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Research Article

Stroke-Like Neurological Manifestations in Hypertensive Emergency: Diagnostic Challenges and Clinical Lessons from a Case Report

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ABSTRACT

Neurologic manifestations in patients with hypertensive emergencies are often stroke-like, and rapid recognition of acute cerebrovascular insults or reversible hypertensive neurologic dysfunction with its implications for clinical management and prognosis continue to pose a great challenge in the field of emergency medicine. Although tremendous progress has been made in the use of neuroimaging, normal initial brain computed tomography (CT) does not rule out diagnostic uncertainty, especially in patients with transient neurological symptoms. The diagnostic difficulty of a stroke-like presentation during a hypertensive emergency is discussed in the context of a representative clinical case and incorporates current evidence on the differential diagnoses, emergency evaluation, neuroimagining protocol, therapeutic management and potential pathophysiological mechanisms. The presented case was a 42-year-old man with uncontrolled hypertension and diabetes mellitus who presented with sudden onset of headache, transient numbness and weakness with marked hypertension (181/110 mmHg). Emergency evaluation, which included a non-contrast brain computed tomography (CT) scan, did not show evidence of acute intracranial hemorrhage, and rapid antihypertensive treatment led to resolution of symptoms and discharge the same day. This is a clinical study with a contemporary evidence-based review that has identified limitations of using early neuroimaging, the importance of structured differential diagnosis, and suggested mechanism(s) for reversible neurological dysfunction in hypertensive emergency. This confirms the importance of timely control of blood pressure, thorough neurological evaluation, and clinical discretion to ensure the best possible outcomes and to avoid misdiagnosis in the emergency setting.

1. INTRODUCTION

Hypertensive emergency is a life-threatening clinical state with the sudden onset of severe hypertension and acute hypertension-related target organ damage to the brain, heart, kidneys, retina, and/or large vessels. Though it is a relatively small percentage of patients with hypertension, hypertensive emergency is one of the most serious cardiovascular conditions seen in the emergency department and if not adequately recognized or managed, can cause irreversible organ damage and death. The diagnosis of hypertensive emergency should be made more with evidence of acute target organ damage and less on the absolute blood pressure level, as the current international guidelines have stated [1, 2].

Neurological damage is one of the more difficult to diagnose types of target organ damage that can occur in a hypertensive emergency. Patients can experience severe headache, dizziness, altered mental status, seizures, visual disturbances, limb weakness, numbness or other focal neurological impairments that mimic acute ischemic or hemorrhagic stroke. These shared clinical characteristics can often lead to initial clinical ambiguity when evaluating a new case of acute stroke and hypertensive encephalopathy, especially when both have very different management strategies. In addition, patients with underlying cardiovascular risk factors like hypertension and diabetes mellitus have a higher risk of both hypertensive crisis and cerebrovascular disease which makes the distinction between the two even more complicated [2-4].

The term stroke mimics has been gaining attention in recent years due to the fact that a significant number of patients who present with acute stroke-like symptoms end up with a different neurological and/or systemic diagnosis. Common diseases that can cause stroke-like neurological symptoms include hypertensive encephalopathy, posterior reversible encephalopathy, metabolic disturbances, seizure, migraine with aura and functional neurological disorders. Misdiagnosis of these disorders as a true cerebrovascular event can result in inappropriate treatment, unnecessary thrombolysis, late initiation of antihypertensive therapy, multiple hospitalizations or missed opportunities for appropriate neurologic

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assessment. Therefore, it is essential to perform a proper differential diagnosis as part of the clinical decision-making process in the emergency room [5-7].

Brain imaging has a crucial role in the diagnosis of patients with acute neurological complaints. Brain computed tomography (CT) is the first imaging test used, as it is quick to obtain and very sensitive to detecting bleeding in the brain. A normal computed tomography (CT) scan, however, will not rule out early ischemic stroke or reversible neurological disorders that may be caused by severe hypertension. Imaging results should always be looked at in context with the patient's clinical presentation, neurological examination, symptom progression, cardiovascular risk profile and treatment response. The integrated approach is also important in patients with very rapid improvement of neurological deficits after blood pressure control, so as to differentiate a transient blood pressure-related neurological dysfunction from evolving cerebrovascular disease during the early stages of evaluation [4,7,8].

Despite significant advances in emergency medicine and neuro-imaging, there are important diagnostic uncertainties to be addressed in patients with transient stroke-like neurological manifestations of hypertensive emergency with normal initial brain imaging. Most of the current literature is based on blood pressure control and acute stroke management, while fewer studies concentrate on the diagnostic reasoning process of patients with reversible neurological symptoms and uncertain neuro-imaging results. The present study, therefore, illustrates a representative case of hypertensive emergency with transient stroke-like neurological symptoms, to show the diagnostic difficulties which one may face in emergency practice. The purpose of this report is to bring together the clinical signs and symptoms with up-to-date evidence and emphasize the need for thorough differential diagnosis, careful interpretation of neuroimaging, and timely clinical decisions for best clinical outcomes while minimizing diagnostic uncertainty.

