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Spontaneous Intracranial Hemorrhage in a 34-Year-Old Male Patient Related to Hypertensive Emergency

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ABSTRACT

Introduction: The incidence of hypertension is currently moving toward younger ages. It's thought that an unhealthy lifestyle serves as a trigger. Hypertension can increase morbidity and mortality related to cardiovascular disease, even at a young age. This case report is intended to report the incidence of spontaneous intracranial hemorrhage in a 34-year-old male patient during a hypertensive emergency. **Case:** A 34-year-old male patient was rushed to the emergency unit at midnight due to a sudden decrease in consciousness. Headaches and seizures occurred before admission. The patient denied having any history of head trauma. The patient's habits included excessive caffeine consumption, frequent late-night sleeping, and extensive smoking. At the time of admission, the patient's vital signs indicated a poor clinical condition: GCS E1V1M1, BP 212/118 mm/Hg, deep irregular rapid breathing, sometimes followed by periodic apnea, RR 28 breath/minute, HR 111 beat/minute, SpO2 50%, T 36.8°C, which gradually developed hyperthermia. Both eyes had constricted and fixed pupils; the light reflexes were negative. A neurological examination revealed the body's left lateralization. The head CT scan without contrast indicated hemorrhage in the right intracerebral, midbrain, pons, intraventricular, and subarachnoid areas, with estimated total volume of about 31 ml. Conservative treatment was chosen due to the bleeding location in the deep brain structure of GCS 3, which was considered to have a poor outcome. **Conclusion:** Hypertension in the younger age group is commonly associated with unhealthy lifestyles, which increase morbidity and mortality related to cardiovascular disease.

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INTRODUCTION

Hypertension is often considered a disease that occurs in older people; however, the incidence of hypertension is currently shifting to younger people.[1] The incidence continues to rise and is associated with an unhealthy lifestyle. Among young adults, the most notable modern-day risk factors are a heavy salt diet, smoking, alcoholism, coffee addiction, an irregular sleep cycle, and sedentary activity.[2]

Guidelines define young adults as those under the age of 30–50 years.[3] In 2023, the World Health Organization (WHO) reported that around 1.28 billion adults aged 30–79 had hypertension.[4] In Saudi Arabia, 15.2% of adults had hypertension.[2] Hypertension affects an estimated 22.4% of people aged 18–39 in the United States. In 2018, the Health Survey in England stated that hypertension affected 2.6% of people aged 16–24 and 12.2% of those aged 35–44.1 In 2018, Indonesian Basic Health Research revealed that hypertension affected 31.6% of people aged 31–44 and 45.3% of those aged 45–54 had hypertension.[5]

Hypertension is commonly known as a silent killer due to its lack of severe symptoms.[2] Untreated chronic hypertension can lead to a hypertensive emergency, which is an acute elevation in blood pressure >180/120 mmHg with target organ damage such as pulmonary oedema, cardiac ischemia, acute renal failure, neurologic deficits, aortic dissection, and eclampsia, which has a high mortality rate even at a young age.[3] This case report aims to report the incidence of spontaneous intracranial hemorrhage in a 34-year-old male patient who was experiencing a hypertensive emergency.

CASE

At midnight, the hospital's emergency unit received a 34-year-old male patient who had suddenly lost consciousness. His colleague stated that the patient had been staying up late playing an online game before the incident. Suddenly, the patient complained of a headache, then had a seizure and went unconscious. The patient denied having any history of head trauma. The patient's medical history was unknown. However, information about the patient's habits revealed that he often stayed up late, was a heavy smoker, and drank a lot of coffee.

The patient's vital signs on admission revealed a poor clinical condition: the Glasgow Coma Scale (GCS) was E1V1M1, the blood pressure (BP) was 212/118 mm/Hg, the breathing pattern showed deep irregular rapid breathing, sometimes followed by periodic apnea, the respiratory rate (RR) was 28 breaths/minute, the heart rate (HR) was 111 beats/minute, the oxygen saturation (SpO2) was 50%, and the body temperature (T) was 36.8°C.

