Management of post-hyperventilation apnea during dental treatment under monitored anesthesia care with propofol

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Abstract

Although hyperventilation syndrome generally carries a good prognosis, it is associated with the risk of developing severe symptoms, such as post-hyperventilation apnea with hypoxemia and loss of consciousness. We experienced a patient who suffered from post-hyperventilation apnea. A 17-year-old female who suffered from hyperventilation syndrome for several years developed post-hyperventilation apnea after treatment using the paper bag rebreathing method and sedative administration during a dental procedure. We subsequently successfully provided her with monitored anesthesia care with propofol. Monitored anesthesia care with propofol may be effective for the general management of patients who have severe hyperventilation attacks and post-hyperventilation apnea. This case demonstrates that appropriate emergency treatment should be available for patients with hyperventilation attacks who are at risk of developing post-hyperventilation apnea associated with hypoxemia and loss of consciousness.

Keywords: Post-hyperventilation apnea, Propofol, Dental treatment

Background

Hyperventilation syndrome in patients with no underlying organic abnormality is frequently observed during medical and dental practice. Although the pathophysiological mechanisms of hyperventilation syndrome are still not fully understood, the relative roles of peripheral and central chemoreceptors in causing hyperventilation attacks have been suggested. Although it is believed that episodes of hyperventilation attacks resolve spontaneously and that the paper-bag rebreathing method or administration of anxiolytic agents may help mitigate an attack, sustained symptoms associated with hypocapnia have been reported to initiate complex clinical complications, such as the delayed occurrence of hypoxemia. Furthermore, several case reports have described the occurrence of post-hyperventilation apnea in association with sustained cyanosis, hypoxemia and loss of consciousness [1-4]. The pathogenesis of post-hyperventilation apnea has been linked to the activity of peripheral chemoreceptors, which may contribute to the susceptibility to apnea during hypoxia or hyperoxia [5]. We provided monitored anesthesia care thrice to a patient who had a history of hyperventilation attacks and post-hyperventilation apnea during dental treatment under sedation with regional anesthesia. The aim of this report is to describe the management of hyperventilation attacks and post-hyperventilation apnea during dental treatment.

Case presentation

First episode (Figure 1)

A female 17-year-old (height 154 cm, weight 56.4 kg) was scheduled for dental treatment (root canal treatment for an infected lower second molar) under regional anesthesia at the dental office of a university hospital. Since she had previously experienced hyperventilation attacks, not only during dental treatment but also in other situations, such as during bus rides, she was prescribed benzodiazepines (alprazolam) and paroxetine hydrochloride hydrate by a psychiatrist for a diagnosis of hyperventilation syndrome. There was no other medical history other than the psychological aspects. During the root canal treatment, the patient had a hyperventilation...
attack after experiencing pain. After fifteen minutes of therapeutic treatment using the rebreathing method with a paper bag did not alleviate her symptoms, our team dental anesthesiologist was asked to support her respiratory condition. As shown in Figure 1, after confirming sustained hyperventilation of respiratory rate 50 ~ 60 breaths/minute with desaturation to 92% associated with blood pressure 130/83 mmHg and pulse rate 115 beats/minute, we decided to provide low dose 2 l/min of oxygen supplementation using a bag-valve-mask instead of using the paper bag. With this, although her oxygen saturation improved to 100%, hyperventilation continued for 20 minutes. Confirming sustained hyperventilation, we decided to administer midazolam intravenously, as generally recommended in the treatment algorithm for hyperventilation syndrome [6]. However, we decided to decrease the dose of intravenous administration of midazolam to 0.5 mg, to minimize the risk of unpredictable changes in respiratory function. Immediately after injection of midazolam the hyperventilation subsided, with her respiratory rate improving to 20 breaths/minute. However, she then lost consciousness and developed complete apnea, resulting in significant desaturation to 88 ~ 76%. We tried to treat her with artificial ventilation using a bag-valve-mask with supplementation of a higher dose of 5 ~ 7 l/min oxygen. Two minutes after continuous mask ventilation of 8 ~ 10 counts/min to treat the complete apnea associated with unconsciousness and cyanosis, she regained consciousness and began to breathe at a respiratory rate of 15 breaths/minute. Her blood pressure was 120/80 mmHg and pulse rate was 72 beats/minute. We continuously monitored her respiratory condition for one hour until full recovery from the symptoms. Thereafter, we informed the patient and her mother about the episode of severe hyperventilation attack and post-hyperventilation apnea during the dental treatment and recommended that she undergo further such treatments under monitored anesthesia, care with spontaneous breathing.

