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Hyperventilation Syndrome After Strabismus Surgery - A Case Report

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Abstract: Hyperventilation syndrome (HVS) mainly occurs in patients under anxiety and stress and is marked by rapid, excessive breathing, resulting in respiratory alkalosis and subsequent hypocalcemia and hypokalemia, causing various physical symptoms, like numbness or tingling sensation, overactive reflexes, muscle cramps, twitches, and in severe cases, acute myocardial infarction or even death, requiring immediate diagnosis and medical intervention. We report a rare case of HVS after strabismus surgery under local anesthesia. The patient is a healthy 31-year-old woman with no psychiatric or other diseases in the past medical history. At the end of the procedure, as soon as the surgeon uncovered the sheet, the patient complained about numbness in the lower limbs, spasm in the upper limbs, muscle rigidity, and double vision. At that time, the electrocardiogram monitoring showed normal blood pressure, heart rate, pulse oxygen saturation, and breathing rate. However, the patient was awake and extremely nervous. Emergency arterial blood gas analysis (ABG) was performed immediately and revealed respiratory alkalosis with significantly reduced $PaCO_2$ and secondary hypocalcemia and hypokalemia. Verbal reassurance, closed-mask inhalation, and electrolyte supplement were administered. HVS gradually subsided. In this context, a prompt differential diagnosis and a high degree of suspicion are essential. Monitoring end-tidal carbon dioxide may also aid in the early detection of HVS, as changes in breathing rate and pattern may be observed before definite signs and symptoms manifest in patients with HVS. However, since surgical drapes and trays cover the torso, detecting any changes in breathing rate and pattern during the surgery may be challenging. This article describes a case of HVS after strabismus surgery under local anesthesia and provides a summary of the occurrence, manifestation, diagnosis, and treatment of HVS. It is hoped that this article will facilitate the prompt identification and appropriate treatment of HVS, thereby preventing any potential negative outcomes for the patients. Additionally, HVS is a rare complication during the perioperative period and has only been reported in a small number of case reports. Therefore, further research is needed to confirm the effectiveness of measures for prevention and treatment mentioned in the text.

Keywords: Hyperventilation Syndrome, Anesthesia, Strabismus Surgery

1. Introduction

Hyperventilation syndrome (HVS) is characterized by excessive and rapid breathing, leading to a decrease in carbon dioxide levels and subsequent respiratory alkalosis, as well as secondary hypocalcemia and hypokalemia, resulting in arrhythmias, and various somatic symptoms [1-3]. For example, paresthesia, hyperreflexia, muscle cramps, and twitches. The symptoms of hyperventilation syndrome are diverse and may be easily confused with those caused by neurological disorders. Failure to handle the condition properly could result in severe consequences such as acute myocardial infarction or even death [4-6], underscoring the importance of timely and accurate identification and treatment of the syndrome. We report a rare case of HVS after strabismus surgery under local anesthesia.

2. Case Report

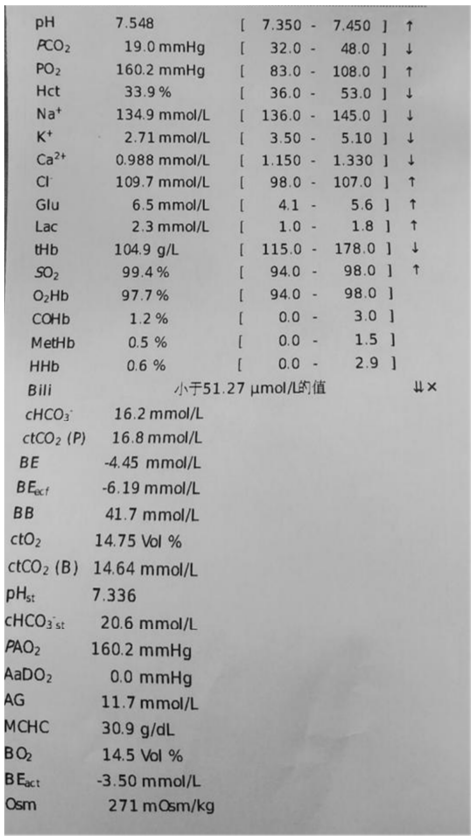
A 31-year-old women with intermittent exotropia was admitted to undergo strabismus correction surgery under topical anesthesia using proparacaine. She had no previous psychiatric illness, such as anxiety or depression, or any other remarkable medical history. Routine blood tests, biochemical tests, coagulation tests, and electrocardiograms did not show any significant abnormalities before the operation. During the procedure, the electrocardiogram, non-invasive blood pressure, and pulse oxygen saturation were routinely monitored. An initial reading of her vital signs showed a blood pressure of 96/60mmHg, a heart rate of 72 breaths per minute, a respiratory rate of 16 breaths per minute, and an oxygen saturation of 100%. The patient received oxygen via a nasal catheter, and 500ml of lactated Ringer's solution was infused intravenously.

