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ABSTRACT

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Background: Studies on the prevalence of chronic kidney disease (CKD) in patients presenting with hypertensive crises are scarce. This study aimed to determine the prevalence of CKD in patients presenting with hypertensive crises (HTN-C) and their 30-day outcomes concerning blood pressure, creatinine level, and mortality.

Methods: Patients admitted with HTN-C were prospectively enrolled. Demographics, examinations, target organ damage (TOD), and investigations were recorded. Patients were classified as having a hypertensive emergency (HTN-E) or hypertensive urgency (HTN-U). Target organ damages included cardiovascular (acute coronary syndromes and pulmonary edema), neurological (stroke and encephalopathy), ocular (retinal hemorrhage and papilledema), renal (acute kidney injury (AKI)), and hematological (microangiopathic hemolytic anemia). The CKD burden was also determined. Blood pressure (BP), creatinine, and mortality were assessed at discharge and after 30 days. Acute kidney injury on CKD was confirmed at follow-up with a change in creatinine from the baseline of ≥25%.

Results: Out of 262 patients, 235 had HTN-E; patients were mostly males. Target organ damages, in decreasing frequency, were cardiovascular (53%), ocular (29%), neurological (26%), and AKI (24%). Patients with CKD were significantly younger, had hypertension treated with ≥2 drugs, and had a higher proportion of TOD. Patients with CKD had significantly higher BP and creatinine levels at discharge and at 30 days. The non-CKD group had a higher inpatient mortality rate and a lower mortality rate at 30 days.

Conclusion: Better community-level support is required for timely assessment of the diagnosis and progression of CKD, recognition and treatment of AKI, BP control, and regular kidney replacement therapy, which may reduce the load on emergency departments.

Keywords: Hypertensive crisis, chronic kidney disease, target organ damage, prevalence, outcomes.

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INTRODUCTION

Hypertension affects approximately 25% of the global populace. It is a leading cause of heart and kidney disease and premature death. The disease burden is higher in developing and underdeveloped countries where awareness, treatment, and control are lower than in high-income countries. At some point in their lifetime, 1%-2% of hypertensives are at risk of experiencing a hypertensive crisis. The estimated incidence rate in the population is approximately 1-2 cases per million per

year.³ A hypertensive crisis is defined as: "systolic blood pressure (SBP) >180 mm Hg and/or diastolic blood pressure (DBP) >120 mm Hg." It can occur with or without target organ damage (TOD), classifying it as hypertensive emergency (HTN-E) or hypertensive urgency (HTN-U), respectively.³ Hypertensive crises are more often seen in the elderly and women.²

Chronic kidney disease (CKD) is defined as kidney damage lasting for more than 3 months. This damage is

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identified by abnormalities in structure or function that may or may not be accompanied by a reduction in glomerular filtration rate (GFR). "Pathological abnormalities or markers of kidney damage, including abnormalities in the composition of blood or urine, or abnormalities in kidney tests, may also be present."

Hypertension is closely related to CKD.4 Reduced GFR, reduced nephron mass, impaired tubular handling of sodium, and activation of the renin–angiotensin–aldosterone system and sympathetic system contribute to arterial hypertension. Despite a normal GFR, the development of arterial hypertension can be attributed to maladaptive alterations in the handling of tubular sodium and chloride.

Therefore, progressive kidney disease worsens poorly controlled hypertension due to increased volume and higher systemic vascular resistance. Glomerular and interstitial fibrosis may occur due to hypertension-related intraglomerular hypertension.⁶ Glomerular damage leads to hyperfiltration and proteinuria.⁷ Arterial elasticity may be affected due to chronic hypertension-related volume overload, which can reduce blood flow to the nephrons.6 Data from 2 major trials showed that blood pressure control was associated with a reduced risk of kidney failure, especially in patients with proteinuria, older individuals, or those with a higher body mass index.8 According to the AProDiTe-2 study, optimal blood pressure management for 12 months in patients demonstrated significantly improved kidney outcomes over 5 years within a CKD cohort from South Korea. Lv et al, in their meta-analysis of 11 trials, found that intensive blood pressure control in CKD patients with proteinuria was protective against kidney failure events, such as the doubling of serum creatinine and a >50% fall in GFR.¹⁰

