (ACEIs, ARBs, and beta-blockers).	Better response to drugs, which are natriuretic and/or vasodilating (CCBs and diuretics).
ACEIs: angiotensin converting enzyme inhibitors ARBs: angiotensin receptor blockers BBs: beta-blockers CCBs: calcium channel blockers	

increases the total BP load. For instance, one study evaluated 62 black and 72 white hypertensives who were matched for the same daytime BP by ambulatory monitoring (37). The mean BP between 1 AM and 5 AM was 7 mmHg higher in the African-American patients versus the European American ones.

Coronary disease and heart failure

Although the overall prevalence of coronary heart disease (CHD) may be lower among US blacks than non-blacks, mortality rates from CHD are similar, probably reflecting more severe disease and/or less adequate health care (38). The major contribution of HTN to heart disease usually involves progressive left ventricular hypertrophy (LVH). By electrocardiography, African-American men and women enrolled in the HDFP had 3-4 times more LVH than did equally hypertensive non-black men and women (39). Heart failure in African-Americans is less often due to associated atherosclerotic coronary artery disease but is more likely associated with HTN leading to increased rates of hospitalization and mortality. That said, the incidence of myocardial infarction in Africans throughout SSA is rising (40) and HTN is the strongest among 6 risk factors, with an astounding odds ratio of 6.99 (41).

Cerebrovascular disease

Another major consequence of HTN is stroke and it is more frequent and more severe in blacks. At the ages of 35-54 years, African-Americans have an approximately four times greater stroke rate than do white Americans (42). Stroke mortality among African-Americans was three-to fourfold higher than for non-blacks in those less than 65 years old (43). In SSA, stroke is also on the increase in direct relation to "westernization", and increased rates of HTN and diabetes. In a study of a South African semirural community, the prevalence of stroke was relatively high, with the rate of stroke disability approaching that in high-income countries (44). Of note, a large percentage, between 30% and 50%, of stroke mortality occurs in those inadequately treated for HTN (45). Yet truly rural populations are still relatively protected (45-47).

Concerning the kind of stroke noted in the Black population, all forms of cerebrovascular disease are more frequent in American blacks, with even greater differences in those strokes that are more tightly connected with HTN, i.e. subarachnoid and intracerebral hemorrhages (48). On the other hand, blacks have less

extracranial carotid and vertebral occlusive vascular disease (49).

Kidney damage leading to ESRD is more frequent in blacks

Although black people make up 13% of the US population, they account for about 30% of the patients on renal replacement therapy, according to the 2004 US Renal Data System (USRDS). With perhaps the exception of atherosclerotic renal disease, black people are at an increased risk of renal diseases from any cause. However, in black americans, HTN occurs earlier, is more severe, and more often associated with ESRD, being its attributed cause in about 37% in black patients versus 26% in white patients.

Other factors that might account for racial disparity in ESRD attributed to HTN include a lack of access to medical care, low socio-economic status (50), low education level, alcohol and drug abuse (51), genetic predisposition, and nephron endowment (52).

The peak age for the development of ESRD in white patients is 65 years and older, while this peak comes at an earlier age (45-65 years) in black people.

As reported earlier, glomerular hyperfiltration could be one of these explanations when high salt intake is combined with a low nephron number (30).

Several renal, hormonal, and physiologic differences, including increased BP, sensitivity to a high-salt diet, increased renal vascular resistance, and decreased renal blood flow, have been suggested as an explanation for the susceptibility of black people to renal HN. As reported previously, a decreased nephron number secondary to low birth weight, which is more common in black people, has also been proposed as an explanation for the increased risk for progressive renal failure in this patient population. However, Li et al. found that the low birth weight was associated with CKD only in men and there was no significant interaction between birth weight and race for either gender (53).

THERAPEUTIC CONSEQUENCES

NON-PHARMACOLOGICAL APPROACH TO TREAT HTN IN BLACKS

Evidence-based, non-pharmacologic therapy for HTN includes dietary salt restriction, higher potassium intake, weight loss in obese patients, avoidance of excess alcohol, and exercise (54).

