The Newcastle upon Tyne Hospitals NHS Foundation Trust

Investigation of Secondary Hypertension in Critical Care

1 Introduction

The majority of patients with hypertension have primary, or essential hypertension. However 5-10% of the hypertensive population have secondary hypertension due to an identifiable, and potentially treatable, cause. In critical care we commonly see patients with end-organ damage from untreated hypertension, usually manifesting as spontaneous intracerebral haemorrhage.

2 Guideline scope

This guideline has been developed in conjunction with the RVI endocrinology department and covers the basic investigations to be requested in critical care patients with suspected secondary causes of hypertension. It should not replace expert guidance from renal, cardiology or endocrine teams.

3 Main body of the guideline

3.1 Who to screen

In all patients ensure common causes of hypertension on ITU (eg pain, agitation, delirium, arousal from sedation, inadvertent overdose of vasopressor/inotrope) are appropriately treated.

All patients with hypertension (BP>140/90) and one or more of the following

- Spontaneous intracerebral haemorrhage or other signs of end organ damage e.g. LVH
- Patients under the age of 30 without other risk factors
- Severe hypertension BP >180/110
- Resistant hypertension despite 3 antihypertensive agents
- Hypokalaemia
- Family history of uncontrolled hypertension

3.2 How to screen

The most common causes of secondary hypertension are

- Obstructive sleep apnoea (>30% of secondary HTN)
- Renal parenchymal disease (2-10%)
- Renal artery stenosis (2.5-20%)
- Primary aldosteronism (Conn's syndrome) (6-23%)

Rarer causes include

- Cushing's disease (<1%)
- Hyper or hypothyroidism (1-3%)

- Phaeochromocytoma (<1%)
- Coarctation of the aorta (<1%)
- Acromegaly

Screening should begin with history and examination

History
- Family history of hypertensive/cardiovascular disease
- Symptoms of OSA: Excessive daytime sleepiness, snoring
- Risk factors for atherosclerotic disease
- Adverse reaction to ACEi (pulmonary odema, renal failure) suggesting renal artery stenosis.
- Symptoms of hyper or hypothyroidism

Examination
- BMI +/- neck circumference
- Fundoscopy: Looking for papilloedema or retinal haemorrhage
- Signs of peripheral vascular disease
- Renal bruits
- Cushingoid appearance
- Signs of hyper or hypothyroidism
- Acromegalic appearance
- Cardiovascular examination ?systolic murmur ?weak femoral pulses

3.3 Investigations

As soon as possible:-
Basic screening investigations should include the following:

U&Es including bicarbonate: Low potassium may indicate Conn's syndrome
Serum creatinine: Screening for renal parenchymal disease
Plasma metanephrines if not on vasopressors
If in AF or other signs of thyrotoxicosis: TSH and T3/T4

Note:-
Random cortisol is unhelpful in the acute setting, especially if the patient has already been given dexamethasone.
Plasma renin and aldosterone ratio is also unhelpful if the patient is on vasopressors or has an electrolyte disturbance. Electrolyte abnormalities should be corrected prior to requesting this investigation.

During admission:-
ECG: Looking for LVH or signs of cardiovascular disease.
Echocardiography: To look for LVH and exclude aortic coarctation.
Urinalysis: Screening for renal parenchymal disease +/- end organ damage
Doppler USS Renal Tract: Assessing renal size and renal artery calibre.

Tests for endocrine causes:

The following are guides for further investigation if requested **after** discussion with the endocrine physicians.

Suspected primary or secondary hyperaldosteronism:

The accurate measurement of plasma renin and aldosterone is important in the investigation of suspected hyperaldosteronism. There are many variables that can affect the results and so it is recommended that patients with unexplained hypokalaemia and hypertension be referred to a Consultant Endocrinologist for further assessment

Suspected Cushing's Syndrome:

The screening test for Cushing's syndrome is the low dose overnight dexamethasone suppression test.
Give 1mg enteral dexamethasone between 23:00 and midnight.

Take cortisol level at 9am the next morning. Normal suppression is indicated by a 9am serum cortisol <50nmol/l.

Failure of cortisol suppression sometimes also occurs in

- Severe endogenous depression
- Alcoholism (pseudo-Cushing's syndrome)
- Severe stressful illness/ infection
- Hepatic enzyme inducing drugs (phenytoin/ rifampicin)
- Oestrogen therapy
- Failure to take dexamethasone or for it to be absorbed
- Glucocorticoid resistance syndrome
- Obesity
- Renal failure
- Steroid containing creams
- Beconase eye drops

Suspected Phaeochromocytoma:

Most phaeochromocytomas diagnosed on ITU will have plasma metanephrine levels >10000 pmol/L and milder elevations may be due to drugs e.g. paracetamol and MAOis, or other causes. These cases should still be discussed with the on call endocrinologist. 24 hour urinary catecholamines and metanephrines is the gold standard test for diagnosis of phaeochromocytomas. 24 hour urine collection bottles containing acid are required from pathology. Paracetamol should ideally be avoided 5 days prior to sample collection.

Suspected Acromegaly:

The following baseline pituitary hormone blood tests should be performed on a 9am sample.

- LH and FSH
- Full thyroid profile (TSH, T4, T3)
- Prolactin
- Testosterone if male
- Oestradiol if female
- Cortisol
- Growth hormone and IGF1

4 Training, Implementation, Resource Implications

No extra training or resources are required to implement this.

5 Monitoring Section

Intermittent audit can be used to look at compliance with this guideline.

6 Evidence Review and Evaluation

Evidence from the references below have been reviewed. This guideline has also been discussed with the endocrinology consultants at the RVI.

7 References

- Secondary hypertension: When, Who and How to Screen? Stefano et al. European Heart Journal 23/12/13
- NICE clinical guideline 127. Hypertension. Clinical management of hypertension in adults.
- 2010 AHA/ASA Recommendations for Treating Elevated BP in SICH
- Discussion of proposed guideline at January 2016 Endocrine PIU Meeting