ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 08 | August - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CLINICAL PROFILE OF HYPERTENSIVE CRISIS: A CROSS-SECTIONAL OBSERVATIONAL STUDY FROM A TERTIARY CARE CENTRE IN CENTRAL INDIA

Medicine

Piyush Jain* Postgraduate Resident, Department of General Medicine, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh, India*Corresponding Author

Ashutosh Kumar Professor, Department of General Medicine, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh, India

D.P Singh H.O.D and Professor, Department of General Medicine, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh, India

Salman Khan Postgraduate Resident, Department of General Medicine, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh, India

Ritendra Patidar Postgraduate Resident, Department of General Medicine, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh, India

Biyani Alkesh Ajay Postgraduate Resident, Department of General Medicine, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh, India

ABSTRACT

Introduction: Hypertensive crisis (blood pressure [BP] ≥180/120 mmHg) is classified as hypertensive emergency (HTN-E) when acute target organ damage (TOD) is present, or hypertensive urgency (HTN-U) when it is absent. Indian data on hypertensive crisis are concentrated in metropolitan centres; central India, despite among the lowest hypertension awareness rates nationally, remains underrepresented. **Aim:** To describe the clinical profile, risk factors, and target organ damage pattern of hypertensive crisis presenting to a tertiary care centre in central India, and to compare HTN-E with HTN-U. **Materials and Methods:** This hospital-based cross-sectional observational study enrolled 100 consecutive adults presenting with BP ≥180/120 mmHg to the Department of Medicine, L.N. Medical College and J.K. Hospital, Bhopal, over 18 months. All patients underwent structured history-taking, examination, 12-lead electrocardiography (ECG), two-dimensional echocardiography, direct fundoscopy (Keith-Wagener-Barker [KWB] grading), biochemical work-up, and neuroimaging or renal Doppler ultrasonography where indicated. Categorical variables were compared using the Chi-square or Fisher's Exact test and continuous variables using Student's t-test or the Mann-Whitney U test; $p<0.05$ was considered significant.**Results:** Of 100 patients, 39 (39%) had HTN-E and 61 (61%) had HTN-U. Patients with HTN-E were significantly older than those with HTN-U (58.1±14.8 vs 52.3±15.2 years; $p=0.044$) and showed a female preponderance (56.4% vs 31.1%; $p=0.022$). Dyslipidaemia (79.5% vs 52.5%; $p=0.012$), diabetes mellitus/diabetes with chronic kidney disease [CKD] (46.2% vs 21.3%; $p<0.05$), CKD alone (17.9% vs 1.6%; $p<0.05$), serum creatinine (1.52±0.71 vs 0.90±0.30 mg/dL; $p<0.001$), Grade III–IV retinopathy (confined to HTN-E; $p<0.001$), and reduced ejection fraction on echocardiography (12.8% vs 3.3%; $p=0.048$) were each significantly associated with HTN-E. Among HTN-E patients, acute stroke was the commonest target organ damage (35.8%), followed by acute pulmonary oedema (25.6%) and coronary artery disease/acute coronary syndrome (17.9%). Medication non-compliance, though numerically higher in HTN-E (62.2% vs 36.4%), did not reach statistical significance ($p=0.125$).**Conclusion:** Hypertensive crisis in this central Indian cohort was characterised by neurological predominance of target organ damage, a strong metabolic comorbidity burden, and older age and female sex as significant correlates of hypertensive emergency. These findings support strengthening community-based hypertension screening and structured post-crisis follow-up in this region.

KEYWORDS

Hypertensive Emergency; Hypertensive Urgency; Target Organ Damage; Hypertensive Retinopathy; Stroke; Central India

INTRODUCTION

Hypertensive crisis is defined as a severe elevation of blood pressure (BP ≥180 mmHg systolic and/or ≥120 mmHg diastolic) requiring prompt clinical evaluation. It is subclassified as hypertensive emergency (HTN-E), in which acute, progressive target organ damage (TOD) is present, or hypertensive urgency (HTN-U), in which severe BP elevation occurs without demonstrable acute TOD.1

Karras et al, in a multicentre emergency department study, showed that individual symptoms have low predictive value for distinguishing HTN-E from HTN-U, underscoring the need for systematic clinical and laboratory evaluation rather than reliance on the absolute BP value.2

Hypertension is the most prevalent chronic non-communicable disease worldwide and the leading modifiable risk factor for cardiovascular mortality. In India, awareness, treatment, and control rates remain poor, and Madhya Pradesh records one of the lowest hypertension awareness rates nationally. Hypertensive crisis complicates a small but clinically important proportion of hypertensive patients and carries substantially higher morbidity and mortality when target organ damage is present.

