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Central Neurogenic Hyperventilation with Acute Respiratory Alkalosis, Transient Lactic Acidosis and Tachycardia Following Endoscopic Third Ventriculostomy in a Child- A Case Report

Fajish Habib [α], Tejas Mehta [σ], Ahamed Lafir Aliyar [ρ], Ahmed Sayed Youssef [ω], Adnan Khan [¥] & Neeraj Kumar [§]

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Outcome: Treatment with sedation and oxygen via rebreathing mask resulted in normalization of symptoms and blood gas.

Message: CNH can occur in children following ETV and should be recognized early. Measurement of ICP during ETV and use of alternative irrigation fluids such as lactated ringer's or artificial CSF may minimize occurrence.

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I. Introduction

Endoscopic third ventriculostomy is a common, minimal-invasive neurosurgical procedure, performed most frequently for patients with obstructive hydrocephalus secondary to impediment of cerebrospinal fluid (CSF) flow across the Aqueduct of Sylvius or the outlets of the fourth ventricle. [1,2]The procedure involves creating a fenestration on the floor of the third ventricle to create a communication between the third ventricle and the basal cisterns as a bypass route for the CSF flow. [3]Complications of ETV include varying degrees of intraventricular hemorrhage, CSF leak, pneumocephalus, arrhythmias (tachycardia and bradycardia) and injury to periventricular structures.[3,4] We present the first pediatric case with intraoperative tachycardia as a result of normal saline irrigation during ETV followed by central neurogenic hyperventilation (CNH) with acute respiratory alkalosis and transient lactic acidosis.

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II. Case Report

a) History and physical examination

A 6-year old, previously healthy girl was admitted to the pediatric ward in Hamad General Hospital with ataxic gait, visual disturbances, difficulty in writing, urinary and fecal incontinence for three months. There was no history of seizures, headache, vomiting or altered sensorium. The child was conscious, alert with intact higher mental functions. Her weight was 25 kg (90th centile), height was 113 cm (50th centile). Heart rate upon presentation was 110 bpm, blood pressure was 100/60 mmHg and respiratory rate was 30 per minute. Examination was significant for Parinaud's syndrome with upward gaze palsy, weakness of the right side with hyperreflexia and positive Babinski's sign on the right side. Fundoscopy revealed Grade 4 papilledema bilaterally but pupils were symmetrical and reactive bilaterally. Urgent MRI of the head (figure 1) revealed a large, irregular, lobular tumor mass in the posterior part of third ventricle and pineal region with involvement of the thalamus and mid brain. There was consequential rapid onset obstructive hydrocephalus. Differential diagnosis included parapinealglioma, pineoblastoma and germinoma. Her serum electrolytes revealed hyponatremia (134mmol/L), which was corrected using normal saline infusion. Other laboratory tests including a complete blood count, coagulation profile, liver and renal functions were within normal limits.

The patient was seen by the neurosurgical team and the decision was taken to perform an endoscopic third ventriculostomy, with an external ventricular drainage catheter insertion followed by biopsy from the tumour mass.

b) Intraoperative course

The patient was intubated, sedated and BP was monitored invasively. Anesthesia was maintained with target -controlled infusion of propofol at 4-5mcg/ml amounting to a total cumulative dose of ~5mg/kg/hr. Intraoperative end-tidal CO_2 was maintained between 32-38mmHg. During the surgical procedure, the

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operative field was being continuously irrigated with normal saline at room temperature. Neuroendoscopic intracranial pressure was not being monitored due to lack of required equipment. From the onset of surgery the patient developed gradual increase in HR from a baseline of 90-100 beats/min to 110-120 beats/min, 1mcg/kg fentanyl was given as a bolus to rule out pain as the cause of tachycardia with no subsequent reductions in heart rate. The intraoperative blood gas (Table1) at this stage revealed pH 7.40, $PaCO_2$ 33mmHg, HCO_3^- 20 mmol/L, base excess -3.7 mmol/L.

