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Cushing Reflex and Importance during Treatment of Cerebrovascular Diseases via Endovascular Route

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Abstract

Aim: Assessment of the Cushing reflex developing during mechanical thrombectomy in a case presenting with acute ischemic stroke.

Case: A 59-year-old male patient had CT angio tests due to speaking disorder and right lateral weakness developing 5 hours earlier. Acute infarctus was observed in the left MCA irrigation area with M1 segment occlusion observed in the left MCA. The decision was made to treat the patient with mechanical thrombectomy and he was transferred to the interventional neuroradiology unit. Under general anesthesia during navigation of the thromboaspiration catheter to the clot localization as the procedure was technically advancing routinely, with sudden development of bradycardia and hypertension the neuroradiology team was warned, and 1 mg IV atropine was administered for bradycardia.

Simultaneously contrast material injection through the guide catheter showed the MCA M1 segment had ruptured and extravasation had developed.

Conclusion: The Cushing reflex is a situation characterized by bradycardia and hypertension in cerebrovascular events causing sudden intracranial pressure increases. The most common situations observed during treatment of cerebrovascular diseases via the endovascular route is intracranial hemorrhage linked to arterial rupture. Prevention of the mortality and morbidity linked to hemorrhagic complications developing during endovascular treatment is based on early awareness and rapid treatment. Additionally, as extravasation is commonly not observed radiologically, the anesthesia team should be on the alert for the Cushing reflex and when variations of hemodynamic parameters in accordance with the Cushing response are observed, informing the operation team immediately has vital importance.

Keywords: Cushing reflex; Cerebrovascular diseases; Endovascular route

Introduction

Due to technologies developing in the last two decades, endovascular methods have gained importance for the treatment of many cerebrovascular diseases. Generally, complications encountered in endovascular treatments may be classified as hemorrhagic and thromboembolic complications. Of these, the hemorrhagic complications have higher mortality and morbidity. Additionally, extravasation during the procedure may not always be identified angiographically, leading to late awareness of hemorrhagic complications and as a result negative effects on survival. In situations where the intracranial pressure (ICP) suddenly increases, hypertension, bradycardia and respiratory irregularity may be observed. Classically, the 'cushing reflex has been reported as the occurrence of hypertension, bradycardia and apnoea following intracranial hypertension [1]. Called the Cushing reflex after the researcher who first observed them, acknowledging these findings and informing the operation team positively changes the progress of hemorrhagic complications encountered during neuroendovascular treatments. Since the introduction of neuroendoscopy in the treatment of cerebral pathology, the problem of early recognition of any sudden increase in intracranial pressure has become crucial as introduction of via the endovascular route in the treatment of cerebral pathology [2]. In this case report, we aim to discuss a case with arterial rupture during mechanical thrombectomy for acute stroke which was not noticed during the procedure. However, after the Cushing reflex was identified by the anesthesia team, the arterial rupture was confirmed angiographically, necessary treatments were applied, and the case survived.

Case Report

A 59-year-old male case applying to the emergency service with aphasia and right-side weakness was determined to have by-pass, aorta valve replacement and to use coumadine on assessment. The patient had blood pressure 165/95 mmHg, pulse 120 beats/min, fever 36.2°C, and respiratory count 18/min.

Neurological examination of the patient found open consciousness, with no cooperation, no response to orders, no verbal output and aphasia. There were no findings of nuchal stiffness and meningeal irritation. Pupils were isochoric and had bilateral light response. Global movements could not be evaluated. There was no nystagmus. Right NLS was faint, uvula and gag reflex could not be assessed. Paresia tests found right upper and lower extremities could not overcome gravity, with muscle power of the right upper and lower 2/5, tonus was normal, and

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senses could not be evaluated. The National Institute of Health stroke scale (NIHSS) was calculated as 16.

Laboratory investigations were AST: 44 U/L(0-35), ALT: 164(0-45) U/L, Glucose 148 (60-100)mg dL-1, creatinine 1.49(0,5-1,4) mg/dL, Urea 35(5,0-21) mg/dL. In venous blood gases pH: 7.40, PO2: 41.6, mmHg, PCO2: 44.9, mmHg, SPO2; 65. Lactate was 2.13 mmol/L. On coagulometry: İNR ;3.34 s N (0.8 – 1.2 s), PtZ; 37.2 s N(10-15))s, aPTT 44s N(23.2- 35.2 sn).

