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Case Report Of New Born With Low Spo2 During Sleep

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ABSTRACT:

CASE:

Baby 1-week-old boy born at 37 weeks' gestational age through spontaneous vaginal delivery. The pregnancy was complicated by maternal gestational diabetes mellitus and pre-eclampsia. The Apgar score was 9 at both 1 minute and 5 minutes after birth. Because of hypoglycemia at delivery that required IV dextrose, he was admitted to the neonatal intensive care unit. His blood glucose levels quickly stabilized, dextrose administration was discontinued, and the patient began breast-feeding. On day 2 of life, the patient began having intermittent oxygen desaturation, with oxygen saturation as measured by pulse oximetry down to 70% while sleeping; Aside from the hypoxia, he was otherwise asymptomatic. He was breast-feeding without difficulty and had no vomiting or gastroesophageal reflux; no cyanosis, stridor, or snoring; and no seizure-like activity or hypertonicity.

Physical Examination:

Baby's temperature was 36.7 C, heart rate was 112 beats per minute, blood pressure was 70/45 mm Hg, respiratory rate was 36 breaths/min, and oxygen saturation was 99% on room air with the patient awake. He was well nourished with no acute distress. His pupils were equal, round, and reactive to light. He was normocephalic, and the anterior fontanelle was open, soft, and flat. The lungs were clear to auscultation bilaterally, with no retractions or adventitious sounds. The heart had a regular rate and rhythm with no murmur. Normal peripheral perfusion was seen. There was normal muscle tone, strength, and range of motion throughout. The abdomen was soft and not tender or distended, and no organomegaly was present.

Diagnostic Studies:

Head ultrasonography, echocardiography, and chest radiography results were all negative. A sepsis workup, including complete blood count, electrolyte panel, and blood culture, was negative. Six-hour polysomnography was performed to further evaluate the desaturation during sleep. Monitoring included electroencephalography, electromyography, electrocardiography, respiratory inductance plethysmography, thermistor, pressure transducer airflow readings, pulse oximetry, and end-tidal CO_2 monitoring.

Discussion

The infant had mixed sleep apnea with a predominance of periodic breathing (approximately 15% of total sleep time) and associated oxygen desaturation.
The apnea hypopnea index (AHI) was 49 events/h, with improvement to six events

per hour when the patient received 0.25 L/min oxygen through a nasal cannula. The American Academy of Sleep Medicine requires four criteria be met for this diagnosis:
(1) gestational age >37 wk,
(2) presence of central apnea,
(3) recurrent prolonged central apnea (>20 s) or periodic breathing 5% of total sleep time on monitoring, and
(4) findings that are not better explained by another disorder or medication. This entity must be distinguished from normal respiratory pauses, either isolated or after sigh, breaths, or movements.
The baby in our study met these four diagnostic criteria.

Primary central sleep apnea of infancy is a well- recognized, though oftentimes forgotten entity in infancy. There is a predominance of central events; however, mixed and obstructive apnea may be present. Sleep apnea during infancy results from the interplay of three important mechanisms. (3)First, in normal breathing, an increase in upper airway muscle tone is required, prior to diaphragmatic contraction for unobstructed inspiration to occur. In newborn infants, this sequence of events may be altered, leading to upper airway collapse and airway obstruction during inspiration. Next, immaturity of respiratory control results in overcompensation for changes in CO_2 and oxygen levels through a mechanism known as "loop gain," which refers to the magnitude of ventilatory response for a given gas exchange disturbance. Those with high loop gain are prone to respiratory instability, whereas those with low loop gain are quite resistant to periodic breathing. Loop gain is affected by many components including the central respiratory controller, the efficiency of CO_2 excretion, and the delays imposed by hemoglobin binding and the circulation.(1) Any of these various "gains" can serve to elevate the overall loop gain and create a propensity for breathing instability. Finally a lower functional residual capacity in infants leaves less available oxygen stores. This, accompanied by a high loop gain, predisposes infants to respiratory instability.

The onset of primary central sleep apnea of infancy is usually in the first weeks to months of life.(1) Workup may include investigation for infection, anemia, metabolic disturbances, medication effects, trauma, seizure, congenital central hypoventilation syndrome, and Chiari malformation. In cases of brief resolved unexplained events, primary central sleep apnea of infancy may be considered in the differential diagnosis. Although it is known that the frequency and prevalence of symptomatic apnea decreases with age, intervention may be necessary to ameliorate the risk for severe oxygen desaturation during apnea in infants with physiologically low functional residual capacity.(2)

First, supplemental oxygen may be used to increase the oxygen reserve and decrease loop gain. However, even oxygen supplementation is not without adverse effects and can become ineffective with dislodgement or mucous plugging of the delivery interface. Also, a recent study in infant rats showed that hyperoxia leads to inflammation, with alveolar perturbation and leukocyte infiltration. CPAP may be used as well. An animal study showed that CPAP ameliorated recurrent central apnea by increasing lung volume and decreasing the cycle of

loop gain, making it a potential option in more severe cases. Another option is to target the neurotransmitters involved in respiratory control(3). Methylxanthines, such as caffeine citrate, have been used in clinical practice to reduce apnea The mechanism of action is uncertain, but possibilities include increase chemoreceptor responsiveness to CO_2, enhanced respiratory muscle performance, and generalized central nervous system excitation.

Overall, the prognosis is good with resolution of findings over time. It is important to note that infant sleep apnea is not an established risk factor for sudden infant death syndrome. Also, persistent apnea may indicate an underlying medical condition requiring further workup.

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