The patient snored, indicating a partial airway obstruction. Both eyes' pupils were constricted and fixed, and light reflexes were negative. A neurological examination revealed the body's left lateralization. To clear the airway, head tilt and chin lift techniques were performed; the Oro Pharyngeal Airway (OPA) was inserted to maintain the clear airway; 15 liters of oxygen supplementation were given via a non-rebreathing mask (NRM); and an intravenous (IV) line was installed. The patient experienced profuse hematemesis, prompting the insertion of a nasogastric tube (NGT).

A head CT scan without contrast revealed the following findings, which the radiologist confirmed (Figure 1): a hyperdense lesion with perifocal oedema in the right internal capsule, midbrain, and pons; a hyperdense lesion in the 3rd and 4th ventricles; a hyperdense lesion in the bilateral sylvian fissure; and a hypodense lesion in the right centrum semiovale, right corona radiata, and left internal capsule. The radiologist noted flattened gyri and narrowed sulci, bilateral lateral ventricle dilatation, especially in the temporal horn, indicating ventriculomegaly, a narrowed subarachnoid space, no midline shift, and an estimated total hemorrhage volume of about 31 ml. The electrocardiography (ECG) examination was done as shown in Figure 2, but other supporting diagnostic examinations were not examined due to the patient's family preference.

Due to the poor prognosis, a neurosurgeon suggested a conservative treatment plan. Citicoline 500 mg IV and piracetam 3 gr IV were given to enhance brain function. Mannitol 100 mL IV drip was used to reduce the intracranial pressure (ICP). Ondansetron 8 mg IV was administered as an antiemetic, ranitidine 50 mg IV to prevent further hematin formation, and paracetamol 1 gr IV drip as an antipyretic. The patient was then transferred to the Intensive Care Unit (ICU). The intensivist planned to perform endotracheal intubation, but the patient's family refused. The patient's vital signs worsened 3 hours after transfer to the ICU, with blood pressure further decreasing to 108/98 mmHg, the body temperature rising to 40°C, deep and shallow breathing, profuse hematemesis, and dilated pupils in both eyes. A moment later, the patient died.