On non-contrast computed cerebral tomography (CT) acute intracranial hemorrhage was not identified (Figures 1A-1C). CT-angiography showed M1 segment occlusion in left MCA. As the INR value was high, intravenous thrombolysis treatment was contraindicated so the decision was made to treat the case with mechanical thrombectomy after consultation with the neuroradiology team.

The patient was taken to anesthesia with ASA Emergency. When the patient was placed on the operating table for intervention venal route was already inserted. After standard monitorization (heart rate (HR), blood pressure (BP), oxygen saturation (SpO_2)) 0.9% saline infusion was begun through the venous access. Before induction blood pressure was 180/100 mmHg, heart rate was 124 beats/min and SpO_2 was 95 and the patient was given 3 mg/ml dose of IV midazolam (Dormicum® Roche-Germany). After administering 2 μgr/kg fentanyl citrate (Fentanyl, Braun, Germany), 1 mg/kg propofol (Propofol® Fresenius container-Germany) and 0.8 mg/kg rocuronium bromide (curon 50 mg/5 ml iv flk), the patient had orotracheal intubation completed with a no. 7.5 endotracheal tube. With the anesthesia device on volume-control mode (Drager RA 2S-Germany), ventilation was maintained with tidal volume of 6 mg/kg.

Anesthesia maintenance used 50% air+50% oxygen and MAC 2 concentration of sevoflurane (Sevoflurane, Abbott, USA) and 0.25-0.5 mcg/kg/min infusion of remifentanil (Ultiva, Glaxo Smith Kline-Italy). During the procedure HR was 80 and mean arterial pressure was set at 70 mmHg. On DSA, left ICA injection showed left MCA M1 segment occlusion (Figure 2). During the routine progress of the procedure with the sudden development of bradycardia (HR 20 beats/min) and hypertension, 1 mg IV atropine was administered, and the neuroradiology team was warned. Contrast material injections demonstrated extravasation due to rupture caused by the navigation of thromboaspiration catheter in the thrombotic segment (Figure 3). At this point revascularization of the thrombosis segment was cancelled and the procedure was ended. With atropine HR rose above 60. After the operation lasting nearly 60 minutes, the intubated patient was sent to neurology intensive care. Follow-up CT imaging demonstrated hemorrhage and contrast material in Sylvian fissure (Figures 4A-4C).

Discussion

Heart rate provides important information about the central nervous system. Tachycardia shows sympathetic discharge. Bradycardia accompanied by high blood pressure may show increased intracranial pressure (ICP) (Cushing reflex). During neuroendoscopic procedures, early recognition of an excessive increase in intracranial pressure, jeopardizing brain perfusion, is of major importance for preserving cerebral homeostasis. The cushing reflex can be seen after profound ischemia in the posterior cerebral circulation with preserved circulation to the forebarain. Ischaemia of the brain stem alone may be sufficient to elicit the Cushing reflex [2-4]. Increased intracranial pressure may be affected by cerebral blood and CSF circulation and many central nervous system problems. It is a common life-threatening complication of many neurologic and non-neurologic diseases. It is related to secondary brain damage. If it is undiagnosed and untreated, it may cause mortal results like brain ischemia and brain stem herniation [5]. In brain damage, increased ICP is an acute situation, reducing blood perfusion and distribution of oxygen in the brain, causing ischemia and may progress to brain tamponade. Recent studies have shown that early diagnosis and treatment of intracranial hypertension in brain-damaged patients