Limited research has been conducted on the occurrence of CKD in patients with hypertensive crises. However, the STAT registry, a retrospective cohort study conducted in the United States, found that 79% of patients who presented with a hypertensive crisis also had CKD.¹¹ Eleven percent of the cohort was dialysis-dependent, and 64% developed acute kidney injury (AKI). According to another study, 27% of patients in Austria who experienced a hypertensive crisis also had kidney impairment.¹² There have been several studies conducted in India on

MAIN POINTS

- Studies on the burden of CKD in patients with hypertensive crises are limited. In our study, the prevalence of CKD in patients who presented with hypertensive crises was 48%.
- Patients with CKD were younger and more likely to have hypertension, with the use of 2 or more antihypertensive agents.
- Both BP and kidney dysfunction were significantly higher in patients with CKD at the time of discharge but differed during follow-up.
- Twenty-seven percent of patients with hypertensive crises did not have any target organ damage at presentation.

hypertensive crises, but none have explored the relationship between hypertensive crises and CKD.¹³⁻¹⁷ In a 2022 review, stroke was found to be the most common presentation of hypertensive emergencies in Asian patients. The review did not examine the presence of CKD.¹⁸ The incidence of CKD was found to be considerably high in individuals who presented with a hypertensive crisis. However, it has not been previously quantified in developing countries such as India, where the burden of CKD is reasonably known. Furthermore, knowledge regarding the relationship between chronic kidney dysfunction and its effects during hypertensive crises is limited. Most studies on CKD and hypertensive crises have been retrospective analyses using large databases. Hence, we prospectively aimed to understand the prevalence and outcomes of CKD in patients presenting with hypertensive crisis.

MATERIAL AND METHODS

The Jawaharlal Institute of Postgraduate Medical Education and Research Ethics Committee (approval number: IEC/2020/097, date: July 24, 2020) approved this study. All participants provided written informed consent before participation. This prospective hospital-based observational study was conducted between July 1, 2020 and July 31, 2022. Patients aged ≥18 years who presented to the emergency medical services (EMS) with a SBP ≥180 mm Hg or DBP ≥120 mm Hg were recruited. Pregnancy and head trauma associated with hypertensive crises were excluded from this study. This study was designed to determine the CKD burden in patients presenting with hypertensive crises and their outcomes at 30 days. Assuming a prevalence of 80% and a precision of 5% to achieve a power of 80%, with an attrition rate of 10% during follow-up, a sample size of 270 was calculated.¹¹

The subjects were screened at the EMS for the diagnosis of a hypertensive crisis (≥180 / ≥120 mm Hg). Supine blood pressure was measured in both arms (or in the non-arteriovenous fistula arm), and a higher reading was obtained. Target organ damage in the form of fundus examination, serum creatinine, urinalysis, electrocardiogram (ECG), and, if required, troponin-T, peripheral smear, ultrasonography (USG) of the abdomen, echocardiography, and computed tomography (CT) of the brain were performed. Patients with any TOD, such as cardiac (acute coronary syndrome (ACS), acute pulmonary edema, or acute heart failure), aortic (aortic dissection), neurological (stroke, hypertensive encephalopathy), kidney (AKI), or retinal (acute hypertensive retinopathy), were considered to have a HTN-E. Patients without any TOD were classified as HTN-U patients.

This study recorded patient details such as demographics, medical history, and treatment for CKD, diabetes, hypertension, ACS, stroke, and drug therapy. The diagnosis of diabetes was based on documented evidence of high blood sugar levels according to ADA criteria, treatment with oral medications or insulin, or diabetic retinopathy.¹9 A history of hypertension was confirmed based on previous recordings (if available) of BP ≥140/90 mm