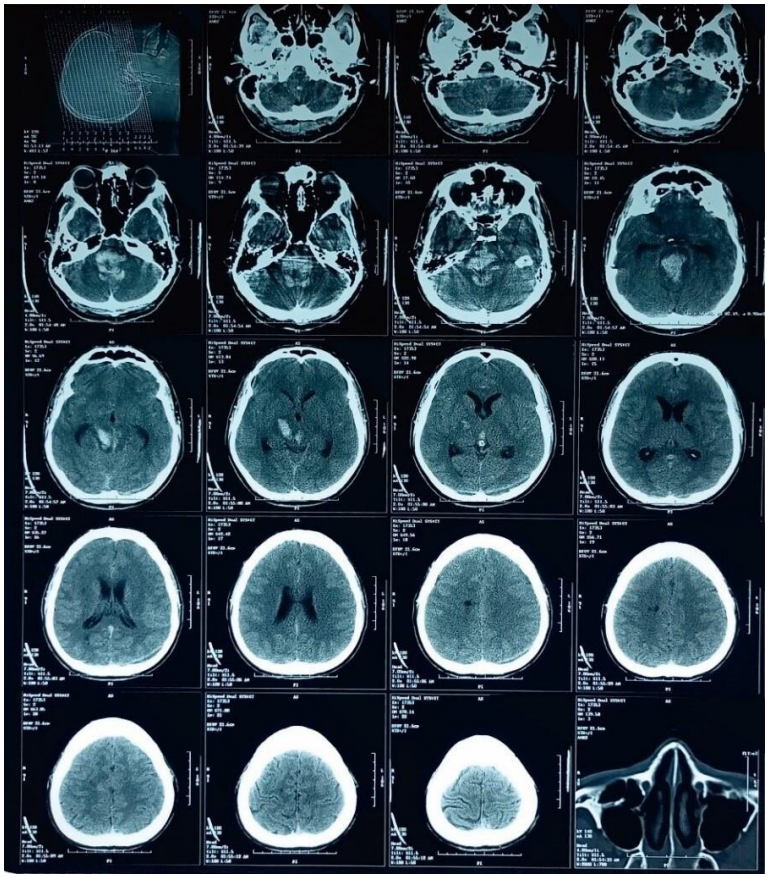

Figure 1. Head CT scan without contrast showed intracerebral and intraventricular hemorrhage with the estimated total hemorrhage volume of about 31 ml

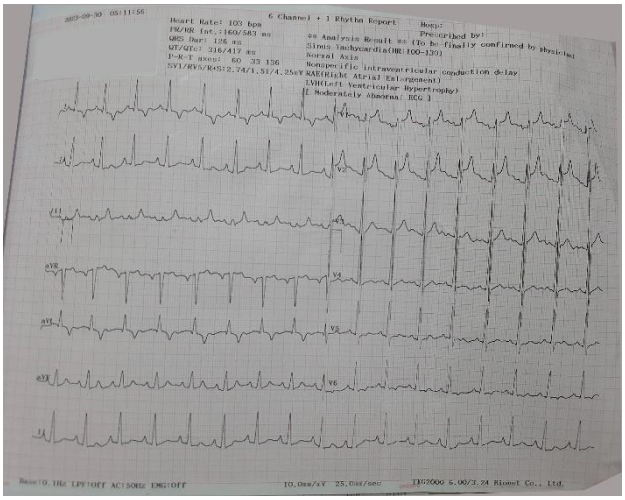

Figure 2. Electrocardiography Examination;
An ECG result showed regular sinus rhythm; heart rate 100 beat/minute; norm axis; P wave in lead II > 2.5 mm tall which indicated right atrial enlargement; the sum of S wave in V1 plus the R wave in V5 or V6 is greater than 35 mm which indicated left ventricular hypertrophy; and T wave inversion in lead I, aVL, V1, V2.

DISCUSSION

Hypertension is frequently associated to old age-related diseases. This is because the aging process involves a variety of physiological changes, including atherosclerosis and vascular changes, that contribute to the incidence of hypertension.[6] However, nowadays, the incidence of hypertension is shifting to younger ages.[1] Young people are susceptible to several risk factors that influence the occurrence of hypertension, including non-modifiable, modifiable, and socioeconomic ones.

Sex is a non-modifiable risk factor. Although hypertension is more common in men, the gradient in its development over time in women is steeper, whereas the blood pressure threshold that causes cardiovascular disease is lower. Sex-related risk factors, such as hypertensive diseases during pregnancy and menopause, may contribute to these differences.[7] A family history of hypertension is another non-modifiable risk factor. There is a significant link between hypertension and genetics; therefore, those with a family history of hypertension are more likely to develop the illness in the future.[2,6]

Lifestyle is a modifiable risk factor. Various unhealthy lifestyles increase the risk of hypertension. A daily salt intake of more than 10 grams significantly increases the risk of developing hypertension.[2,6] This is due to the fact that excess natrium in the body causes water retention, increases systemic peripheral resistance, alters endothelial function, changes the

structure and function of large elastic arteries, modifies sympathetic activity, and alters autonomic nerve function.[2,6,8] A recent study revealed a correlation between smoking status, coffee intake and the incidence of hypertension.[9] Nicotine, the main component in cigarettes, stimulates the sympathetic nervous system and epinephrine release, resulting in higher blood pressure.[2,6,10] Caffeine contained in coffee raises blood pressure by blocking adenosine receptors in vascular tissue, causing vasoconstriction, and increasing the plasma level of stress hormones.[9] Nowaday, alcoholic beverages are consumed regularly by most of the societies. Several studies revealed the association between alcohol consumption and hypertension. Alcohol stimulates the Renin Angiotensin Aldosterone System (RAAS), which makes more powerful vasoconstrictor hormones that help the natrium and water retention. This can change blood pressure and cause hypertension in young adult.[2,6,11]

