Perioperative Management of a Patient With Idiopathic Pulmonary Hypertension and a History of Syncope: A Case Report

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Pulmonary hypertension is characterized by higher-than-normal pulmonary arterial pressures. This case report describes the perioperative management of a male patient with idiopathic pulmonary hypertension and a history of vasovagal syncope during previous dental extractions with local anesthesia. He underwent successful extraction of a single tooth with intravenous moderate sedation using dexmedetomidine and midazolam as well as prilocaine with felypressin for local anesthesia. There are many considerations surrounding the anesthetic management of patients with pulmonary hypertension, including the need to maintain systemic blood pressure, avoid hypoxemia and hypercapnia, and ensure adequate analgesia.

Key Words: Pulmonary hypertension; Idiopathic pulmonary hypertension; Dexmedetomidine.

Pulmonary hypertension (PH) is a condition wherein the pulmonary artery pressure (PAP) is abnormally high (normal: mean PAP <25 mm Hg). Key points in anesthetic management of patients with PH include maintaining systemic blood pressure (BP), avoiding hypoxemia and hypercapnia, and providing adequate analgesia.[1] An individualized preoperative risk assessment and treatment optimization along with advanced perioperative planning may improve anesthetic management outcomes for patients with PH.[2]

We present the case of a patient with idiopathic pulmonary arterial hypertension (IPAH) and a history of vasovagal syncope (VVS) scheduled to undergo tooth extraction under intravenous (IV) moderate sedation. The patient was effectively sedated using IV dexmedetomidine and midazolam. Written informed consent was obtained from the patient to share the details of this case report, and efforts were made to protect personal information and other ethical considerations.

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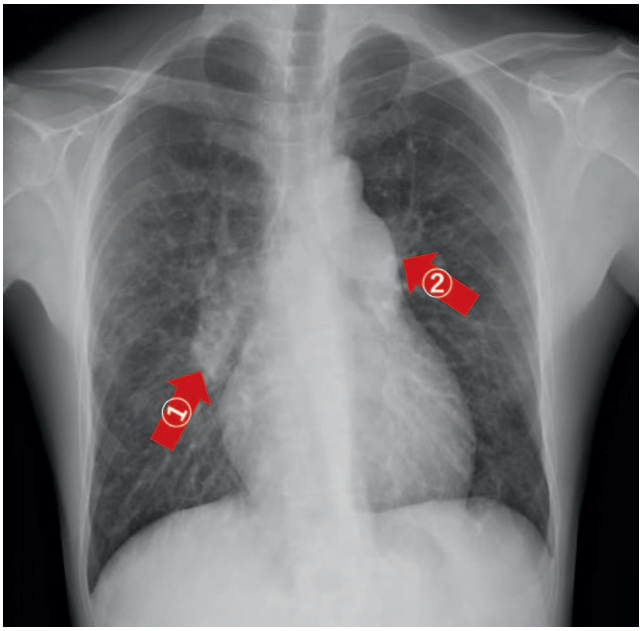
CASE PRESENTATION

A 47-year-old man (height 164 cm, weight 54 kg, body mass index 20.1 kg/m^2) was scheduled to undergo extraction of his right mandibular first molar under moderate sedation with IV dexmedetomidine and midazolam. His past medical history included IPAH and nonsustained ventricular tachycardia (NVT) starting in 2005 as well as a cerebral hemorrhage in 2018 with residual right upper extremity paresis. He also reported several episodes of VVS during previous dental extractions that were primarily associated with delivery of local anesthesia. His reported daily medications included riociguat 3 mg/d, ambrisentan 5 mg/d, and selexipag 0.4 mg/d for IPAH and mexiletine 300 mg/d for NVT. The patient denied any surgical history, notable family history, or allergies. His exercise tolerance approximated