Hg, treatment with antihypertensives, left ventricular hypertrophy on electrocardiogram/echocardiography, or a combination of these factors. ST-elevation myocardial infarction, non-ST-elevation myocardial infarction, and unstable angina were considered as acute coronary syndromes. These patients were diagnosed in the EMS by the presence of angina or anginal equivalents, along with diagnostic ECG changes and/or troponin T levels. Stroke was defined as a history of transient ischemic attack, hemiplegia, or hemiparesis, with or without cranial nerve deficits, or CT evidence of ischemia or hemorrhage. Acute kidney injury was diagnosed according to the KDIGO 2012 criteria.²⁰ Acute kidney injury on CKD was diagnosed only on followup when the serum creatinine level showed a change of at least ≥25% compared to that on admission. Those without such a reduction (or a lack of prior creatinine values) were considered to have probable underlying CKD. A diagnosis of CKD was considered in patients with a previous diagnosis during follow-up at either the Department of Nephrology or Medicine. For those patients visiting the institution for the first time, CKD was diagnosed according to KDIGO criteria.4

The examination findings included temperature, vital signs, pallor, and elevated jugular venous pulse. The cardiorespiratory examination included crackles, S3 and S4, murmurs, and rubs. The neurological examination included focal neurological deficits and altered consciousness. The following parameters were measured: glomerular filtration rate (CKD-EPI), complete blood count with or without peripheral smear, urea, creatinine, urinalysis, liver function tests, blood culture, USG KUB, chest radiography, electrocardiogram, echocardiography, arterial Doppler/CT angiography, and CT brain. Information related to CKD included etiology, duration, treatment (antihypertensive number, dose, compliance, and control, hematinics, statins, and others), and dialysis (presence of fistula/catheter, number of days on dialysis, number of dialyses, and duration per week).

Patients who experienced a hypertensive crisis were examined in the EMS department during their initial visit and monitored until discharge from the hospital. The cause of the hypertensive crisis, whether it was related to CKD, the duration of intravenous antihypertensive therapy, number of oral medications prescribed, number and type of TOD, and whether the patient died due to the illness were all recorded. A follow-up appointment was scheduled 30 days after discharge to assess kidney function (serum creatinine) and blood pressure readings. Patients who did not attend the follow-up appointment were contacted by phone to determine the reasons for their absence.

Statistical Analysis

Continuous variables such as age, duration of CKD, blood pressure, serum creatinine, blood urea, and estimated GFR were summarized using means or medians, depending on the distribution of the data. Categorical data, such as sex, CKD stage, kidney replacement therapy, presence of diabetes mellitus,

hypertension, stroke, AKI, blood pressure control, and survival until discharge and 30 days, were summarized using frequency and percentage. To determine the association between the presence of CKD, hypertensive crisis, and categorical variables, the chi-squared test or Fisher's exact test was used. To compare the mean and median of continuous variables between patients with and without CKD, independent sample t-tests and Mann-Whitney U-tests were performed. All statistical tests were performed at the 5% significance level.

The dataset is available at 10.6084/m9.figshare.21820632.²¹

RESULTS

There were 262 patients, with 71% males, in this study. The most common complaint was breathlessness, followed by limb weakness (Table 1). More than half of the patients were taking ≥2 antihypertensives (Table 1). Twenty-seven patients did not exhibit target organ damage (hypertensive urgency). Deranged 29 kidney function was observed in 66% of patients, with prior CKD in 36% (n = 93), of whom 53 were undergoing kidney replacement therapy (KRT). Ninety percent of the patients had evidence of TOD, with pulmonary edema being the most typical manifestation (Table 2).

The prevalence of CKD was 48% in patients who experienced a hypertensive crisis. In nearly half of the cases, the etiology of CKD was unknown, and almost 50% of the patients with CKD had kidney failure (Figure 1A). Patients with CKD were younger and more likely to have hypertension treated with 2 or more antihypertensive agents (Table 2).

Only the duration of symptoms was significantly longer in patients with hypertensive urgency than in those with HTN-E (Table 3). Thirty-four (34/127) patients were newly diagnosed with CKD at admission, compared to 93 (93/127) who already had CKD. Prior hypertension and clinical findings, such as crepitations and altered consciousness, differed significantly between patients with prior and newly diagnosed CKD (Table 4).

Both BP and kidney dysfunction were significantly higher in the CKD group at discharge. However, the same was not reflected 1 month after discharge (Table 5).