Available data suggest that non pharmacologic therapies have a similar benefit in black patients as they do in whites (55,56), especially restriction of sodium intake.

The following reports illustrate some of the benefits of non-drug treatment:

One study randomized 46 African American men with severe untreated HTN to antihypertensive therapy alone or with added thrice weekly exercise (57). The exercise group showed a further 5mmHg reduction in BP and significant regression of LVH, which were not seen in the control group.

In another trial of 40 American blacks, four weeks of a low sodium diet was associated with a reduction in the BP from 159/101 to 151/98 mmHg (58).

PHARMACOLOGICAL APPROACH

Considerations regarding the use of antihypertensive drug classes in African American patients with high BP are outlined in Table 2. We have divided this chapter into two categories: uncomplicated and complicated HTN.

TREATMENT OF UNCOMPLICATED ESSENTIAL HTN IN BLACKS

First choice of antihypertensive drugs

Overall, the choice of antihypertensive drugs in black patients is similar to that in other groups, but as reminded by Lemogoum et al. (56), it should preferentially begin with low dose thiazide therapy.

a) Thiazide diuretics

A number of comparative studies have demonstrated that black patients generally respond well to diuretic therapy, with the fall in BP exceeding that of monotherapy with an ACEI or a beta blocker (59-61).

The ALLHAT trial of US patients with mild HTN showed that low dose chlorthalidone (12.5 to 25 mg/day) was the first-line drug to be preferred in almost all patients because of fewer cardiovascular complications compared to amlodipine and lisinopril (62). In this study (63), doxazosin (an alpha blocker) was harmful.

Subset analysis showed that these benefits were as or more prominent in black patients. Further prespecified analyses on subgroup of blacks also reported specific benefits with the diuretic (64), especially for stroke and combined outcomes.

b) Potassium –sparing diuretics

Some black patients, particularly, those with low PRA, appear to have increased activity of the aldosterone-sensitive epithelial sodium channel (ENaC) in the collecting tubule (65). This suggests that aldosterone-antagonism or ENaC blockade may provide additional antihypertensive benefits in blacks.

The antihypertensive effects of amiloride, spironolactone and their combination were therefore examined in 98 US black hypertensive subjects with low PRA who were already receiving a diuretic (thiazide or loop diuretic) and a calcium channel blocker (66). Concomitant ACEI use was not allowed given concerns regarding hyperkalemia. Amiloride, spironolac-

Table 2 : Data regarding use of antihypertensive classes in African-American patients with high blood pressure

Class	Comments	References
Diuretics		
Thiazide diuretics	Chlorthalidone demonstrated cardiovascular benefits in ALLHAT.	(62, 89)
Potassium-sparing diuretics	Evidence for BP reduction with amiloride, spironolactone. Recent evidence for BP-lowering efficacy with a selective aldosterone blocker (eplerenone).	(66, 67)
Beta-blockers	Evidence of less BP-lowering efficacy as mono-therapy versus white patients. Evidence of benefits in post-myocardial infarction.	(78,82,89,90)
Alpha-Antagonists	Negative data reported in ALLHAT for doxazosin	(63,89,90,)
CCBs	Evidence of efficacy and benefits. But, AASK found less renoprotective effect than ACE Inhibitors in hypertensive renal insufficiency.	(68,69,84,89,90)
ACE Inhibitors	Some evidence of less BP-lowering efficacy as monotherapy versus in white patients. Some evidence of more cough and angioedema compared with white patients. Strong evidence of target-organ protection (AASK).	(73-75,84)
ARBs	Small studies show BP- lowering efficacy particularly in combination with hydrochlorothiazide.	(71)

Abbreviations: AASK, African-American Study of Kidney Disease and Hypertension; ACE, angiotensin-converting enzyme; ALLHAT, Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial; ARB, angiotensin II receptor blocker; BP, blood pressure; CCB, calcium channel blocker; RAS, renin-angiotensin system.