**Second episode (Figure 2)**

Several weeks later, the same patient was scheduled for dental treatment under monitored anesthesia care with spontaneous breathing plus regional anesthesia. We induced and maintained anesthesia for one hour with a target controlled infusion of propofol (TCI level = 1.1 ~ 1.2 μg/ml) after intravenous administration of 1 mg midazolam. At the end of treatment, propofol was discontinued and the patient was carefully observed by the anesthesiologist until return of consciousness. Although she regained consciousness 20 minutes after stopping the propofol infusion, hyperventilation with a respiratory rate of 40 breaths/minute occurred and continued for several minutes. Immediately after the hyperventilation subsided spontaneously, post-hyperventilation apnea with desaturation to 86% occurred in association with loss of consciousness. However, the patient regained consciousness within one minute without any therapeutic intervention. She was discharged after confirming full recovery from the unstable respiratory condition.
Third episode (Figure 3)
The patient was scheduled for the third dental treatment session under monitored anesthesia care with spontaneous breathing plus regional anesthesia. As with the previous anesthetic management, we induced without any premedication and maintained anesthesia with a TCI of propofol (TCI level = 1.2 ~ 1.5 μg/ml) for the fifty minute treatment period. At the end of the treatment, propofol was discontinued and the anesthesiologist carefully observed the patient until recovery of consciousness. She did not develop any symptoms of hyperventilation, even 10 minutes after regaining consciousness and was later discharged after an uneventful recovery period.

Discussion
It is known that some patients with hyperventilation syndrome develop post-hyperventilation apnea or hypoxia with cyanosis and loss of consciousness, resulting in severe complications, including death [7,8]. The pathogenesis of post-hyperventilation apnea has only been partly understood as being due to the activity of peripheral chemoreceptors that may contribute to the susceptibility to apnea.
had a low PaCO2 and high PaO2. In such a situation, the
after the hyperventilation diminished, the patient probably
during artificial respiration with bag-valve-mask, even
In our patient, because oxygen continued to be supplied
imbalance in the levels of both PaO2 and PaCO2 would
accumulated to reach the threshold to stimulate adequate
compensatory apnea reaction until sufficient carbon dioxide
have resulted in the peripheral chemoreceptors initiating a
suggested that apnea secondary to hyperventilation occurs
during both hypoxia and hyperoxia [2,5,9]. Ogawa et al.
supported that apnea secondary to hyperventilation occurs
because following the wash out of carbon dioxide from
the body during hyperventilation an extended period of
time is required for it to accumulate adequately enough to
reach the threshold required to stimulate breathing [10].
In our patient, because oxygen continued to be supplied
during artificial respiration with bag-valve-mask, even
after the hyperventilation diminished, the patient probably
had a low PaCO2 and high PaO2. In such a situation, the
imbalance in the levels of both PaO2 and PaCO2 would
have resulted in the peripheral chemoreceptors initiating a
compensatory apnea reaction until sufficient carbon dioxide
accumulated to reach the threshold to stimulate adequate
breathing. Although, usually, a vicarious arousal reaction to
the apnea can occur, in this case this arousal reaction was
probably inhibited by the sedative effect of the small dose of
midazolam used in first episode and second episodes.

