Childhood Hypertension: A Review

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Abstract

Prevalence of hypertension in children has increased significantly in recent times, in part related to the epidemic of childhood obesity. Identification and treatment of hypertension in childhood is likely to favorably impact on cardiovascular disease in adulthood. Identification of hypertensive children continues to be problematic because of incomplete blood pressure screening during routine pediatric clinical visits. The blood pressure norms are based on age, gender and height specific values in contradistinction to adults where a single value suffices. Childhood hypertension is either primary or secondary and is categorized as prehypertension (between 90th to 95th percentile), stage 1 (95th to 99th percentile plus 5 mmHg) and stage 2 (≥ 99th percentile plus 5 mmHg) hypertension. Ambulatory blood pressure monitoring is useful in confirming the diagnosis and in helping diagnose white coat and masked hypertension. Once diagnosed as definitive hypertension, the causes of secondary hypertension should be determined and appropriately treated. In children with primary hypertension, a combination of life-style changes (diet and exercise) and drug therapy should be instituted depending upon the stage of the hypertension. Continued follow-up to ensure compliance with treatment regimen and to monitor blood pressure control is mandatory.

Introduction

Hypertension is one of the major contributors of cardiovascular disease in adults, accounting for approximately one-half of all strokes and ischemic heart disease worldwide [1]. Existing evidence suggests that hypertension in adult originates in childhood because childhood Blood Pressure (BP) predicts BP in the adult [2]. Consequently, early identification and treatment of hypertension in childhood are likely to have important impact on long-term outcomes of hypertensive cardiovascular disease. However, none of the studies are of long enough duration to demonstrate the effect of BP reduction in childhood on cardiovascular disease in adults. But, several studies have suggested that elevated BP in childhood correlates with atherosclerosis in adulthood and carotid-medial thickness (marker of hypertension end organ damage) of young adults [3,4].

Prevalence of children with systemic hypertension appears to be increasing and is estimated to be 2 to 5%, especially with the growing population of children with obesity [5]. The prevalence of overweight children in the United States in the 1990s was 11% which is more than double the prevalence of 5% in the 1960s [6]. In a 2002 school-based hypertension and obesity screening study, the prevalence of hypertension was 3 times greater in obese (33%) compared with non-obese adolescents (11%). The prevalence of obesity itself is high at 23% [7]. There has also been a shift in primary hypertension becoming more evident in late childhood and early adolescence which may be related to relatively recent epidemic of childhood obesity. The purpose of this review is to discuss important aspects of hypertension in pediatric patients.

Definition and Diagnosis of Hypertension

In children, it is not possible to use a single BP level that is used in adults to define hypertension since the BP in children increases with increasing age and body size. The definition of elevated BP throughout childhood is based on the percentiles of BP specific to age, gender and height. The current categorization of BP in children and adolescents is based on the Fourth Report on the Diagnosis, Evaluation and Treatment of High Blood Pressure in Children and Adolescents by the National

Heart, Lung and Blood Institute, which is derived from analysis of extensive data of more than 70,000 children in the United States [8].

According to these guidelines, all children and adolescents older than 3 years-old should have their BP measured annually. Children younger than three years old should have their BPs measured if they are at risk of having hypertension such as history of neonatal complications requiring neonatal intensive care, congenital heart disease, recurrent urinary tract infection, hematuria, proteinuria, known renal or urologic disease, family history of congenital renal disease, solid organ or bone marrow transplantation, malignancy, treatment with drugs known to raise BP, other systemic illnesses associated with hypertension and evidence of increased intracranial pressure.