The landmark Italian series by Zampaglione et al reported an HTN-U:HTN-E ratio of approximately 76:24 among 1,396 patients, with cardiac manifestations predominating among emergencies.3

Indian series, in contrast, consistently report a higher proportion of HTN-E and a predominance of neurological over cardiac target organ damage, a pattern attributed to a greater burden of undetected hypertension, later presentation, and a higher prevalence of metabolic comorbidities.4-8

The Dutch/ESC-ESH position statement on hypertensive emergencies emphasises that classification of HTN-E versus HTN-U is a clinical determination requiring systematic assessment — fundoscopy, electrocardiography, echocardiography, renal function, and neuroimaging where indicated — rather than reliance on the absolute BP reading.9

Published Indian data on hypertensive crisis are concentrated in metropolitan referral centres; central India — despite documented low hypertension awareness (30.7% per the National Family Health Survey-5) — remains substantially underrepresented. This study was therefore undertaken to describe the clinical profile, risk factor pattern, and spectrum of target organ damage of hypertensive crisis presenting to a tertiary care hospital in central India, and to compare hypertensive emergency with hypertensive urgency across demographic, biochemical, and investigational parameters.

MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of Medicine, L.N. Medical College and J.K. Hospital, Bhopal, Madhya Pradesh — a tertiary care teaching hospital serving both urban and rural populations of central India — over a period of 18 months. The study was approved by the

Institutional Ethics Committee of L.N. Medical College, and written informed consent was obtained from all participants or their legally authorised representatives prior to enrolment, in accordance with the Declaration of Helsinki (as revised in 2013).

A total of 100 consecutive patients aged ≥18 years, admitted to the medicine ward or emergency department with a presenting systolic BP ≥180 mmHg and/or diastolic BP ≥120 mmHg on two consecutive readings taken ≥5 minutes apart in the sitting position, were enrolled by purposive consecutive sampling. Patients or attendants who declined consent, and those below 18 years of age, were excluded.

All patients were evaluated using a structured, pre-tested case record proforma capturing sociodemographic details (age, sex, residence, educational status, modified Prasad socioeconomic classification), history (presenting complaints, known duration of hypertension, antihypertensive regimen and compliance status, comorbidities, smoking), and a standardised general and systemic examination. Non-compliance was defined as self-reported irregular intake of prescribed antihypertensive medication in the four weeks preceding presentation. Investigations performed in all patients included complete blood count, renal and liver function tests, fasting glucose and glycated haemoglobin, serum electrolytes, fasting lipid profile, cardiac biomarkers (CK-MB, Troponin-I), urinalysis, 12-lead ECG, chest radiograph, direct fundoscopy, and two-dimensional echocardiography. Hypertensive retinopathy was graded using the Keith-Wagener-Barker (KWB) classification (Grade I–IV). Left ventricular hypertrophy (LVH) on ECG was defined by Sokolov-Lyon voltage criteria; echocardiographic LVH was defined as left ventricular mass index >115 g/m² in men and >95 g/m² in women. Non-contrast CT brain (with MRI in selected cases) was performed in patients with neurological deficit, altered sensorium, or clinical suspicion of intracranial pathology; renal Doppler ultrasonography was performed to screen for renal artery stenosis (peak systolic velocity >180 cm/s, renal-aortic ratio >3.5).

Data were entered in Microsoft Excel and analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables are expressed as mean ± standard deviation and compared using Student's independent-samples t-test (or the Mann-Whitney U test for non-normally distributed data, assessed by the Kolmogorov-Smirnov test); categorical variables are expressed as frequencies and percentages and compared using the Chi-square test, or Fisher's Exact test where expected cell counts were <5. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Of 100 patients presenting with hypertensive crisis, 39 (39%) fulfilled criteria for hypertensive emergency (HTN-E) and 61 (61%) for hypertensive urgency (HTN-U).

