Did the infant have respiratory depression after sedation with chloral hydrate?

To the Editor,

Dr. Çeçen and his colleagues[1] presented a four-month-old girl with mild hyperventilation (respiratory rate 40/min, hyperpnea) tachycardia with cyanosis and coma, which developed about 10 minutes after oral administration of chloral hydrate (CH).

The infant had severe acidosis (pH: 7.05, HCO_3: 22 mEq/L), although the authors stated respiratory acidosis because of pCO_2: 80 mm/Hg and PO_2: 48 mm/Hg. I am curious about the mixed condition and would like to learn the Na+, K+ and Cl values.

Respiratory alkalosis is expected in the presence of hyperpnea (respiration rate 40/min; not suppressed) and she had acidosis (pH: 7.05). In my opinion, it is hard to explain this acidosis with the accumulation of trichloroacetic acid degradation by the CH metabolism since it occurred within 10 or 15 minutes after the drug administration. Because of the HCO_3 level, a metabolic acidosis with respiratory alkalosis combination might also be considered. How was the acidosis treated?

I would also question the sedation therapy for jitteriness due to vaccination[7]. Was something the cause of all of these symptoms? Were toxicologic studies carried out?

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Reply

We read with great interest the comments of Prof. Şinasi Özsoylu about our article[1]. To this point, in our opinion, detailed information should be given about the first physical examination and the laboratory findings. After the patient's first treatment, we contacted the doctor who administered the chloral hydrate and learned that no convulsion was present other than jitteriness, agitation and crying. In addition to the physical examination findings in our article, the patient had hypoxic gasping and subcostal/suprasternal retractions. If not treated, the patient would probably have reached the agonal gasping in a short time and then the apnea period. There was no clinical or laboratory finding of alkalosis with hyperpnea as mentioned by Prof. Özsoylu. In the first blood gas analysis, severe acidosis (pH: 7.05, HCO_3: 22 mEq/L; pCO_2: 80 mm/Hg and PO_2: 48 mm/Hg) was noted and after intubation, the blood gas analysis started to improve (15^{th} minute pH: 7.16, HCO_3 23 mEq/L; pCO_2: 66.4 mm/Hg and PO_2: 66 mm/Hg). The blood gas analysis returned to normal 30 minutes after the intubation and the oxygen saturation was 99%. During this period, no other treatment such as sodium bicarbonate was given besides intravenous fluid and ventilation therapy. For this reason, respiratory alkalosis was not considered to explain the respiratory acidosis and hyperpnea. The serum electrolyte levels were normal (Na: 130 mEq/L, K: 4.7 mEq/L, Cl: 102 mEq/L) but the blood glucose level was 508 mg/dl. The blood glucose levels were measured hourly and found as 343, 279, 169, 120 mg/dl, respectively, and were finally normal at the fourth hour. The hyperglycemia was connected to stress and no insulin treatment was given. Because respiratory depression and miosis are possible side effects of chloral hydrate, no toxicology test was suggested by the toxicology department. At the 8^{th} hour, the patient was extubated and nebulized steroid was used because of the inspiratory stridor. During the intubation, the larynx was edematous and laryngeal obstruction was noted. Thus, the inspiratory stridor may have been the result of this condition. The immediate arrest of spontaneous breathing and the coma (E1M1V1) period after the intubation suggested central nervous system depression. There was severe hypoxia in our patient and if not treated the spontaneous breathing would have rapidly worsened and the patient would have reached the apnea period. Gasping is a strong indicator of hypoxia. In response to hypoxia, there is an initial period of arousal and hyperpnea, then primary apnea lasting seconds or minutes, then a gasping stage. The gasps become progressively weaker and finally result

in terminal apnea unless external support is provided[2]. In the literature, although chloral hydrate commonly results in innocent hypoxia requiring minimal or no treatment, severe side effects including death are reported[3,4]. Our patient was discharged without any sequelae, but the family made a criminal complaint regarding the doctor applying the chloral hydrate and a legal investigation was performed.

To conclude, we reported this patient to remind of the possible side effects of chloral hydrate, which is used commonly and sometimes unnecessarily, and to point out the importance of precaution in all anesthetic drugs. We thank Prof. Özsoylu for his comments and the editor for giving us this opportunity to reply.

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