Paediatric hypertension – evaluation and management

Shenal Thalgahagoda

Mohan Shenoy

Abstract

Hypertension (HT) is an increasing problem in children, mainly because of the increasing prevalence of obesity. Unlike adults, the diagnosis of HT in children is based on the age, sex and height of the child. The overall prevalence is thought to be 1–5%. Measurement of blood pressure can be challenging in infants and young children. BP must be measured in the upper arm using an appropriate sized cuff. It is important to confirm automated oscillometric readings by a manual auscultatory method or by using a Doppler. The latter is particularly useful in infants. There is an increasing role for ambulatory BP monitoring in the diagnosis and also assessment of BP control.

The aetiology of HT varies depending upon the age of the child, with renal parenchymal pathology being the predominant pathology in young children while obesity is the main cause in older children. The baseline investigations in all children include electrolytes, creatinine, urine dipstick and an ultrasound scan of the renal tract. Therapeutic lifestyle measures would be the initial treatment of choice for all patients with stage I hypertension. Pharmacotherapy is indicated if these measures fail or if there is stage II HT or evidence of target organ damage.

Keywords ABPM; evaluation; hypertension; obesity; treatment

Introduction

Hypertension (HT) in children is an increasing problem. While previously secondary HT (SHT), mainly of renal origin, was the predominant aetiology, primary HT (PHT) is now quite common, mainly due to the epidemic of obesity worldwide. However, SHT continues to be the dominant entity, especially in children under the age of 5.

HT is a major risk factor for atherosclerosis and cardiovascular disease in adults. Its origins may even be present even in childhood and adolescence. Children who have elevated blood pressure (BP) readings are at increased risk of developing HT as adults (BP tracking phenomenon). HT is also responsible for the progression of renal impairment, a fact of importance considering the high incidence of renal parenchymal and renovascular HT in children with chronic kidney disease (CKD). Early identification and treatment is therefore of paramount importance. Prompt recognition of the hypertensive child, defining aetiology based on selected investigations and therapeutic strategies based on aetiology, encompass the management of the childhood hypertensive.

The American Academy of Pediatricians (AAP) published its latest guidelines for the screening and management of HT in children and adolescents in September, 2017. These are an update to the Fourth Report of 2014. Two of the key differences in these guidelines are that the term 'pre-HT' has been replaced by the term 'elevated BP' and that new normative BP tables that are based on BP values of normal-weight children and adolescents have been included. The latter is a change from the tables in the 4th Report which included the BP values of overweight and obese individuals as well.

Classification of hypertension in children and adolescents

Class	Children 1–13 years of age	Children >13 years of age
Normal	<90th centile	<120/<80 mmHg
Elevated BP	90th centile to <95th centile >120/80 mmHg even if below 90th centile	120/<80 to 129/<80 mmHg
Stage 1	95th centile to 99th centile + 12 mmHg or 130/80 to 139/89 mmHg (whichever is lower)	130/80 to 139/89 mmHg
Stage 2	>99th centile + 12 mmHg or ≥140/90 mm Hg (whichever is lower)	≥140/90 mmHg

Table 1

Definition

Paediatric HT differs from adult HT in that while the definition of adult HT is based on its effects on target organ damage (TOD), the definition of paediatric HT is based on population statistics. Accordingly, paediatric HT is defined as the average systolic and/or diastolic BP that is equal to or greater than the 95th percentile for age, gender and height. Measurements should be obtained on three separate occasions. For classification of paediatric HT see Table 1.

The term 'white coat HT' is used when a patient has a BP above the 95th centile in a clinical setting but below the 90th centile in other settings. Although previously thought to be benign, recent evidence suggests that this may actually be a pre-hypertensive state, with some patients progressing to sustained HT. Masked HT is essentially the opposite where the BP is above the 95th centile outside a clinical setting but is below the 90th centile when checked in clinic. Masked HT is also associated with progression to sustained HT and development of left ventricular hypertrophy (LVH). Ambulatory BP monitoring (ABPM) is required to diagnose both these conditions (see Table 2).

It is difficult to estimate the exact prevalence of hypertension in children, but various studies state a prevalence of 1–5%

Shenal Thalgahagoda *MBBS DCH MD MRCPCH is a Consultant Paediatric Nephrologist at the University of Peradeniya, Sri Lanka. Conflict of interest: none declared.*

Mohan Shenoy *MBBS DCH MRCPCH is a Consultant Paediatric Nephrologist at Royal Manchester Children's Hospital, Manchester, UK. Conflict of interest: none declared.*

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Aetiology of hypertension depending on age

Age	Aetiology
Neonatal period to 1 year	Renal artery stenosis, coarctation of the aorta, autosomal recessive polycystic kidney disease, renal parenchymal disease
1 to 5 years	Renal parenchymal disease; reno-vascular disease; endocrine causes; coarctation of the aorta; EHT
5 to 10 years	Renal parenchymal disease; PHT; reno-vascular disease; endocrine causes; coarctation of the aorta
10 to 20 years	PHT; renal parenchymal disease; reno-vascular disease; endocrine causes; coarctation of the aorta

Table 2

depending upon the population studied and the techniques used to measure BP.