tone and their combination successfully reduced BP by 12/4, 7/3 and 14/2 mmHg, respectively, after nine weeks of treatment. According to the authors, the slight benefit of combination therapy with spironolactone and amiloride does not justify the increased risk of hyperkalemia which developed only in patients on both diuretics. Further data are required to determine whether the addition of amiloride or spironolactone to an antihypertensive regimen yields superior outcomes compared to more traditional strategies. Flack et al. showed that eplerenone, an aldosterone antagonist, was successful in hypertensive black patients compared to losartan (67). This again highly suggests the role of salt in the physiopathogenesis of HTN in blacks.

c) Calcium channel blockers (CCB)

Black patients are also more responsive to CCB than to monotherapy with ACEIs or beta blockers (68). In a randomized, open-label comparative study performed in 409 South African black men and women, the dihydropyridine CCB (nifedipine GITS) provided a statistically greater reduction in BP and control rate of HTN than did a diuretic (hydrochlorothiazide), a non dihydropyridine CCB (sustained-release verapamil), or an ACEI (enalapril) (68).

One very informative trial in the US evaluated the efficacy of monotherapy with six different antihypertensive drugs in almost 1300 men with initial diastolic blood pressures between 95 and 109 mmHg; a successful response was defined as a fall in diastolic pressure below 90 mmHg at the end of the tritration phase and below 95 mmHg at one year (61). The response rates in older blacks were 85 percent for diltiazem, 64 percent for hydrochlorothiazide, and only 33 percent for captopril. Although, other studies have not documented such a prominent preferential response to a CCB (69), this relative advantage may apply only to the use of CCB as monotherapy. Numerous studies have shown amlodipine to be effective for the treatment of HTN in black and non black patients. In comparative studies, amlodipine and nifedipine demonstrated similar antihypertensive efficacy in black patients (70). Moreover, in a randomized, double-blind, parallel-group, multicenter study in black patients with stage 1 or 2 HTN, amlodipine monotherapy at 10 mg had efficacy similar to that of a combination of valsartan/hydrochlorothiazide 160 mg/12.5 mg, but valsartan/hydrochlorothiazide was better tolerated (71). In a more recently-reported study and based on both clin-

ic BP measurements and ambulatory BP monitoring (ABPM) data, amlodipine and valsartan produced similar reductions in BP in African-American patients with uncomplicated HTN (72).

d) ACE inhibitors (ACEIs) and Angiotensin II receptor blockers (ARBs)

Although ACEIs are established as a major drug class in the treatment of HTN, the perception in clinical practice is that ACEIs are less effective as monotherapy in African-Americans compared to whites because of low renin levels and high degree of salt sensitivity.

Indeed, black patients do not respond well to ACEIs when given as monotherapy (73) and can develop cough or angioedema more frequently than in whites (74,75). One explanation could be the higher dose of ACEI used to obtain the target of BP in blacks.

However, these drugs are effective when given in combination with a thiazide diuretic (76). In one study, for example, enalapril alone produced only a minimal antihypertensive effect, whereas combination therapy with 12.5 mg of hydrochlorothiazide lowered the BP from 157/101 to 132/86 mmHg. Another study demonstrated similar BP lowering, as reported above, with combination valsartan/hydrochlorothiazide versus amlodipine (16/10 and 15/9 mmHg, respectively) (71).

The issue of why black patients respond poorly to monotherapy with ACEIs was addressed by comparing the response to different doses of an ACEI, trandolapril of 91 black patients to 207 white patients with mild to moderate HTN (77).