DISCUSSION

Hypertensive emergencies occur when blood pressure readings reach or exceed 180/120 mm Hg, leading to new or worsening damage to target organs.²² The condition known as HTN-E was initially reported by Volhard and Fahr in 1914. They categorized it as a disorder with severe accelerated hypertension, associated with kidney disease and vascular injury to the heart, brain, retina, and kidney, ending fatally.23 It is estimated that 1% of people with high blood pressure may experience a hypertensive crisis at some point. Chronic kidney disease is a significant global public health concern. The SEEK study found that 17.2% of the Indian population has CKD.²⁴

Table 1. Baseline Characteristics	
Parameter (n = 262)	Mean ± SD/ Percentage
Age (years)	52.9 ± 13.4
Male gender	187 (71%)
Blood pressure at presentation Systolic BP ≥180 mm Hg Diastolic BP ≥120 mm Hg	262 (100%) 56 (21%)
Presenting complaints Breathlessness Weakness of limbs Altered consciousness Chest pain	126 (48%) 48 (18%) 26 (10%) 23 (9%)
Duration of symptoms (days)	3.7 ± 7.5
Duration of hospital stay (days)	10.5 ± 8.7
Deranged kidney function (at admission)	173 (66%)
Co-morbidities	
Prior hypertension Duration of hypertension (years) Number of anti-hypertensives prior to admission 1 2 3 4 5 Compliance with anti-hypertensives	173 (66%) 5.6 ± 5.0 1.9 ± 0.8 51 (30%) 92 (53%) 23 (13%) 5 (3%) 2 (1%) 118 (68%)
Prior CKD Duration of CKD (years) On kidney replacement therapy	93 (36%) 2.5 ± 2.1 53 (57%)
Prior diabetes mellitus Duration (years)	98 (37%) 9.0 ± 6.7
Prior coronary artery disease Duration (years) New acute coronary syndrome	21 (8%) 4.0 ± 3.2 34 (13%)
Prior stroke Duration (years) New stroke	5 (2%) 7.0 ± 4.7 64 (24%)

The prevalence of hypertension in CKD varied in many studies, ranging from 35.8% in stage 1 to 84.5% in stage 5, often affecting up to 100% of patients and contributing to disease progression. Renovascular diseases had a higher prevalence of hypertension. The population-level prevalence of CKD in adults with hypertension in South Asia was 27% (CI: 20%-35%). One-quarter to two-fifths patients with CKD have resistant hypertension. A reduction in GFR is associated with increases in nocturnal BP and albuminuria.

Hsu et al studied 316675 patients without baseline kidney disease and observed kidney failure in 1149 subjects over 8210431 person-years of follow-up; the study showed a graded relationship between hypertension and kidney failure. The risk of developing kidney failure was 1.6-fold for blood

pressure 120-129/80-84 mm Hg and 4.2 times for blood pressure $\geq 210/120$ mm Hg compared to subjects who had blood pressure <120/80 mm Hg. 28 Between 2006 and 2013, hypertensive emergencies occurred in about 2 of every 1000 emergency room visits in the United States, according to data from the Nationwide Emergency Department Sample. 29 The lack of a primary care physician and failure to comply with prescribed antihypertensive regimens were major risk factors for hypertensive emergencies in another study of a minority population. 30 The median survival for untreated hypertensive emergencies is 10.4 months, with a death rate of over 79% within a year. 31

A study using the National Inpatient Sample in the United States found that 15% of hypertensive emergencies had a coexisting diagnosis of kidney failure.³² Hospitalizations with coexisting kidney failure had higher odds for pulmonary edema and cardiac arrest compared to those without, but there was no significant difference in inpatient mortality rates. In contrast, the current study showed that 48.4% had CKD (prior and newly diagnosed) and 20.2% had kidney failure (dialysis-dependent). A higher mortality in the non-CKD group due to hemorrhagic strokes and acute coronary syndrome was observed; a higher proportion of pulmonary edema was seen in the CKD group, similar to the National Inpatient Sample (Tables 2 and 5).