Aetiology

Paediatric HT can be classified as primary or essential when no identifiable cause exists, or as secondary when it occurs due to a specific, potentially correctable cause. While SHT predominates in infancy and childhood, the incidence of PHT increases with age. A general rule to follow is that the younger the child and the more severe the HT, the greater the likelihood of there being a secondary cause.

Secondary causes of HT also vary with the age of the patient. In the neonatal period reno-vascular HT, usually secondary to umbilical artery catheterisation, and renal parenchymal HT due to structural renal anomalies, account for the majority of cases. Coarctation of the aorta is another cause that may be picked up by identification of weak femoral pulses at routine neonatal examination.

In early childhood renal parenchymal HT secondary to glomerulonephritis, hydronephrosis, renal scarring, renal dysplasia and renal cystic disease predominate, while reno-vascular HT and coarctation of the aorta remain important entities. The same trend continues into late childhood, however, with PHT gradually gaining importance. In adolescence PHT becomes the most frequent cause, especially in those with obesity and mild HT.

Monogenic HT

Monogenic forms of HT, although rare, must be considered in the differential diagnosis in patients with early onset, severe HT. They may have a family history of relatives with early onset, difficult to control HT. Monogenic HT is associated with low plasma renin activity (PRA) and various electrolyte and acid base anomalies. These conditions, as the name implies, are due to a single genetic mutation. Apparent mineralocorticoid excess, Gordon syndrome, Liddle syndrome and glucocorticoid responsive aldosteronism are examples of monogenic HT.

Clinical features

The history and examination in a hypertensive patient should be focused to identify a possible aetiology and to look for complications of the disease. An important point to reiterate here is that the higher the degree of HT and the younger the patient the more likely it is due to a secondary cause. It is only moderate to severe or sustained HT that gives rise to symptoms. As such, most patients with PHT remain asymptomatic and are identified during routine examination.

In the current history, features of HT like headache, visual disturbances, vertigo and epistaxis should be sought. Dyspnoea on exertion, facial palsy and seizures would indicate target organ involvement. Haematuria, oliguria, polyuria, nocturia, oedema, fatigue and growth failure are all suggestive of a renal aetiology.

A past history of recurrent urinary tract infections or urinary tract anomalies would suggest renal scarring, obstructive uropathy or reflux nephropathy as the cause. A history of low birth weight and prematurity would be important as would neonatal intensive care admission and umbilical arterial catheterisation.

The patient with PHT often has a family history of HT, hyperlipidaemia, diabetes mellitus, cardiovascular disease or stroke. Hereditary renal diseases like polycystic kidney disease and endocrine diseases like pheochromocytoma, multiple endocrine neoplasia type 2 and von Hippel–Lindau syndrome could present with a positive family history, as would syndromes like neurofibromatosis and the monogenic forms of HT.

A drug history should include both prescription medication (e.g. corticosteroids, methylphenidate, cyclosporine etc.) and over the counter medication (e.g. decongestants, ibuprofen etc.). Illicit drug use should also be probed. Table 3 shows features to look for in the examination.

Measurement of BP

In accordance with current recommendations, BP should be routinely measured annually in all children three years and above. In those with risk factors for HT like obesity, diabetes, renal disease, coarctation of aorta/repaired coarctation and those on medication known to cause HT, BP should be checked at every healthcare encounter. In younger children, BP should be measured in patients with a history of prematurity, low birth weight and who have required intensive care in the neonatal period, patients with cardiac disease, established renal disease, patients with conditions associated with HT, elevated intracranial pressure and patients on medication known to cause HT.

During BP measurement, **choosing the correct cuff size is of utmost importance**. This should be one where the inflatable bladder width is at least 40% of the upper arm circumference at a point midway between the olecranon and the acromion. The cuff bladder length should cover 80–100% of the circumference of the arm. An undersized cuff overestimates BP and, theoretically, an oversized cuff underestimates it. If an appropriate cuff size is not available however, the next largest size should be used.

If BP is found to be above the 90th centile by either auscultation or oscillometric measurement, two further measurements should be obtained and the average used to classify the patient's blood pressure category. Oscillometric measurements should be confirmed by auscultation. A Doppler is often helpful to measure the systolic BP, particularly in neonates and in infancy. In order to diagnose a patient as hypertensive three measurements on separate occasions are required. All measurements should be compared with the BP tables published by the Clinical Practice

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