The Clinical Presentation of Hypertensive Crisis in the Emergency Department of Baghdad Teaching Hospital

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ABSTRACT:

BACKGROUND:

Uncontrolled hypertension can progress to a hypertensive crisis defined as a systolic blood pressure ≥180 mm Hg or a diastolic blood pressure ≥120 mm Hg which is potentially fatal. It can manifest as either hypertensive emergency or urgency, depending on end-organ damage including cardiac, renal, retinal and neurologic injury.

OBJECTIVE:

To analyze the clinical presentation, and characteristics of patients presented with hypertensive crisis and to assess the frequency of the target organs involved.

METHODS:

An 8 months cross sectional study in which 306 patients older than 18 years who attended the medical emergency department of Baghdad Teaching Hospital with a hypertensive crisis were selected; excluding cases of preeclampsia and eclampsia. The criteria that were used were based on the 7th Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure which was the latest report at the time of the study. Patients were differentiated into hypertensive emergency or urgency, and were divided according to age into five groups: ≤30y, 30-39y, 40-49y, 50-59y, ≥60y. From each patient, a clinical history, physical examination including fundoscopy, blood analysis, electrocardiogram, and a chest X-ray were obtained. Computarised tomography was done when indicated.

RESULTS:

Hypertensive emergency was found in 60.8% and hypertensive urgency in 39.2% of cases. Of total; 56.2% were males. Mean age of total sample was 48.7 ± 16.2y, but patients with a hypertensive emergency were older with statistically significant difference. Hypertension was unknown to 48% of patients.Twenty-four percent of patients were diabetics. Smoking and dyslipidemia were significant risk factors. Hypertensive emergency presented with dyspnea in (30%), and papilledema was found in (18.63%) with statistically significant difference. Patients with hypertensive urgency presented with epistaxis in (11.90%), and papilledema was found in (9.80%). Most cases of hypertensive emergency corresponded to acute myocardial infarction (25.3%) followed by encephalopathy (21.0%), renal failure (20.4%), heart failure (17.2%), and cerebrovascular accident (11.3%).

CONCLUSION:

Hypertensive emergency was more frequent, males were more frequently affected and were significantly older than those with a hypertensive urgency. Smoking & dyslipidemia were significant risk factors. Hypertension was unknown to about half of patients. The most frequent clinical manifestations were papilledema and dyspnea. Acute myocardial infarction, encephalopathy, and renal failure were the most frequent target organ lesions in hypertensive emergency.

KEY WORDS: hypertensive crisis, hypertensive emergency, hypertensive urgency.

INTRODUCTION:

Systemic hypertension (HTN) is a highly prevalent disease, affecting about one billion individuals worldwide and is responsible for approximately 7.1 million deaths per year.[1] http://journal.publications.chestnet.org/article.aspx?articleid=1085166 - b2Patients with a systolic BP (SBP) ≥ 180 mm Hg or a diastolic BP (DBP) ≥ 120 mm Hg are usually considered to be having a hypertensive crisis according to the 7th report of

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Joint National Committee (JNC). [2,3,4] It is cassified into hypertensive emergency (HTN-E) when there is evidence of progressive end organ damage, or hypertensive urgency (HTN- U) in the absence of organ failure.[5,6] End organ damage may include the following : hypertensive encephalopathy, cerebral infarction, subarachnoid hemorrhage, intracranial hemorrhage, myocardial infarction (MI), acute pulmonary edema, aortic dissection, unstable angina pectoris, acute renal failure, retinopathy, eclampsia, and microangiopathic hemolytic anemia.[7]

Pathophysiology. An abrupt rise in BP causes endothelial injury in vascular smooth muscle cells, causing inability of arteries to appropriately dilate in response to increased blood flow and acute increase in systemic vascular resistance resulting in hypertensive crisis.[8] This leads to increased permeability, activation of platelets and coagulation and fibrinoid necrosis. The renin-angiotensin system is often stimulated leading to vasoconstriction and the production of cytokines such as interleukin-6 resulting in damage to the endothelium of blood vessels.[9] Other factors are hypovolemia due to natriuresis and catecholamine excess. The end result is end-organs hypoperfusion and ischemia and organ dysfunction [10].