The overall mean age was 55.4±15.1 years (range 25–78). Patients with HTN-E were significantly older (58.1±14.8 years) than those with HTN-U (52.3±15.2 years; p=0.044). Males comprised 59% of the cohort overall; however, HTN-E showed a significant female preponderance (56.4% female vs 43.6% male) whereas HTN-U was predominantly male (68.9%; p=0.022 for the sex-subtype association). Rural residents constituted 57% of the cohort, with a higher proportion in HTN-E (64.1%) than HTN-U (52.5%), though this difference was not statistically significant (p=0.347). Thirty-two percent of patients were illiterate or had no formal schooling. Mean body mass index (BMI) was 24.0±4.3 kg/m² and did not differ significantly between subtypes (25.0±4.8 vs 23.4±3.9 kg/m²; p=0.073) (Table 1).

Table 1: Baseline demographic and anthropometric characteristics by hypertensive crisis subtype

Variable	HTN-E (n=39)	HTN-U (n=61)	Total (n=100)	p-value
Mean age, years (±SD)	58.1 ± 14.8	52.3 ± 15.2	55.4 ± 15.1	0.044*
Male sex	17 (43.6%)	42 (68.9%)	59 (59.0%)	0.022*
Female sex	22 (56.4%)	19 (31.1%)	41 (41.0%)	0.022*
Rural residence	25 (64.1%)	32 (52.5%)	57 (57.0%)	0.347
Illiterate / no formal schooling	10 (25.6%)	22 (36.1%)	32 (32.0%)	—
Mean BMI, kg/m² (±SD)	25.0 ± 4.8	23.4 ± 3.9	24.0 ± 4.3	0.073

Dyslipidaemia was the most prevalent comorbidity (63%) and was significantly more common in HTN-E than HTN-U (79.5% vs 52.5%; p=0.012). Diabetes mellitus, alone or with CKD, was present in 31% overall and was significantly more frequent in HTN-E (46.2% vs 21.3%; p<0.05), as was CKD alone (17.9% vs 1.6%; p<0.05). Known hypertension was documented in 65% of patients and newly diagnosed hypertension in 35%; this difference between subtypes was not statistically significant (p=0.176). A positive family history of hypertension (54%) and smoking (37%) were common but did not differ significantly between subtypes (p=0.521 and p=0.213, respectively). Among the 66 patients with documented compliance status, 43 (65.2%) were non-compliant with antihypertensive therapy; non-compliance was numerically higher in HTN-E (62.2%) than HTN-U (36.4%), but this difference did not reach statistical significance (p=0.125) (Table 2).

Table 2: Risk factors, comorbidities, and medication compliance by hypertensive crisis subtype

Variable	HTN-E (n=39)	HTN-U (n=61)	Total (n=100)	p-value
Dyslipidaemia	31 (79.5%)	32 (52.5%)	63 (63.0%)	0.012*
Diabetes mellitus / DM+CKD	18 (46.2%)	13 (21.3%)	31 (31.0%)	<0.05*
CKD alone	7 (17.9%)	1 (1.6%)	8 (8.0%)	<0.05*
Known hypertension	29 (74.4%)	36 (59.0%)	65 (65.0%)	0.176
Family history of hypertension	19 (48.7%)	35 (57.4%)	54 (54.0%)	0.521
Smoking	11 (28.2%)	26 (42.6%)	37 (37.0%)	0.213
Medication non-compliance*†	23/30 (62.2%)	20/36 (36.4%)	43/66 (65.2%)	0.125

† Denominator restricted to the subset of patients with documented compliance status among known/newly compliant-assessed hypertensives (n=66).

Severe headache was the commonest overall presenting complaint (36%) and was predominant in HTN-U (52.4% vs 10.2% in HTN-E). Hemiparesis was the dominant presenting complaint in HTN-E (35.8% vs 14.8% in HTN-U), reflecting the high prevalence of stroke as target organ damage. Dyspnoea (23.0% vs 6.6%) and chest pain/palpitation (15.3% vs 4.9%) were also more frequent in HTN-E. Among the 39 patients with HTN-E, acute stroke (ischaemic or haemorrhagic) was the commonest target organ damage (14 patients, 35.8%), followed by acute pulmonary oedema (10, 25.6%), coronary artery disease/acute coronary syndrome (7, 17.9%), hypertensive retinopathy with papilloedema (5, 12.8%), and hypertensive nephropathy/CKD as the primary target organ (2, 5.1%) (Table 3).

