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CATECHOLAMINE TOXICITY AFTER CRANIOTOMY EVACUATION CRANIOTOMY AND EVACUATION OF THE ABSCESS

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ABSTRACT

Background: Increasing levels of endogenous catecholamines occur acutely to provide short-time adaptation to stressful conditions, known as the fight-or-fly response. Catecholamine toxicity requires multidisciplinary management

Method: This was a retrospective case report, after analysis of patient clinical data. The patient provided written informed consent to publish their case details and any accompanying images. The study protocol complies with the requirements of the institute's committee of Haji Adam Malik Hospital, Medan, Indonesia.

Results: In this case, the patient is diagnosed with a brain abscess since birth. According of the history, physical examination and investigations, it was concluded that the diagnosis of epidural abscess with abscess evacuation craniotomy and PS ASA 2 (leukocytosis) with GA-ETT anesthesia. The operation is carried out with a duration of 4 hours. Vital sign monitoring obtained blood pressure sp108 – 125 62 - 90 mmHg, heart frequency 90 - 120 times per minute, 99% oxygen saturation. When in the recovery room, the patient experiences cardiac arrest, this is thought to result from catecholamine toxicity.

Conclusion: Patients are treated as resuscitation in accordance with the algorithm of cardiac arrest in children. Patients experienced a response of spontaneous circulation (ROSC) and performed vital sign monitoring.

Keywords

Catecholamine Toxicity, Massive Abscess Evacuation, Cardiac Arrest

INTRODUCTION

Increased levels of endogenous catecholamines occur acutely to provide short-term adaptation to stressful conditions, known as the fight-or-fly. Likewise, administration of exogenous catecholamines is a lifesaver in clinical situations associated with a decrease in cardiac output and / or hypotension. In contrast to these short-term benefits, continuous increase in catecholamine harms the cardiovascular system by initiating significant cardiotoxicity, as observed in chronic heart failure, pheochromocytoma, stress-triggered cardiomyopathy ("takotsubo") and during prolonged therapy with high-dose exogenous catecholamines . According to this latest condition, the administration of isoproterenol synthetic b-adrenoceptor agonists to laboratory animals has been used for decades as a model of known heart injury (1).

Catecholamine poisoning requires cross-discipline in the complex management of catecholamines and clearly highlights the value of invasive monitoring to guide appropriate fluid

resuscitation and alpha blockade in this rare but challenging group of patients. A review of recent literature conducted by Whitelaw et al. examined alpha-blockade use for the treatment of catecholamine crises and found that mortality was significantly higher in those who did not receive treatment with alpha-blockade vs. those performed (99% vs. 40%). Of course, the use of alpha-blockade may have been contraindicated in some cases which were reviewed because hypotension coincided with cardiogenic shock and / or intravascular depletion. As described in the case we describe, careful monitoring of fluid status can guide appropriate fluid resuscitation and allow careful introduction of alpha blockade in high dependency units. Researchers recommend that if possible, patients should be monitored in units with expertise and access to mechanical circulation support if needed. The use of intra-arterial balloon counter-pulsation or extra-physical life support has shown efficacy in cases of refractory catecholamine crisis associated with hypotension and can act as a connecting therapy to prevent a catecholamine crisis subsiding or provide the circulation support needed to introduce alpha-blockade (2).

The same researchers have also shown that initial test doses of short-acting alpha-blockers, such as phentolamine, can provide valuable information about cardiovascular reserves in these patients and careful fluid resuscitation may be sufficient to allow optimal alpha blockade (2).

METHOD

This was a retrospective case report, after analysis of patient clinical data. The patient provided written informed consent to publish their case details and any accompanying images. The study protocol complies with the requirements of the institute's committee of Haji Adam Malik Hospital, Medan, Indonesia.