Cardiovascular diseases like hypertension are associated with chronic sleep irregularities, both in duration (less than 7 hours per day) and timing. During the non-rapid eye movement (NREM) sleep phase, there is a lower level of sympathetic nerve activity. When entering the Rapid Eye Movement (REM) sleep phase, which usually occurs in the second half of the night, there is an increase in sympathetic nerve activity, resulting in surges in blood pressure.[12]

As a result of the rapid development of technology, convenience causes sedentary behavior. Lack of exercise causes obesity, overweight, and a higher Body Mass Index (BMI), which are risk factors for hypertension and other non-communicable diseases. Low-socioeconomic status individuals are often more susceptible to hypertension due to their limited knowledge and limited access to health facilities.[2,6]

In this case, there are risk factors that support the occurrence of hypertension in the patient, specifically the male gender, the habit of staying up late, smoking, and drinking coffee. The patient's is a high school graduate, and his original residence is in a remote area, which indicates low socioeconomic status.

Intracerebral hemorrhage (ICH) is commonly divided into traumatic and non-traumatic. Non-traumatic ICH is divided into primary (spontaneous) and secondary. Primary ICH occurs when small arteries rupture due to chronic hypertension (incidence 60%) or amyloid angiopathy (incidence 30%). Secondary ICH can refer to several factors, including aneurysms, vascular malformations, vasculitis, hemorrhagic conversion of infarcts, and drug side effects. Primary ICH is associated with hypertension; over 60% of cases occur in deep brain structures such as the posterior fossa, pons, basal ganglia, and thalamus. Amyloid angiopathy is associated with ICH, primarily affecting the lobar area of the brain.[13,14] Based on the location of the hemorrhage and the previously mentioned prevalence, we suspected spontaneous ICH due to chronic hypertension, as the patient denied any trauma history. This is supported by the ECG result support this, which meets the Sokolow-Lyon Criteria for left ventricular hypertension (LVH), where the sum of the S wave in V1 and the R wave in V5 or V6 is greater than 35 mm.

According to Monro Kellie's law, ICH causes a sudden increase in brain parenchyma mass and directly increases intracranial pressure (ICP) because the skull is a rigid space. The normal ICP pressure in adults is 5–15 mmHg.[15] The high ICP is directly proportional to the volume of blood. An increase in ICP compresses and disrupts the surrounding neuronal tissue. This causes a focal neurological deficit, lower cerebral perfusion pressure (CPP), and brain herniation.[13,14]

Increased ICP symptoms include altered levels of consciousness, headaches, nausea, projectile vomiting, the onset of a new neurological deficit, and altered visual function.[16] The typical signs of increased ICP include high blood pressure, bradycardia, and irregular respiration known as Cushing's triad, which indicate an impending brain herniation. In the first stage, blood pressure and heart rate rise in response to sympathetic nerve activity in order to overcome high ICP. Rising blood pressure serves to maintain CPP and prevent further brain ischemia. In the second stage, high blood pressure persists, but heart rate decreases. High blood pressure causes bradycardia by activating baroreceptors in the aortic arch, which subsequently stimulates the parasympathetic nerve. Bradycardia is also believed to be caused by intracranial vagal nerve impairment. Intracranial vagal nerve compression also compromises the brainstem, which affects respiration.[15,16] The clinical manifestation depends on the hemorrhage location. If the hemorrhage location is in the brainstem, which is responsible for many vital functions of life, the clinical manifestation can be a decrease in consciousness, cardiorespiratory distress, or even arrest.[13,14]