Figure 1. Preoperative AP Chest Radiograph



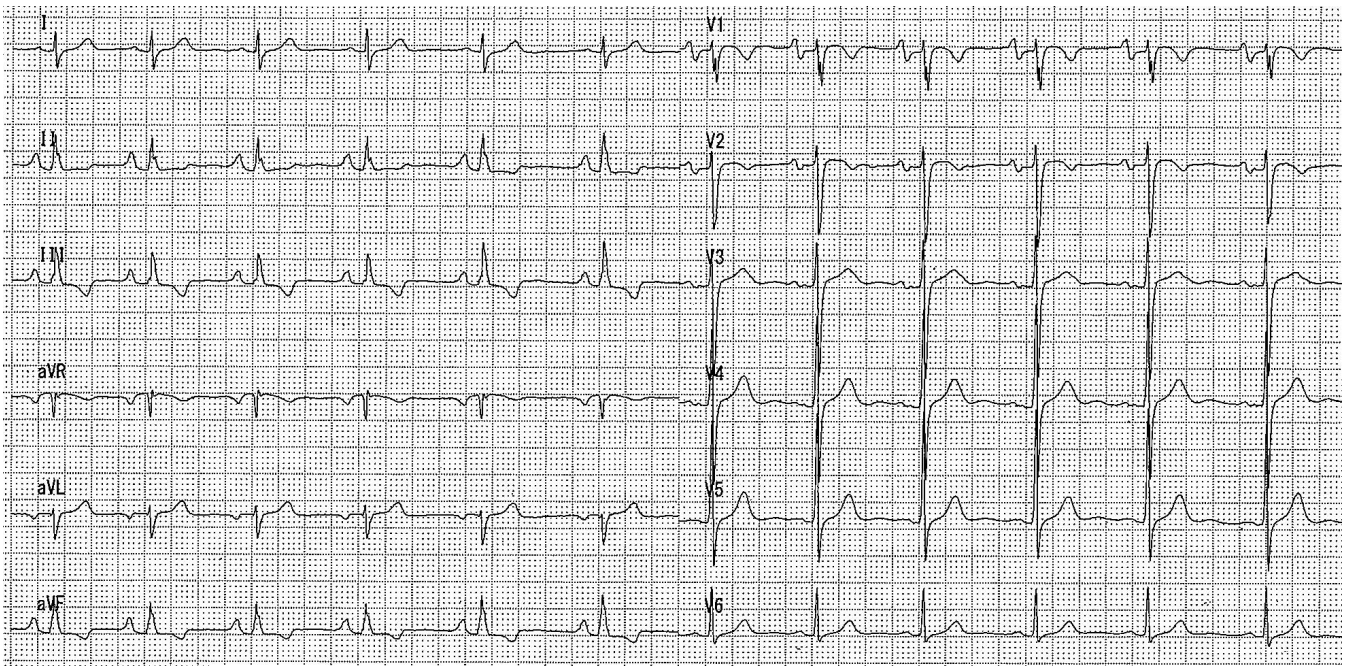
The primary trunk of the pulmonary artery is dilated. (1) Inferior branch of the right pulmonary artery. (2) Left main pulmonary artery.

3 metabolic equivalents, and he reported being able to ascend and descend stairs slowly.

Preoperative testing 2 weeks prior to surgery included echocardiography, an anterior posterior (AP) chest radiograph, a 12-lead electrocardiogram (ECG), and routine blood tests. Echocardiography indicated decreased right ventricular wall motion, main pulmonary artery widening, slightly decreased left ventricular diastolic function, and mild tricuspid valve narrowing. The estimated PAP was 37 mm Hg (normal: PAP <25 mm Hg), indicating mild PH. The chest film also showed dilatation of the main pulmonary artery (Figure 1), and the ECG showed nonspecific T-wave abnormalities and a right axis deviation (Figure 2). His blood tests were unremarkable. Consultation with the patient's cardiology team indicated that his PH was stable.

