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Case Report

Hypertensive emergency presenting with acute spontaneous subdural hematoma

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ABSTRACT

Hypertensive crisis is a serious medical condition defined as severely elevated blood pressure; typically the systolic blood pressure is above 180 mmHg, and/or the diastolic blood pressure is above 120 mmHg. Hypertensive crises are divided into two categories: hypertensive urgency and hypertensive emergency. Hypertensive urgency is asymptomatic while hypertensive emergency presents with end-organ damage requiring more aggressive blood-pressure lowering. The common presentations for neurological end-organ dysfunction in conjunction with hypertensive emergency include ischemic strokes, intracranial hemorrhage, subarachnoid hemorrhage, head trauma, and hypertensive encephalopathy. The occurrence of acute spontaneous subdural hematoma (SDH) as a neurological end-organ damage complicating hypertensive crisis is rare and should receive tight blood-pressure lowering to prevent further bleeding. We present a case of hypertensive emergency complicated with acute spontaneous SDH.

<**Learning objective:** Acute spontaneous subdural hematoma as a neurological end-organ damage complicating hypertensive crisis is a rare entity. Hypertensive crisis presenting with acute spontaneous subdural hematoma prompts tight blood pressure control in timely manner to prevent permanent neurological sequalae. Rapid and severe elevation in the blood pressure might be a potential etiology of spontaneous bleeding into the subdural space. Further studies are needed to investigate this finding>.

Introduction

Hypertensive crisis is a critical medical condition that englobes two entities: urgency and emergency. Both presenting with significantly elevated blood pressure (systolic pressure ≥180 mmHg and/or diastolic pressure ≥120 mmHg), however, with differing symptomatology. Hypertensive urgency is asymptomatic, while hypertensive emergency is associated with end-organ damage, such as cardiovascular, pulmonary, renal, sympathetic overactivity, and neurological end-organ dysfunction. Hypertensive emergency requires prompt blood pressure control, within minutes to hours, by intravenous blood pressure medications and critical care attention to avoid further end-organ damage [1]. Hypertensive emergencies complicated with acute neurological end-organ damage account for 30% of hospitalized patients due to severe hypertension and the majority of those who die [2]. Acute neurological manifestations in the setting of significantly elevated blood pressure include ischemic strokes, intracranial hemorrhage, subarachnoid hemorrhage, head trauma, and hypertensive encephalopathy. The occurrence of acute spontaneous subdural hematoma (SDH) is uncommon and limited to case reports and case series. These reported cases describe many etiologies for the spontaneous bleeding into the subdural space (e.g. arteriovenous fistula [3], arteriovenous malformation, arachnoid cyst, coagulopathy, meningioma [4], cocaine [5], and bleeding from small cortical branches of the middle cerebral artery [6]). We report a rare case where the acute SDH presented with hypertensive emergency.

Case report

We present the case of a 53-year-old male with past medical history of hypertension, who presented to the emergency department with a 3-day history of left-sided frontotemporal headache of sudden onset. The headache was described as pressure-like, constant, 8/10 in intensity, radiated to the left peri-orbital area, without alleviating or aggravating factors. It was associated with nausea and dizziness upon standing from supine

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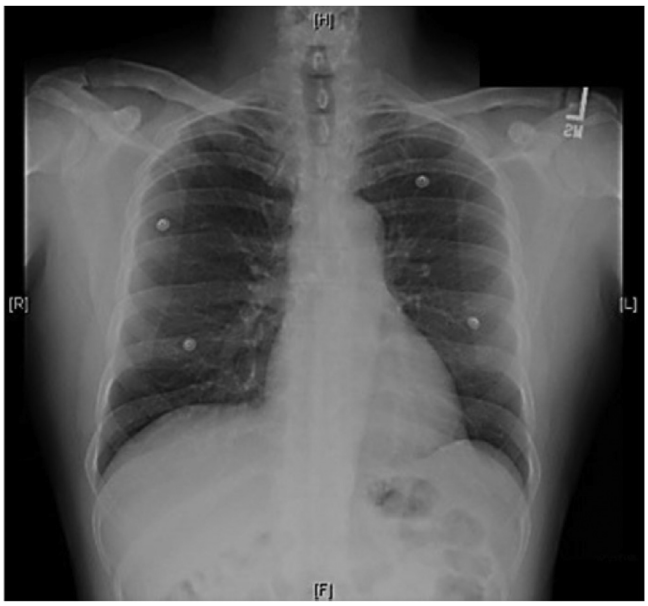

Fig. 1. Chest X-ray showing normal cardiac shadow and no pulmonary disease.