Use of appropriate technique of BP measurement is essential. Ideally the subject should not have consumed stimulant drugs or foods, have been sitting quietly for 5 minutes and seated with his or her back supported with feet on the floor and right arm supported. Infants and small children may assume supine position for BP measurement. Blood pressure should be recorded in the right arm (the left arm may give falsely low readings in association with aortic coarctation) with the antecubital fossa at the heart level and with an appropriate sized cuff for measured arm circumference. The width of the cuff's inflatable bladder should be at least 40% of arm circumference midway between the olecranon and the acromion. The inflatable bladder length should cover 80-100% of the arm circumference. If a cuff is too small, the next largest cuff should be used, even if it appears large. Measurement should be performed by auscultation using a standard

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clinical sphygmomanometer. An oscillometric device is preferred to screen initial BP in neonates and infants. However, if the blood pressure value exceeds the 90th percentile by oscillometry, it should be confirmed by an auscultatory measurement. In the initial evaluation for hypertension, BP measurements are obtained in both upper and lower extremities to detect coarctation of the aorta since it is one of the more common treatable causes of hypertension. Elevated BP must be confirmed on at least two additional occasions to confirm the diagnosis of hypertension. With only a single measurement, it may be categorized as pre-hypertension [8].

Hypertension in the pediatric patient is categorized into several types [8]: Pre-hypertension - when the Systolic Blood Pressure (SBP) or Diastolic Blood Pressure (DBP) is between 90th and 95th percentile or greater than 120/80 mmHg, Stage 1 hypertension - when the SBP or DBP is between 95th and 99th percentile plus 5 mmHg, or if the BP exceeds 140/90 mmHg in adolescents even if the value is <95th percentile, Stage 2 hypertension - when the SBP is > 99th percentile plus 5 mmHg. If there is a disparity between the SBP and DBP, the higher percentile value of either of these determines the BP category. For the interested reader, the tables of normative values and percentiles based on age, gender and height can be obtained from reference 8. In a further study of the National Childhood Blood Pressure database, 14 percent of pre-hypertensive male and 12 percent of pre-hypertensive female adolescents became hypertensive two years later [9]. As mentioned above, confirmed pediatric hypertension is divided into two stages (1 & 2); this is clinically useful to help guide the evaluation and management. Stage 2 hypertension is more severe and therefore, needs to be evaluated for secondary hypertension and managed more aggressively than the other categories [8].

The frequency of hypertension screening nationally was evaluated recently by Shapiro et al. [10]. They found that BP screening has taken place in only two-thirds of routine pediatric visits, one third of ambulatory pediatric visits and that 20% of overweight and obese children are not being screened at their routine visits. The study also revealed that younger children, who are more likely to have secondary hypertension, are commonly not being screened as often as older children. Even when blood pressures are measured, 75% cases of hypertension and 90% cases of pre-hypertension in children and adolescents remain uninvestigated [11].

Ambulatory Blood Pressure Monitoring (ABPM) helps monitor BP changes with daily physiologic activity and environmental stimuli during both sleep and wake periods and may serve to better assess dynamic BPs. A portable device, worn by the patient, records BP every 20 minutes during a 24-hour period. The mean 24-hour blood pressure and mean day time and night time SBP and DBP are obtained and compared against gender and height specific 99th percentiles based on normative ambulatory BP monitoring data. Blood pressure load (% of BP values exceeding the 95th percentile over the 24-hour period) is also obtained. Although data is more limited in children compared to adults, the ABPM has an important role in the evaluation of the various types of hypertension.

ABPM is helpful in the initial evaluation of elevated BP to confirm hypertension, to identify patients at risk and in assessing response to treatment. Categories for ABPM are as follows: Ambulatory hypertension - mean ambulatory SBP or DBP >95th percentile and SBP or DBP load of 25 to 50%, Pre-hypertension - mean ambulatory SBP or DBP <95th percentile and ambulatory SBP or DBP load of 25 to 50% percentile, White Coat Hypertension (WCH) - 24-hour SBP and DBP less than 95th percentile and BP load less than 25%. ABPM is particularly useful in diagnosing white coat hypertension and masked hypertension [12]. The designation of WCH is used when office BP measurements lead to a diagnosis of hypertension while in fact the blood pressures are normal. WCH may be seen in up to 60% of children referred for evaluation of high blood pressure and may not require further work up. However, a retrospective study in children has suggested that WCH is possibly a pre-hypertensive condition with increased LV mass with progression to persistent hypertension [13]. Alternatively, the term "masked hypertension" is when the office blood pressure measurements are normal, but the patient is truly hypertensive based on finding of ABPM. Diagnosing masked hypertension remains a clinical challenge since there is no predictable way to identify these patients during screening. Studies have shown that masked hypertension may be associated with development of sustained hypertension and end organ damage such as left ventricular hypertrophy [13].