In a retrospective study conducted in Korea over a 3-year period, among those with an eGFR <30 mL/kg/min, HTN-E and HTN-U contributed to 46.2% and 34.9% of mortality, respectively.33 The 1-month mortality in those with a GFR <90 mL/kg/ min was 6.2%, slightly less than that of the current study (n = 8 / 105; 7.6%). The inpatient mortality in the CKD group was 6.8% (n = 8/116) compared to 17.4% in those without CKD, contributed mainly by strokes (5 vs. 59) and ACS (3 vs. 32). Mortality rates in the non-CKD group at follow-up were not significantly different from those with CKD, ostensibly due to improvements in kidney function and control of blood pressure (7% vs. 7.6%). Emergency department readmissions within 1 month were higher in patients with a lower GFR.³³ The current study showed that in patients experiencing a hypertensive crisis, 48% had CKD in comparison to 79% from the STAT registry, a retrospective cohort study in the US population. 11 Higher blood pressure and cardiac dysfunction were associated with worse CKD at presentation, with a higher proportion of cardiac-related TOD was found. Similarly, the proportion of pulmonary edema (98.7% vs. 64.3%) was higher in the CKD group of the current study, but with less ACS (2.4% vs. 57.1%).

Pothuru et al³⁴ studied 680 333 patients with hypertensive crisis from the Nationwide Emergency Department Sample database. They found that only 11.4% and 6.6% had CKD and kidney failure, respectively. Among 979 Austrian patients admitted with hypertensive crisis, kidney dysfunction was observed in 27%; however, CKD was not identified in this study. Kidney dysfunction was linked to an increased risk of HTN-E and a higher number of hypertensive medications.¹²

Table 2. Comparison between Patients With and Without CKD			
Parameter	CKD Group (n = 127)/Mean ± SD/ Percentage	Non-CKD Group (n = 135)/Mean ± SD/Percentage	P
Age (years)	49.4 ± 13.4	56.1 ± 12.6	<.01
Gender Male	94(74)	93(69)	.36
Complaints Breathlessness Chest pain Altered sensorium Weakness of limbs	96(76) 1(1) 7(5.5) 7(5.5)	30(22) 22(16) 19(14) 41(30)	<.01
Duration of illness (days)	4.3 ± 6.2	3.0 ± 8.6	.16
Duration of hospital stay (days)	10.6 ± 8.7	10.5 ± 8.8	.93
Hypertensive crisis (n) Emergency Urgency	111(87) 16(13)	124(92) 11(8)	.24
Co-morbidities Prior hypertension Duration of hypertension (years) Anti-hypertensives at admission (n) Compliance to therapy	103(81) 5.1 ± 4.9 2.2 ± 0.8 83(81)	70(52) 6.2 ± 5.1 1.6 ± 0.6 35(50)	<.01 .16 <.01 <.01
Prior diabetes mellitus	42(33)	56(42)	.16
Prior coronary artery disease	9(7)	12(9)	.59
Prior stroke	3(2)	2(2)	.60
Target organ damage	126(93.3)	109(85.8)	.04
Eye	31(24)	44(33)	.14
Renal (acute kidney injury)	44(35)	20(15)	<.01
Cardiovascular Acute coronary syndrome (ACS) ACS and pulmonary edema Pulmonary edema	82(65) 1 2 79	56(42) 20 12 24	<.01
Central nervous system Hemorrhagic stroke Ischemic stroke Hypertensive encephalopathy Posterior reversible encephalopathy syndrome	7(1) 5 0 1	62(46) 40 19 3 0	<.01
Pulse rate (per minute)	103 ± 82	91 ± 17	.09
Respiratory rate (per minute)	26 ± 6	20 ± 6.5	<.01
Elevated jugular venous pulse	35(28)	14(10)	<.01
Crepitations	99(78)	37(27)	<.01
Altered sensorium	12(9)	48(36)	<.01
Focal neurological deficit	7(6)	56(42)	<.01
Hemoglobin (g/dL)	7.2 ± 1.7	12.4 ± 2.9	<.01
White blood cells (per mm³)	11.20 ± 6.03	13.03 ± 4.54	<.01
Urea (mg/dL)	125 ± 68	47 ± 39	<.01
Serum creatinine (mg/dL)	8.9 ± 4.9	1.6 ± 1.9	<.01