Common Causes. Medication noncompliance, drugs (cocaine, oral contraceptives, cyclosporin), the withdrawal of beta-blockers and alpha-stimulants, renal parenchymal & renovascular disease, collagen vascular diseases, Cushing's disease, pheochromocytomas, hemolytic uremic syndrome, pre- eclampsia, eclampsia and postoperative HTN[11].

Management. The history should focus on the presence of end-organ dysfunction. The physical examination should focus on proper BP measurement in both upper limbs. Pulses should be palpated and compared in the upper, femoral, and lower extremities. Carotid and abdominal arteries should be auscultated for bruits. Check for JVP, third heart sound, and/or pulmonary rales [10]. Fundoscopic examination must be done for the presence of hemorrhages, papilledema, or exudates and a thorough neurological examination must be done to assess for focal deficit [12].Laboratory work should include a serum test for potassium, renal and hepatic function tests, CBC and blood film, and urine examination. An ECG, CXR, head CT or MRI for mental status changes, or headaches and a chest CT scan or transesophageal echocardiogram should be also performed [13]. In patients with HTN- U, utilizing oral medications to lower the BP gradually over 24 to 48 h is the best approach to management. Rapid reduction of BP may be associated with significant morbidity due to a rightward shift in the pressure/flow autoregulatory curve in critical arterial beds [14]. In HTN-E, patients require treatment in an ICU with titratable, short-acting IV antihypertensive agent.

The BP should be lowered within minutes to an hour by no more than 25%, and then lowered to 160/100-110mm Hg within the next 2-6 hours. Nicardipine, fenoldopam, ACE inhibitors, angiotensin receptor blockers are used in emergency cases. Recently, clevidipine, a third-generation dihydropyridine calcium-channel blocker was shown in clinical trials to reduce mortality when compared with nitroprusside [15].

AIM OF THE STUDY:

To analyze the clinical presentation, and characteristics of patients presented to the ED with hypertensive crisis and to assess the frequency of the end target organs involved.

PATIENTS & METHODS:

This is a cross-sectional study conducted at the medical emergency department (ED) of Baghdad Teaching Hospital, during the period (Feb. to Sep. 2013) including (306) patients older than 18y admitted with a hypertensive crisis, cases of preeclampsia and eclampsia were excluded. The criteria that were used to determine hypertensive crisis were according to the 7th report of (JNC),[6] which includes values of SBP ≥180 mm Hg or DBP ≥120 mm Hg at the time of admission. This report was the latest report at the time of the study. Patients were differentiated into HTN- E and HTN- U. Hypertensive urgencies are those situations associated with severe HTN without target organ dysfunction. HTN- E was diagnosed when damage to any target organ was evident specifically, the brain, heart, aorta, kidneys, and eyes [16]. Patients were further divided according to age into 5 groups as shown in table 3. Diagnosis was based on clinical history, physical examination, and diagnostic tests [CBP, RBS, (Blood urea, creatinine, cholesterol, TG), troponin, funduscopy, ECG, chest X-ray]. CT- scan of the brain was sent for when necessary. The interpretation of X- ray & CT- scan films was confirmed by a radiologist. The BP level of each patient was measured bilaterally using a mercury-column

sphygmomanometer in supine and standing positions. While waiting for the results; patients were resuscitated and treated according to the type of hypertensive crisis.

Definitions:

- HTN is considered if the patient was on current antihypertensive drugs [17].
- Diabetes melitus is defined as the use diet, insulin or glucose-lowering medication on admission, documented in medical history [18].
- Chronic kidney disease (CKD) is defined as either kidney damage or GFR < 60mL/ min/ 1.73m$^{(2)}$ for ≥ 3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies[19], which were used in our study.
- Acute coronary syndrome (ACS): Presence of precordial pain, ECG changes, and or enzymatic elevation indicative of myocardial damage [20].
- Acute heart failure (AHF): Presence of dyspnea, crackles on thoracic auscultation, and radiographic evidence suggestive of pulmonary venocapillary hypertension [21].
- Ischemic or hemorrhagic stroke: Presence of altered mental state and evidence of ischemic or hemorrhagic events on tomography [22].

Statistical analysis. Data of all patients were entered and analyzed by using SPSS software (version 20). Descriptive statistics for categorical data were presented as frequencies (No.) and proportions (%) and as mean ± standard deviation (SD) for continuous variables. Fisher's exact tests were used to assess the significance of association between types of HTN and age, gender and presentation of the patients. Student's t test was used to compare means. Level of significance (*P*- value) ≤ 0.05 was considered as significant association. Results were presented in tables and or figures.