position. The patient denied fever, vomiting, or photophobia. On presentation, his blood pressure was 255/130 mmHg, pulse 83 beats per minute, respiratory rate 16 breaths per minute, and temperature 37.2 °C. He was diagnosed with hypertension three years before this presentation and admitted to being completely noncompliant with his medications, which included: amlodipine 10 mg daily and hydrochlorothiazide 25 mg daily. Complete blood count, comprehensive metabolic panel, kidney function, thyroid function, and cardiac enzymes were within normal limits. Urine toxicology screen was negative and chest X-ray did not show any pulmonary disease or cardiomegaly (Fig. 1). The electrocardiogram (ECG) did not satisfy any criteria for left ventricular hypertrophy, and it also did not show any ischemic changes or arrhythmias (Fig. 2). Transthoracic echocardiography showed mild left ventricular hypertrophy but otherwise it was within normal limits. Head computed tomography (CT) without contrast performed in the emergency room revealed an acute small SDH above the left frontal cortex without signs of increased intracranial pressure (Fig. 3).

Interestingly, the patient denied any recent or even remote history of head trauma, so the SDH was deemed to be spontaneous. He also denied the use of non-steroidal anti-inflammatory drugs, aspirin, warfarin, or other anticoagulation or antiplatelet medication. The presence of spontaneous SDH in the setting of severely elevated blood pressure was considered as a sign of end-organ damage secondary to hypertensive emergency. In the emergency room, the patient received a total of four doses of 20 mg of intravenous labetalol every 10–15 min with a subsequent reduction in blood pressure from 255/130 mmHg–180/113 mmHg and a pulse rate of 61 beats per minute. He was also given 1 g of levetiracetam for seizure prophylaxis and desmopressin 40 μg intravenously to prevent further bleeding. Repeat head CT 6 h after the first one showed stable SDH without any interval changes. CT angiography of the head and neck performed the next day of admission ruled out any vascular aneurysms or arteriovenous malformations. A neurosurgeon was consulted who recommended close observation in the neurosurgical unit. Since the subdural hematoma was small, non-expanding, and not associated with elevated intracranial pressure or focal neurological deficits, the decision was made to treat medically with close observation, aggressive blood pressure control, and frequent neurological examinations. Thirteen hours later the patient was started on oral blood-pressure lowering medications including amlodipine 10 mg and hydrochlorothiazide 25 mg daily. Blood pressure further decreased to 160/105 mmHg over the next 3 days after admission. Oral labetalol 100 mg 3 times a day was also started during the second day and was increased to 200 mg 3 times a day on day 3. On day 5, the patient was discharged home with a blood pressure of 130/85 mmHg and a pulse rate 66 beats per minute. A trend of systolic blood pressure, diastolic blood pressure, and heart rate along with the medications administered is illustrated in Table 1

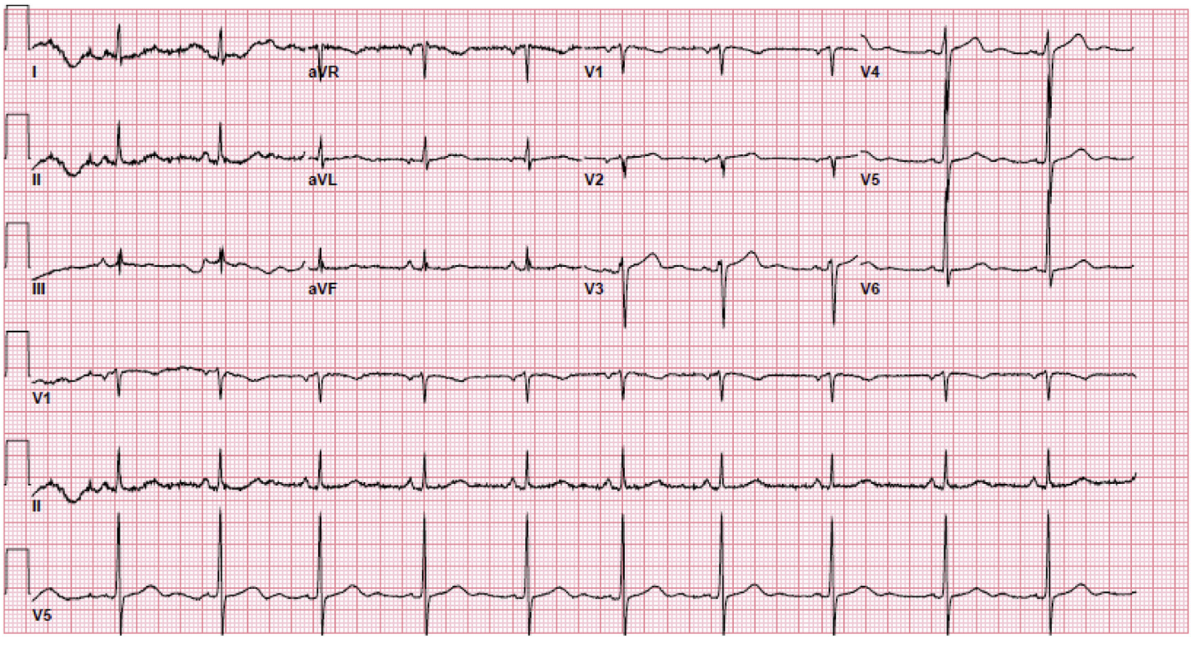

Fig. 2. Electrocardiogram.