2. CASE PRESENTATION

Sudden onset of severe generalized headache associated with numbness and weakness of both upper and lower limbs following emotional stress after argument with his son, in a 42-year-old man presented to the emergency department. The patient complained of increasing lethargy and nausea, but did not experience any chest pain, visual disturbances, abdominal pain, seizures, loss of consciousness or any head trauma in the recent past. His past medical history included long-standing hypertension, type 2 diabetes mellitus, which was treated regularly with antihypertensive agents Angizar H. No history of previous cerebrovascular disease, epilepsy, chronic kidney disease or other neurological disorders was documented (As shown in the Table I).

TABLE I. SUMMARY OF THE PATIENT'S CLINICAL CHARACTERISTICS AND EMERGENCY MANAGEMENT.

Parameter	Findings
Age	42 years
Sex	Male
Past medical history	Hypertension, Type 2 diabetes mellitus
Regular medication	Angizar H
Triggering event	Emotional stress following an argument
Chief complaints	Severe generalized headache, upper and lower limb numbness, limb weakness, nausea, lethargy
Blood pressure	181/110 mmHg
Pulse rate	91 beats/min
Oxygen saturation	96% (room air)
Level of consciousness	Alert and oriented
Chest examination	Clear bilateral breath sounds
Abdominal examination	Soft, non-tender
Peripheral edema	Absent
Initial clinical impression	Hypertensive emergency with suspected cerebrovascular involvement
Emergency treatment	Oral captopril (100 mg), intravenous labetalol, intravenous furosemide (Lasix)
Brain CT findings	No intracranial hemorrhage, mass lesion, midline shift, or acute intracranial abnormality
Clinical outcome	Complete resolution of neurological symptoms after blood pressure control
Disposition	Discharged home on the same day with continuation of antihypertensive therapy

At admission the patient was conscious, alert and fully oriented. Physical examination showed no signs of respiratory distress and auscultation of the chest showed no abnormalities in the bilateral breath sounds. No acute abnormalities were seen in cardiovascular examination and abdominal examination was non-tender and soft. There was no peripheral edema noted. Neurological examination gave a result of subjective numbness and weakness in the limbs but did not reveal any ongoing loss of consciousness or any seizure activity.

Given the significantly high blood pressure reading coupled with acute neurological symptoms, the diagnosis of hypertensive emergency with possible cerebrovascular involvement was made. Oral captopril (100 mg), intravenous labetalol and intravenous furosemide (Lasix) were immediately administered, while the blood pressure was monitored continuously and clinical management was closely monitored.

An urgent non-contrast computed tomography (CT) scan of the brain was performed to rule-out acute intracranial pathology. The examination showed no evidence of intracranial haemorrhage, space occupying lesion, mass effect or midline shift and there was no acute intracranial abnormality, the ventricular size and configuration were normal.
Blood pressure started to gradually improve after the antihypertensive therapy and headache and limb numbness and weakness were completely resolved during the observation period. He was discharged on the same day in good condition with his usual antihypertensive drugs (Angizar H) without radiological evidence of acute intracranial disease and after complete neurological recovery. The treating physician suggested outpatient follow up to optimize blood pressure control and suggested that changes to the antihypertensive regimen may be warranted during the next clinical evaluation. During this encounter at the emergency department, no magnetic resonance imaging (MRI), laboratory investigations or neurological consultation was done.

3. DIAGNOSTIC ASSESSMENT

3.1 Initial Clinical Impression

On arrival at the emergency department, blood pressure was much elevated at 181/110 mmHg and the patient had also presented with a sudden onset of severe generalized headache, bilateral limb numbness, weakness and nausea along with lethargy, which immediately raised concerns of a hypertensive emergency with possible acute neurological involvement. Given the neurological symptoms in the context of elevated blood pressure, the importance of obtaining prompt evaluation to assess for acute hypertension mediated target organ damage to the central nervous system [9].
Acute cerebrovascular disease was an important initial diagnosis that was entertained because of the rapid onset of neurological symptoms. Patients with known vascular risk factors, including hypertension and diabetes mellitus may have a headache, motor weakness, sensory disturbances and altered neurological function with an ischemic stroke or intracerebral hemorrhage. The potential life-threatening situations therefore necessitated immediate exclusion prior to making definitive management decisions [10].
One of the main differential diagnoses considered intracerebral hemorrhage, as uncontrolled hypertension is one of strongest risk factors for spontaneous cerebral bleeding. Given the patient's history of life-threatening hypertension and acute neurologic symptoms, an urgent neuro-imaging was undertaken to rule out a blood pressure-mediated hemorrhagic stroke before the additional treatment measures were started [11].
The initial clinical findings led to a working diagnosis in the ED of hypertensive emergency with suspected cerebrovascular involvement. This initial evaluation signaled the need to quickly lower the blood pressure, closely monitor the neurological status to identify or rule out acute intracranial pathology, and adopt urgent non-contrast computed tomography (CT) of the brain.