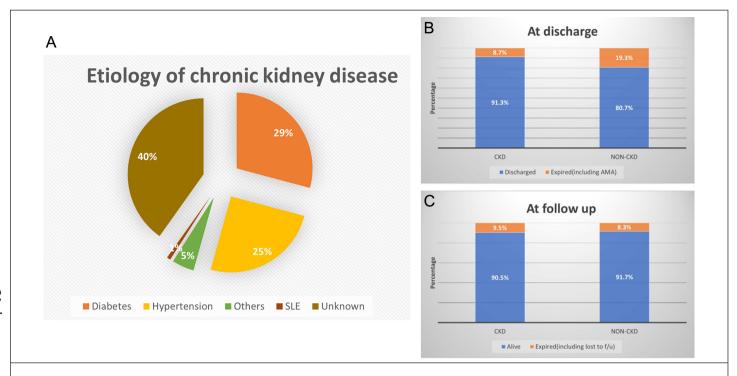


Figure 1. Etiology of chronic kidney disease.

There were 8 studies from India on hypertensive crises (range: 30-100 patients), with only 1 study having follow-up data. 13-17,35-³⁷ Kidney dysfunction was not evaluated in 4 studies. ^{13,16,17,37} Anitha et al³⁵ conducted a study on 30 patients who had acute severe hypertension and kidney dysfunction and underwent kidney biopsy. Of these patients, 6 had CKD, and 1 of them was on KRT. Over the course of 10 years, 6 of the remaining 25 patients developed kidney failure. Unfortunately, 3 patients died. According to a retrospective study by Singh et al in Haryana, 8% of patients (8/100) had CKD, and 24 had kidney dysfunction overall.14 About 12% of patients admitted to the intensive care unit (ICU) for HTN-E (6/50) had CKD in a study from Maharashtra. 15 Only HTN-E was studied; in contrast, 13% (16/127) of the CKD group in the current study had HTN-U. The second ICU study had kidney involvement in 24% (n = 14), but the type of dysfunction was not characterized.³⁶

Stroke is the most common TOD in Asians; 18 in contrast, cardiovascular (53%) and ocular (29%) manifestations were more common in our study. Cardiovascular dysfunction was observed in 56% (n = 28) and 69% (n = 138) of patients from Visakhapatnam and Gujarat, respectively. Acute (n = 15) and LVF (n = 13) were the most common types of cardiovascular dysfunction. 16,17 The Haryana (36%) and Nellore (56%) cohorts had more neurological complications. 13,14 In 2 ICU studies, retinopathy was found to be the most common TOD (88%, n = 44) in the first study, while cardiac and neurological complications were equally prevalent in 67% (n = 39) in the second study. 15,36 In Zaheerabad, Telangana, retinopathy (55, 36.6%) was the most frequent complication. 37

Deshmukh et al² analyzed Nationwide Inpatient Sample data from 2000 to 2007 to investigate hypertensive emergencies. They found that hospitalization rates increased by an average of 10%, with a larger proportion of black and female patients hospitalized. However, mortality rates decreased after 2005. Additionally, being uninsured, male, older, and having multiple comorbidities were all factors that increased the likelihood of mortality. In the group of patients without CKD, there was a significantly higher rate of mortality during hospitalization compared to the CKD group (19.2% vs. 8.7%) (Figure 1B and C). The difference in the number of patients with stroke and ACS between the CKD and non-CKD groups may have influenced our results. However, this difference was not observed during the 1-month follow-up. According to Pothuru et al, patients with CKD had a higher proportion of patients with HTN-E (38%) as compared to the non-CKD group (22.4%), and a higher mortality rate of 0.3% as compared to 0.1% in the non-CKD group.34 There was no follow-up data. The corresponding figures for the current study were 87% vs. 92% and 17.3% vs. 25.9%, respectively. The mortality rate of patients with HTN-E was 14% in a study by Rao et al. Of the 50 patients studied, 5 had intracerebral hemorrhage and 2 had LVF. 16 Salagre et al 36 showed a mortality rate of 15.8%, all within 72 hours of admission. Eighteen patients (9%) died in the Gujarat study.¹⁷ Outcomes in terms of mortality were not studied in the remaining 4 studies. 13-15,37