The tumor from the right side was biopsied and specimens were obtained for histopathological diagnosis. Septostomy was performed using bipolar and bleeding was encountered from the edges. After the septostomy, normal saline at room temperature was used to irrigate the ventricles under high pressure using a 50cc syringe to minimise the hemostasis, which resulted in a marked increase in the heart rate to 150-160 beats/min. Blood pressure during the time rose to 140-130/70-80. The surgeons were informed, and the scopes were immediately withdrawn and drainage of CSF was done, which drained visibly under very high pressure. Following the sudden drainage of CSF, the heart rate dropped down to 130-140 and the ETCO2 dipped transiently to 24 from 32 mmHg. Arterial blood gas at this stage revealed a pH 7.31, $PaCO_2$ 39.1 mmHg, HCO_3^- 19.9mmol/L, base excess -7 mmol/L. The patient received yet another bolus of fentanyl 1mcg/kg for the possibility of pain induced tachycardia, but there was no response. The total duration of surgery was 4 hours 30 minutes and the patient was successfully extubated at the operating theatre after ensuring adequate voluntary respiration and was then transferred to the PICU for further observation.

c) Postoperative Course

The patient continued to have tachycardia with HR ranging 140-150/min. Her BP was 120/70 mmHg and the patient was tachypneoic with RR reaching 50 /min. ABG done 1 hour after the procedure revealed fully compensated respiratory alkalosis with metabolic acidosis pH 7.43, PaCO2 13.0 mmHg, HCO_3^- 9.1mmol/L, base excess -13.6 mmol/L, glucose 14.9 mmol/L and lactate 4.3 mmol/L. Although the patient was fully conscious, alert and euvolemic, a bolus of normal saline was administered to see if there would be any change in HR or RR and there was none. The patient was still hyperventilating with tachycardia and an ABG repeated 2 hours from surgery showed no change in the respiratory alkalosis and metabolic acidosis with pH 7.38 $PaCO_2$ 13.2 mmHg, HCO_3^- 8.0mmol/L, base excess -13.1 mmol/L, lactic acid 4.9 mmol/L. As the cause for the hyperventilation and lactic acidosis was unclear a dose of 1mEq/kg of 8.4% sodium bicarbonate was administered as symptomatic treatment. The HR dropped to 120-130 per minute and RR to 35-40 per minute. Follow-up ABG, 6 hours from the procedure showed an uncompensated respiratory alkalosis with pH 7.51, $PaCO_2$ 16.6mmHg, HCO_3^- 13.5 mmol/L, base excess -8 mmol/L and lactic acid 1.4 mmol/L. The child was more irritable and had started to complain of fear and nightmares. She also developed fever with a core temperature of 38.5^0C. A suspicion of central neurogenic hyperventilation was raised at this stage and the patient was put on oxygen via a rebreathing facemask. The patient was sedated with lorazepam of 0.1mg/kg intravenously. ABG after 8 hours from the procedure showed an improvement in the respiratory alkalosis with pH 7.46, $PaCO_2$ 26.6 mmHg, HCO_3^- 21.1 mmol/L, base excess -4 mmol/L and lactic acid 1.0 mmol/L. HR dropped to 110-120 per minute and RR to 25-30 per minute, almost 12 hours after the surgery. 16 hours from surgery her ABG was back to normal with pH 7.42, $PaCO_2$ 32.2 mmHg, HCO_3^- 22.0 mmol/L, base excess-2.7 mmol/L, lactic acid 0.4 mmol/L. The patient was transferred to the pediatric ward on the second post-operative day. The biopsy revealed a final diagnosis of WHO grade 1 Pilocytic Astrocytoma.

III. Discussion

Hyperventilation can be central or peripheral. However, when it does occur, peripheral causes have to be ruled out first. There are numerous causes for peripheral hyperventilation such as fever, pain, asthma, pneumothorax, pulmonary embolism, drugs, alcohol withdrawal, ischemic heart disease and congestive heart failure, hyperthyroidism.[5] Reported first by Plum and Swanson in 1959, central neurogenic hyperventilation (CNH) is a rare respiratory syndromedefined as an abnormally regular, rapid (>25 to 30 breaths/min) breathing pattern that cannot be explained by hypoxemia.[6]CNH can either be persistent or transient. Persistent CNH is seen most commonly due to tumors, especially with pontine involvement.[7] Of the various mechanisms that have been described, CNH has been thought to occur mainly due to the disconnection between pontine and medullary respiratory centers leading to unopposed stimulation of the latter or from acidosis due to lactate production by tumor mass, thereby activating the brainstem chemosensitive respiratory neurons.[7]

