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Case Report OMICS International

Successful Management of Hypotension Induced by Low Intracranial Pressure with Intravenous Administration of Lidocaine

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Abstract

A man suffered severe hypotension down to 20~30 mmHg when he received a surgical remove of glioma. This hypotension was hard to treat with many ways due to the blood pressure did not show any response to many vasoconstrictors, such as: adrenaline, norepinephrine, dopamine, and Aramine etc. A rapid injection of lidocaine 200 mg from right femoral vessel showed an efficient effect on promoting blood pressure when combined with other vasoconstrictors.

Keywords: Hypotension; Severe; Lidocaine; Intravenous; Low intracranial pressure

Introduction

Hypotention induced by leakage of cerebrospinal fluid was reported occasionally [1]. Small dose of leakage may be the potential cause of intracranial hypotension, and some treatments worked as patch on epidural were also reported [2-4]. Normally, the leakage of CBF was small and slow, and severe degrees of intracranial hypotension were related with the speed or the amount with CBF leakage [4]. A large amount of CBF leakage in a short time could be very dangerous to the patient, and it is hard to treat [5]. Here we reported our successful treatment about a severe hypotention induced by low intracranial pressure with lidocaine during the surgery.

Case Report

A 43-year-old male patient received surgical remove of glioma at the parietal lobe underwent the general anesthesia after being diagnosed. Preoperative examination revealed that the functions of the heart, lung and kidneys were normal. The patient was monitored by ECG, IBP, SpO2, $P_{et}Co2$; prior to the operation, continuous lumbar punctures were carried out, and an indwelling tube was secured in place to drain the cerebrospinal fluid, the purpose of which is to reduce the intracranial pressure for better exposure of the surgical field; after anesthetic induction, artificial airway was established, anesthesia was maintained with intravenous continuous infusion of Propofol, Remifentanil, and muscle relaxation with Atracuriun. The operation was begun half an hour later, and after the craniotomy and the opening of the dural mater, the blood pressure of the patient was found to drop drastically from 123/68 mmHg to 54/32 mmHg about 3 min after the skin incision was finished, and drop continuously later; the author ruled out the mechanic malfunction and enormous blood loss, and determined that it may be caused by the drainage of cerebrospinal fluid up to 140 mL. Intravenous administration of Dopamine at the dosage of 6 mg, 20 mg, and 20 mg again cannot reverse the drop of the blood pressure, neither did the intervention with combined use of Aramine (Metaraminol), Adrenaline, Norepinephrine and Dexamethasone. One hour after the drop of the blood pressure, the fluid we infused into the patient include 2500 mL colloid, 2000 mL crystalloid, and 4 U red blood cells, in addition to 80 mL normal saline injected into the subarachnoid space. Despite all these efforts, the blood pressure of the patient was barely keep at 20~23/10~12 mmHg, and heart rate hovering around 120 bpm, with oxygen saturation status unable to detect. The cardiovascular expert was invited into the operating room for consultation, whose advice were tried but still with futile efforts to bring up the blood pressure. In consideration of the urgent situation, the author suggested the remedy of intravenous infusion of large dose of Lidocaine, after obtaining the approval of the ethic committee of the hospital, 200 mg of Lidocaine was rapidly injected through the femoral vessel; in the initial 2 min after the injection, no obvious change of blood pressure was detected; 3 min after the injection, the monitoring screen demonstrated the slow rise of the blood pressure, returning from 22/12 mmHg to 35/18 mmHg, 104/65 mmHg, 146/75 mmHg at 5 min, 10 min and 15 min, respectively. Then, a norepinephrine was i.v. injected continually at 1.6 mg/h to maintain the blood pressure of the patient at 130/70 mmHg and escort him back to the intensive care unit for further treatments and observations. Unfortunately, this patient was diagnosis as (Multiple Organ Failure) MODS in Intensive care unit (ICU) at the next day: Acute Renal Failure (ARF), with Blood Urea Nitrogen (BUN) up to 51.4 mmol/L and Creatinine up to 632 μmol/L. Acute Respiratory Distress Syndrome (ARDS), with PaCO2 up to 96 mmHg, PaO2 down to 36 mmHg, and died 4 days later.

Discussion

Lidocaine is the commonly used local anesthetics, which is often adopted in local anesthesia [6], intravenous regional anesthesia [7], and epidural anesthesia [8,9]; meanwhile, it is also used as an antiarrhythmic drug to treat ventricular arrhythmia [10]. Due to its excellent efficacy of surface anesthesia, it has been reported to be used to attenuate the injection pain caused by Propofol and Etomidate with good results [11,12]; it also has been used to prevent the endotracheal suctioning-induced cardiovascular reactions through endotracheal instillation [13]; other reports suggested that Lidocaine by intrathecal

injection can relieve the vasoparesis caused by acute neurogenic hypotension and the occurrence of myocardial dysfunction [14]. Sharp drop of blood pressure often occurs in acute brain trauma managed by craniotomic decompression, especially when the dural mater is breached, possibly related to Cushing reaction [15]. In our reported case, after the opening of the dural mater and drainage of large amounts of cerebrospinal fluid, the intracranial pressure dropped sharply, followed by reactive drastic decline of blood pressure, which cannot be addressed by fluid infusion of large volume and injection of various vasoactive drugs, possibly attributable to sympathetic paralysis. When we took consideration of that Lidocaine can attenuate the vasoparesis induced by neurogenic hypotension and that it is one of the local anesthetics which can be used intravenously and is with effect of surface anesthesia, we gave the patient rapid injection of 200 mg of lidocaine, achieving obvious effects and winning a second operative chance for the patient. In review of what happened in our case and what has been reported in literature, we hypothesize that the phenomenon in our case is related to the surface anesthesia by lidocaine: after rapid injection of lidocaine, anesthesia of the internal vascular wall can reduce the intensity or totally block Cushing reaction, bring back the blood pressure. However, since it is still less known about target sites in the reflex arc of Cushing reaction, the exact mechanism of the effect of intravenous injection of lidocaine on Cushing reaction still waits for further study.

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