Sustained hypertension can cause kidney function to worsen, and declining kidney function can lead to poor blood pressure control.⁴ The CKD group in this study had poorer blood pressure control and kidney function at discharge and at 1-month

.39

Parameter	Hypertensive Emergency (n = 235) Mean ± SD/	Hypertensive Urgency (n = 27) Mean ± SD/	P
Males (n)	168	19	.90
Age (years)	52.93 ± 13.1	52.33 ± 15.2	.82
Duration of illness (days)	2.8 ± 3.7	10.4 ± 19.7	<.001
Prior hypertension (n)	150	23	.02
Duration of hypertension (years)	5.6 ± 4.9	4.9 ± 5.5	.50
Compliance to therapy (n)	104	14	.41
Prior CKD (n)	80	13	.14
Duration of CKD (years)	2.2 ± 1.8	3.5 ± 2.9	.16
Hemodialysis (n)	44	9	.33
Duration of hemodialysis (months)	13.9 ± 16.7	22.2 ± 24.2	.21
Prior diabetes (n)	85 ± 3.5	13	.23
Systolic blood pressure SBP (mm Hg)	193.9 ± 17.2	187.0 ± 10.3	.04
Diastolic blood pressure DBP (mm Hg)	106.7 ± 12.1	103.4 ± 10.6	.17
Hemoglobin (g)	10.0	8.3 ± 2.6	.02
Echocardiography left ventricular hypertrophy (n)	126	14	.86
Duration of hospital stay (days)	10.6 ± 8.9	9.7 ± 6.4	.63
In-hospital death (n)	26	0	.08
SBP at discharge (mm Hg)	136.3 ± 7.4	136.8 ± 6.0	.75
DBP at discharge (mm Hg)	90.2 ± 7.7	89.0 ± 7.8	.45
Death within 1 month (n)	15	0	.13
SBP at 1month (mm Hg)	136.8 ± 12.2	137.4 ± 8.5	.80
DBP at 1 month (mm Hg)	88.6 ± 8.8	89.4 ± 7.5	.63

 2.8 ± 2.5

follow-up compared to the non-CKD group, despite better compliance with anti-hypertensive therapy (Table 5). Blood pressure at discharge and during follow-up has not been evaluated in other studies conducted in India or elsewhere.

Strengths and Limitations

Creatinine at 1 month follow-up (mg/dL)

We conducted a prospective study with a larger sample size than similar studies. The participants were recruited and underwent a thorough examination to identify any TOD, which was not performed in larger studies. A 1-month follow-up was performed to record blood pressure and serum creatinine levels. The purpose of this follow-up was mainly to differentiate between AKI on CKD and newly diagnosed CKD in patients presenting with hypertensive crises.

We are a tertiary teaching institute that offers nephrology and transplant services. As a result, we have a large number of patients with CKD who are under follow-up. Our study found that this factor may have played a role in the higher prevalence observed. However, it is important to note that our study was conducted in a single center; therefore, the results may not be representative of the entire Indian population. As we included only admitted patients, we may have missed a larger number of patients with hypertensive urgency who sought treatment in the outpatient department or were discharged directly from EMS. In addition, we could not differentiate between acute pulmonary edema due to heart failure, volume overload related to kidney disease, or a combination of both.

 3.3 ± 2.7

Unfortunately, we did not have data on how many participants with CKD were advised to undergo KRT but did not initiate it and therefore presented with a hypertensive crisis due to the delay in initiation. Additionally, it is possible that we missed some patients with AKI among those undergoing maintenance dialysis, as a marginal elevation in creatinine levels may have been attributed to a delay or inadequate dialysis. Finally, owing to