Our case is an example of transient CNH, which is one of the rare but documented complications following endoscopic third ventriculostomy. [4,8-10]As reported previously, the proposed mechanism of CNH following ETV is due to a transient hypothalamic dysfunction caused by unrecognized rise in intra-cranial pressure while irrigating the third ventricle with normal saline under high pressure.[8,11,12] The floor of the third ventricle is formed by a part of the hypothalamus and it is this hypothalamic dysfunction, which was responsible for the tachycardia, hypertension and

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hyperthermia that was seen in our patient as a part of the midbrain dysregulation syndrome reported previously by Pranzatelli et al.[12] The initial management of CNH is with delivery of oxygen via a rebreather mask and use of benzodiazepines to sedate the patient. In severe cases, where patients can have irritability and altered sensorium, mechanical ventilation maybe required to control the hyperventilation.[8,9]

Although uncommon, it is pertinent that the anesthesiologists, neurosurgeons and critical care teams involved be aware of such significant intra and post-operative hemodynamic disturbances which can occur following an ETV. Such a dramatic clinical presentation, as in our case, can pose quite a difficult diagnostic challenge to pinpoint the exact etiology unless it is specifically looked for. Again, this case, along with those reported previously, highlights the necessity of intraoperative intracranial pressure monitoring during neurosurgical interventions.[8,9]

The transient lactic acidosis associated with the CNH and acute respiratory alkalosis is a novel feature of our case, as none of the previously reported cases in literature have suggested a transient serum lactic acidosis as secondary sequelae of the CNH, which in turn can occur as a complication of ETV. We were unable to attribute a cause for the elevated serum lactate initially; however, there have been detailed studies, which looked into the association of hyperventilation with lactic acidosis and found that there is a strong correlation.[13-15]Hyperventilation has been shown to increase the basal concentration and reduce the elimination of lactic acid, thereby inducing lactic acidosis throughout the period of hyperventilation.[13] This happened to be the case in our patient who had elevated serum lactate as long as she was hyperventilating, following which the levels returned to normal. We recommend measurement of lactic acid levels in all patients with CNH to gain more insight, as there have been no previous reports of an association between CNH and lactic acidosis per se.

The type of neurosurgical fluid used for irrigation during neurosurgical procedures has been the subject of debate for a long time. The detrimental effects of normal saline on neural tissue especially in neuroendoscopic surgeries, due to the large volume of saline used for irrigation in a closed space, has been highlighted in a review by Syed et al.[16] They have suggested, based on previous physiological studies, that normal saline has a much different composition to CSF, which is the natural irrigant. Composition of normal saline is different form CSF in terms of pH, osmolality and presence of bicarbonate-which is the key buffer in normal CSF. The continuous loss of carbon dioxide normally from neural cells, combined with the absence of the buffering action offered by bicarbonate and slightly acidic pH (6.4) of normal saline may lead to brain damage. Moreover, the lack of K^+, Ca^{2+}, and Mg^{2+} in normal saline may contribute to its unfavorable effects on neural tissue.[16-19] Artificial CSF followed by lactated ringer's solution share the most similarities in terms of physiological resemblance (pH, osmolality and inorganic ions content) to CSF and are the fluids of choice for various authors for brain irrigation during neuroendoscopic procedures.[16-21] The hyperventilation associated with our case could be partly associated with CSF acidosis induced by normal saline used for brain irrigation during the ETV.[1] Hence, more detailed studies are needed to look into usage of artificial CSF and lactated ringer's as substitutes for normal saline as the irrigating fluids of choice, especially for neuroendoscopic procedures.

In conclusion, ETV may cause intra-operative hemodynamic disturbances such as tachycardia, hypertension and hyperthermia followed by post-operative transient hypothalamic dysfunction and CSF acidosis leading to sequelae of central neurogenic hyperventilation with acute respiratory alkalosis and transient lactic acidosis. We would like to emphasize the importance of ICP monitoring during neuroendoscopic procedures, as an in advertant rise in ICP appears to be the central factor leading to the various ill effects encountered both intra and post operatively. Moreover, normal saline has been the irrigation fluid of choice for neurosurgeons, although a multitude of laboratory studies suggest normal saline being less than ideal for the purpose. It might be prudent to look into alternatives, namely artificial CSF and ringer's lactate.

Disclosure

The authors report no conflict of interest concerning the findings specified in this paper.