CASE DESCRIPTION

A 9-month-old male patient with a body weight of 6.1 kg entered the emergency room with a reduction in awareness by diagnosing brain abscesses. Patients are diagnosed with a patient's brain abscess from birth. A history of fainting was denied, a history of seizures was denied. A history of fever is denied. History of trauma is denied. Patients experience difficulties during labor. Obstetric history, the patient is the first child, when the patient is experiencing cyanosis, apnea and cardiac arrest.

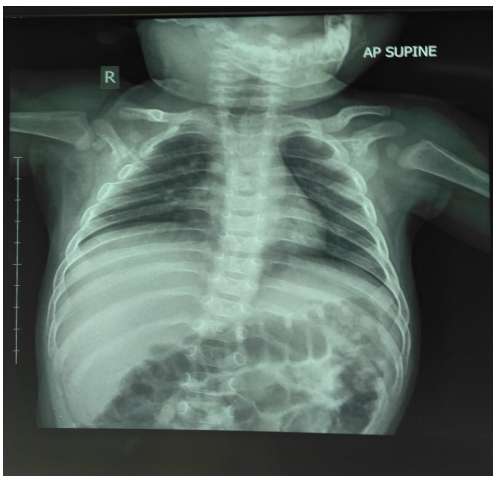

Figure 1. Chest X-ray

From the physical examination in the room on 7/25/2018 Airway clear was found, no snoring, gurgling, and crowing were found, with a frequency of breathing 20 times per minute, vesicular breath

sounds, no additional breath sounds and 99% oxygen saturation. Circulation is found Akral warm, red, dry, with heart frequencies 114 times per minute, regular, with strong pulse pressure and sufficient volume. The patient's consciousness was compos mentis, GCS 15, with isocorous pupils Ø 3 mm right and left and positive light reflexes. Positive urine output. Abdomen soepel and peristaltic are positive normal. No edema and fractures of the extremities were found.

Laboratory tests on 24-07-2018 obtained hemoglobin (13.4 gr / dl), hematocrit (42%), leukocytes (13,910 gr / dl), platelets (393,000 / ul), sodium (137 mmol / L), potassium (4.3 mmol / L), chloride (106 mmol / L), blood glucose level (114 mg / dl), urea (40 mg / dl), creatinine (0.27 mg / dl). Radiological examination revealed chest radiographs within normal limits. Radiological examination obtained a CT scan of the head with a brain abscess in the right hemisphere..