It has also been suggested that administration of benzo-
diazepine drugs, such as diazepam or midazolam, may
contribute to increasing the threshold for respiratory
stimulation. In other words, reduction of respiratory con-
trol related to the sedative effects of drugs might decrease
the respiratory stimulant effect of arterial carbon dioxide
wash out [11]. They suggest that 5-hydroxytryptamine 1A
agonists can provide adequate anxiolysis in patients who
have frequent occurrence of post-hyperventilation apnea.
These studies indicate that the drugs used to manage and
treat patients with hyperventilation syndrome should be
carefully chosen, due to the risk of occurrence of post-
hyperventilation apnea. In our case, we used midazolam
followed by propofol, a short acting intravenous anesthetic
agent, during the second episode. However, when we did
not use midazolam in the third episode, there were
no symptoms of post-hyperventilation apnea after propo-
ofol anesthesia. We cannot deny that the small dose
of midazolam administered may have caused the post-
hyperventilation apnea due to its sedative effect. However,
it is difficult to believe that the effects of 1 mg administra-
tion of midazolam would remain active for the 1 hour
20 minutes before the emergence of post-hyperventilation
apnea. The exact influence of propofol on the occurrence
of post-hyperventilation apnea is not known. Previously,
Tomoka et al. reported that propofol was not effective in
preventing hyperventilation syndrome [12]. However, based
on the third episode in our patient, in which she regained
consciousness from propofol anesthesia without any symp-
toms of hyperventilation and/or post-hyperventilation apnea,
we believe that an adequate dose of propofol is useful
in patients who frequently experience hyperventilation
syndrome.

The timing of drug usage for sustained hyperventila-
tion in the case of drugs that influence the occurrence
of apnea also needs discussion. The duration for which
carbon dioxide rebreathing using a paper bag should be
attempted before further intervention should be deter-
minal. Treatment using a rebreathing bag for a prolonged
period may result in a decrease in oxygen content. In our
case, a cause of the desaturation (92%) might be related to
reducing the oxygen supply by inappropriate continuation
of rebreathing paper bag for a prolonged period of 15 mi-
utes before the arrival of our team. Callaham et al. re-
vealed that the paper bag rebreathing method seems to be a
potentially risky procedure, because death, severe hypoxia
and collapse have been reported to occur with paper bag re-
brreathing [7]. The need for sedative/anxiolytic drug therapy
in the early stages of hyperventilation should be considered,
even though sedative and anxiolytic agents have the poten-
tial to depress respiratory control. Another unanswered
question is the need for oxygen supplementation if desatur-
ation is observed during rebreathing therapy, and whether
the arterial PO2 level directly alters the compensatory re-
sponse to hyperventilation. Previous reports have suggested
that it might be desirable to provide a proper amount of
oxygen, because hypoxemia is detrimental for the brain and
hypoxemia during hypocapnia does not stimulate respir-
ation [1]. Our experience seems to indicate that respiratory
assistance using a bag and mask should be performed in
critical cases of post-hyperventilation apnea with hypoxemia
and loss of consciousness. It should be noted that most pa-
tients with post-hyperventilation apnea recover spontane-
ously in a short time without any treatment. Therefore, we
can wait for spontaneous recovery for some time without
the use of a bag-valve-mask under full monitoring of vital
signs related to cardio-respiratory function. However, as in-
dicated in a previous case report by Munemoto T et al. [1],
if prolonged post-hyperventilation apnea associated with se-
vere hypoxemia persists, as a precautionary measure it is
necessary to immediately perform respiratory assistance by
bag-valve-mask. Routine monitoring of SpO2 and other
vital signs, such as heart rate and blood pressure, should be
available for patients with hyperventilation syndrome.

**Conclusion**

We experienced a case of post-hyperventilation apnea
during dental treatment. Our experience suggests that
monitored anesthesia care with propofol may be effective
in the general management of patients with severe hyper-
ventilation attacks and post-hyperventilation apnea. This
case highlights the fact that proper emergency treatment
should be available for patients with hyperventilation syn-
drome who might develop post-hyperventilation apnea
associated with hypoxemia and loss of consciousness.

**Consent**

Written informed consent was obtained from the pa-
tient's parent for the publication of this case report and
accompanying images.
Competing interests
The authors declare that they have no competing interests.

Authors’ contributions
MK, KS, and TA all were involved in the management of this patient and the preparation of this case report. All authors read and approved the final manuscript.

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