Although there was a possibility of an acute increase in PH, as indicated by syncope, hypotension, or cardiac arrest, which could result from perioperative stressors, the surgery proceeded as planned. Because of his past complex medical history and previous VVS episodes, a dental anesthesiologist was consulted to provide advanced anesthetic management, such as IV sedation or general anesthesia. The planned procedure was anticipated to be short (extraction of 1 tooth), and there was potential for large hemodynamic changes during general anesthesia, so the anesthetic plan was to perform IV moderate sedation. Dexmedetomidine and midazolam were selected as the anesthetic agents.

Figure 2. Preoperative 12-Lead Electrocardiogram



Electrocardiogram showed nonspecific T-wave abnormalities and a right axis deviation.

On the day of surgery, the patient presented having appropriately fasted and taken all his usual daily medications. Standard anesthesia monitors (eg, an ECG, noninvasive BP cuff, and pulse oximeter) were applied in the operating room. Capnometry via a nasal cannula was also used to help monitor the patient's ventilatory status. His baseline preoperative vital signs at that time were as follows: percutaneous oxygen saturation (SpO_2) 93% on room air, heart rate (HR) 65 beats per minute (bpm), and BP 96/60 mm Hg. Supplemental oxygen 3 L/min was then administered through the nasal cannula, and a 22-gauge IV catheter was placed in the left dorsal hand. An IV bolus of midazolam 1 mg was administered rather than a loading dose of dexmedetomidine. Approximately 3 minutes after the midazolam bolus, a continuous infusion of dexmedetomidine 0.4 μg/kg/h was started. The patient became sufficiently sedated roughly 15 minutes after starting the dexmedetomidine and had stable breathing. His intraoperative bispectral index was 70 to 75 and Ramsay sedation score was 2 to 3, all consistent with moderate sedation.

Because of the patient's history of NVT, we opted for a prilocaine preparation containing felypressin (Dental Citanest-Octapressin Cartridge, Dentsply Sirona Japan) for subperiosteal local infiltration. There were no changes in vital signs after local anesthesia, and his intraoperative HR (60 bpm), BP (90-100/50-60 mm Hg), end-tidal CO_2 (40-45 mm Hg), and SpO_2 (96% under 3 L/min oxygen) were all withing acceptable limits and remained stable throughout. The surgery was completed without any major issues and lasted for 15 minutes. The total anesthesia time was 30 minutes. The total doses of dexmedetomidine and midazolam were 10 μg and 1 mg respectively.

DISCUSSION

Patients with PH are classified into 5 clinical groups according to hemodynamic, etiologic, and pathologic characteristics[3]:

1. Pulmonary arterial hypertension (PAH), with the most common subgroups being IPAH, connective tissue disease–associated PAH, and congenital heart disease (mainly Eisenmenger syndrome).
2. PH associated with increased pulmonary capillary wedge pressure (postcapillary PH) due to left-sided heart disease.
3. Lung disease and chronic hypoxia-related PH.
4. Predominantly chronic thromboembolic PH.
5. PH caused by multisystemic disorders or multiple/unknown mechanisms, including sarcoidosis and hematological conditions.

PAH therapy has improved dramatically because of the introduction of several new therapeutic agents. The 5-year survival rate of PAH has improved from 40% before 2000 to 90% in recent years.[4,5] When managing anesthesia in patients with PAH, a critical goal is to ensure stability of the patient's cardiovascular and respiratory status perioperatively to prevent the onset of right heart failure. Although our patient did not have a severe case of PAH, perioperative mortality is 6% in severe cases, with a PAP of 46 mm Hg, and the presence of right heart failure, low exercise capacity, and emergency surgery further increase that risk.[6] It is important that patients with PAH undergoing surgery be treated at a facility (ie, hospital) specializing in PH whenever possible rather than an outpatient dental clinic or surgery center and that the best possible perioperative care be provided.[7]