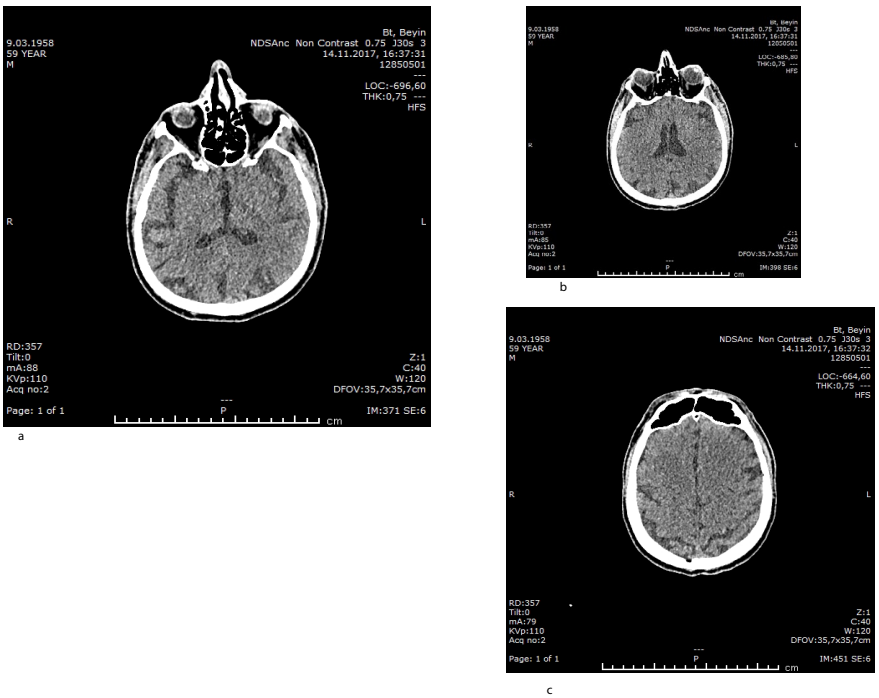

Figure 1: Acute intracranial hemorrhage was not identified.

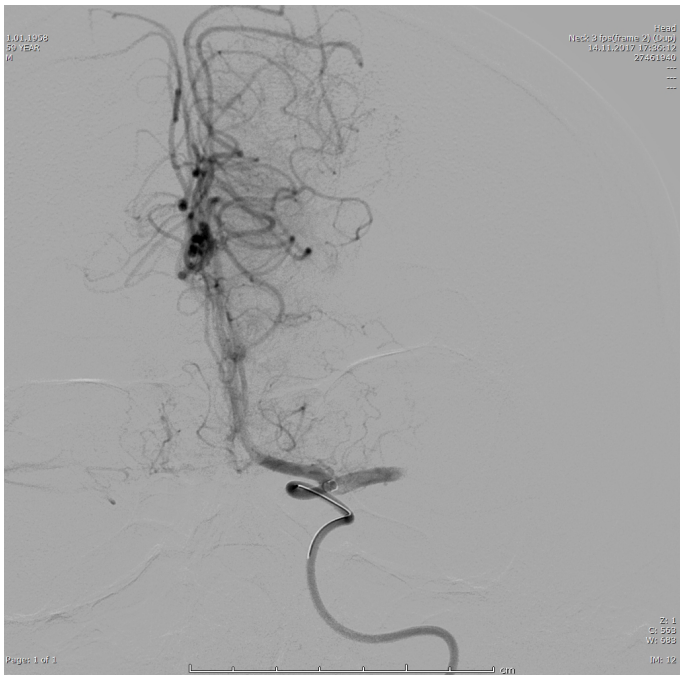

Figure 2: On DSA, left ICA injection showed left MCA M1 segment occlusion.

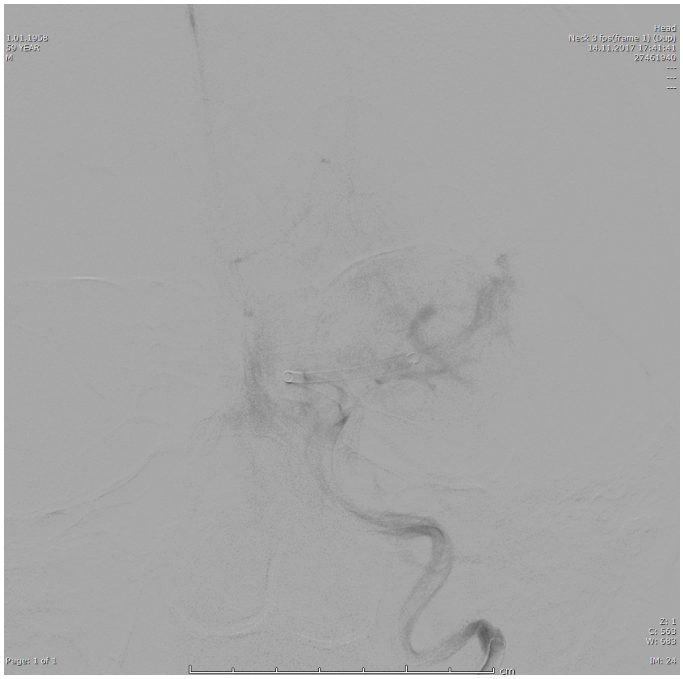

Figure 3: Follow-up CT imaging demonstrated hemorrhage and contrast material in Sylvian fissure.