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Figure Legend

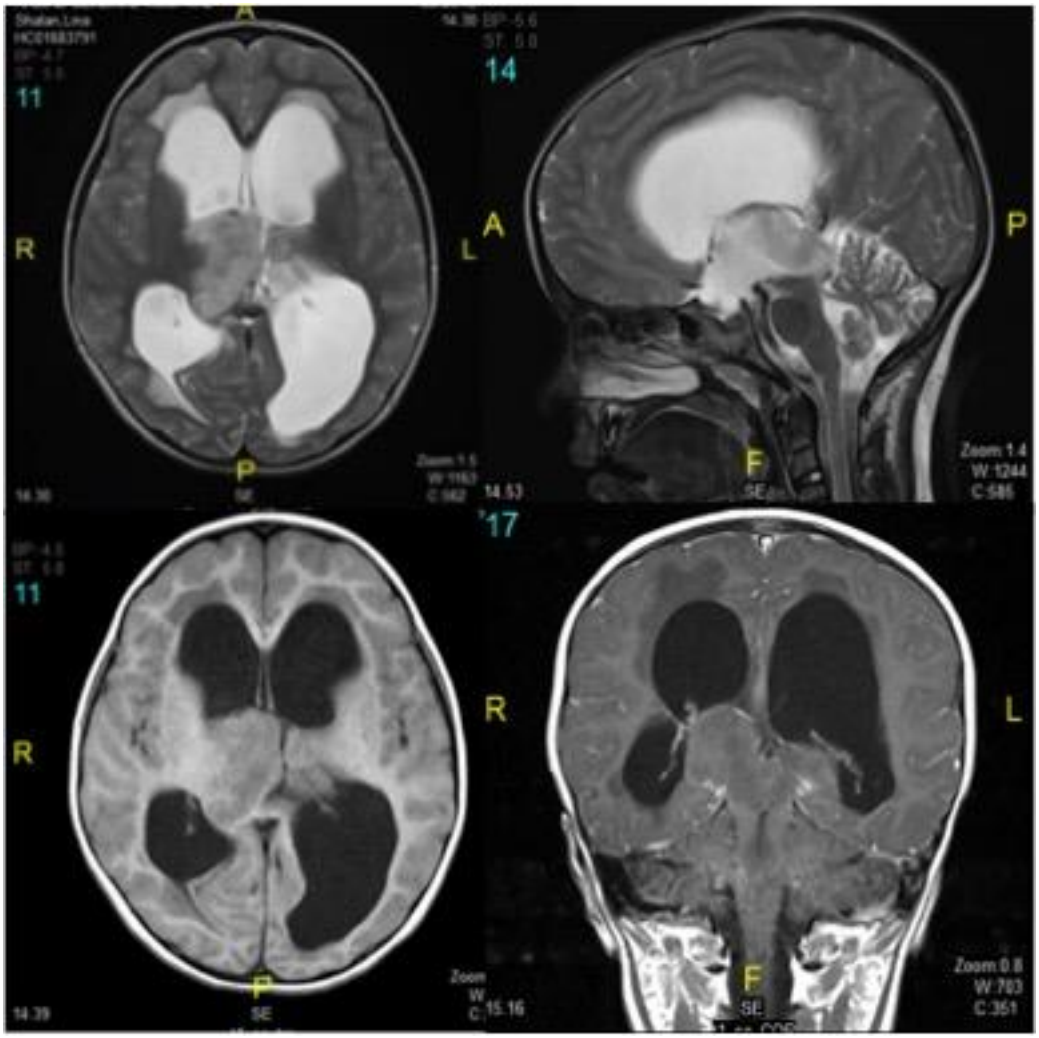

Figure 1: Preoperative MRI showing a large, irregular, lobular tumor mass in the posterior part of third ventricle and pineal region with involvement of the thalamus and mid brain

Table 1: Arterial blood gas analysis

	pH	$PaCO_2$ (mmHg)	HCO_3^- (mmol/L)	B.E (mmol/L)	Lactate (mmol/L)
90 min into surgery	7.40	32.9	20.6	-3.7	0.4
4 hours into surgery	7.31	39.1	19.9	-7.0	1.5
1 hour post-surgery	7.43	13.0	9.1	-13.6	4.3
2 hours post-surgery	7.38	13.6	8.1	-15.4	4.9
6 hours post surgery	7.51	16.6	13.5	-8.4	1.4
8 hours post surgery	7.46	26.6	21.1	-4.0	1.0
16 hours post surgery	7.42	32.6	22	-2.7	0.4

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