Utilization of sedation or general anesthesia often depends heavily on a variety of factors. An advantage of general anesthesia is that ventilatory control is assured. Disadvantages include anesthetic-induced suppression of cardiac contractility, increased pulmonary vascular resistance, and the effects of positive-pressure ventilation on pulmonary circulation. Inhaled anesthetics in general and propofol in particular all markedly reduce right ventricular contractility. Nitrous oxide, isoflurane, and desflurane increase pulmonary vascular resistance.[7] Although opioids are often a mainstay of general anesthesia, they are less likely to be used in sedation because of their effects on ventilation. In our case, we decided to manage the patient with moderate sedation without opioids because of the anticipated short operative time and the cardiovascular effects of general anesthesia. Respiratory depression and insufficient analgesia can both worsen PH during sedation, and in this case specifically, we focused on maintaining systemic BP, avoiding hypoxemia and hypercapnia, and providing adequate analgesia while avoiding respiratory depression. Right atrial and ventricular pressures are usually elevated in patients with PH because of incremental increases in pulmonary vascular resistance. Consequently, decreased systemic vascular resistance may significantly reduce coronary blood flow and lead to right heart failure. Ultimately, strict cardiovascular/hemodynamic control including controlling BP and HR is always ideal, but in these cases, the goal is to avoid significant hypertension and hypotension. Hypotension is particularly worrisome because of its potential for negatively impacting perfusion of the right ventricle.

Midazolam, propofol, and dexmedetomidine are commonly used IV anesthetic agents for dental surgery.[8,9] Midazolam is considered to be relatively safe for use in patients in poor general health, as it has been reported that a 0.2 mg/kg dose in patients with an American Society of Anesthesiology physical status classification of 3 to 4 resulted in mild hypotension

and conversely increased cardiac index.[10] Propofol is a strong respiratory depressant and tends to cause dose-dependent decreases in cardiac output and systemic vascular resistance. Reports of bradyarrhythmias exist, presumably resulting from suppression of the sympathetic nervous system.[11] Dexmedetomidine has little impact on ventilation,[12] has analgesic effects,[13,14] and has many advantages, such as lowering delirium,[15] lowering shivering threshold,[16] neuroprotection,[18] and avoiding decreases in BP.[18]

After careful consideration, we decided to use dexmedetomidine in combination with midazolam. Because of cardiovascular considerations, we started the patient on the maintenance dose of dexmedetomidine 0.4 μg/kg/h rather than an initial loading dose of 6 μg/kg/h to reduce the chance of hemodynamic fluctuations (ie, hypertension followed by hypotension) upon administration of the initial loading dose. In this case, a small dose of midazolam, 0.2 mg/kg, was used to achieve adequate sedation relatively quickly without the use of a typical loading dose of dexmedetomidine, and no significant hemodynamic changes were observed.

Hypoxemia and hypercapnia should be avoided because both will lead to vasoconstriction of the pulmonary arteries and aggravate PH. To achieve these goals under IV moderate sedation, it is necessary to preserve adequate spontaneous ventilation and oxygenation. Dexmedetomidine has little effect on the respiratory drive, which is beneficial in this type of scenario as we want to preserve baseline pulmonary function as much as possible. Although midazolam can depress the respiratory drive and relax the muscles of the airway, leading to airway obstruction, these effects are typically seen in higher doses or when combining midazolam with other central nervous system depressants (opioids, propofol, etc).

Lidocaine with epinephrine is often used for local anesthesia in dentistry and oral and maxillofacial surgery to ensure adequate anesthetic effects. Because the patient had a history of NVT and because there were concerns that the increased sympathetic tone induced by epinephrine would increase pulmonary vascular resistance, we avoided epinephrine altogether. Consequently, prilocaine with felypressin was used as the local anesthetic, with small doses administered over time, and was well tolerated. It is possible that the analgesic effect of dexmedetomidine may have also played a minor role.

CONCLUSION

This case report describes the perioperative management of a patient with IPAH and a history of VVS who underwent IV moderate sedation for a tooth extraction. There are many considerations surrounding the anesthetic management of patients with PH. It is very important to maintain systemic BP, avoid hypoxemia and hypercapnia, and ensure adequate analgesia. Our patient was managed effectively with IV midazolam and dexmedetomidine along with local anesthesia using prilocaine with felypressin, allowing the tooth extraction to be performed without any problems.

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