3.2 Differential Diagnosis

The patient's presentation was very difficult to make a diagnosis and a number of neurological disorders can have acute neurological deficits in the context of markedly elevated blood pressure. The patient's symptoms cleared up after control of blood pressure, and the initial non-contrast computed tomography of the brain was normal, but several important differential diagnoses were entertained during the diagnostic evaluation.
Acute severe hypertension with headache, nausea, lethargy and transient neurological symptoms were taken into consideration and were called hypertensive encephalopathy. There are no other significant clinical signs other than these which can be attributed to a failure of cerebral autoregulation due to poorly controlled blood pressure. In the absence of magnetic resonance imaging (MRI), however, the diagnosis was not definitive, as this imaging technique might be able to show the characteristic reversible vasogenic edema. In addition, there was no neurological consultation during evaluation in the emergency department. Hence, the diagnosis of hypertensive encephalopathy was still a possibility, which may not have been confirmed [12].
The other important point was transient ischemic attack (TIA), which was of critical importance as the patient did not have any remaining neurological deficit after the symptoms had completely abated within a relatively short time. However, diffusion-weighted MRI and vascular imaging and a full neurological examination were not available, so the diagnosis or exclusion of TIA was not possible. Thus, the diagnosis was still considered to be speculative, with only the clinical data available [13].
Acute ischemic stroke was also taken into consideration due to the fact that hypertension and diabetes mellitus are well established cerebrovascular risk factors. The patient's clinical presentation was initially suggestive of ischemic stroke, but was reassuringly rapidly improving and had no acute abnormalities on non-contrast CT, making it less likely to be a major ischemic event. However, the use of CT alone would not have ruled out early ischemic stroke completely, especially in the hyperacute phase, and MRI would have been more sensitive [14,15].
The other salient and important differential diagnosis was intracranial hemorrhage, as one of the most important factors for spontaneous cerebral hemorrhage is severe hypertension. This possibility was appropriately explored with the use of emergency non-contrast computed tomography (CT) which showed no evidence of intracranial bleeding, mass effect or

midline shift. Thus, during the emergency evaluation, intracranial hemorrhage was effectively ruled out [16]. The migraine with neurological symptoms sometimes resembles acute cerebrovascular disease, with transient sensory disturbances or motor symptoms with headache. The patient did not have migraine or aura or have a history of similar attacks, however, reducing the likelihood of the diagnosis [17].

Functional neurological disorder was also thought of as a possibility, as functional neurological symptoms can mimic the symptoms of acute stroke. However, the fact that blood pressure was quite high and that the other symptoms were resolved when the antihypertensive medications were started supported organic rather than functional causes. In addition, there were no clinical features indicative of a functional neurological disorder recorded during the examination [18].

Another neurological syndrome associated with hypertension is posterior reversible encephalopathy that presents with headache, altered mental status, seizures, visual disturbances, and focal neurologic deficits. While there were some common clinical characteristics, the current case did not have brain MRI which is the imaging modality of choice to demonstrate posterior vasogenic edema and thus PRES was not confirmed. Thus, this diagnosis was still in the differential diagnosis, but not confirmed [19].

Last, metabolic disorders such as hypoglycemia, electrolyte disturbances, and other systemic disorders should be always kept in mind in the patients with acute neurological symptoms. But no laboratory studies were done at the time of the emergency department visit. This made the determination of metabolic causes impractical and not conclusive, therefore, and a limitation of the diagnostic assessment.

Overall, the working diagnosis was not altered by the available clinical information, emergency neuroimaging, and the patient's good response to antihypertensive therapy, which remained a hypertensive emergency with a transient neurological manifestation. However, due to the lack of advanced neuroimaging and laboratory testing, certain neurological disorders were also unable to be ruled out such as hypertensive encephalopathy, early ischemic stroke, posterior reversible encephalopathy and transient ischemic attack (As shown in the Table II)**.**

