Cushing Reflex Variant: A case report

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Abstract

In our case, we report a 67-year-old male admitted with lower limb ischemia for angiography under vascular surgery. During the procedure, he exhibited a sudden change in consciousness level, an abnormal breathing pattern, and bradycardia associated with normal blood pressure. This case represents a rare and detrimental manifestation of the Cushing reflex present with isolated bradycardia. This case report concluded a possible early haemorrhagic insult on top of chronic infarction. To direct the patient to the appropriate course of action, they must be monitored in an intensive care unit and have their intracranial pressure continuously checked.

Keywords

Cushing reflex; Bradycardia; Early haemorrhagic insult; Raised intracranial pressure; Chronic infarction

Imprint


Abbreviation

CT angiography = Computed tomography angiography, MAP = Mean arterial pressure, ECG = Echocardiogram, CT brain = Computed tomography brain, ICP = Intracranial pressure IV = intravenous, CK-MB = Creatine kinase-MB, CK = Creatine kinase, GCS = Glasgow coma score, ACLS = Advanced cardiac life support.

Introduction

Cushing's Triad is defined as respiratory irregularity, bradycardia, and hypertension. Hypertension is considered the most common presentation, whereas hypotension is rare and associated with detrimental consequences [1]. Originally, Cushing’s Reflex was first described in 1903 as acute hemodynamic changes in response to intracerebral haemorrhage [2, 3]. Later, this brain-heart interaction was studied with a signaling pathway that may indicate acute brain insult [4]. In 1977, Dr. Fitch demonstrated that an increase in blood pressure was the last of the six indices related to increased intracranial pressure [5]. Cushing’s reflex is a protective mechanism and possibly a sign of fatal brain insult brought during critical brain ischemia, which can lead to a release of catecholamines that increases MAP (mean arterial blood pressure) in response to the rising intracranial pressure [6]. There is a paradoxical response in Cushing’s Triad as whenever there is a release in catecholamine, which leads to a rise in blood pressure, surprisingly, it is not accompanied by tachycardia as expected. The main feature is the bradycardia that reflects the vago-sympathetic response [7]. Cushing's Reflex is considered a late and ominous phenomenon that presents before circulatory failure. When brain insult occurs, brain edema is initiated, which progress till it compromises the brain blood vessel causing compression and eventually ischemia. The body’s response to stroke activates the sympathetic nervous system, leading to a catecholamine flare state [8]. Peripheral vasoconstriction leads to a raised blood pressure level of more than 20% above the baseline to keep the mean arterial blood pressure above intracranial pressure to overcome the pressure gradient and maintain adequate brain perfusion [9]. Activation of the sympathetic nervous system and increased systemic blood pressure stimulate Baroreceptors in the carotid bodies. This leads to a drastic slowing in heart rate till bradycardia [10, 11]. Irregular breathing patterns are related to reduced brainstem perfusion, where the body's autonomic functions are controlled and homeostasis is maintained [12]. Cushing’s Reflex necessitates a rapid evaluation with a CT brain following a discussion with the neurosurgical team for acute intervention with decompressive craniotomy [13]. The patient will require monitoring in an intensive care unit and continuous intracranial pressure monitoring to guide them to proper action [14, 15]. Although Cushing’s Reflex is a defense mechanism to save brain tissue, it is a late sign of increased intracranial pressure. Following the present case, the patient’s complete clinical presentation indicates the
possible cause for bradycardia is a Cushing reflex related to intracranial pressure.

Case presentation
A 67-year-old Bahraini male known case of Hypertension, Diabetes, Chronic Kidney Disease, and a history of a stroke three years ago was admitted with dry gangrene in the second toe of the right foot. On 14 January 2020, the patient underwent amputation of the right second metatarsal under local anaesthe sia, proceeded by a CT angiography. On 18 January 2020, the patient developed a sudden drop in Glasgow coma score to 13/15 and an abnormal irregular breathing pattern. The heart rate dropped 30-40/min from a previous 80-90/min range. ECG showed bradycardia with a heart rate of 37/min with prolonged QTc 576 msec. However, the blood pressure was maintained at 115/59-120/60. Venous blood gases were reported as pH 7.32, Pco2 35.4 mmHg, Po2 24.2 mmHg, HCO3 18 mmol, glucose 13.5 mmol, lactate 0.88 mmol/l. One dose of intravenous (IV) Atropine 1 mg was administered. However, with no improvement, so dopamine infusion was commenced. On admission, an echocardiogram (ECG) showed a normal left ventricular cavity, poor global systolic function, and an estimated ejection fraction of 20-25%. Pulmonary artery pressure was 26 mmHg. Cardiac enzymes revealed a mild elevation in troponin at 0.12 ng/ml. CKMB and CK were normal. The change in the mental status was thought to be attributed initially to bradycardia. However, the Cushing reflex was taken into consideration. CT’s brain reported a hyper-dense shadow at the left temporal region with a surrounding area of hypodensity, dilatation of the temporal horn of the left lateral ventricle, and mild dilatation of the supra-tentorial ventricular system. The study concluded a possible early hemorrhagic insult on top of chronic infarction. The patient got a rapid drop in glass to grow the coma scale to 4/15 and was arrested.