Table 3: Spectrum of target organ damage in hypertensive emergency (n=39)

Target organ damage	n	% of HTN-E
Acute stroke (ischaemic/haemorrhagic)	14	35.8%
Acute pulmonary oedema	10	25.6%
Coronary artery disease / acute coronary syndrome	7	17.9%
Hypertensive retinopathy with papilloedema	5	12.8%
Hypertensive nephropathy / CKD (primary)	2	5.1%
Total	39	100.0%

On neuroimaging performed in HTN-E patients with neurological presentations, acute ischaemic infarction was the commonest finding (28.2% of HTN-E), followed by haemorrhagic stroke (7.7%) and old ischaemic changes (7.7%); the ischaemic-to-haemorrhagic stroke ratio was approximately 3.7:1.

ECG abnormalities were frequent (LVH by Sokolov-Lyon criteria in 34%, ST-T changes in 14%, arrhythmia in 8%) but did not differ significantly between subtypes (all p>0.05). Echocardiographic LVH of any grade was present in 60% of patients — substantially more sensitive than ECG — and reduced ejection fraction (<50%) was significantly more common in HTN-E (12.8% vs 3.3%; p=0.048). Hypertensive retinopathy of any grade was present in 69% of patients; Grade III and IV retinopathy were confined exclusively to HTN-E (17.9% and 12.8% of HTN-E respectively; combined p<0.001), confirming fundoscopy as a reliable bedside discriminator of target

organ damage. Serum creatinine was significantly higher in HTN-E than HTN-U (1.52±0.71 vs 0.90±0.30 mg/dL; p<0.001), while serum urea showed a similar but non-significant trend (44.8±22.1 vs 38.2±15.6 mg/dL; p=0.096). Renal artery stenosis was detected on Doppler ultrasonography in 8% of the total cohort (Table 4).

Table 4: Investigational findings by hypertensive crisis subtype

Investigation	HTN-E (n=39)	HTN-U (n=61)	Total (n=100)	
ECG — LVH (Sokolov-Lyon)	15 (38.5%)	19 (31.1%)	34 (34.0%)	0.584
ECG — ST-T changes	5 (12.8%)	9 (14.8%)	14 (14.0%)	0.793
Echo — LVH, any grade	22 (56.4%)	38 (62.3%)	60 (60.0%)	—
Echo — Reduced EF (<50%)	5 (12.8%)	2 (3.3%)	7 (7.0%)	0.048*
Retinopathy — Grade III	7 (17.9%)	0 (0.0%)	7 (7.0%)	<0.001 *
Retinopathy — Grade IV (papilloedema)	5 (12.8%)	0 (0.0%)	5 (5.0%)	<0.001 *
Serum creatinine, mg/dL (±SD)	1.52 ± 0.71	0.90 ± 0.30	—	<0.001 *
Renal artery stenosis (Doppler)	—	—	8 (8.0%)	—

Table 5 summarises the parameters that differed significantly between HTN-E and HTN-U.

Table 5: Summary of parameters significantly associated with hypertensive emergency

Parameter	HTN-E	HTN-U	p-value
Mean age, years	58.1 ± 14.8	52.3 ± 15.2	0.044*
Female sex	56.4%	31.1%	0.022*
Dyslipidaemia	79.5%	52.5%	0.012*
Diabetes mellitus / DM+CKD	46.2%	21.3%	<0.05*
CKD alone	17.9%	1.6%	<0.05*
Serum creatinine, mg/dL	1.52 ± 0.71	0.90 ± 0.30	<0.001*
Grade III–IV retinopathy	30.8%	0%	<0.001*
Reduced ejection fraction (<50%)	12.8%	3.3%	0.048*

DISCUSSION

In the present cohort, HTN-E accounted for 39% of hypertensive crisis presentations, considerably higher than the 24.4% reported by Zampaglione et al in their large Turin series3 but comparable to the 35–42% range reported across several Indian tertiary-care series.4-6

This pattern is consistent with the referral bias of a tertiary-care emergency department and with the well-documented gap in hypertension awareness in Madhya Pradesh, where fewer than one-third of hypertensive individuals are aware of their diagnosis.10

The mean age of our cohort (55.4±15.1 years) was closely comparable to that reported by Chaudhary and Lakhani in a similarly designed Indian study, and patients with HTN-E were significantly older than those with HTN-U in our series — consistent with the concept that longstanding hypertension and cumulative vascular injury increase susceptibility to acute target organ damage during a further pressure surge.