TABLE II. DIFFERENTIAL DIAGNOSIS CONSIDERED DURING THE DIAGNOSTIC ASSESSMENT

Differential diagnosis	Supporting findings	Findings against diagnosis / Limitations	Final assessment
Hypertensive encephalopathy	Severe hypertension, headache, nausea, transient neurological symptoms	MRI not performed; no neurological consultation	Possible but unconfirmed
Transient ischemic attack (TIA)	Complete recovery of neurological symptoms	No MRI or vascular imaging	Cannot be excluded
Acute ischemic stroke	Hypertension, diabetes, acute neurological symptoms	Normal CT; rapid recovery; MRI unavailable	Less likely but not excluded
Intracranial hemorrhage	Severe hypertension with acute symptoms	CT showed no hemorrhage	Excluded
Migraine with neurological symptoms	Headache with transient neurological symptoms	No migraine history	Unlikely
Functional neurological disorder	Stroke-like presentation	Clinical findings favored an organic cause	Unlikely
Posterior reversible encephalopathy	Severe hypertension with neurological symptoms	MRI not performed	Possible but unconfirmed
Metabolic causes	May mimic acute neurological disease	Laboratory investigations unavailable	Cannot be excluded

3.3 Neuroimaging Assessment

The patient's presentation of acute neurological symptoms and significant elevation of blood pressure, made neuroimaging a priority in order to rule out life-threatening intracranial pathology. The first-line imaging modality to evaluate suspected cerebrovascular events was computed tomography (CT) scan of the brain, which was performed immediately after admission. The use of non-contrast computed tomography (CT) is preferable in the emergency room setting as this is a highly sensitive modality and is readily available [12].

The CT exam was negative for the presence of intracranial hemorrhage, intracranial mass lesion, mass effect or midline shift. Ventricular size and configuration were normal and no acute abnormalities were found in the brain. The results are of great importance to decrease the risk of a hemorrhagic stroke and other space-occupying intracranial lesions in which immediate intervention is required.

However, the CT images were reassuring, but did not rule out early ischemic stroke or early reversible disorders of the brain due to severe hypertension. The sensitivity of the non-contrast CT for hyperacute cerebral ischemia is limited, especially in the first hours after symptom onset. For this reason, the imaging results were correlated with the patient's clinical features, neurological examination, and subsequent clinical outcome with antihypertensive treatment [13,14] (As shown in the Table III).

TABLE III. SUMMARY OF BRAIN CT FINDINGS

CT Parameter	Finding	Clinical Interpretation
Intracranial hemorrhage	Not detected	Hemorrhagic stroke unlikely
Space-occupying lesion	Not detected	No evidence of intracranial mass
Mass effect	Absent	No significant cerebral compression

Midline shift	Absent	No intracranial displacement
Ventricular system	Normal size and configuration	No hydrocephalus or ventricular distortion
Overall CT interpretation	No acute intracranial abnormality	Further diagnosis should rely on clinical assessment and additional imaging if indicated

3.4 Diagnostic Challenges

The present case represents a common, yet clinically relevant diagnostic challenge in emergency medicine: A stroke like presentation without an initial abnormal computed tomography (CT) scan of the brain. The patient's severe hypertension and the transient neurological deficits were initially concerning for acute cerebrovascular disease, but the emergency neuroimaging did not show any signs of an acute structural problem, including intracranial hemorrhage.

One of the major issues in this case involved deciding if the neurological symptoms were a reversible effect of severe hypertension or an early ischemic event that was not apparent on non-contrast computed tomography (CT). Although blood pressure reduction led to rapid improvement, a transient neurological process related to hypertension, the transient hyperacute phase of early ischemic stroke cannot be ruled out due to the limited hyperacute period sensitivity of CT. A major drawback of the diagnostics was the lack of magnetic resonance imaging (MRI). In addition to the increased sensitivity of DW MRI to detect acute cerebral ischemia, it is also the preferred imaging modality to diagnose cases of posterior reversible encephalopathy and confirm or exclude hypertensive encephalopathy when clinically suspected [16,17].

In addition, a laboratory examination and neurological consult were not carried out at the ED visit. The possibility of alternative causes of transient neurological dysfunction, however, such as metabolic abnormalities or other neurological disorders, was not completely assessed. This constriction caused the inability to definitively diagnose one of the following neurological conditions. Given these limitations in diagnosis, the patient's full recovery after antihypertensive therapy and the lack of acute abnormalities in the CT scan of the brain supported a clinical diagnosis of hypertensive emergency with transient neurologic symptoms. This case emphasizes the need to consider clinical data, emergency neuroimaging, and continued neurological evaluation to determine the cause of severe hypertension and acute neurological symptoms, not just a normal CT scan.

To summarize the clinical reasoning and clinical management used in the present case, a timeline of the patients' clinical course is shown in Figure 1 from presentation in the emergency room to the era of neuroimaging, antihypertensive therapy, improvement, and discharge. The flowchart shows the important decision steps used in the diagnostic evaluation.