RESULTS:

Table 1 shows that 186 patients (60.8%) had HTN- E and 120 patients (39.2%) had HTN- U. As shown in table 2; there were 172 (56.2%) males and 134 (43.8%) females, mean age of total sample was (48.7 ± 16.2) years with a range of (21- 86) years. Patients with a HTN- E had greater mean age (56.7± 14.7y) than those with a HTN- U (37.1± 9.7y) with statistically significant difference (*P*- value <0.001), but there was no significant difference regarding gender. Only 34.3% of total sample were current smokers but about 40% of patients with HTN- E were current smokers compared to 27.5% of patients with HTN- U with statistically significant difference (*P*- value 0.034). Half of patient with HTN- U and 46.8 % of patients with HTN- E were known hypertensive on treatment. So 48% of patients with hypertensive crisis ignored their hypertensive status prior to the study. Twenty one percent of patients with HTN- E were known diabetics on treatment. Dyslipidemia was significantly found in 40.3% of patients with HTN- E; while CKD and HF were more prevalent in patients with HTN- U than those with HTN- E; (15% Vs. 8.1%) and (15% Vs. 6.5%) respectively with statistically significant difference (*P*- value 0.028 & 0.014). In HTN- E cases; 14.5 % had history of IHD, and 4.8% had previous CVA but the differences were statistically nonsignificant. Patients with HTN- E had significantly greater mean SBP (204.9± 20.2mmHg) than those with HTN- U (195.0± 10.3mmHg) with (*P*- value <0.001).

Table 3 shows that about 10% of total sample were ≤ 30y while 26.5% were ≥ 60y. There was a direct association between age and both types of hypertensive crisis with statistically significant difference (*P*- value <0.001). Table 4 shows that in HTN- U; males were more frequently affected than females as age increases while in HTN- E; females were more frequently affected after the age of 40y. Mean age of males was more than that of females in both types of HT with statistically significant difference (*P*- value <0.001).

Testing patients with hypertensive crisis showed the following results: mean RBS (142.1± 90.8 mg/dl), mean TG (204.9±33.1mg/dl), mean cholesterol (240.7 ± 55.1 mg/dl), mean Bl. urea (67.5±42.7 mg/dl), and mean creatinine (2.1±0.12 mg/dl). Troponin test was positive in 14.7%, ischemic ECG changes were found in 23.7% and LVH in 2.9%. Cardiomegaly by CXR was found in 18.6% of patients. CT- scans showed ischemic changes in 4.8% while hemorrhage was seen in 2% of patients. CT- scan was normal in 92.2% as shown in table 5.

Patients with HTN- E presented with: Dyspnea in (30%), epistaxis in (29.03%), decrease urine output (UOP) in (14.52%), and disturbed consciousness in (6.45%); with statistically significant difference as shown in table 6. While patients with HTN- U presented with: Headache in (30%), chest pain in (27.50%), vomiting in (25%), with statistically significant difference.

Fundoscopic examination showed papilledema in 30.65% of patients with HTN- E compared to 25% of those with HTN- U with statistically significant difference (*P*- value < 0.001). Table 7 shows the distribution of cases of HTN- E in the population studied. Most cases of HTN- E corresponded to acute MI (25.3%) followed by encephalopathy (21%), renal failure (20.2%) which included acute renal failure and CKD, HF (17.2%), and CVA (11.3%).

Table 1: Distribution of Types of Hypertensive Crisis among Study Sample.

Type of Hypertension	No.	% from sample (N=306)
Hypertensive Emergency	186	60.8
Hypertensive Urgency	120	39.2
Total	306	100%

Table 2: Baseline and clinical characteristics of patients.

Variable	Hypertensive Crisis (n=306)	Urgencies (n=120)	Emergencies (n=186)	*P*- value
Age, y (mean± SD**)	48.7 ± 16.2	37.1 ± 9.7	56.7 ± 14.7	<0.001*
Male, %	56.2	55.0	57.0	0.91
Female, %	43.8	45.0	43.0	
Medical history				
Current smoker, %	34.3	27.5	39.3	0.034*
Hypertension, %	48	50.0	46.8	0.58
Diabetes, %	22.5	25.0	21.0	0.45
Dyslipidemia, %	35.7	28.6	40.3	0.01*
Heart failure, %	9.8	15.0	6.5	0.014*
CKD, %	10.8	15.0	8.1	0.028*
IHD, %	17.6	22.5	14.5	0.082
CVA, %	5	5.0	4.8	0.92
Presentation				
SBP, mm Hg (mean± SD)	201± 20.2	195.0± 10.3	204.9± 20.2	<0.001*
DBP, mm Hg (mean± SD)	131.3± 10.7	120.7± 13.5	121.6± 14.4	0.45

* Statistically significant.
** SD, standard deviation.