Discussion
Due to the initial absence of hypertension, Cushing’s Triad, the initial impression of this patient’s bradycardia was thought to be related to acute kidney injury and hyperkalemia. The expected potassium-inducing cardiac changes in the atrium range between 5.5 to 6 mmol/L, but this patient’s potassium level did not exceed 5.2 mmol/L. The initial features of hyperkalemia on ECG usually involve narrowing the T wave, flattening the P wave, and widening the QRS complex. This was not evidenced on the patient’s ECG. In addition, the patient was not on antihypertensive or rate-controlling medications that could have caused bradycardia. Given sepsis, the pH level from the patient’s venous blood gas results does not indicate a level of acidosis critical enough to affect the heart rate. The patient’s bradycardia did not recover following two doses of atropine, as the heart rate was 45 bpm. In

Figure 1. Abnormal Echocardiogram of the patient.
addition, the patient's cardiac enzymes and echocardiogram confirmed a reduced ejection fraction. As the patient had dementia, mental status did not help much in the assessment. The patient's complete clinical picture indicates that the possible cause for bradycardia is a reflex related to intracranial pressure. The findings confirmed that the heart rate is not improving to atropine, the change in patient interaction level, abnormal breathing pattern, and confirmation of the hemorrhagic insult on the CT brain. The presence of normal blood pressure and later hypotension caused this sign to be neglected in the evaluation, and the patient's condition worsened. The question is: would this patient have benefited from an acute intervention such as a decompressive craniotomy? It has been reported that early neurosurgical intervention would improve severe traumatic brain injury outcomes in younger patients with higher GCS scores[16, 17].

The fact is that Cushing's triad is a late response to an increase in intracranial pressure on the medullary centres. Intracranial pressure can be monitored through intraventricular, intraparenchymal, subdural, and epidural catheters[18]. However, the decision to do the monitoring is based on the underlying progress. Neurosurgical guidelines recommend ICP monitoring for patients with post-traumatic brain injury presenting with GCS of 3-8, abnormal CT brain, and at least one of the following criteria: age more than 40 years, systolic blood pressure less than 90, or abnormal posturing[14]. Besides this, the indication for ICP monitoring is not well-established in non-traumatic cases[18]. Generally, patients with rapid neurological deterioration from focal space-occupying lesions may benefit from surgical decompression and diffuse brain swelling[19]. It has been reported that patients with the impaired left ventricular function will consequently have lower blood pressure despite catecholamine release. Other reasons include severe sepsis and subclinical adrenal insufficiency[20].

It can be concluded that Cushing's reflex is a late mechanism that led the patient brain to accommodate the increase in intracranial pressure[6]. It may be a time for life-saving interventions. Hypertension is a common manifestation of Cushing's reflex and purpose to maintain cerebral perfusion[21]. However, hypotension is considered a rare and detrimental manifestation which can make Cushing Reflex less to be considered[22]. Normal blood pressure should not rule out the possibility of Cushing's reflex, especially in extreme age, severe atherosclerotic disease, cardiomyopathy, reduced ejection fraction, and cardiac output[23]. Bradycardia can cause altered mental status due to reduced cardiac output[24]. This can be treated as per ACLS guidelines, and focusing on bradycardia alone may lead to missed brain insult [25, 26].

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Availability of data and materials
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Conflict of Interest
The author declares that there is no conflict of interest.

Consent for Publication
After reading the clinical information in this report, the patient provided their informed permission. The author attests that the patient was fully informed about all elements of the study, including its goals, methods, and therapies, without incurring any additional costs or fees. In addition, the research complied with all necessary institutional and international standards.

References
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