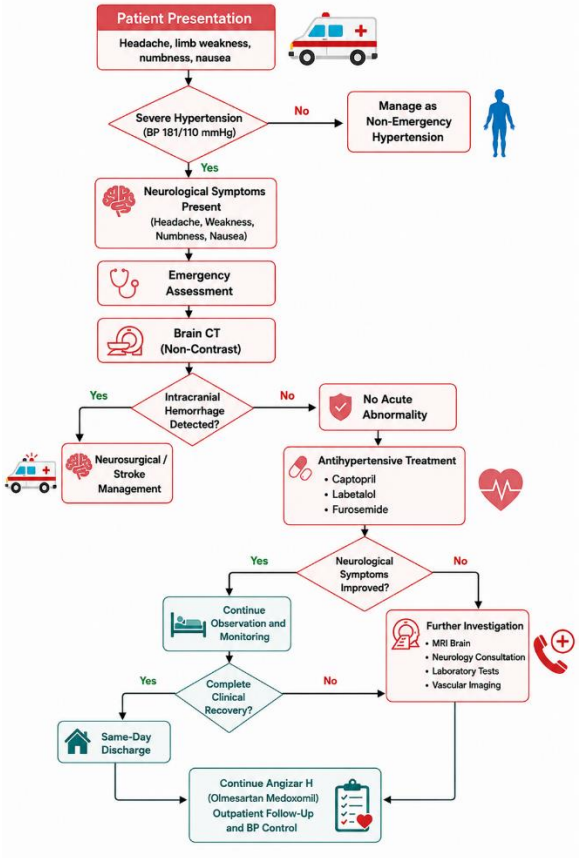

Fig. 1. Diagnostic and clinical management pathway of the presented case of hypertensive emergency with transient stroke-like neurological manifestations.

4. THERAPEUTIC INTERVENTION

4.1 Emergency Treatment

Upon a first clinical evaluation, immediate treatment was started to obtain controlled BP lowering and stop the progression of target-organ damage caused by hypertension. Prompt treatment for the patient's extremely high blood pressure (181/110 mmHg), with the presence of acute neurological symptoms, led to the immediate initiation of antihypertensive therapy in the emergency room.

Labetalol (4 mL) intravenously was administered to the patient to allow controlled blood pressure reduction, and oral captopril (100 mg) was used as the initial antihypertensive. In addition, emergency management included administration of 1 ampoule of intravenous furosemide (Lasix). Blood pressure, neurological status and vital signs were monitored continuously during the observation period to assess the patient's response to treatment and to detect any clinical deterioration. Antihypertensive treatment led to a progressive clinical improvement. His headache slowly faded and the neurological symptoms (numbness and weakness in the limbs) were completely gone throughout the observation period. No toxicity or other problems with the treatment were recorded (As shown in the Table IV).

TABLE IV. EMERGENCY MEDICATIONS ADMINISTERED

Medication	Dose	Route of Administration	Clinical Purpose
Captopril	100 mg	Oral	Initial blood pressure reduction
Furosemide (Lasix)	1 ampoule	Intravenous	Adjunctive management of hypertensive emergency
Labetalol	4 mL	Intravenous	Controlled reduction of blood pressure

5. CLINICAL OUTCOME AND FOLLOW-UP

5.1 Clinical Outcome

Good antihypertensive therapy began and the patient's blood pressure continued to fall over a period of time within the emergency department. Progressive improvement of his neurologic symptoms followed this. The pain in the headache was decreased in severity, and the numbness and weakness of the upper and lower limbs were completely resolved over the observation period. The patient was awake and alert, and had no neurological deterioration or new symptoms during his hospitalization. With a good clinical response and no acute intracranial abnormalities seen on non-contrast brain CT scan, the conservative management and close clinical observation was recommended. There were no adverse events or complications reported as a result of treatment in the emergency department.

5.2 Discharge and Follow-Up

After full clinical recovery and stabilization patient was sent home on the same day. He was advised to keep taking his regular anti-hypertensive treatment (Angizar H) and to have regular checks of his blood pressure and long-term treatment in the out-patient department. Modification of the antihypertensive regimen was suggested if appropriate for future follow-up based on the BP control and clinical condition of the patient. At this presentation to the emergency department, no magnetic resonance imaging (MRI), laboratory or vascular investigations or neurological consultation were carried out. The patient recovered fully, but definitive diagnosis/exclusion of some neurological diseases was not possible with the available clinical assessment (As shown in the Table V).