The baseline PRA was modestly but not significantly lower in the black patients. Accordingly, they required two to four times the dose of trandolapril to achieve the same reduction in BP. This occurred despite no differences between the groups in the degree of ACE inhibition at the same dose. These observations confirm that the HTN is not as angiotensin II-dependent in blacks and that ACEIs in blacks at higher doses may act by a mechanism that could not be angiotensin-II dependent.

e) Beta-blockers (BB)

In general, black compared to non-black hypertensives are less responsive to BB therapy. The fall in BP following administration of a BB is roughly half of that expected from diuretic monotherapy (78). Also, black patients tend to require a higher dose of BB to achieve the goal BP (79). Two studies have made a direct comparison of the antihypertensive effects of BB therapy in

hypertensive blacks and whites (78, 80). The VA study on propranolol showed a greater therapeutic response in whites ($> 5/3.1$ mmHg). In blacks, hydrochlorothiazide lowered systolic BP by 20.3 ± 14.3 mmHg versus 8.2 ± 12.2 mmHg for propranolol; hydrochlorothiazide reduced diastolic BP by 13.0 ± 7.0 mmHg versus 9.5 ± 7.0 for propranolol. In whites, the systolic BP reductions were 15.3 ± 12.0 mmHg for hydrochlorothiazide versus 13.2 ± 13.1 mmHg for propranolol; diastolic BPs were 10.9 ± 5.7 mmHg for hydrochlorothiazide and 12.6 ± 6.6 mmHg for propranolol. In the other VA study, nadolol showed a greater efficacy in whites ($> 11.4/6.0$ mmHg).

Less than a third of US blacks achieved satisfactory control of their BP, contrasted with a responder rate of 80% in the white subjects. The reasons for this observation (=less response to BB) may relate to higher plasma volume and lower renin level in blacks. The black-white differences in therapeutic response to BB can be eliminated by the addition of diuretic. While hypertensive African-Americans often respond poorly to BB monotherapy, compared with whites, there is evidence, however, that this response may be different if BB with vasodilating effects are used. A novel BB, nebivolol, a cardioselective beta 1-blocker with vasodilating effects has been shown to restore nitric oxide bioavailability in African-Americans and may be very effective in high-risk African-American hypertensive patients (81).

TREATMENT OF HYPERTENSIVE COMPLICATIONS IN BLACKS

As already said, HTN is a major problem in black patients who are much more prone to the associated cardiovascular complications. As a result, effective antihypertensive therapy is particularly important in these patients.

After **myocardial infarction**, or **heart failure**, BB or ACEI use is suggested in all patients, either whites or blacks (82,83).

In **chronic renal failure**, despite the observation that blacks are generally more responsive to diuretics and CCB than to monotherapy with ACEIs, there are clear benefits of ACEIs in black patients with chronic renal failure, at least in case of benign HN associated with proteinuria, illustrated in the African American Study of Kidney disease and Hypertension (AASK) (84).

We thus advise that an ACEI should be the first drug used in black subjects of African descent with proteinuric CKD. However, despite the benefits of RAAS-blocking therapy on CKD progression, most African Americans with hypertensive CKD who are treated with currently recommended BP therapy continue to progress during the long term (29).

PRACTICAL RECOMMENDATIONS FOR TREATING HTN IN BLACKS

Because of the high prevalence of cardiovascular risk factors in African-Americans such as obesity, cigarette smoking, type 2 diabetes as well as sensitivity to salt intake, diet and lifestyle modifications are particularly important.

Goal blood pressure:

Although the optimal BP in black subjects is uncertain, in 2003, a consensus statement concerning the management of high BP in African-Americans was published by the Hypertension in African-Americans Working Group of the International Society on Hypertension in blacks (85). To lower the risk of a cardiovascular event and to inhibit progression of renal disease, the following recommendations for goal BP were proposed:

For those with uncomplicated essential HTN, lowering the BP to less than 140/90 mmHg is a reasonable target.

Further reduction to less than 130/80 mmHg is indicated in those with diabetes or nondiabetic renal disease with significant proteinuria (86). This lower goal may also be preferred in patients with end-organ damage including microalbuminuria, a history of a cardiovascular event or stroke or being at high risk for coronary artery disease. In addition, the 2004 K/DOQI guidelines suggest that an even lower SBP may be more effective in slowing progressive renal disease in patients with a spot urine total protein-to-creatinine ratio $> \text{or} = 1000$ mg/g. These recommendations do not differ from those for white patients with HTN (60, 87).