The significant female preponderance observed in our HTN-E group (56.4%) contrasts with the overall male predominance of the cohort and with several published series in which males predominate across both subtypes.7 Possible explanations include post-menopausal metabolic changes, a higher comorbidity burden among affected women, and delayed healthcare-seeking behaviour among women in rural central India — an area that merits focused prospective evaluation.

Dyslipidaemia, diabetes mellitus (alone or with CKD), and CKD were each significantly more prevalent in HTN-E than HTN-U in our study, supporting their role as pathophysiological amplifiers of end-organ vulnerability during acute BP surges through endothelial dysfunction, accelerated arteriosclerosis, and impaired renal autoregulation.

This concurs with the mechanistic role of diabetes-related endothelial injury described by Sowers and Epstein11 and with the role of microalbuminuria and glomerular arteriolar injury as both a consequence and a predictor of hypertensive crisis described by Palatini.12

Medication non-compliance was numerically more frequent in HTN-E than HTN-U in our cohort (62.2% vs 36.4%), and the overall non-compliance rate (65.2% of patients with documented compliance status) was among the highest reported in Indian literature; however, this difference between subtypes did not reach statistical significance in our sample (p=0.125), and should be interpreted as a numerically prominent but not independently confirmed precipitant in this cohort. Larger, adequately powered studies are needed to establish this association formally.

Nonetheless, medication non-adherence has been identified as the principal precipitant of hypertensive crisis in several Indian series4,13, and the structural barriers identified in our cohort — rural residence, low educational attainment, and economic constraints on drug procurement — mirror those described in comparable central and north Indian populations.

Acute stroke was the leading target organ damage among HTN-E patients in our study (35.8%), a pattern that distinguishes Indian hypertensive crisis cohorts from Western series, where cardiac manifestations — acute pulmonary oedema and acute coronary syndrome — typically predominate.3,5,6,8

This neurological predominance is consistent with reports from other Indian tertiary centres and reflects the substantial burden of hypertension-attributable stroke in the Indian population, alongside later presentation and a higher prevalence of newly diagnosed hypertension with intact but vulnerable cerebral autoregulation.

Fundoscopy proved a valuable, cost-free bedside discriminator in our study: Grade III and IV hypertensive retinopathy were found exclusively among HTN-E patients (p<0.001), corroborating the established role of the Keith-Wagener-Barker classification in target organ damage assessment. Echocardiography was substantially more sensitive than ECG for detecting LVH (60% vs 34%), consistent with the well-recognised superior sensitivity of echocardiographic over electrocardiographic criteria, and reduced ejection fraction was significantly more common in HTN-E — reflecting acute afterload-related left ventricular dysfunction superimposed on a background of chronic hypertensive heart disease. Serum creatinine was significantly higher in HTN-E, in keeping with acute renal arteriolar injury and glomerular ischaemia during hypertensive emergency; renal artery stenosis, detected in 8% of our cohort on Doppler ultrasonography, carries direct implications for antihypertensive drug selection given the relative contraindication of ACE inhibitors and angiotensin receptor blockers in bilateral disease.

These findings align broadly with the ESC/ESH position statement on hypertensive emergencies, which emphasises systematic multi-organ assessment — rather than the absolute BP value — as the basis for accurate classification and risk stratification.9

Strengths and Limitations

This study's principal strength is its comprehensive, uniformly applied evaluation protocol — ECG, echocardiography, fundoscopy, renal function testing, and neuroimaging or Doppler ultrasonography as indicated — performed in all enrolled patients, enabling robust target organ damage ascertainment. It also contributes original data from central India, a region underrepresented in the published literature on hypertensive crisis.

The cross-sectional design precludes causal inference and does not permit assessment of in-hospital mortality, 30-day outcomes, or the efficacy of antihypertensive regimen modification. The single-centre, referral-based sample of 100 patients may over-represent severe presentations and limits statistical power for subgroup analyses; several comparisons in this study, including medication compliance, may consequently have been underpowered to detect true associations. Medication compliance was assessed by self-report without objective corroboration. Quantitative lipid and urinary microalbumin data and a normotensive or non-crisis hypertensive control group were not available, limiting formal risk-factor comparison. These limitations should be borne in mind when interpreting and generalising the present

findings, and prospective, adequately powered, multicentric studies are recommended to confirm them.

