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Annals of Pediatrics & Child Health

Case Report

Neurologic Recovery after an Exam Consistent With Brain Death in an Adolescent with Severe Asthma

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Submitted: 12 November 2014

Accepted: 11 March 2015

Published: 14 March 2015

Keywords

- Asthma
- Hypercapnia
- Supercarbia
- Status asthmaticus

Abstract

This report describes a 13 year-old female with a history of asthma who presented in acute respiratory failure with profound hypercarbia secondary to status asthmaticus. On admission to our PICU, her neurologic exam was consistent with brain death. Her respiratory symptoms were treated with bronchodilating agents including terbutaline, magnesium, aminophylline and epinephrine infusions, as well as inhaled sevoflurane. Upon resolution of the severe hypercarbia, her neurologic status improved. She was ultimately extubated on day six of admission and discharged to an inpatient rehabilitation facility approximately one week later. At one year follow up the patient was back to school and had made a full recovery with no detectable neurologic deficits. Clinical and neuroradiographic changes consistent with brain death may be associated with high PaCO2 levels and amenable to treatments directed against the bronchospasm and respiratory failure. Only after the hypercarbia has resolved can the patient's neurologic outcome be accurately prognosticated.

ABBREVIATIONS

PICU: Pediatric Intensive Care Unit.

INTRODUCTION

Changes in neurologic examination in the setting of extreme hypercapnia have not been well documented, especially in the pediatric population. We report one patient who presented to our ICU with respiratory failure and extreme hypercapnia secondary to status asthmaticus with a neurologic exam consistent with brain death, including bilateral fixed and dilated pupils, absent gag, cough and corneal reflexes.

CASE PRESENTATION

A 13 year old African American female with a past medical history significant for asthma presented to an outside emergency department with a one day history of increased difficulty breathing and cough despite nebulized albuterol treatments at home. Upon arrival to the emergency department at a referring institution, she was wheezing, tachypneic and in moderate respiratory distress. A chest radiograph performed at this time was interpreted as normal. Treatment commenced with continuous B-agonist therapy, intravenous steroids, and antibiotics. She was subsequently admitted to the pediatric intensive care unit (PICU) at the referring institution where she was placed on bilevel positive airway pressure (BiPAP) for worsening respiratory distress. While on BiPAP she had an episode of emesis with aspiration which required emergent endotracheal intubation. A repeat chest radiograph following endotracheal intubation demonstrated patchy infiltrates, extensive subcutaneous emphysema, pneumomediastinum, and small bilateral apical pneumothoraces. Due to increasing difficulty with ventilation and oxygenation despite maximal ventilatory support, the patient was transferred to our facility for possible Extracorporeal Life Support (ECLS) therapy.

Upon arrival of our transport team to the referring institution the physical exam was notable for continued wheezing with markedly diminished air entry bilaterally. During transport to our facility, her pupils were found to be sluggish and unequal. Bronchodilating measures including intravenous terbutaline and magnesium were continued.

Approximately ten hours after her initial presentation at the referring institution, she arrived to our PICU with a significant air leak thought to be due to a malfunctioning endotracheal tube cuff. The endotracheal tube was exchanged without incident. Oxygen saturation at this time was in the upper 80's, however slowly improved. The first arterial blood gas revealed a pH

6.73, $paCO_2$ 183 mmHg, paO_2 162 mmHg and a BE of -12. The measured A-a gradient was 323 and oxygenation index was 6.1. Initial ventilator settings (Avea ventilator, Carefusion, San Diego, California, USA) included a PIP 60 cmH_2O to achieve a TV 3 ml/kg, PEEP 2 cmH_2O, RR 8, T_i 1.1, and a FiO_2 of 100%. Additionally, on arrival to the PICU, the patient's left pupil became non-reactive. Bolus doses of mannitol and hypertonic saline were administered and an infusion of hypertonic saline was initiated. Despite these treatments, the right pupillary exam did not change, and the left pupil became non-reactive. An emergent portable head CT was performed which demonstrated preserved gray-white matter differentiation, sinusitis, and a small hyperdense area in the right parietal lobe, concerning for a small petechial hemorrhage.

Despite maximal bronchodilating measures including terbutaline, magnesium, aminophylline and epinephrine infusions, the hypercarbia persisted. An anesthesia consult was called and treatment with inhaled sevoflurane was initiated in addition to bronchodilating therapies already in place. An infusion of norepinephrine was started to treat mild hypotension associated with the initiation of inhaled sevoflurane. All intravenous sedatives and paralytics were discontinued with the initiation of inhaled anesthetic therapy. Her neurologic exam continued to be consistent with that of brain death, with absent pupillary and corneal reflexes, no cough or gag reflex, and a negative dolls-eye.

