Severe hypoxia following local anesthesia in a sedated patient: case report

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One of the most serious adverse reactions to benzodiazepines is their potential respiratory depressant effect on the subcortical levels of the central nervous system. When used in the accepted oral therapeutic dosage required for healthy patients, this drug does not usually produce any clinically significant respiratory depression and does not potentiate the depressant effects of opiates. In addition, benzodiazepines produce virtually no changes in cardiovascular function.[1] Ataxia and sedation develop only at doses above those required for anxiolytic effects. The reason for this is that the benzodiazepines depress the limbic system, which affects the emotions and behavior at much lower dosages than drugs that depress the reticular activating system and the cerebral cortex. This fact gives the benzodiazepines a very wide margin of safety between the therapeutic and toxic doses.[1]

The most frequently reported adverse reactions following oral administration of benzodiazepines for anxiety reduction include transient drowsiness, fatigue, and ataxia. Paradoxical reaction, though rare, may occur and is manifested by excitement, hallucinations, and rage. Discontinuation of drug administration will terminate these reactions.[1]

The aim of this report is to describe an unexpected adverse reaction to local anesthesia in a sedated patient during dental treatment in order to increase the awareness to the possible side effects associated with this situation.

Case report

A healthy, 12-year-old boy weighing 37 kg (25th percentile) was referred to the children's dental clinic for treatment because of lack of cooperation. His past medical history was unremarkable with no systemic diseases or any medication (ASA I), except for his dental problem. One week earlier, the child was treated in our department using 10 mg diazepam orally 1 h before treatment in conjunction with 40% N_2O/60% O_2 without negative consequences. A week later, 1 h before treatment, when his baseline vital signs were a pulse rate of 70 and 98% oxygen saturation, he received 10 mg diazepam (0.27 mg/kg) P.O. Inhalation of 45% N_2O/55% O_2 was started 5 min before treatment and the patient was connected to a pulse oximeter. The patient received a mandibular block injection (36 mg of lidocaine with 36 µg norepinephrine [0.002%]) following aspiration which was done as a routine procedure, and a rubber dam was placed. When cavity preparation (DO) on the left mandibular first molar commenced, the patient complained of pain. Therefore, a second cartridge (another 36 mg lidocaine and 36 µg norepinephrine) was added following aspiration, about 15 min after the first one. Immediately following the second injection, the patient complained of nausea and dizziness, and vomited. His pulse decreased from 70 to 47–52/min, and his oxygen saturation dropped from 97–98% to 75–87% intermittently. His pulse was very weak, and blood pressure could not be measured. His pupils were equal and responded to light, but he did not lose consciousness. Immediately, N_2O flow was stopped, 100% oxygen was delivered, and the patient was placed in the Trendelenburg position. When we noticed that his oxygen saturation had dropped because of the lowered rate and depth of his breaths, we were prepared to inject flumazenil to reverse the diazepam effect. The child objected to another injection. When he saw the syringe, the rate and depth of his breaths increased and immediately the saturation values returned to normal for a few minutes. During the entire episode, the patient was fully conscious with normal protective reflexes and his temperature was normal, but he was very pale. When oxygen saturation and breathing continued to be labile for 1 h, 25 min, emergency care was summoned. The patient's blood pressure was 120/80 mm Hg and his pulse had returned to 70, baseline values. The EKG done by the emergency care's staff showed sinus arrhythmia with bradycardia. The patient was transferred to a hospital for continued supervision. Three hours following the

beginning of this episode his pulse rose to 80 and his saturation and blood pressure spontaneously returned to normal ranges, and the patient was released.

For completion of dental care, the patient was referred to the hospital for general anesthesia, which was performed uneventfully a few weeks later. Blood tests (Complete blood count, PTand PTT), urine tests, and a physical examination performed a few days before general anesthesia were unremarkable and within normal ranges.

Discussion

We present a report of a 12-year-old boy who received diazepam (0.27 mg/kg) and 45% N_2O/55% O_2 for anxiety relief to facilitate dental treatment. After injection of 72 mg of lidocaine for local anesthesia, the patient developed bradycardia, his oxygen saturation fell to 75% (PO_2 = 40 mm Hg),[2] and his pulse was labile 47–52/min. The reason for this reaction is not completely clear. One possibility is that the combination of the sedative agents—diazepam and N_2O/O_2 in conjunction with two cartridges of local anesthesia—caused this adverse reaction.[3] The timing of the adverse reaction, immediately after the second injection, might suggest inadvertent injection into a blood vessel. This could have caused transient overdose although the total amount injected (72 mg) was far from the recommended limit of 4.4 mg/kg,[3,4] which would have been 163 mg for this patient.[3,4] The length of the reaction supports this possibility, as the recovery period lasted about 3 h, which is the amount of time required to metabolize lidocaine (half-life is 1 1/2 h).[4] The most likely explanation, therefore, is that overdose of lidocaine was caused by IV injection, and together with the diazepam administration in therapeutic dose, caused this severe hypoxemia.

The role of N_2O/O_2 in inducing this respiratory depression is probably negligible, as N_2O at therapeutic levels does not exert any respiratory depression of the central nervous system. In addition, because N_2O is not metabolized in the body, the gas is rapidly and virtually completely eliminated from the body within a brief period of time (3–5 min).[1] In our case, the patient recovered 3 h after the injection, although N_2O was stopped immediately after respiratory depression was observed. The only possible role of N_2O in this episode could be intensifying the changes induced by diazepam in respiratory rate and depth, which is more likely to result from its sedative relief of anxiety than its having a direct effect on the respiratory system.

Another possibility is that the patient developed a vaso-vagal reaction during the second injection, triggered by the edges of the rubber dam which was not removed completely or by the fear of the second injection.[5] This reaction is characterized by nausea or vomiting, pallor, perspiration, yawning, epigastric distress, hyperpnea, weakness, confusion, and pupillary dilation. There is initially tachycardia and decreased blood pressure. This is followed by bradycardia, pupillary constriction, and syncope. Removing the offending stimulus will restore consciousness, with recovery within a few minutes.[5,6] We observed bradypnea with no tachypnea in the beginning of the episode and no perspiration or pupillary dilatation, however, the recovery period was 2–3 h. These findings seem to at least partially negate this possibility.

The emergency care in this case was called after 1 h, 25 min. We did not call sooner, because it seemed that, during the period of observation, when the patient's labile pulse and O_2 saturation readings were noted, that he would stabilize and recover. Therefore, our decision to call for emergency care was repeatedly delayed. In retrospect, this decision should have been made much earlier.

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