Etiology

Pediatric Hypertension is categorized into two major types, primary (essential) and secondary hypertension. The true cause of primary hypertension in children is not known and is generally considered multifactorial. Essential (or primary) hypertension is often associated with a family history of hypertension or obesity. Family history of hypertension is present in nearly 80% of patients and is thought to be due to multiple genetic and environmental factors. Obesity is a key factor in the development and persistence of primary hypertension in childhood and is present in 35-50% of hypertensive adolescents. The mechanism of hypertension related to obesity is complex and may involve insulin resistance, sodium retention, increased sympathetic activity and hyperuricemia. Obesity is also associated with metabolic syndrome, which has been identified as a strong independent predictor of cardiovascular events in adults with primary hypertension [14]. Other comorbidities associated with primary hypertension which increase the risk of cardiovascular disease include abnormal lipid profile, glucose intolerance and sleep-disordered breathing [8]. Sleep disordered breathing, with the most severe form being obstructive sleep apnea, is associated with primary hypertension especially in overweight children. The prevalence of primary hypertension is higher in Native American, Hispanic and African American children and lower in Asian children, when compared with Caucasian children [15-17].

The causes of secondary hypertension vary with the age of the child. Overall, renal parenchymal or renal vascular causes of hypertension account for 70-90% of secondary causes with 2% from coarctation of the aorta (Table 1). In the neonatal intensive care unit, the most common cause is related to renal vascular disease due to umbilical artery-related thromboembolism [18]. From neonates to age 6 years, the most common causes are renal parenchymal, renal vascular disorders and coarctation of the aorta. From ages 6 through 10 years, renal parenchymal disease, such as glomerulonephritis, renal scarring and polycystic kidney disease predominate [17,19]. After 10 years of age, the most common cause of hypertension shifts to essential hypertension. The other rare causes of secondary hypertension are systemic arteritis (example, Takayasu arteritis, Henoch-Schonlein purpura), tumors (example, pheochromocytoma, neuroblastoma, Wilm's tumor), endocrine dysfunction (example, hyperthyroidism, polycystic ovary syndrome, Cushing syndrome, congenital adrenal hyperplasia) and neurologic disorders (increased intracranial pressure, Guillan-Barre' syndrome, dysautonomia). Other rare causes of hypertension are monogenic disorders which are due to functional mutations. Most of these result in overproduction of mineralocorticoids or increased mineralocorticoid activity and include Liddle's syndrome, Pseudohypoaldosteronism type

Renovascular hypertension	Renal artery or vein stenosis or thrombosis Fibromuscular dysplasia Arteritis (Takayasu's, Kawasaki, Moyamoya) Renal vessel compression – tumor, post-trauma, or surgery
Renal Parenchymal Diseases	Congenital renal malformations Glomerulonephritis Systemic vasculitis (SLE, HSP) Acute or chronic renal failure
Cardiovascular	Coarctation of the aorta Mid aortic hypoplasia Syndromes associated with increased risk for aortic hypoplasia (William, Turner's)
Endocrine	Catecholamine excess (Pheochromocytoma, Neuroblastoma) Corticosteroid excess (Iatrogenic, Cushing's disease, CAH) Hyperthyroidism Hypercalcemia (malignancy, hyperparathyroidism)
Medications	Steroids Cyclosporine, tacrolimus ADHD Medications Oral contraceptives Erythropoietin Illicit Drugs
Miscellaneous	Neurologic (Elevated ICP, seizures, Guillan Barre, Dysautonomia) Post-ECMO Chronic Lung Disease Obstructive Sleep Apnea

Table 1: Causes of secondary hypertension in children ADHD, Attention deficit hyperactivity disorder; CAH, Congenital adrenal hyperplasia; ECMO, Extra-corporeal membrane oxygenation; HSP, Henoch-Schonlein purpura ICP, Intra cranial pressure; SLE, Systemic lupus erythematosis.