Over the next 12 hours, ventilation slowly improved with $paCO_2$ levels decreasing to 60-70 mm Hg. At this time sevoflurane administration was discontinued. Other bronchodilating therapies were continued at this time. She began to exhibit spontaneous respirations and was subsequently switched to a mode of pressure supported ventilation. Her pupils were at this time both sluggishly reactive to light. Over the course of the day she became increasingly responsive with spontaneous movements and gestures. Within days, the patient's neurologic status greatly improved as she was able to follow commands and communicate with her mother and staff. Mechanical ventilatory support and bronchodilator therapies were weaned. On hospital day 5, an MRI of the brain was performed, which showed changes within the corpus callosum consistent with infarction or ischemia. Interval follow-up was recommended. On hospital day six, the patient was extubated. On hospital day 10 the patient was off all bronchodilator infusions and was taken off BiPAP. On hospital day 14 the patient was discharged to an inpatient rehabilitation facility. Upon social work follow up one year later the mother reported that the patient was back to school and had made a full recovery with no detectable neurologic deficits.

DISCUSSION

The differential diagnosis for dilated and nonreactive pupils include: direct trauma to the eye or the sympathetic trunk, accidental contamination with or direct instillation of anti-cholinergic or alpha-adrenergic agents, cranial nerve III injury or compression from acute intracranial hypertension [1,2]. There have been case reports of both adult and pediatric patients with asthma who were found to have anisocoria secondary to local contamination of the eye with aerosolized anticholinergics [3-5]. In our case, the patient had not received anticholinergic agents prior to the pupillary changes; nor was there any history of trauma reported upon initial presentation or throughout her hospital course that would explain the bilateral dilated, non-reactive pupils. Additionally, the absence of cranial nerve function could not be explained by any history at home, events in the hospital, or the normal appearance of the brain on CT scan.

To our knowledge, there is currently no data on the effects of hypercapnia on the pupillary response or cranial nerve exam of pediatric patients with asthma. There has been a small number of case reports in adults describing reversible neurologic sequelae of hypercapnia associated with status asthmaticus [1,6,7]. However, CT findings in those cases, unlike our patient, were consistent with intracranial hypertension, cerebral edema, and in one case, subarachnoid hemorrhage. In each case, the resolution of neurologic findings on physical exam and normalization of the CT scan correlated directly with reduction in $PaCO_2$ levels.

It is well known that cerebral vasodilation occurs in response to changes in $PaCO_2$ mediated locally by changes in perivascular pH [8,9]. The relationship between cerebral blood flow and $PaCO_2$ is linear, increasing by 4 ml/100g/min for every rise of $paCO_2$ by 1 mmHg up to 120 mmHg, at which point maximal cerebreal vasodilation is achieved [9,10]. At extreme $PaCO_2$ levels, as seen in our patient, we would expect maximal cerebral vasodilation, leading to increased intracranial pressure (ICP). This rise in intracranial pressure could also have been exacerbated by poor cerebral venous drainage secondary to high intrathoracic pressures generated both by dynamic hyperinflation associated with asthma and by our treatment with high ventilator settings [1,6,11]. Although our patient's CT scan did not demonstrate evidence of cerebral edema or intracranial hypertension, it is plausible that these changes were reversed by aggressive medical treatment. Another possibility includes the decrease in neuronal activity in the cortex and reticulo-activating system that occurs at $paCO_2$ levels above 200 mmHg [10]. As the highest recorded $paCO_2$ in our patient was 183 mmHg, it is likely that $paCO_2$ levels were greater than 200 mmHg for some period of time, which may have contributed to the absence and return of cranial nerve function with improvement in clinical status.

It is important to note that despite extreme hypercapnia; neurologically intact survival is possible as long as tissue anoxia and ischemia are prevented [10-12]. Numerous case reports in adult patients detail full neurologic recovery in patients who suffer from severe hypercapnia in the setting of status asthmaticus or hypoventilation [1,7,10]. Children have been found to recover neurologically as well. In a retrospective chart review of PICU patients with supercarbia ($Paco_2$ > 150 torr), there was no evidence to suggest long term impaired development [13]. There is one reported case of an 8 year old patient with asthma who suffered a prolonged period of profound hypercarbia (293 mmHg) yet survived with no neurologic sequelae [12]. Our case is the first reported case of a patient exhibiting a neurologic exam consistent with brain death secondary to hypercapnia. More importantly however, is the full recovery she accomplished with the help of physical and rehabilitative therapy, and its implications for future rescue treatments such as inhalational anesthetics and ECMO in similar patients.

In summary our patient's clinical course was unique in that her physical exam was consistent with brain death, yet

a complete recovery occurred with no lasting neurocognitive effects. Although there have been reports of both pediatric and adult asthmatics with supercarbia (Pco_2 >/=150mmHg) and unresponsive pupillary exams, none of the patients described had a lack of brainstem reflexes [7,12,13]. It is important for clinicians caring for patients with supercarbia to be aware of the possible temporary, and reversible clinical and neuroradiographic changes associated with high $PaCO_2$ levels, as medical aggressive treatment should continue to treat the bronchospasm and respiratory failure. Only once the hypercarbia has resolved can the patient's neurologic outcome be accurately prognosticated.

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Cite this article

Glater-Welt LB, Kessel A (2015) Neurologic Recovery after an Exam Consistent With Brain Death in an Adolescent with Severe Asthma. Ann Pediatr Child Health 3(3): 1057.