CONCLUSION

In this central Indian tertiary-care cohort, hypertensive urgency was more common than hypertensive emergency, and older age and female sex were significantly associated with hypertensive emergency. Dyslipidaemia, diabetes mellitus, CKD, elevated serum creatinine, advanced hypertensive retinopathy, and reduced ejection fraction were each significantly associated with hypertensive emergency, while medication non-compliance, though numerically prominent, was not statistically confirmed as a discriminating factor in this sample. Acute stroke was the leading target organ damage in hypertensive emergency, distinguishing this cohort from predominantly cardiac Western series. These findings support the case for strengthened community-based hypertension screening, structured medication adherence support, and readily accessible stroke imaging services in central India, and highlight the value of fundoscopy and echocardiography as accessible bedside tools for target organ damage assessment in resource-limited settings.

ACKNOWLEDGEMENT

The authors thank the Department of Community Medicine, L.N. Medical College, Bhopal, for statistical guidance, and the patients who consented to participate in this study.

REFERENCES

1. Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA. 2003;289(19):2560–2572.
2. Karras DJ, Kruus LK, Cienki JJ, et al. Evaluation and treatment of patients with severely elevated blood pressure in academic emergency departments: a multicenter study. Ann Emerg Med. 2006;47(3):230–236.
3. Zampaglione B, Pascale C, Marchisio M, Cavallo-Perin P. Hypertensive urgencies and emergencies: prevalence and clinical presentation. Hypertension. 1996;27(1):144–147.
4. Gupta R, Sharma SK, Bhardwaj S. Hypertensive emergencies and urgencies: a clinical profile. J Assoc Physicians India. 2012;60:12–16.
5. Rajan S, Bhanu S, Krishnamurthy S. Clinical profile of hypertensive crisis — a study from south India. Int J Sci Res Pub. 2013;3(7):1–6.
6. Vasan G, Bhuvaneswari V, Premkumar A. Clinical profile of hypertensive crisis. Int J Adv Med. 2014;1(1):38–43.
7. Sharma R, Singh AK. Clinical profile and outcomes of hypertensive emergencies in a tertiary care centre. J Clin Diagn Res. 2018;12(1):OC01–OC05.
8. Chaudhary S, Lakhani JD. Clinical profile of hypertensive crisis — a study of 200 patients. Int J Sci Res. 2019;8(7):G1–G9.
9. van den Born BJH, Beutler JJ, Gaillard CAJM, et al. Dutch guideline for the management of hypertensive crisis — 2010 revision. Neth J Med. 2011;69(5):248–255.
10. International Institute for Population Sciences (IIPS). National Family Health Survey (NFHS-5), 2019–21: India Fact Sheet. Mumbai: IIPS; 2021.
11. Sowers JR, Epstein M. Diabetes mellitus and associated hypertension, vascular disease, and nephropathy — an update. Hypertension. 1995;26(6):869–879.
12. Palatini P. Microalbuminuria in hypertension. Curr Hypertens Rep. 2003;5(3):208–214.
13. Mehta A, Patel R, Merchant S. Hypertensive crisis — retrospective audit of internal medicine emergency admissions. KEM Hospital Annual Report. Mumbai: KEM Hospital; 2015.
14. Krishnamurthy S, Rao S, Suresh K. Hypertension in acute neurological admissions — a NIMHANS experience. Neurol India. 2022;70(4):1420–1427.
15. Pinna G, Pascale C, Fornengo P, et al. Hospital admissions for hypertensive crisis in the emergency departments: a large multicenter Italian study. PLoS One. 2014;9(4):e93542.
16. Kannel WB. Framingham study insights into hypertensive risk of cardiovascular disease. Hypertens Res. 1995;18(3):181–196.
17. Webster J, Petrie JC, Jeffers TA, Lovell HG. Accelerated hypertension — patterns of mortality and clinical factors affecting outcome in treated patients. QJM. 1993;86(8):485–493.
18. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018;39(33):3021–3104.
19. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. J Am Coll Cardiol. 2018;71(19):e127–e248.