2, familial hyperaldosteronism type I, congenital adrenal hyperplasia and syndrome of apparent mineralocorticoid excess.

Evaluation

Identification and thorough evaluation of children with primary and secondary hypertension is a clinical challenge and is vital for successful treatment. In the initial evaluation of the hypertensive patient, a thorough search for an underlying cause of secondary hypertension must always be undertaken along with comorbid risk factors such as obesity, diabetes and kidney disease. A detailed history and physical examination are essential for securing key information to unveil the type of hypertension and presence of a systemic disorder [8].

Secondary hypertension must be distinguished from primary hypertension, especially because addressing the underlying cause may result in a cure. Important aspects of history taking include birth history (gestational age, umbilical artery catheterization), family history and symptoms suggestive of target end organ damage such as visual changes, headaches and chest pain. History of symptoms such as sweating, palpitations, flushing, intermittent claudication, medication use, and sleep history should also be obtained.

On physical exam, any clues for secondary causes (four extremity BPs or target organ damage, such as hypertensive retinal changes via fundoscopic exam) should be searched for. The initial work up for children with persistent blood pressure ≥ 95th percentile should include Blood Urea Nitrogen (BUN), creatinine, electrolytes, urinalysis, renal ultrasound and echocardiogram. Electrolytes help screen for hypokalemia or alkalosis which are present in monogenic causes of hypertension. Abnormalities in renal function, renal ultrasound or urinalysis may point toward a renal etiology of hypertension. Whenever secondary causes are suspected, advanced laboratory and renovascular imaging should be considered. For example, if there is any suspicion for hypertension related to hormones, plasma renin, aldosterone, 24 hour urine collection for catecholamine, aldosterone, cortisol and thyroid function tests should be performed. If renovascular disease is strongly suspected, nephrology consultation should be sought and conventional or intra-arterial digital-subtraction angiography or scintigraphy

is recommended. Newer techniques such as magnetic resonance angiography and 3D or spiral computed tomography may be useful in children, but more data are needed to assess their effectiveness [8].

Primary hypertension has a higher association with obesity and positive family history of hypertension. A sleep history is important since obstructive sleep apnea and other sleep disorders have been associated with hypertension, especially in overweight children [16]. Sleep may be evaluated using the BEARS (bedtime problems, excessive daytime sleepiness, awakenings during night and sleep disordered breathing such as snoring) survey [8]. If abnormalities are found, polysomnography may be useful and should be performed [20]. Comprehensive evaluation is needed looking for additional cardiovascular risk factors which overlap with primary hypertension, such as low plasma HDL-C, elevated plasma triglyceride, abnormal glucose tolerance, elevated fasting plasma insulin. If family history is positive for type 2 diabetes, hemoglobin A1c or glucose tolerance test should be considered. A fasting lipid panel and fasting glucose should also be performed in children who are overweight with blood pressure between the 90th to 94th percentile, in all children with blood pressure ≥ 95th percentile and patients with family history of hypertension, cardiovascular disease, or a child with chronic renal disease [8].

ABPM should be performed to confirm the diagnosis of hypertension or if white coat hypertension is suspected. ABPM may also help differentiate secondary hypertension in adolescents since they have been shown to have greater nocturnal systolic BP loads and daytime and nocturnal diastolic BP loads than in children with primary hypertension at similar ages [21].