Table 3: Distribution of types of hypertensive crisis according to age groups.

Age group (y)	Hypertensive Crisis		HTN- U		HTN- E		*P*- value
	No.	%	No.	%	No.	%	
≤ 30	30	9.8	10	8.3	20	10.8	<0.001*
30-39	42	13.7	20	16.7	22	11.8	
40-49	72	23.5	27	22.5	45	24.2	
50-59	81	26.5	33	27.5	48	25.8	
≥ 60	81	26.5	30	25.0	51	27.4	
Total	306	100	120	100	186	100	

* Statistically significant

Table 4: Association between type of hypertensive crisis and age distributed by gender.

Age group (y)	HTN- Urgency (No. = 120)		HTN- Emergency (No. = 186)		*P*- value
	Male No (%)	Female No (%)	Male No (%)	Female No (%)	
≤ 30	10(8.3)	0(0.0)	15(8.1)	5(2.7)	
30-39	12(10.0)	8(6.6)	17(9.1)	5(2.7)	
40-49	15(12.5)	12(10.0)	21(11.3)	24(13.0)	
50-59	17(14.2)	16(13.3)	23(12.4)	25(13.4)	
≥ 60	12(10.0)	18(15.0)	30(16.1)	21(11.3)	
Total	66(55.0)	54(45.0)	106(57.0)	80(43.0)	
Mean age (y) ± SD**	49.2 ± 11.7	35.6 ± 9.1	59.1 ± 14.2	54.9 ± 13.3	<0.001*

* Statistically significant
** SD, standard deviation.

Table 5: Results of investigations performed for patients with hypertensive crisis.

Investigation	Mean ± SD	No.	%
RBS (mg/dl) *	142.1 ± 90.8	-	-
TG (mg/dl) **	204.9 ± 33.1	-	-
Total serum cholesterol (mg/dl)	240.7 ± 55.1	-	-
Blood urea (mg/dl)	67.5 ± 42.7	-	-
Serum creatinine (mg/dl)	2.1 ± 0.12	-	-
Troponin	Positive	47	15.4%
	Negative	259	84.6%
	Ischemic changes	71	23.2%
	Sinus tachycardia	24	7.8%
ECG findings	LVH	9	2.9%
	Tented T- wave	18	5.9%
	AF	12	3.9%
	LBBB	3	1.0%
	Normal	168	54.9%
	Cardiomegaly	57	18.6%
Chest X-ray findings	Lobar pneumonia	6	2.0%
	Normal	243	79.4%
	Ischemic changes	15	4.8%
CT- scan findings	Hemorrhage	6	2.0%
	Brain atrophy	3	1.0%
	Normal	282	92.2%

* RBS, random blood sugar.
** TG, triglyceride.

Table 6: Correlation of Type of Hypertensive Crisis and Clinical Presentation of Patients.

Presentation	HTN-U (N=120)	HTN-E (N=186)	*P*- Value
Chest pain	27.50%	17.74%	0.016*
Palpitation	2.50%	3.23%	NS
Gallop	5.00%	4.84%	
Dyspnea	17.50%	30.00%	0.001*
Orthopnea	0.00%	1.61%	
Hemoptysis	0.00%	1.61%	
Epistaxis	11.90%	29.03%	0.032*
Basal crepitation	7.50%	14.52%	0.003*
Decrease UOP	7.50%	14.52%	<0.001*
Earthy face	7.50%	14.52%	
Vomiting	25.00%	21.22%	0.001*
Anorexia	0.00%	5.00%	
Headache	30.00%	17.74%	0.004*
Syncope	0.00%	3.23%	0.016*
Disturbed level of	5.00	6.45%	0.004*
Weakness	2.94%	6.45%	
Fit	0.00%	1.61%	
Confusion	0.00%	1.61%	
Fundoscopic Examination			
Papilledema	25.00%	30.65%	< 0.001*
Retinal edema	2.50%	11.29%	
Flame shape hemorrhage	5.00%	1.61%	
Cotton wool	5.00%	0.00%	
Optic disk atrophy	0.00%	1.61%	
Silk wiring	2.50%	0.00%	

* Statistically significant NS, statistically nonsignificant

Table 7: Distribution of Cases of Hypertensive Emergency.