Fig. 3. Computed tomography of the head without contrast showing an acute subdural hematoma over the left cerebral frontal cortex.

and Fig. 4. The patient was instructed to take amlodipine 10 mg daily, hydrochlorothiazide 25 mg daily, and labetalol 200 mg 3 times a day for blood pressure control. A follow up head CT 2 weeks after discharge showed a stable hematoma in size without any signs of expansion or increased intracranial pressure. His blood systolic blood pressure was controlled within an adequate range.

Discussion

Hypertensive emergency must be rapidly recognized and the reduction in blood pressure by 10–20% during the first hour should be achieved by intravenous antihypertensive medications [1]. In this case, the patient presented without laboratory, imaging, or ECG findings indicating target organ damage. His hypertensive crisis appeared to be due to worsened essential hypertension from complete noncompliance with his medications rather than secondary hypertension. This is also reflected on the mild left ventricular hypertrophy found on his echocardiogram, which likely developed over the three-year period after being diagnosed with hypertension and not taking any medications, the rapid response to blood-pressure lowering agents which again favors worsened essential hypertension, and normal laboratory results (electrolytes, kidney function, complete blood count, and thyroid function) which potentially ruled out secondary hypertension. The symptoms of headache, nausea, and dizziness lead to the decision of performing a CT scan without contrast of the head. The finding of an acute spontaneous SDH prompted the diversion in treatment plan and to recognize this SDH as an end-organ damage complicating the hypertensive crisis; hence, labeling it as a hypertensive emergency rather than urgency. Most cases of hypertensive emergencies with central nervous system (CNS) involvement present with ischemic stroke, intracerebral hemorrhage, subarachnoid hemorrhage, or hypertensive encephalopathy. Acute spontaneous SDH is an entity of rare presentation, and yet is even rarer to present as a neurological end-organ damage with hypertensive crisis. It is unclear whether the hypertensive crisis happened secondary to the pain from the subdural bleeding, or that the hypertensive crisis caused the spontaneous SDH. In our

Table 1 Vital signs and medications administered during hospital course. HCTZ, hydrochlorothiazide; PO, oral route; IV, intravenous route.

Flow sheet	Presentation	Day 1 AM	Day 1 PM	Day 2 AM	Day 2 PM	Day 3 AM	Day 3 PM	Day 4 AM	Day 4 PM
Systolic blood pressure (mmHg)	255	180	173	167	155	160	151	143	137
Diastolic blood pressure (mmHg)	130	113	96	91	84	105	90	82	93
Heart rate (beat per minute)	83	61	63	72	66	69	62	70	67
Medications	• Labetalol 20 mg IV 3 dose • Desmopressin 40 mcg IV ×1 • Levetiracetam 1 g IV ×1	Amlodipine 10 mg ×1 HCTZ 25 mg ×1 Levetiracetam 750 mg PO ×1	• Labetalol 10 mg IV ×1 • Levetiracetam 750 mg PO ×1	• Amlodipine 10 mg ×1 • HCTZ 25 mg ×1 • Labetalol 100 mg PO ×1 • Levetiracetam 750 mg PO ×1	• Labetalol 100 mg PO ×2 • Levetiracetam 750 mg PO ×1	• Amlodipine 10 mg ×1 • HCTZ 25 mg ×1 • Labetalol 200 mg PO ×1 • Levetiracetam 750 mg PO ×1	• Labetalol 200 mg PO ×2 • Levetiracetam 750 mg PO ×1	• Amlodipine 10 mg ×1 • HCTZ 25 mg ×1 • Labetalol 200 mg PO ×1 • Levetiracetam 750 mg PO ×1	• Labetalol 200 mg PO ×2 • Levetiracetam 750 mg PO ×1