Once hypertension is confirmed, it is important to assess target-organ damage. Elevated blood pressure has been shown to have damaging vascular consequences early in life, as evidenced by its associations with surrogate markers of vascular injury: for example, increased arterial intima-media thickness [3], impaired arterial compliance [22] or retinal arteriolar narrowing [23]. Target-organ damage may be found even in very mild stages of hypertension. One of the strongest predictors of cardiovascular morbidity and mortality in adults with hypertension is left ventricular hypertrophy (LVH). Following evaluation of 130 children and adolescents with persistent

BP elevation, Daniels et al found that 55% of hypertensive children had Left Ventricular Mass Indices (LVMI) above the 99th percentile and 14% had LVMI above 51 g/m (exponent 2.7), a value associated with a fourfold increase in risk of adverse cardiovascular outcomes [24]. LVH is the most common form of end organ damage caused by hypertension in children and adolescents. Echocardiography is sensitive in calculating LVMI using the Devereux equation, LV mass divided by height to the 2.7 power. LVMI equal to or above the 95th percentile based on normative pediatric data is considered LVH [24]. In addition, echocardiography is helpful in confirming coarctation of the aorta. The Fourth Report recommends echocardiography to diagnose and periodically monitor LVH in all hypertensive children and adolescents [8]. Renal damage may be assessed by creatinine, glomerular filtration rate and elevated Urinary Albumin Excretion (UAE). Small amounts of urinary albumin have been shown to be associated with progression of nephropathy and higher cardiovascular risk [25]. At this time, there are no recommendations on the routine evaluation of urine microalbumin in children with hypertension.

Treatment

Although there is a lack of long-term data showing that improving blood pressure by initiating therapy in pediatric patients with persistent hypertension lowers the risk of cardiovascular disease in adults, there is convincing evidence that hypertension in childhood and adolescence contributes to premature atherosclerosis and the early development of cardiovascular disease.

Primary hypertension is a diagnosis of exclusion after secondary causes (Table 1) are excluded. It is typically mild (stage 1 hypertension) and more common in males and older children/adolescents than secondary hypertension. In secondary hypertension, an organic cause is identified and it is more common in infants and preschool age children. Therefore, severe hypertension in a younger child is more likely related to secondary hypertension and should undergo comprehensive evaluation searching for underlying causes. Once the cause of secondary hypertension is identified, it should be treated accordingly and further discussion of this subject is beyond the scope of this review.

The Fourth Report contains the best evidence for the treatment of high blood pressure in the pediatric population and when such evidence is not available, consensus recommendations from experts in the field are given [8]. The approach stresses the importance of reducing the elevated BP with the least possible adverse side effects. It has an emphasis on the use of non-pharmacologic interventions first and pharmacologic treatment for those who fail to respond and those that have certain criteria. The treatment approach accounts for BP severity, secondary vs. primary hypertension, target-organ abnormalities and presence of underlying co-morbidities such as obesity, diabetes and kidney disease. The Fourth Report includes a treatment algorithm which provides a detailed overview of the management [8].

The initial approach for children and adolescents with mild hypertension and no hypertensive target-organ disease is to incorporate therapeutic lifestyle changes with a focus on diet modification and exercise. Emphasis on weight loss and increasing physical activity is of vital importance, especially with the growing childhood obesity epidemic. Diet modification with the Dietary Approach to Stopping Hypertension (DASH) diet is recommended for all patients. The DASH diet stresses the importance of vegetables, fruits, low-fat dairy and whole grains and reduced intake of foods high in saturated fat and refined sugar. Reduction of sodium in the diet has been shown to have a reduction the BP in the range of 1-3 mmHg. Current recommendations for sodium intake are 1.2 g/day for children aged 4 to 8 years, 1.5 g/day for 8 to 16 year-old and 2.4 g/day for adults [8]. However, in some young athletes, a significant total-body sodium deficit can develop as a result of extensive sweating during extended exercise. Therefore, rehydration in this setting often requires deliberate, concomitant intake of additional salt-containing fluids and foods to ensure a greater body-water retention and distribution to all fluid compartments.