Choice of agent:

Based upon ALLHAT and cost, antihypertensive therapy in blacks with uncomplicated essential HTN should be initiated with dietary salt restriction and a thiazide diuretic, preferably beginning at a low dose

(e.g.: 12.5 mg of hydrochlorothiazide or chlorthalidone), or perhaps even better with a thiazide plus a potassium-sparing drug. If goal pressure is not reached with such monotherapy, a CCB, ACEI/ARB, or BB can then be added or substituted (88). If a thiazide cannot be taken as the first choice, a CCB is likely to be most effective. Multidrug therapy is usually required to successfully treat patients with moderate to severe HTN, which occurs with increased frequency in blacks, as well as in those with complicated HTN. The optimal choice of combination therapy will vary based upon initial response and type of co-morbid disease.

Among those with HTN and CKD or heart failure, an agent for renal and cardiovascular protection is preferred, specifically ACEI.

A diuretic should however be included in the combination therapy.

CONCLUSIONS AND PERSPECTIVES

It is clear that HTN is a major public health problem in the black people worldwide who represent a special subset of hypertensive patients. They have a higher prevalence of HTN and its complications. They respond distinctively to certain antihypertensive therapies. The susceptibility of blacks to HTN and its complications relates to socio-economic, environmental and genetic factors. The main characteristics of HTN in blacks, being salt retention with a low plasma renin activity not only reflect its particular pathophysiology but also have practical implications. Indeed, it is known that in monotherapy, diuretics or CCB are more effective than others in lowering BP in blacks compared to whites. But, BB, ACEI and ARB can be used in particular situations such as cardiovascular and/or renal disease and are more effective and well tolerated in combination with diuretics in hypertensive blacks.

There is however an urgent need to improve education for HTN detection and its dangers and to encourage healthy diet and lifestyle as means for primary prevention. We must also identify barriers to treatment and good control of HTN and find ways to remove them.

RÉSUMÉ

L'hypertension artérielle du sujet de race noire présente des particularités épidémiologiques, cliniques, physiopathologiques et thérapeutiques. Elle est plus

fréquente et plus sévère. Elle se développe plus précocement et se caractérise par un pourcentage plus élevé de complications cardiovasculaires et rénales que chez le sujet d'origine caucasienne. Le diagnostic est souvent porté avec retard (à l'occasion de ces complications).

Ces patients sont plus souvent sensibles au sel et ont une activité rénine plasmatique plus basse que les sujets de race blanche. Malgré des progrès substantiels dans la compréhension et le traitement des maladies cardiovasculaires, les sujets noirs continuent à présenter une morbi-mortalité accrue suite aux complications liées à l'hypertension. Les explications restent incomplètes. Participent des influences génétiques auxquelles s'ajoutent des facteurs socioéconomiques et environnementaux. La première approche thérapeutique qui s'ensuit est de diminuer l'apport en sodium alimentaire et d'accroître celui en potassium. Une moins bonne réponse à certains agents antihypertenseurs a été observée. Les diurétiques (thiazides et d'épargne potassique) et les antagonistes calciques constituent la première ligne de traitement médicamenteux antihypertenseur. Les inhibiteurs de l'enzyme de conversion, les bloqueurs de récepteur de l'angiotensine II, et les bêtabloquants apparaissent moins efficaces dans le contexte d'une hypertension non compliquée, surtout chez le sujet âgé mais l'ajout d'une dose minime de diurétique améliore leur efficacité. Quoiqu'il en soit, ces combinaisons sont à préférer chez les patients hypertendus développant une insuffisance rénale chronique ou une décompensation cardiaque.

La cible tensionnelle à atteindre est la même que chez les blancs : une pression artérielle inférieure à 140/90 mmHg en présence d'une hypertension non compliquée et inférieure à 130/80 mmHg si diabète ou insuffisance rénale chronique.

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