TABLE V. CLINICAL OUTCOME AND FOLLOW-UP SUMMARY

Parameter	Outcome
Blood pressure	Improved following emergency treatment
Headache	Completely resolved
Limb numbness	Completely resolved
Limb weakness	Completely resolved
Level of consciousness	Remained alert and oriented
Neurological deterioration	None observed
CT findings	No acute intracranial abnormalities
MRI performed	No
Laboratory investigations	Not performed
Neurology consultation	Not performed
Hospital disposition	Discharged home on the same day
Follow-up recommendation	Continue Angizar H and attend outpatient follow-up

6. DISCUSSION

The diagnostic dilemma is often encountered with patients with hypertensive emergency who present with acute neurological symptoms and signs that are similar to those of cerebrovascular disease as in the present case. The presence of marked HTN, generalized headache, transient (20 minutes) weakness and numbness, nausea and lethargy immediately

triggered the suspicion of acute HTN-mediated neurological injury. The 2024 European Society of Cardiology (ESC) Guidelines classify hypertensive emergency as a condition of severe hypertension and acute target-organ damage, which can be one of the most severe clinical presentations. Headache, lethargy, and neurologic symptoms should be evaluated promptly as they could be signs of hypertensive encephalopathy, ischemic stroke or intracranial hemorrhage. Importantly, the guidelines also add that in addition to the presence of persistent focal neurological deficits, one should be more suspicious of cerebrovascular disease rather than of hypertensive encephalopathy [18].

The main diagnostic dilemma in the current case comprised the ability to distinguish between a number of neurological disorders which may produce similar clinical presentation. The patient made a complete neurologic recovery after lowering the BP, but the available investigations did not allow a definite neurologic diagnosis to be made. Rather, the diagnosis was made based on the clinical history, neuroimaging, responsiveness to treatment and the absence of immediately life-threatening conditions (As shown in the Table VI).

TABLE VI. DIFFERENTIAL DIAGNOSTIC CONSIDERATIONS IN THE PRESENT CASE

Condition	Supporting Findings	Findings Against / Limitations	Diagnostic Status
Hypertensive encephalopathy	Severe hypertension, headache, lethargy, transient neurological symptoms	MRI unavailable; no neurological consultation	Possible but unconfirmed
Transient ischemic attack (TIA)	Complete neurological recovery	No MRI or vascular imaging	Cannot be excluded
Acute ischemic stroke	Sudden neurological deficit with vascular risk factors	Normal CT; rapid recovery; MRI unavailable	Less likely but not excluded
Intracranial hemorrhage	Severe hypertension with acute neurological symptoms	CT showed no hemorrhage	Effectively excluded
Posterior reversible encephalopathy	Severe hypertension with headache and neurological manifestations	MRI not performed	Possible but unconfirmed
Migraine with neurological symptoms	Headache and transient symptoms	No migraine history	Unlikely
Functional neurological disorder	Stroke-like presentation	Clinical findings favored an organic process	Unlikely
Metabolic disorders	May produce transient neurological deficits	Laboratory investigations unavailable	Cannot be excluded

The acute neurological symptoms despite a normal non-contrast brain computed tomography (CT) examination was particularly significant with this case. This observation should NOT be taken to mean that the symptoms were not related to neurological dysfunction in the patient. Instead, it underlines one of the significant drawbacks of emergency neuro-imaging. For patients with suspected stroke or hypertensive emergency, non-contrast computed tomography (CT) is the best initial imaging test to rule out a primary intracranial hemorrhage and other acute structural causes of the symptoms. The sensitivity of CT for hyperacute ischemic stroke, however, is low and it is not useful to diagnose hypertensive encephalopathy or posterior reversible encephalopathy. MRI, especially diffusion-weighted imaging and fluid-attenuated inversion recovery (FLAIR) sequences, offer significantly higher sensitivity for such conditions, and may show changes that are not seen on CT [19].

Favorable clinical response was also a major part of the process of diagnostic reasoning. Blood pressure control was associated with complete resolution of neurological symptoms, including headache, weakness of limbs and sensory symptoms, which indicated that these symptoms were likely reversible and closely linked to the hypertensive crisis itself. However, resolution of symptoms should not be considered diagnostic of the underlying condition as TIA and some stroke mimics will also show complete clinical resolution. The lack of MRI, vascular imaging, laboratory tests and specialist neurological assessment is therefore an important limitation of the diagnostic evaluation and does not allow for definitive statements to be made or excluded with regard to a number of competing diagnoses (As shown in the Table VII).

TABLE VII. COMPARISON OF THE PRESENT CASE WITH CURRENT EVIDENCE

Clinical Feature	Present Case	Current Evidence
Severe hypertension	Present	Common feature of hypertensive emergency
Acute neurological manifestations	Present	Frequently reported in hypertensive emergencies and acute stroke
Initial brain CT	Normal	Useful for excluding hemorrhage but limited for early ischemia and PRES
MRI performed	No	Recommended when diagnostic uncertainty persists
Response to antihypertensive therapy	Complete clinical recovery	Frequently observed in reversible hypertension-related neurological disorders
Final diagnosis	Hypertensive emergency with transient neurological manifestations	Consistent with a clinical diagnosis after exclusion of immediate life-threatening pathology, while acknowledging diagnostic uncertainty

This case is similar to the diagnostic uncertainty that often occurs in emergency medicine, as compared to other literature from the same time period. Recent reviews have highlighted the need for patients with severe hypertension and acute neurological symptoms to be quickly classified as either hypertensive emergency, ischemic stroke, intracranial haemorrhage or stroke mimics as the treatment is markedly different. The current recommendations recommend a prompt clinical evaluation, prompt neuroimaging, and careful blood pressure management and further neurological evaluation if there is any doubt after initial evaluation [20].

