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Education-Family Physician Corner

Severe Asymptomatic Hypertensive: Considerations for Out-patient Practice

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Physicians in clinical practice are likely to encounter patients with hypertensive crisis, such as systolic blood pressure of 180 mmHg or greater, or diastolic blood pressure of 110 mmHg or greater; physicians need to distinguish between hypertensive emergency from severely asymptomatic hypertension (classified as hypertensive urgency or severe uncontrolled hypertension).

Severe asymptomatic hypertension should be treated progressively with continuous followup over weeks to months to reach the anticipated blood pressure target.

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INTRODUCTION

Hypertension remains an important risk factor for high morbidity and mortality for heart disease and stroke; currently, the global hypertension prevalence is increasing from 40% to 50% in 2025^{1-3} .

Mortality in the Middle East could be attributed mainly to chronic, non-communicable diseases (NCD), such as cardiovascular disease, diabetes, and high blood pressure. NCDs are associated with prevalence of serious risk factors (e.g. obesity, physical inactivity, high salt and energy-dense food). Most hypertension patients remain undiagnosed, while patients with end organ damage (heart diseases, stroke, blindness, and renal disease) could be prevented by adequate blood pressure control⁴⁻⁸.

Hypertensive crises include both hypertensive emergencies and severe asymptomatic hypertension; hypertensive crisis means sustained elevation of systolic blood pressure of more than 179 mmHg or diastolic blood pressure more than 119 mmHg. If a patient presents with symptomatic, end-organ damage, it means hypertensive emergency, if not, it means severe asymptomatic hypertension; it could be categorized as hypertensive urgency or severe uncontrolled hypertension⁹. Presence of risk factors for progressive end-organ damage (e.g. history of congestive heart failure, unstable angina, or preexisting renal insufficiency) is called hypertensive urgency; absence of these risk factors is called severe uncontrolled hypertension¹⁰.

Physicians in clinical practice are likely to encounter patients with hypertensive crisis, which sometimes reaches up to 25% of all patients presenting to emergency departments¹¹. One year and 5 year mortality rates following untreated hypertensive emergency were 80% and 100%.

Consequently, with adequate blood pressure treatment, those mortality rates will decrease to 25% and $50\%^{12}$.

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In 2007, the prevalence of hypertension among Bahrainis was 40% in males and 33.7% in females¹³. Moosa et al found an increase in the prevalence of common risk factors for hypertension in Bahrain, see figure 1.



Figure 1: Leading Risk Factors for Hypertension in Bahrain¹⁴

In 2012, NCD was the leading cause of mortality in Bahrain. It reached to one-third of all-cause mortality¹⁵.

Al-Banny et al found that the prevalence of hypertension emergency was 35% in females and 65% in males; consequently, most middle aged patients (45-65 years) were affected¹⁶. There was statistical significance associated with shortness of breath and neurological deficit in hypertensive emergency; headache and blurring of vision in hypertensive urgency. Chmiela et al found smoking, high body mass index (BMI) and patients on single therapy were strong independent risk factors for hypertensive crisis¹⁷.

The following patient is an example of everyday practice in family medicine. Pre-employment people are presenting asymptomatically for screening of possible chronic diseases. This patient was chosen to highlight the importance of the following:

- Physician should confirm diagnosis of severe uncontrolled hypertension.
- Physician should stratify patient's risk factors.
- Physician should encourage patient for better lifestyle modification.
- Physician should choose proper evidence-based antihypertensive suitable for patient's condition.

A thirty-year-old housemaid attended the clinic for pre-employment screening. The blood pressure (BP) readings were 210/123; the reading was confirmed with mercury device monitoring.

The patient had no significant history of chronic disease; she had negative signs and symptoms of end-organ damage (shortness of breath, chest pain, numbness/weakness back pain and difficulty in vision or speech). There was no history of using drug inducing Hypertension, see table 1.