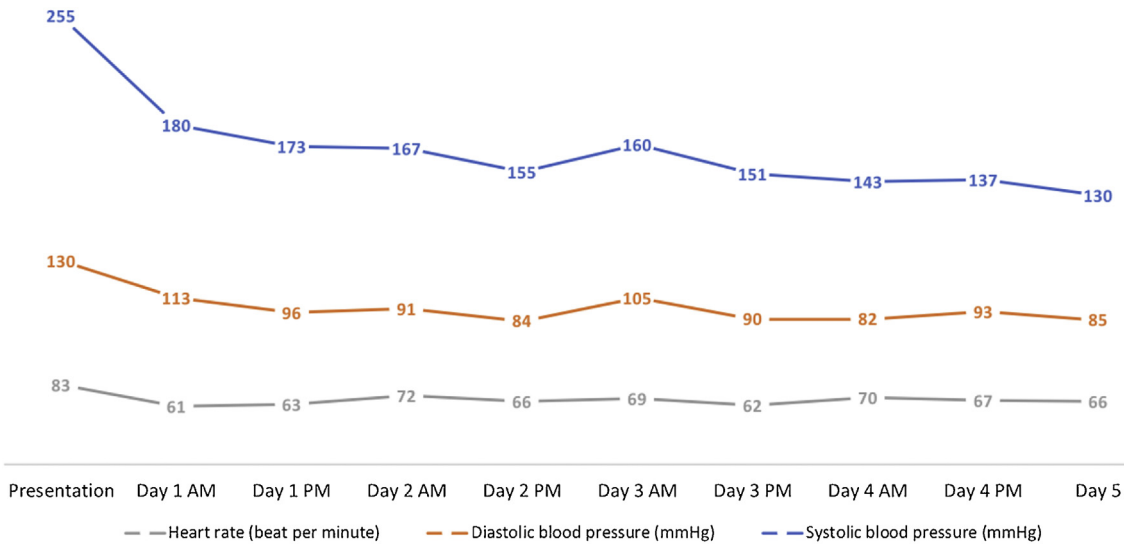

Fig. 4. Graph representing vital signs trend during hospital course.

opinion, we suggest that the acute spontaneous SDH is the result of the hypertensive crisis. This consideration is supported by a similar pathophysiology that possibly explains cocaine-induced spontaneous SDH. In this theory, a severe elevation of the blood pressure, mainly from cocaine-induced sympathetic system overactivity, results in adaptive vasoconstriction of the cerebral blood vessels, and consequently, a hemorrhage results from chronic ischemia and rupture of the blood vessels wall [5]. To date, there are no clear guidelines or clinical trials that address the target blood pressure goal in acute spontaneous SDH, mostly because it is a rare entity and is limited to case reports. In one randomized clinical trial, the INTERACT2 trial, which randomly assigned 2839 patients with acute (within 6 h) intracerebral hemorrhage and elevated blood pressure to either intensive blood-pressure lowering (target systolic blood pressure $<$140 mmHg within one hour) versus traditional management (target systolic blood pressure $<$180 mmHg) found that intensive blood-pressure lowering was associated with improved measures of disability [7]. However, the INTERACT2 trial studied spontaneous intracerebral hemorrhage rather than subdural hematoma. In our patient, intensive blood-pressure lowering measures resulted in stabilization of the subdural hematoma and prevented further neurological damage. We learned from our case that the importance of rapidly identifying the acute spontaneous SDH as an end-organ damage is to divert the management to a more intensive and rapid approach, similar to other common end-organ dysfunctions happening with hypertensive emergencies, in order to prevent further expansion of the hematoma and to avoid permanent neurological sequelae.

Conclusion

Hypertensive crisis presenting with acute spontaneous SDH is a rare entity and should be identified as hypertensive emergency rather than urgency, which prompts tight blood-pressure control in a timely manner.

Conflict of interest and financial disclosure

The authors confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

Consent

Written and documented consent has been obtained from the patient to publish his case report.

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References

[1] Elliott WJ. Clinical features in the management of selected hypertensive emergencies. Prog Cardiovasc Dis 2006;48:316–25.
[2] Mayer SA, Kurtz P, Wyman A, Sung GY, Multz AS, Varon J, et al. Clinical practices, complications, and mortality in neurological patients with acute severe hypertension: the Studying the Treatment of Acute hyperTension registry. Crit Care Med 2011;39:2330–6.
[3] De Aguiar GB, Veiga JCE, De Almeida Silva JM, Conti MLM. Spontaneous acute subdural hematoma: a rare presentation of a dural intracranial fistula. J Clin Neurosci 2016;25:159–60.
[4] Okuno S, Touho H, Ohnishi H, Karasawa J. Falx meningioma presenting as acute subdural hematoma: case report. Surg Neurol 1999;52:180–4.
[5] Alves ÓL, Gomes Ó. Cocaine-related acute subdural hematoma: an emergent cause of cerebrovascular accident. Acta Neurochir (Wien) 2000;142:819–21.
[6] Mulcahy MJ, Chaganti J, Dower A, Al-Khawaja D. Spontaneous acute arterial subdural hematoma. World Neurosurg 2018;110:403–6.
[7] Anderson CS, Heeley E, Huang Y, Wang J, Stapf C, Delcourt C, et al. Rapid blood-pressure lowering in patients with acute intracerebral hemorrhage. N Engl J Med 2013;368:2355–65.