Increasing physical activity (30-60 minutes per day) is recommended because it has been shown to reduce blood pressure, especially in overweight children [26]. However, there are limitations on the types of activities children with uncontrolled stage 2 hypertension should participate in. These patients should avoid competitive and high static resistance sports, including, but not limited to, activities such as gymnastics, water skiing, weight lifting and wrestling. In addition, when hypertension and other cardiovascular diseases coexist, the eligibility of participation in competitive athletics should usually be based on the type and severity of the other cardiovascular disease [27]. In order to encourage physical activity, sedentary time should be limited to 2 hours per day spent on television, computers and video games. If the conservative management of hypertension is not successful, then pharmacologic intervention may be considered, however the emphasis of therapeutic lifestyle changes should be maintained even after adding drug therapy [8].

Pharmacologic therapy is indicated for patients with 1. Stage 1 hypertension that persists despite a trial of four to six months of non-pharmacologic (diet and exercise) therapy, 2. Stage 2 hypertension, 3. Hypertension patients who are symptomatic, 4. Secondary hypertension and 5. Hypertensive target-organ damage [8]. Antihypertensive drugs are also considered in the presence of multiple cardiovascular risk factors such as chronic kidney disease, diabetes mellitus, dyslipidemia, etc. because these factors increase cardiovascular risk exponentially [28]. Since the long term consequences of untreated hypertension are unknown and there is no data on the long term effects of antihypertensive drugs on the growth and development of children, definite indications for pharmacologic therapy should be assured.

A variety of medication classes (Table 2) are currently available for treatment of hypertension in children and include diuretics, calcium channel blockers, Angiotensin-Converting Enzyme Inhibitors (ACEI), Angiotensin-Receptor Blockers (ARB) and beta-blockers. The choice of initial medication is largely based on the presence of an underlying disease and physician preference. This latter is because of lack of long-term clinical outcome trials which evaluated the comparable effectiveness of specific antihypertensive drugs in the pediatric patient. In adults, long term clinical data from randomized trials such as the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) support the use of a diuretic or a beta-blocker as the initial choice in the medication [29]. This type of evidence is lacking for pediatric patients. In some cases of an underlying renal, endocrine or cardiac disease, a specific medication is warranted. For example, in coarctation of the aorta, beta-blockade is preferred since hypertension tends to persist after surgery [30]. In order to help prevent or delay the progression of diabetic nephropathy and decrease albuminuria, adolescents with type 1 or type 2 diabetes mellitus should be treated with ACEI or ARB [31]. ACEI and ARB are also indicated in patients with glomerulonephritis, chronic kidney disease and other types of renal disease to help prevent renal failure. Since ACEI and ARB may have adverse effects on the fetus, and females of childbearing age should

Class	Drug	Starting dose	Dosage Interval	Maximum dose
ARAs	Spironolactone	1 mg/kg/day	QD–BID	3.3 mg/kg/day (100 mg/day)
ARBs	Candesartan	1–6 years: 0.2 mg/kg/day;	QD	1–6 years: 0.4 mg/kg/day
		6–17 years: <50 kg 4-8 mg	QD	6–17 year: <50 kg 16 mg daily
		>50 kg: 8-16 mg	QD	>50 kg: 32 mg daily
	Losartan	0.75 mg/kg/day(max 50 mg/day)	QD	1.4 mg/kg/day (100 mg QD)
	Valsartan	<6 years: 5-10 mg/day	QD	<6 years: 80 mg QD
		6–17 years: 1.3 mg/kg/day(max 40 mg/day)	QD	6–17 years: 2.7 mg/kg/day(160 mg/day)
ACE inhibitors	Captopril	0.3–0.5 mg/kg/dose	BID–TID	0.6 mg/kg/day (450 mg/day)
	Enalapril	0.08 mg/kg/day	QD–BID	0.6 mg/kg/day (40 mg/day)
	Lisinopril	0.07 mg/kg/day(max 5 mg/day)	QD	0.6 mg/kg/day (40 mg/day)
α- and β-Adrenergic antagonist	Labetalol	2-3 mg/kg/day	BID	10-12 mg/kg/day (1.2 g/day)
Adrenergic antagonists	Atenolol	0.5-1 mg/kg/day	QD	2 mg/kg/day (100 mg/day)
	Metoprolol	1-2 mg/kg/day	BID	6 mg/kg/day (200 mg/day)
	Propranolol	1 mg/kg/day	BID–QID	8 mg/kg/day (640 mg/day)
CCBs	Amlodipine	0.06 mg/kg/day	QD	0.3 mg/kg/day (10 mg/day)
	Nifedipine ER	0.25-0.5 mg/kg/day	QD–BID	3 mg/kg/day (120 mg/day)
Central α -agonist	Clonidine	5-20 mcg/kg/day	QD–BID	25 mcg/kg/day (0.9 mg/day)
Diuretics	Chlorthalidone	0.3 mg/kg/day	QD	2 mg/kg/day (50 mg/day)
	Furosemide	0.5-2 mg/kg/dose	QD–BID	6 mg/kg/day
	HCTZ	0.5-1 mg/kg/day	QD	3 mg/kg/day (50 mg/day)
Vasodilators	Hydralazine	0.25 mg/kg/dose	TID–QID	7.5 mg/kg/day (200 mg/day)
	Minoxidil	0.1-0.2 mg/kg/day	BID–TID	1 mg/kg/day (50 mg/day)