Table 1: Drug Induced Hypertention¹⁸

Prescription: ADHD medications (e.g. Methylphenidate), antidepressants (e.g. Venlafaxine, Bupropion, Desipramine), calcineurin inhibitors (e.g. Cyclosporine, Tacrolimus), corticosteroids, estrogens, midodrine, NSAIDs (e.g. ASA, Ibuprofen, Naproxen, Diclofenac, Celecoxib), testosterone, triptans

Non-Prescription: decongestants (e.g. Pseudoephedrine, Phenylephrine), NSAIDs (Ibuprofen, Naproxen), topical ASA or Diclofenac

Herbal: black licorice root, Ginkgo Biloba, St. John's Wort

Recreational: stimulants (e.g. amphetamines like crystal meth or ecstasy), anabolic steroids, caffeine, cocaine, phencyclidine.

Energy drinks containing taurine, guarana root, yerba mate, Glucuronolactone, etc.

Measurement of this patient's cardiovascular risk factor based on the National Cholesterol Education Program (NCEP) risk calculator was below 1% of cardiovascular (CVS) event in the next 10 years¹⁹.

Patient's examination revealed no significance; only her body mass index (BMI) was 27. Other potential causes of secondary hypertension had been excluded. This patient is a case of uncontrolled severe hypertensive (without end-organ damage, no risk factor for CVS)¹⁰.

The patient was managed by reassurance of the nature of her disease; she was advised to use the 'DASH' (Dietary Approaches to Stop Hypertension). The diet includes three servings of low-fat dairy foods and eight to ten servings of fruits and vegetables, which was shown to lower the blood pressure and may have another lifesaving benefit and protection against heart disease^{20,21}.

Severe uncontrolled hypertensive is not an emergency and its management is less aggressive; our overall goal of management was to reduce her BP by 25% over 24-48 to keep her BP below 195/110mmHg. The patient was sent home on Thiazide plus ACE inhibitor²².

Patient's blood glucose, electrolytes, TSH, CBC, ECG, urinalysis, renal function were normal.

If a patient presented with severe asymptomatic hypertensive crisis with comorbid conditions, we should consider the use of long-term BP lowering agents, see table 2.

 Table 2: Drug Considerations for Long-Term BP Lowering in Severe Asymptomatic

 Hypertensive^{23, 24}

| Comorbid Conditions | Initial Therapy Options | | |
|--------------------------------|--|--|--|
| Heart Failure | ACE Inhibitor (or ARB), Beta Blocker (bisoprolol, carvedilol), | | |
| | Aldosterone Antagonist (spironolactone) ; Thiazide | | |
| Post-Myocardial Infarction | ACE Inhibitor (or ARB), Beta Blocker | | |
| Isolated Systolic Hypertension | Thiazide, Calcium Channel Blocker; ACE Inhibitor or ARB | | |
| Diabetes | ACE Inhibitor (or ARB), Cardio Selective Beta Blocker (if age \leq | | |
| | 60), Thiazide, Calcium Channel Blocker | | |
| Chronic Kidney Disease | ACE Inhibitor (or ARB) | | |

But if a patient presented with hypertensive urgency, we should consider the use of short-term BP lowering agents for a short time followed by long-term BP lowering agents, see table 3.

| Table 3: Drug Considerations for Short-Term BP | Lowering in Hypertensive Urgency ^{23, 2} | 4 |
|--|---|---|
|--|---|---|

