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Editorial Correspondence

Severe Respiratory Acidosis-induced Involuntary Hyper defecation: The Role of Medullary Caudal Raphe Nuclei.

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Dear Editor,

We report a case of a 9-year-old male child who presented to us with progressive quadriparesis over the past 6 months. Clinical examination and imaging studies revealed congenital atlantoaxial dislocation. He was thus posted for posterior fixation. Early on the morning of the surgery, the child developed sudden bradypnea (respiratory rate – 10-12 breaths/min), tachycardia (165-170 beats/min), high blood pressure (systolic 140 mm Hg), oxygen desaturation (SpO_2 – 90-92%), and responsive only to painful stimuli (Glasgow Coma Scale: E1V1M2). This respiratory depression was secondary to the compression of the cervico-medullary junction and spinal cord edema (Figure. 1).

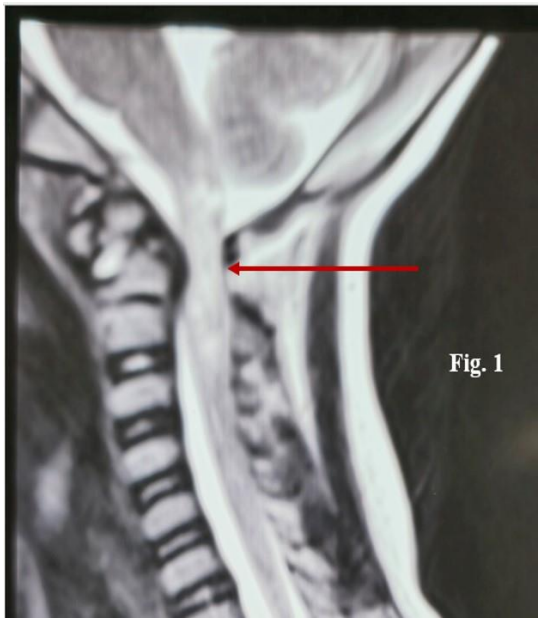

Figure 1. T2 weighted magnetic resonance imaging of cranio-vertebral junction and upper cervical spine (sagittal view) showing retroflexion of odontoid process with ventral compression at the cervico-medullary junction. There is T2 hyperintensity with edema extending from the cervical spinal cord (C3-C4) region to the middle portion of the medulla oblongata (marked by a red arrow).

He also developed involuntary hyper defecation during this time which was unusual. Oxygen inhalation by facemask was initiated at 10 litres/min along with placement of an oropharyngeal airway. After rapid cleaning of the stool and application of new pads, he was immediately rushed to the operating room and his airway was secured by endotracheal intubation. The first end-tidal carbon dioxide ($EtCO_2$) reading was 101 mm Hg. One arterial blood gas (ABG) analysis immediately thereafter showed $PaCO_2$ of 110 mm Hg and severe respiratory acidosis (pH = 6.8). We then hyperventilated his lungs, and over the next 20 mins, $EtCO_2$ was stabilised to values between 35-40 mm Hg. The surgery started after all the parameters were stable. He remained hemodynamically stable during the entire surgery. He was electively ventilated after the surgery where he regained consciousness and remained so throughout the rest of his stay in the intensive care unit.

Through this report, we would like to convey that involuntary hyper defecation is probably a feature of severe respiratory acidosis. Colorectal motility is regulated by two defecation centers viz. the supraspinal center (mainly located in the pons) and the lumbosacral defecation center (L6–S1).[1] The supraspinal defecation center controls the lumbosacral defecation center to modulate the colorectal motility, and outflows from the lumbosacral defecation center project to the enteric nervous system of the colorectum via colonic nerves and pelvic nerves. [2] Nakamori et al[3]had demonstrated in anesthetized rats that stimulation of the medullary caudal raphe nuclei (comprising of raphe pallidus, raphe magnus, and raphe obscurus) enhances colorectal motility through activation of the serotonergic system in the lumbosacral defecation center. Previous studies have clearly demonstrated that serotonergic neurons of the medullary caudal raphe nuclei are stimulated by hypercapnia in vivo, and their disruption results in a blunted hypercapnic ventilatory response.[4, 5] It could very well happen in our case that hypercapnic respiratory acidosis stimulated the serotonergic neurons of the medullary caudal raphe nuclei which in turn enhanced the colorectal motility through activation of the serotonergic system in the lumbosacral defecation center leading to involuntary hyper defecation (Figure. 2).

However, large studies are needed to establish this postulate into concrete evidence.

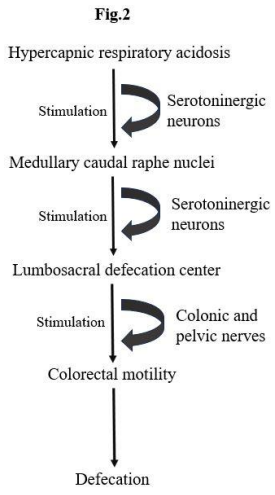

Figure 2. Schematic representation of the mechanism of hypercapnic respiratory acidosis-induced hyper defecation.

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