Diagnosis	No.	%
Acute myocardial infarction	47	25.3
Encephalopathy	39	21.0
Renal failure	38	20.2
Acute heart failure	32	17.2
CVA*	21	11.3
Unstable angina	9	5.0
Total	186	100

CVA*, cerebrovascular accident

DISCUSSION:

In this study, the frequency of HTN- E was more than HTN- U (58.8%, 41.2%). Similar finding was found in Vilela JF study[23](63.8%, 36.2%) while Al-Bannay R[24] reported 36% HTN- E. Men had a greater frequency of hypertensive crises than did women. Similar finding was found in Al-Bannay R[24] study where the male to female ratio was 2:1. Our study shows a direct association between age of men and frequency of both types of hypertensive crisis but women were more affected after the age of 50y.

As revealed in the Framingham study, which showed that the incidence of coronary arterial disease in men increased in an almost linear mode as age increased [25], differently from that observed in women, who are protected until menopause. In other words, the morbidity and mortality rates associated with any level of BP are lower in women than in men up to 45 years of age [26].

Current smoking was evident in 34.3% of the study group but it was a statistically significant in HTN- E compared to HTN- U. Still tobacco has acute and chronic hypertensive actions, such as inhibition of endothelial cyclooxygenase, leading to a reduction in the production of prostacyclin and an increase in the synthesis of thromboxane, a situation that may cause chronic vasoconstriction and damage to the endothelial cell [27].

In this study, DM was found in about one fourth of patients with hypertensive crises but it did not reach the level of significance. In Roubsanthisuk W[28] study, nearly half of 184 patients studied had DM. The etiological link between insulin resistance and HTN becomes progressively greater. Metabolic abnormalities (hyperglycemia, hyperinsulinemia, and dyslipidemias) may play a role in the pathogenesis and complications of HTN. Mechanisms of diabetes-induced endothelial dysfunction include the production of prostanoid vasoconstrictors and degradation of the vasodilator nitric oxide, this increases vascular resistance and promotes atherogenesis and HTN in this group [29].

This study shows that dyslipidemias was a significant risk factor. It is one of the strong predictors of cardiovascular disease, causes endothelial damage, loss of physiological vasomotor activity, increased oxidative stress, progression of atherosclerosis, and ends up with a major CV event that may manifest as severe HTN [30].

In this study; 48% of patients with hypertensive crises ignored their HTN status before presentation to ED, this is why only 2.9% of them had LVH as a sign of chronic HTN. In the study by Martin JFV [31], only 18% were not hypertensives. The JNC 7 noted that approximately 30% of adults were unaware of their HTN; up to 40% of people with HTN were not receiving treatment; and of those treated, up to 67% did not have their BP controlled to less than 140/90mm Hg.[4] Controlling arterial HTN is not an easy task, as its chronic insidious character contributes to non-compliance with treatment. Estimates indicate that two-thirds of the people utilizing basic health care services do not keep their BP within desirable levels [32].

The present study shows that in HTN- E, the most frequent symptom and sign were dyspnea and papilledema. Similar result was reported in the review of severe HT by John DB.[33] In HTN-U, the most common clinical complaints were headache and chest pain, as also reported by Bender SR [34]. The present study shows that, among HTN- E, cardiovascular lesions were the most common, and among the latter, acute MI was the most frequent, followed by left ventricular failure. Similar findings were reported by Cerrillo MR et al[35]. In our study, encephalopathy was the second common target lesion followed by renal failure.

CONCLUSION:

In our institution, HTN- E was more common than HTN- U. Males were more frequently affected, and patients with HTN- E had a significantly greater mean age than those with a HTN- U. Smoking & dyslipidemia were significant risk factors for HTN- E. HTN was unknown to 48% of patients. The most frequent clinical manifestations of HTN- E were dyspnea and papilledema. Acute MI, encephalopathy, followed by renal failure were the most frequent target organ lesions in HTN- E.

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