| Drug | Dose | Advantage | Contraindications (CI)/ | | |
|------------------|------------------------------|--------------------------------|---|--|--|
| | | | Adverse Effects (AE)/ | | |
| | | | Drug Interactions (DI)/ | | |
| | | | Comments | | |
| Captopril | Acute Dose: 12.5 mg | Benefits for cerebral auto | CI: bilateral renal artery stenosis; | | |
| (CAPOTEN) | oral/sublingual | regulation and blood flow. | immune-mediated diseases; pregnancy. | | |
| 6.25, 12.5, 25, | (repeat 1-2 times at a | Favorable effect on regional | AE: cough, rash, dizzy, fatigue, | | |
| 50, 100 mg | 30-60min | myocardial perfusion. | angioedema, increase K+, abnormal taste | | |
| tablets | interval) | Reduces pre and afterload. | DI: Diuretic K+ sparing, NSAID, | | |
| | Max: 150mg TID for | No fluid retention. | Bactrim, Spironolactone. | | |
| | hypertension | Suitable to be used in chronic | Caution in volume depleted patients and | | |
| | Onset: | management of | high renin states (patients on diuretics) | | |
| | 10-15 minutes (sublingual); | HF and scleroderma. | If >65 years old, consider a low starting | | |
| | 15-30 minutes (oral) | | dose and titrate. | | |
| | Peak Effect: 1 hour | | For chronic therapy switch to an ACE-I | | |
| | (sublingual); | | requiring less frequent dosing is often | | |
| | 1-2 hours (oral) | | advantageous. | | |
| | Duration : 4-8 hours | | | | |
| Clonidine | Acute Dose: 0.1-0.2 mg | Decreases heart rate (in | CI: 2nd/3rd degree heart block; caution in | | |
| (CATAPRES) | orally | about 4% of patients). | HF (due to potential decrease cardiac | | |
| 0.1, 0.2, 0.3 mg | (can repeat 1-2 hours) | No increase in myocardial | output). | | |
| tablets | Maximum: 0.6-0.8 orally | oxygen consumption. | AE: Sedation (up to 50%); orthostatic | | |
| | mg/day for acute use | | hypotension; dramatic decrease cerebral | | |
| | Onset : 30-60 minutes | | blood flow | | |
| | Peak Effect: 2-4 hours | | DI: cyclosporine, mirtazapine, TCAs, | | |
| | Duration : 3-12 hours | | beta blockers | | |
| | | | ↑rebound hypertension | | |
| | | | ↑risk of falls | | |
| | | | Decrease dose if: >65 years old. | | |
| Labetalol | Acute Dose: 200-400 mg | Favorable cardiac and | CI: HF; reactive airway disease; 2nd/3rd | | |
| (TRANDATE) | orally | possible central nervous | degree heart block | | |
| 100, 200 mg | (can repeat 6-12 hours | system effects. | AE: fatigue, insomnia, decrease HR, | | |
| tablets; | PRN) | Mixed alpha/beta antagonist | impotence, decrease exercise tolerance, | | |
| 5mg/mL vial | Maximum: 1200 mg/day | Used in pregnancy. | dizzy, cold extremity, bronchospasm, | | |
| | orally for | | mask & delay symptoms of | | |
| | hypertension | | hypoglycemia, decrease HDL | | |

| Onset : variable (30-120 | DI : anti-diabetics: CCB s: clonidine: |
|---------------------------------|---|
| minutes) | digoxin; fluconazole, insulin, NSAIDS; |
| Peak Effect: 3-4 hours | phenobarbital, cimetidine. |
| Duration : 8-12 hours | Beta blockers, in general, may have |
| | decreased efficacy in the elderly. |

Rapidly lowering blood pressure in the primary care should be avoided because it is usually unnecessary in asymptomatic patients and may be harmful²⁵.

Repeated follow-up and observation are essential in severe uncontrolled hypertension to reach desired blood pressure goals. The physician should monitor the blood pressure reduction, evaluate signs of hypertension or hypotension, enforce lifestyle interventions and assess the medication adherence or side effect. The blood pressure of the patient showed gradual decrease in the next consecutive weeks, see table 4.

Table 4: Blood Pressure Measurement over one Month Follow-Up²⁶

| Day | 1 st visit | 2 nd visit (3 rd day) | 3 rd visit (7 th day) | 4 th visit (2 nd week) | 5 th visit (4 th Week) |
|-----|-----------------------|--|--|---|---|
| BP | 220/123 | 190/105 | 180/100 | 170/100 | 130/80 |

CONCLUSION

Physician should distinguish severe asymptomatic hypertension from hypertensive emergency.

Physician should evaluate patient's cardiovascular risk factors and treat patient less aggressively with fewer risk factors.

Physician should initiate oral medication in patients with severe asymptomatic hypertension before patient discharge.

Physician should not expect patient's blood pressure to decrease to the desired level during the initial visit.

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