At the beginning of the operation, the patient complained of pain, and after intravenous administration of 50mg flurbiprofen axetil injection, the patient rested quietly. Fifteen minutes later, the patient complained of pain again, prompting the administration of a 5ug sufentanil citrate hydrochloride injected Intravenously, which effectively relieved the patient. Five minutes before the end of the operation, the patient reported nausea, which was relieved after intravenous injection of 8mg ondansetron.

Following the surgery, as soon as the surgeon uncovered the sheet, the patient complained about numbness in the lower limbs, spasm in the upper limbs, muscle rigidity, and double vision. The electrocardiogram monitoring revealed blood pressure of 103/50 mmHg, heart rate of 64 beats per minute, pulse oxygen saturation was 100%, and respiratory rate of 14 breaths per minute. The patient was awake and extremely nervous.

Arterial blood gas (ABG) analysis was performed immediately by radial artery puncture. The ABG analysis revealed severe respiratory alkalosis with pH 7.548, pCO_2 19 mmHg, pO_2 160.2 mmHg, HCO_3^- 16.2 mmol/L, base excess − 3.5 mmol/L and lactate 2.3mmol/L. Electrolytes and blood glucose were within normal range. Hyperventilation syndrome was considered on the basis of postsurgical respiratory alkalosis with hypocalcemia and hypokalemia and somatic symptoms.

The treatment involved providing verbal comfort to the patient and administering oxygen inhalation with a closed mask. Additionally, the patient received an intravenous injection of 10% calcium gluconate to correct hypocalcemia and potassium magnesium aspartate 50ml was pumped intravenously to correct hypokalemia. Wrist stiffness and spasticity were gradually relieved. The patient was then sent to the postanesthesia care unit (PACU) for continued observation before being discharged from the hospital on the same day. A subsequent telephone interview was unremarkable.



pH	7.548	[7.350 -	7.450]	↑
PCO_2	19.0 mmHg	[32.0 -	48.0]	↓
PO_2	160.2 mmHg	[83.0 -	108.0]	↑
Hct	33.9 %	[36.0 -	53.0]	↓
Na^+	134.9 mmol/L	[136.0 -	145.0]	↓
K^+	2.71 mmol/L	[3.50 -	5.10]	↓
Ca^{2+}	0.988 mmol/L	[1.150 -	1.330]	↓
Cl^-	109.7 mmol/L	[98.0 -	107.0]	↑
Glu	6.5 mmol/L	[4.1 -	5.6]	↑
Lac	2.3 mmol/L	[1.0 -	1.8]	↑
tHb	104.9 g/L	[115.0 -	178.0]	↓
SO_2	99.4 %	[94.0 -	98.0]	↑
O_2Hb	97.7 %	[94.0 -	98.0]	
COHb	1.2 %	[0.0 -	3.0]	
MetHb	0.5 %	[0.0 -	1.5]	
HHb	0.6 %	[0.0 -	2.9]	
Bili	小于51.27 μmol/L的值			⇊×
$cHCO_3^-$	16.2 mmol/L			
$ctCO_2$ (P)	16.8 mmol/L			
BE	-4.45 mmol/L			
BE_{ecf}	-6.19 mmol/L			
BB	41.7 mmol/L			
ctO_2	14.75 Vol %			
$ctCO_2$ (B)	14.64 mmol/L			
pH_{st}	7.336			
$cHCO_{3\,st}^-$	20.6 mmol/L			
PAO_2	160.2 mmHg			
$AaDO_2$	0.0 mmHg			
AG	11.7 mmol/L			
MCHC	30.9 g/dL			
BO_2	14.5 Vol %			
BE_{act}	-3.50 mmol/L			
Osm	271 mOsm/kg			