In clinical terms, this case serves as a reminder of some key points. First, in a patient with a hypertensive emergency, a normal non-contrast computed tomography (CT) examination should not rule out all neurological pathology. Secondly, clinical judgment is crucial in the interpretation of imaging results, especially in those cases where neurological symptoms improve very quickly following treatment. Third, MRI and expert neurological evaluation should be considered when there is any doubt regarding the diagnosis despite CT scan-negative results. Lastly, the key to successful management of a hypertensive emergency is to do more than rely on any one diagnostic tool, but to integrate the patient's history, physical examination, neuroimaging, treatment response, and follow-up evaluation. Similar clinical situations may benefit from the use of these principles for enhanced diagnostic precision, fewer unnecessary interventions, and more favorable outcomes for patients [21,22].

There are some limitations in this study. First, MRI was not performed and therefore early ischemic stroke, hypertensive encephalopathy or PRES could not be confirmed or excluded. Second, there were no lab investigations or vascular imaging. Thirdly, this report presents a case study and thus only provides a limited generalisability. However, these constraints provide useful examples of important items that can be referred to in emergency clinical settings.

7. CONCLUSION

This case illustrates the diagnostic challenges of a case of acute stroke-like neurologic symptoms with hypertensive emergency. Headache, weakness, numbness and change in neurological symptoms are concerning features in severe hypertension and may be a sign of acute hypertension-mediated target-organ damage or acute serious cerebrovascular disease, so these should not be overlooked. The need for clinical assessment, speedy BP control, and timely neuroimaging for morbidity reduction and appropriate therapeutic interventions remain essential aspects of emergency management. Non-contrast brain CT was effective in ruling out intracranial hemorrhage and other acute structural abnormalities which allowed immediate assessment of the life-threatening conditions. But the continued uncertainty of a diagnosis despite normal CT suggests that there is a significant restriction on emergency imaging. Because of lack of advanced testing (magnetic resonance imaging, vascular imaging, laboratory studies and specialist neurological assessment), it was not possible to confirm or rule out early ischemic stroke, transient ischemic attack, hypertensive encephalopathy and posterior reversible encephalopathy syndrome. The final clinical assessment, therefore, remained unchanged, and the patient was diagnosed as having hypertensive emergency with transient neurological manifestations, but with the understanding that other neurological causes could not be excluded. The patient's complete neurological recovery after the controlled antihypertensive therapy highlights the need for early treatment and careful clinical monitoring in patients with high blood pressure and neurological symptoms. In this case, however, the clinical improvement does not prove enough to make a definite neurological diagnosis. A thorough diagnostic evaluation is still critical whenever there is a clinical suspicion that may not be proved initially by imaging. The overall message of this report is that an integrated diagnostic strategy, including patient history, physical examination, neuroimaging, treatment response and appropriate follow-up is necessary. The clinician should be aware that in a patient with a normal initial CT scan, neurologic disease cannot be ruled out and should have a wide differential diagnosis for the evaluation of patients with hypertensive emergency. Because larger patient populations are needed to further develop diagnostic algorithms and to optimize the management of patients with severe hypertension with transient neurological manifestations, further studies are warranted.

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Conflicts of Interest:

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References

[1] C. P. McCarthy, R. M. Bruno, J. W. McEvoy, and R. M. Touyz, “2024 ESC Guidelines for the management of elevated blood pressure and hypertension: What is new in pharmacotherapy?” 2025.
[2] E. Berge, W. Whiteley, H. Audebert, G. M. De Marchis, A. C. Fonseca, C. Padiglioni, et al., “European Stroke Organization (ESO) guidelines on intravenous thrombolysis for acute ischaemic stroke,” European Stroke Journal, vol. 6, no. 1, pp. I–LXII, 2021.
[3] S. M. Greenberg, W. C. Ziai, C. Cordonnier, D. Dowlatshahi, B. Francis, J. N. Goldstein, et al., “2022 guideline for the management of patients with spontaneous intracerebral hemorrhage: A guideline from the American Heart Association/American Stroke Association,” Stroke, vol. 53, no. 7, pp. e282–e361, 2022.
[4] T. Potter, A. Agarwal, and T. J. Schaefer, “Hypertensive encephalopathy,” in StatPearls [Internet]. Treasure Island, FL, USA: StatPearls Publishing, 2024.