Table 4. Comparison Between Patients with Prior and Newly Diagnosed CKD

Parameter	Newly Diagnosed CKD (n = 34) Mean ± SD	Known CKD (n = 93) Mean ± SD	P
Age (years)	49.9 ± 10.8	49.2 ± 14.2	.78
Gender (n)	28	66	.19
Duration of illness (days)	5.9 ± 3.4	3.7 ± 5.3	.07
Duration of hospital stay (days)	8.6 ± 3.4	11.2 ± 9.9	.03
Prior hypertension	15	88	<.01
Hypertensive emergency at admission (n)	31	80	.43
Duration of hypertension (years)	7.2 ± 5.7	4.7 ± 4.6	.06
Compliance to antihypertensives (n)	10	73	.14
Systolic blood pressure SBP (mm Hg)	195.1 ± 19.3	194.9 ± 17.3	.96
Diastolic blood pressure DBP (mm Hg)	110.3 ± 13.4	107.5 ± 10.9	.23
Pulse rate (beats/min)	92.5 ± 16.8	107.0 ± 95.2	.38
Crepitations (n)	21	78	.008
Altered consciousness (n)	6	6	.05
Hemoglobin (g/dL)	6.9 ± 1.7	7.2 ± 1.7	.35
Urea (mg/dL)	161.0 ± 79.1	111.5 ± 58.2	.002
Creatinine (mg/dL)	10.6 ± 4.9	8.3 ± 4.7	.01
In-hospital death (n)	2	1	.07
SBP at discharge (mm Hg)	138.1 ± 6.9	139.6 ± 6.1	.25
DBP at discharge (mm Hg)	92.9 ± 7.8	93.4 ± 6.2	.74
Death within 1 month (n)	2	6	.91
SBP at 1 month (mm Hg)	146.6 ± 12.6	144.3 ± 10.3	.35
DBP at 1 month (mm Hg)	96.2 ± 6.4	94.6 ± 6.8	.29
Creatinine at 1 month follow-up (mg/dL)	5.5 ± 2.1	4.6 ± 2.1	.06

the COVID-19 pandemic, it was difficult to follow up with some patients. As a result, serum creatinine values were obtained from different laboratories, which may have introduced some variability to our data. Additionally, we did not have access to readmission data within 1 month of discharge.

Two-thirds of patients admitted with acute severe hypertension had kidney dysfunction. Nearly half of the patients with a hypertensive crisis had underlying CKD. The mortality rate during hospitalization was higher in patients with CKD. Additionally, individuals with CKD exhibited higher BP both at the time of

Table 5. Outcomes of Patient			1
	Mean ± SD (n)/Frequency (%)		
	CKD	Non-CKD	P
At admission	n = 127	n = 135	
Systolic BP (mm Hg)	195 ± 17.8	192 ± 15.6	.1
Diastolic BP (mm Hg)	108 ± 11.7	104 ± 12.1	.01
At discharge	n = 116	n = 109	
Systolic BP (mm Hg)	139.2 ± 6.4	133.5 ± 7.1	<.01
Diastolic BP (mm Hg)	93.3 ± 7.1	86.7 ± 7.0	<.01
Serum creatinine (mg/dL)	5.4 ± 2.5	1.0 ± 0.9	<.01
Alive	116(91.3)	109(80.7)	.02
Died and left against medical advice	11(8+3) (8.7)	26(18+8) (19.2)	.02
At follow-up	n = 105	n = 100	
Systolic BP (mm Hg)	145.0 ± 11.0	128.4 ± 4.7	<.01
Diastolic BP (mm Hg)	95.1 ± 6.8	82.0 ± 4.4	<.01
Serum creatinine (mg/dL)	4.9 ± 2.1	0.8 ± 0.4	<.01
Alive	105(82.6)	100(74.0)	.75
Died and lost to follow-up	11(8+3) (8.7)	9(7+2) (6.6)	
BP, blood pressure.			

discharge and during follow-up. Better support at the community level, including physicians, nephrologists, and dialysis centers, is needed for the judicious assessment and treatment of AKI, CKD, and hypertension. This could help to reduce the number of patients seeking emergency care for hypertensive crises.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Ethics Committee Approval: Ethics committee approval was received for this study from the Ethics Committee of Jawaharlal Institute of Postgraduate Medical Education and Research (approval number: IEC/2020/097, date: July 24, 2020).

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

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