Figure 1. *Arterial blood gas (ABG) immediately after the scenario. The ABG analysis revealed severe respiratory alkalosis with pH 7.548, pCO_2 19 mmHg.*

3. Discussion

HVS is mainly caused by excessive breathing in stressful situations, leading to hypocapnia and secondary electrolyte disturbance inducing various systemic symptoms. During and after hyperventilation, tingling sensation of the hand and numbness of the foot, sweating and central nervous system symptoms can be manifested, and even abnormal electrocardiogram, manifested as atrioventricular block and T wave changes [2, 6]. Anxiety and fear are thought to be the main causes of HVS, especially in young women [7]. Asthma, chronic bronchitis, emphysema, and chronic obstructive pulmonary disease (COPD), as well as less frequently pulmonary embolism, encephalitis, brain tumors, and hypoparathyroidism, may also occur. The onset of HVS can also appear following a surgical intervention on the brain. The case reported by Paquin-Lanthier indicates that localized increases in tissue lactate related to a tumor metabolism may stimulate the brainstem respiratory centers and trigger hyperventilation [8]. Similarly, Park et. al reported a case of HVS caused by extensive intracranial pneumatosis after third ventriculostomy [9]. In our case, the patient is a middle-aged healthy woman with no history of respiratory or pulmonary diseases or any other physical diseases. The preoperative electrocardiogram and chest X-ray were completely normal, so we inferred that the patient's anxiety and inadequate analgesia during the surgical procedure may be the primary reason for the occurrence of HVS. Therefore, we suggest that

for patients who frequently complain of pain during local anesthesia surgery, the use of benzodiazepines such as midazolam or diazepam may help prevent the occurrence of HVS and improve patient satisfaction. However, further research is needed to confirm the effectiveness of this approach.

Environmental changes such as anxiety and psychological trauma during anesthesia and surgery have been identified as the main causes of HVS. As seen in several previous reports, HVS developed rapidly within several minutes after removal of the endotracheal tube [7, 10, 11]. Theoretically, changes in breathing rate and pattern should be observed before definite signs and symptoms manifest in patients with HVS. In this patient, the respiratory rate and respiratory mobility were completely normal when the symptoms of limb rigidity and wrist spasticity developed, so hyperventilation was considered to have occurred during the procedure. During ophthalmic surgery, changes in the patient's respiration were not detected timely since surgical drapes and trays covered the torso, and end-tidal carbon dioxide concentration was not monitored during the operation, so it was not detected in time.

In this case, the patient underwent ophthalmic surgery under local anesthesia, without routine monitoring of end-tidal carbon dioxide. This resulted in delayed detection of abnormalities in respiration. Attention to patients' breathing and end-tidal carbon dioxide levels during the perioperative period is crucial for early detection of hyperventilation and timely intervention.

Management of the hyperventilation syndrome includes inhalation with a closed mask to increase carbon dioxide inhalation. However, caution should be exercised when this approach is used in patients with hypoxia and dyspnea. Patients experiencing anxiety may benefit from verbal comforting, and anxiolytics can be used for sedation when necessary [12]. Notably, propofol seems not effective in the treatment of HVS [11, 13, 14]. For patients with increased sympathetic nervous system excitability, beta-blockers are recommended, but care must be taken when treating patients at risk of bronchial obstructive diseases such as bronchial asthma. Additionally, dexmedetomidine has been found to be effective in treating hyperventilation syndrome by activating α2-adrenergic receptors, inhibiting the release of norepinephrine and sympathetic nerve activity, and ultimately producing sedative, analgesic and anti-anxiety effects [15]. However, further studies are required to confirm the usefulness of dexmedetomidine for HVS.

4. Conclusion

HVS is a rare complication during the perioperative period, anesthesiologists should be familiar with the manifestation of HVS and pay attention to distinguishing it from other organic diseases as well as be vigilant and closely monitor patients' vital signs and breathing during surgery, even under local anesthesia. Remember, there is no such thing as minor anesthesia when it comes to surgery. Additionally, further research is needed to confirm effective measures for its prevention and treatment.

Conflict of Interests

All the authors do not have any possible conflicts of interest.

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