[5] S. J. Mendelson and S. Prabhakaran, "Diagnosis and management of transient ischemic attack and acute ischemic stroke: A review," JAMA, vol. 325, no. 11, pp. 1088–1098, 2021.
[6] E. H. Tedyanto, K. Tini, and N. A. K. Pramana, "Magnetic resonance imaging in acute ischemic stroke," Cureus, vol. 14, no. 7, 2022.
[7] M. Duering, G. J. Biessels, A. Brodtmann, C. Chen, C. Cordonnier, F. E. de Leeuw, et al., "Neuroimaging standards for research into small vessel disease—Advances since 2013," The Lancet Neurology, vol. 22, no. 7, pp. 602–618, 2023.
[8] L. Puy, A. R. Parry-Jones, E. C. Sandset, D. Dowlatshahi, W. Ziai, and C. Cordonnier, "Intracerebral haemorrhage," Nature Reviews Disease Primers, vol. 9, no. 1, p. 14, 2023.
[9] J. Olesen, "The international classification of headache disorders: History and future perspectives," Cephalalgia, vol. 44, no. 1, Art. no. 03331024231214731, 2024.
[10] K. Bennett, C. Diamond, I. Hoeritzauer, P. Gardiner, L. McWhirter, A. Carson, and J. Stone, "A practical review of functional neurological disorder (FND) for the general physician," Clinical Medicine, vol. 21, no. 1, pp. 28–36, 2021.
[11] A. N. Gewirtz, V. Gao, S. C. Parauda, and M. S. Robbins, "Posterior reversible encephalopathy syndrome," Current Pain and Headache Reports, vol. 25, no. 3, p. 19, 2021.
[12] S. Qiu and Y. Xu, "Guidelines for acute ischemic stroke treatment," Neuroscience Bulletin, vol. 36, no. 10, pp. 1229–1232, 2020.
[13] M. Abdalkader, J. E. Siegler, J. S. Lee, S. Yaghi, Z. Qiu, X. Huo, et al., "Neuroimaging of acute ischemic stroke: Multimodal imaging approach for acute endovascular therapy," Journal of Stroke, vol. 25, no. 1, pp. 55–71, 2023.
[14] U. Fischer, M. Branca, L. H. Bonati, E. Carrera, M. I. Vargas, A. Platon, et al., "Magnetic resonance imaging or computed tomography for suspected acute stroke: Association of admission image modality with acute recanalization therapies, workflow metrics, and outcomes," Annals of Neurology, vol. 92, no. 2, pp. 184–194, 2022.
[15] A. L. Czap and S. A. Sheth, "Overview of imaging modalities in stroke," Neurology, vol. 97, no. 20, Suppl. 2, pp. S42–S51, 2021.
[16] Y. K. Cetinoglu, I. O. Koska, M. E. Uluc, and M. F. Gelal, "Detection and vascular territorial classification of stroke on diffusion-weighted MRI by deep learning," European Journal of Radiology, vol. 145, Art. no. 110050, 2021.
[17] R. G. Nogueira, D. C. Haussen, D. Liebeskind, T. G. Jovin, R. Gupta, A. Jadhav, et al., "Stroke imaging selection modality and endovascular therapy outcomes in the early and extended time windows," Stroke, vol. 52, no. 2, pp. 491–497, 2021.
[18] A. Astarita, M. Covella, F. Vallelonga, M. Cesareo, S. Totaro, L. Ventre, et al., "Hypertensive emergencies and urgencies in emergency departments: A systematic review and meta-analysis," Journal of Hypertension, vol. 38, no. 7, pp. 1203–1210, 2020.
[19] M. A. Akbarzadeh, S. Sanaie, M. Kuchaki Rafsanjani, and M. S. Hosseini, "Role of imaging in early diagnosis of acute ischemic stroke: A literature review," The Egyptian Journal of Neurology, Psychiatry and Neurosurgery, vol. 57, no. 1, p. 175, 2021.
[20] Y. Tang, Atlas of Emergency Neurovascular Imaging. Cham, Switzerland: Springer International Publishing, 2020, pp. 21–32.
[21] B. H. Buck, N. Akhtar, A. Alrohimi, K. Khan, and A. Shuaib, "Stroke mimics: Incidence, aetiology, clinical features and treatment," Annals of Medicine, vol. 53, no. 1, pp. 420–436, 2021.
[22] T. Unger, C. Borghi, F. Charchar, N. A. Khan, N. R. Poulter, D. Prabhakaran, et al., "2020 International Society of Hypertension global hypertension practice guidelines," Hypertension, vol. 75, no. 6, pp. 1334–1357, 2020.