HCTZ: Hydrochlorothiazide; ARA: Aldosterone Receptor Blockers; ARBs: Angiotensin ii Receptor Blockers; ACE: Angiotensin Converting Enzyme; CCBs: Calcium Channel Blockers; ER: Extended Release; QD: Daily; BID: Twice Daily; TID: Three Times Daily; QID: Four Times Daily

Table 2: Recommended doses for selected antihypertensive agents for use in pediatric hypertension.

be counseled regarding the importance of effective contraception [32] during the treatment with these classes of medications.

The last three pediatric working groups of the National High Blood Pressure Education Program endorsed an individualized stepped-care approach in the use of anti-hypertensive agents [8,33,34]. In this approach, initial treatment is a single drug (mono-therapy regimen) is added to non-pharmacologic (diet & exercise) measures. Therapy should start with a low dose of the initial agent and titrated up until target BP is achieved or the highest recommended dose of that particular drug is reached. If the target BP is still not achieved, a second drug with a low dose from a different class should be added after the initial drug dose reaches the highest recommended level or if the patient begins to experience side effects from the initial drug. If target BP control is still not achieved, a third anti-hypertensive agent may be added. Referral to an expert on hypertension should also be considered. Continued follow up is essential in order to ensure BP control and monitor compliance and adverse side effects. In particular, adolescents have a high tendency to be non-compliant and every effort should be made to ensure compliance. Ideally, treatment should be with a single drug regimen once daily in order to increase chances of compliance [8].

As recommended by the Fourth Report, in children and adolescents with uncomplicated primary hypertension and no evidence of target-organ damage, the targeted goal for BP should be less than the 95th percentile for age, height, and gender. If there are comorbid cardiovascular disease risk factors such as diabetes, dyslipidemia, or chronic kidney disease, the targeted BP goal should be lowered to below the 90th percentile for age, height, and gender [8].

Blood pressure should be monitored every 2 to 4 weeks in the office setting until good control is achieved and thereafter, every 3 to 4 months. Home blood pressure measurement may improve compliance [32]. Gradual reduction of medications should be considered after an extended period of good BP control. Such is most likely to be possible in children with mild initial hypertension who are well controlled on a single drug and who can be maintained on non-pharmacologic therapy. The eventual goal should be to completely discontinue the drug therapy and continue blood pressure monitoring along with non-pharmacologic treatment because the hypertension may recur [8].

Conclusions

Increasing prevalence of elevated BP and obesity in children and adolescents is likely to adversely impact on the hypertensive cardiovascular disease in adulthood. Secondary hypertension should be treated as and when the cause is identified. With regard to primary hypertension, prompt identification, exclusion of secondary hypertension, non-pharmacological (diet and exercise) intervention followed by drug therapy, as necessary are essential to address pediatric hypertension. Continued follow-up is mandatory in most patients.

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