is related to better survival [6]. Brain edema begins to compress the blood vessels supplying the brain. This compression results in reduced blood flow to the brain and ultimately brain ischemia. The ischemia will then cause the arteries leading to the brain to dilate, causing an additional increase in capillary pressure and a further increase in intracranial pressure. The increased arterial blood pressure caused by the CNS ischemic response stimulates the baroreceptors in the carotid bodies, thus slowing the heart rate drastically often to the point of a bradycardia [7]. The normal range for ICP varies with age. Values for pediatric subjects are not as well established. Normal values are less than 10 to 15 mm Hg for adults and older children, 3 to 7 mm Hg for young children, and 1.5 to 6 mm Hg for term infants. ICP values greater than 20 to 25 mm Hg require treatment in most circumstances. Sustained ICP values of greater than 40 mm Hg indicate severe, life-threatening intracranial hypertension. A decrease in the cerebral perfusion pressure to 15-30 mm Hg often results in a Cushing reflex, but frequently causes hypertension without tachycardia or bradycardia [2].

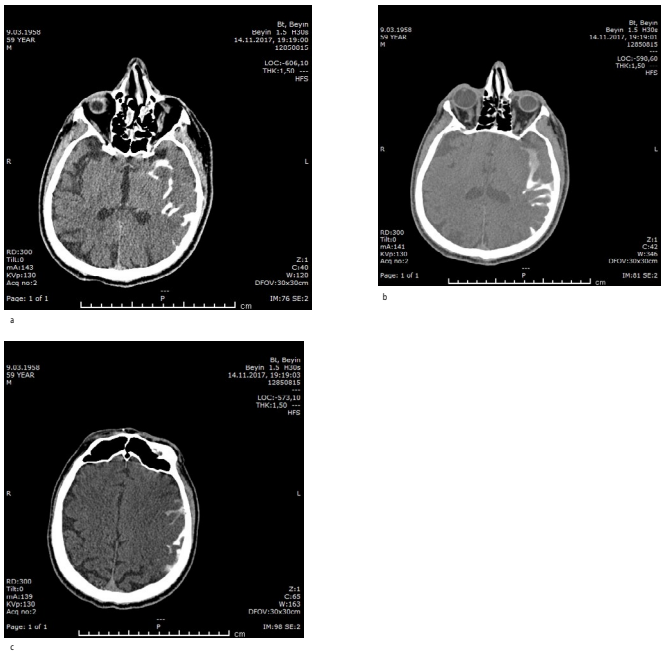

Figure 4: CT imaging demonstrated hemorrhage and contrast material in Sylvian fissure.

In our case, anesthesia maintenance was continuing in a manner that did not disrupt the hemodynamics of the patient under general anesthesia. As the procedure was technically progressing routinely during navigation of the thromboaspiration catheter to the clot localization, with the development of sudden bradycardia and hypertension, the interventional neuroradiology team was warned, and 1 mg IV atropine was administered for bradycardia. Simultaneously, contrast material injection through the guide catheter showed rupture of the MCA M1 segment and it was understood extravasation had developed.

Conclusion

The Cushing reflex is a situation characterized by bradycardia and hypertension in cerebrovascular events causing sudden intracranial pressure increases. The most common situations observed during treatment of cerebrovascular diseases via the endovascular route is intracranial hemorrhage linked to arterial rupture. Prevention of the mortality and morbidity linked to hemorrhagic complications developing during endovascular treatment is based on early awareness and rapid treatment. Additionally, as extravasation is commonly not observed radiologically, the anesthesia team should be on the alert for the Cushing reflex and when variations of hemodynamic parameters in accordance with the Cushing response are observed, informing the operation team immediately has vital importance.

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