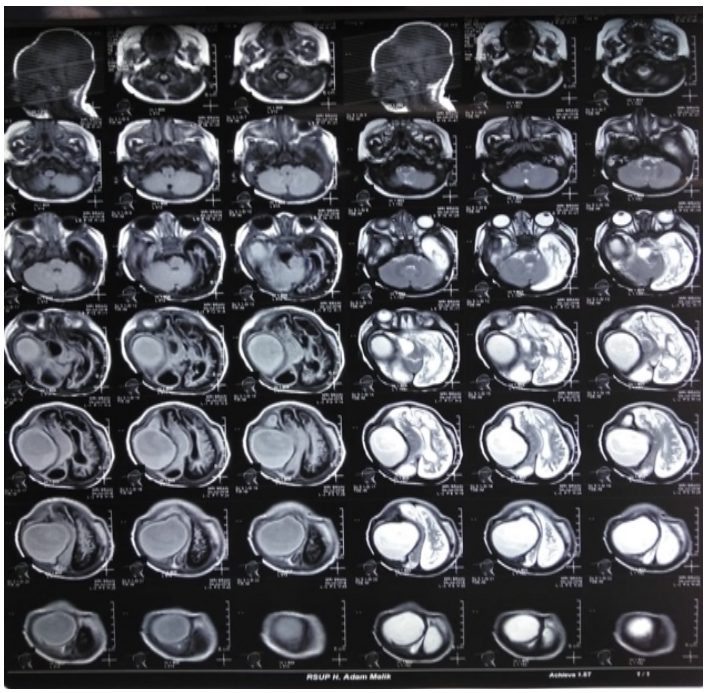

Figure 2. X-ray head CT scan

From the history, physical examination and investigations it was concluded that the diagnosis of epidural abscess with abscess evacuation craniotomy and PS ASA 2 (leukocytosis) with GA-ETT anesthesia. Patients were preoperative 24 hours before the operation was carried out by giving education to patients, making anesthesia permits and filling out informed consent, patients were fasted 6-8 hours before surgery, installation of IV Line 24 G and Ensuring Smoothness, injection of Cefazolin 200 mg 1 hour before surgery, preparation blood 1 bag 25cc, head hair shaving, oral and personal hygiene, and patients are recommended to pray. Anesthetic technique, carried out active suction, Pre-oxygenation O2 8 liters per minute in 3-5 minutes. Injection of midazolam 2 mg (intravenously), injection of Fentanyl 60 mcg (intravenously), Sellick maneuver and Induction Propofol 40 mg (intravenously) with evaluation of negative eyelids, Injection of Rocuronium 20 mg / (intravenous) with evaluation until positive sleep apnea. Maintenance is given Water: O2 = 2 liters: 2 liters, Isofluran 0.8 - 1%, Maintenance Rocuronium 5 mg / 30 minutes, Maintenance Analgetic fentanyl 20 mcg according to hemodynamics.

The operation is carried out with a duration of 4 hours. Vital sign monitoring obtained blood pressure 108 - 125/62 - 90 mmHg, heart frequency 90 - 120 times per minute, 99% oxygen saturation. Balance fluid during preoperation with ringer lactate fluid (500 ml), operating durante ringer lactate (1500 ml), bleeding + 300 ml, evaporation plus maintenance: (2 + 8) x 50 kg = 500 ml / hour. Urine

output 50 ml / hour. Postoperative therapy is done bed rest, head up 30º, given O2 3 liters per minute via nasal canul, TPN diet, IVFD RL 30 drops per minute, injection of ceftriaxone 1 gr / 12 hours (intravenously), injection of fentanyl 200 mcg dissolved in 50 ml 0.9% NaCl was given 6 ml / hour, injection of ranitidine 50 mg / 12 hours (intravenously).

DISCUSSION

Catecholamines, released from the adrenal medulla and from the central nervous system and sympathetic, function as hormones and neurotransmitters play an important role in the cardiovascular system. Structurally, catecholamines include dihydroxybenzene (katekol) rings and nitrogen (amine) groups. They are produced from aminoacid L-tyrosine through hydroxylation, decarboxylation and successive methylation steps which result in sequential formation of dihydroxyphenylalanine (L-Dopa), dopamine, norepinephrine (NE, noradrenaline) and epinephrine (Epi, adrenaline) (1).

Catecholamines given in high concentrations produce myocardial damage in some mammalian species. Histological changes are similar to those found in patients given large amounts of pressor agents and in those who develop pheochromocytomas. They include myofiber necrosis, myofibrillar degeneration, and mononuclear leukocyte infiltration. Heart function is significantly impaired. Endogenous release of catecholamine can also cause myocardial injury in rabbit patients infused with tyramine. Anatomical and functional abnormalities described in various models of catecholamine cardiomyopathy are summarized. Some of the main theories about pathogenesis are reviewed. The latest data shows that the generation of free radicals originating from 02 is involved discussed (1).

Increased levels of endogenous catecholamines occur acutely to provide short-term adaptation to stressful conditions, known as the fight-or-fly. Likewise, administration of exogenous catecholamines is a lifesaver in clinical situations associated with a decrease in cardiac output and / or hypotension. In contrast to these short-term benefits, continuous increase in catecholamine harms the cardiovascular system by initiating significant cardiotoxicity, as observed in chronic heart failure, pheochromocytoma, stress-triggered cardiomyopathy (" takotsubo ") and during prolonged therapy with high-dose exogenous catecholamines . According to this latest condition, the administration of isoproterenol synthetic b-adrenoceptor agonists to laboratory animals has been used for decades as a model of known heart injury (3).