The development of fixed pupils and non-reactive pupillary light reflexes following a neurological test is associated with increased ICP. The location of the pupillomotor nuclei in the dorsal midbrain and the efferent oculomotor nerve running from the midbrain to the superior orbital fissure become impaired due to descending transtentorial herniation and brainstem compression.[14,16] An increased ICP also overstimulates the vagus nerve, causing an increase in gastric acid secretion, which leads to gastroduodenal ulcer formation known as Cushing's ulcer. The mixture of gastric acid and blood causes hematin formation, and later hematemesis can occur.[17]

Central hyperthermia is characterized by an

elevated setting of the thermoregulatory center that is related to cytokines and doesn't respond to antipyretic treatments. Central hyperthermia is diagnosed when the body temperature is ≥38.3°C after the onset of ICH and there are no signs of infection or a negative workup examination. The thermoregulatory centers that are located in the midbrain, hypothalamus, and spinal cord can be impaired due to ICH. The mortality rate is significant due to complications from a high temperature.[18] In this case, an intracranial hemorrhage in the deep brain structures with a total volume of about 31 ml clearly increased ICP. Despite the lack of direct ICP measurement due to invasive procedures in the poor prognosis case and cost concerns, the patient's clinical manifestations clearly indicated an increase in ICP. There was a sudden onset of headache, a sudden neurological deficit such as seizure and left lateralization of the body, a sudden decrease in unconsciousness, and a fixed non-reactive pupillary light reflex, all of which indicate an increase in ICP. The high blood pressure, along with tachycardia in this case, suggested the first stage of Cushing's triad. The patient's head CT scan showed flattened gyri, narrowed sulci, and bilateral lateral ventricle dilatation, especially in the temporal horn, suggesting increased ICP.[16] T wave inversion in lead I, aVL, V1, and V2 of the patient's ECG examination can also be an indication of increased ICP. According to the study, one of the differential diagnoses of inverted T waves on the 12-lead ECG is an increased ICP.[19] This patient was suspected of having central hyperthermia since his temperature climbed quickly after ICH occurrence and he did not respond to the antipyretic drug. However, the cause of the infection could not be ruled out because the history of previous infections was not known and a laboratory examination for infection was not carried out.

The choice of treatment plan, whether surgical or conservative, depends on the site and size of the bleeding. A Cochrane database meta-analysis found that surgical treatment in patients with ICH located in the deep brain, along with intraventricular hemorrhage (IVH) that leads to hydrocephalus, had poor outcomes. Meanwhile, patients whose bleeding location was less than 1 cm from the cerebral cortex's surface had better outcomes.[13] Conservative treatment in IVH has a better outcome compared with surgical treatment using an external ventricular drain (EVD) in patients with a GCS > 10, and it can prevent unwanted complications of EVD such as cerebrospinal fluid infection.[20] Based on the STICH II trial, surgical treatment may have benefits in patients aged between 50 and 69 years, GCS > 9, a bleeding volume of 20–60 ml, or surgical evacuation within 8 hours of ictus.[13] In this case, conservative treatment was chosen due to the bleeding location in the deep brain structure of GCS 3, which was considered to have a poor outcome.

This case report was limited by a lack of adequate information and supporting laboratory or radiology examinations. So, the primary disease that causes hypertension cannot be excluded, as well as secondary causes of ICH such as aneurysm, vascular malformation, vasculitis, and a history of long-term substance consumption that increases the risk of bleeding. Future research must be more comprehensive to bolster the findings of this case report.

CONCLUSION

In younger age groups, unhealthy lifestyles are believed to trigger hypertension, which can increase morbidity and mortality related to cardiovascular disease, including ICH.

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Conflict of Interest

The authors have no conflicts of interest to disclose.

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Author Contributions

MN contributed to the data collection and wrote the manuscript. DW was a senior author, manuscript conceptor, and manuscript reviewer.

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