The pathophysiological events triggered by an increase in [Ca2 +] M are significantly strengthened by oxidative stress which develops on sustained exposure to high levels of catecholamines, due to the mechanism identified below. First, MAO-dependent oxidative deformation of catecholamines forms hydrogen peroxide (H2O2), which can be converted into highly reactive hydroxyl radicals (OH-) through metal catalysis (Fenton chemistry). Second, activation of A1-adrenoceptors by catecholamine induces activation of NADPH oxidase, with the next generation of superoxide (O2-) anion radicals in cardiac myocytes. Third, and most importantly, catecholamines are easily oxidized to toxic compounds called " aminochromes " (because of their colored appearance in solution). This process occurs spontaneously at a low level (autooksidation), but this is markedly accelerated in the presence of oxidants and free radicals such as O2-, redox metals (especially iron and copper) and by enzymatic catalysis (mainly by xanthine oxidase, myeloperoxidase and cytochrome oxidase) (2).

In short, sustained high level catecholamines can cause major toxic effects on the myocardium, which cause morphological changes similar to those produced by myocardial infarction, including cell death especially cardiomyocytes and progressive myocardial focal fibrosis. The toxicity comes from a variety of catecholamine actions in the heart, the most significant being excess calcium, oxidative stress and mitochondrial dysfunction (1,4).

Clinical manifestations caused in the form of paroxysimal hypertension. For example in patients with pheochromocytoma, episodes of hypertension are usually sudden onset and often accompanied by physical symptoms, such as headache, dizziness, nausea, diaphoresis, chest pain, and palpitations. The frequency of these episodes ranges from every day to less than once per month, and the duration of each episode can range from minutes to days. This disorder is more common in women. A potential explanation for this can be increased activation of specific cardiac sympathetic nervous systems in women compared to men (5).

There is no specific therapeutic approach for catomolamine-induced cardiomipathy until now. Management of catecholamine-induced cardiomyopathy should include a reduction in sympathetic activation by using α-and β-adrenergic receptor inhibitors and administration of diuretics in case of excessive volume. Emergency management of arrhythmias, pulmonary edema, and cardiogenic shock based on conventional symptomatic treatment, such as antiarrhythmics and inotropics, vasoactive electric shock and / or mechanical circulation support (1).

CONCLUSION

Increased levels of endogenous catecholamines occur acutely to provide short-term adaptation to stressful conditions, known as the fight-or-fly. There is no specific therapeutic approach for catomolamine-induced cardiomipathy until now. Management of catecholamine-induced cardiomyopathy should include a reduction in sympathetic activation by using α-and β-adrenergic receptor inhibitors and administration of diuretics in case of excessive volume. Emergency management of arrhythmias, pulmonary edema, and cardiogenic shock are based on conventional symptomatic treatment, such as antiarrhythmia and inotropics, vasoactive electric shock and / or mechanical circulation support. In this case, massive abscess evacuation in patients can cause catecholamine toxicity.

ABBREVIATIONS

H_2O_2, Hydrogen peroxide; AKI, Acute Kidney Injury; ROSC, response of spontaneous circulation.

ETHICS APPROVAL AND INFORMED CONSENT

The study protocol complies with the requirements of the institute's committee of Haji Adam Malik Hospital, Medan, Indonesia.

CONSENT FOR PUBLICATION

The Authors agree to publication in Journal of Society Medicine.

DATA AVAILABILITY

The datasets generated during and/or analyzed during the current study are available from the corresponding author upon reasonable request.

FUNDING

None.

COMPETING INTERESTS

None.

AUTHORS' CONTRIBUTIONS

All authors significantly contribute to the work reported, whether in the conception, study design, execution, acquisition of data, analysis, and interpretation, or in all these areas. Contribute to drafting, revising, or critically reviewing the article. Approved the final version to be published, agreed on the journal to be submitted, and agreed to be accountable for all aspects of the work.

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