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Unexpected Atrial Fibrillation During Tooth Extraction in a Sedated Elderly Patient

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A case is reported of unexpected atrial fibrillation in response to tooth extraction under intravenous sedation in a 70-yr-old patient with thoracic aneurysm of the aorta. Atrial fibrillation developed after the additional injection of a 2% solution of lidocaine containing 1:200,000 epinephrine. After 20 min, the arrhythmia disappeared spontaneously. The arrhythmia was associated with insufficient analgesia for tooth extraction, epinephrine in the local anesthetic, decreased blood pressure, and the presence of cardiovascular disease. Even when a low concentration of epinephrine is employed, caution should be paid to development of unexpected cardiovascular reactions in elderly patients with severe cardiovascular disease. We conclude that an electrocardiogram, blood pressure device, and pulse oximeter should be used in high-risk patients in order to prevent and detect potentially dangerous cardiovascular emergencies, even if dental treatment is scheduled under local anesthesia.

Circulatory abnormalities that sometimes occur during dental treatment include changes in blood pressure (BP), arrhythmias and myocardial ischemia.[1–8] In relation to arrhythmias, occasional or monofocal ventricular premature beats[1,2] and supraventricular premature beats are often observed on the electrocardiogram (ECG). Most cases of arrhythmia and myocardial ischemia are transient, and treatment for these conditions is seldom required. Occasionally, however, severe arrhythmias,[2,3] such as multifocal ventricular premature beats and paroxysmal supraventricular tachycardia, develop. Patients with cardiovascular disease appear to be especially prone to severe arrhythmias as a result of dental stress or local anesthesia.[9]

We report a patient with a thoracic aneurysm of the aorta who developed unexpected transient atrial fibrillation during tooth extraction under intravenous sedation.

CASE REPORT

A 70-yr-old man visited the gerodontic clinic with the chief complaint of ill-fitting dentures. Oral examination revealed the need to extract four teeth. At 60 yr of age, he was diagnosed as having hypertension, and he was given antihypertensive drugs. At the age of 65, he was diagnosed with a thoracic aneurysm of the aorta and has been taking 10 mg of nifedipine to prevent rupture of the aneurysm. Systolic BP has been well controlled (below 130 mm Hg) by administration of nifedipine. At 69 yr of age, he was admitted to the hospital for 3 days with a diagnosis of transient ischemic brain attack.

No neurologic deficits, such as unilateral paralysis, numbness, sensory deficit, diplopia, or dizziness, remained when the patient visited our clinic. In the preoperative examination, an incomplete right bundle branch block was observed on the ECG. Chest radiographs showed a widened aortomediastinal shadow. Arterial BP and heart rate (HR) were 120/70 mm Hg and 88 beats/min, respectively. In daily life the patient experienced shortness of breath, palpitation, and pericordial discomfort at work.

On the day of tooth extraction, after the patient had rested for 30 min in the waiting room, he was seated on the dental chair in a semisupine position, and an automated BP recording device, ECG, and pulse oximeter were applied. At this time his vital signs were stable: BP, 120/64 mm Hg; HR, 84 beats/min; oxygen saturation (SpO_2) 97%; and respiration rate (RR), 20 breaths/min.

An indwelling intravenous catheter was inserted into an antecubital vein, and then 3 mg of midazolam was in-

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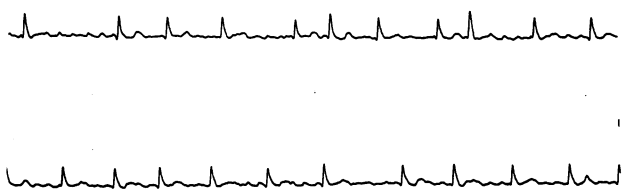
jected carefully while monitoring the patient's vital signs. After sedation was obtained, 3.6 mL of a 2% solution of lidocaine containing 1:200,000 epinephrine was injected around the left mandibular premolar and lateral incisor tooth and the left maxillary canine. At this time, the BP decreased to 110/60 mm Hg. Other vital signs were normal and stable: HR, 86 beats/min; RR, 20 breaths/min; and SpO_2, 96%. As soon as extraction of the left mandibular premolar tooth was started (5 min after local anesthesia), the patient complained of slight pain. The HR increased to 96 beats/min, and ventricular premature beats appeared on the ECG. The procedure was interrupted immediately. After 5 min the arrhythmia disappeared spontaneously. An additional 0.9 mL of the same lidocaine solution was then injected around the left mandibular premolar tooth. When one tooth had been extracted, atrial fibrillation developed (Figure 1). The BP decreased to 106/56 mm Hg, HR increased to 104 beats/min, RR was 16 breaths/min, and SpO_2 was 96%. At this time we decided to discontinue tooth extraction. Atrial fibrillation continued for 20 min. During this episode, the patient retained clear consciousness and had no complaint of pericardial discomfort, headache, nausea, or paresthesia of limbs. The BP was 100/54 mm Hg. The SpO_2 was normal and stable in spite of the tachycardia (96 to 104 beats/min) with atrial fibrillation. Although digitalis and an extracorporeal cardiac pacemaker were prepared for use, the arrhythmia disappeared spontaneously (with a concomitant reduction in HR) without employing these emergency measures. Figure 2 shows the waveform of the ECG 20 min after the development of atrial fibrillation, when it returned to the same waveform as shown by preoperative ECG. There were no complaints of palpitation, shortness of breath, nausea, or pericardial discomfort 90 min after the occurrence of atrial fibrillation. The BP was 122/60 mm Hg, and the HR 60 beats/min. He could walk by himself, and he was subsequently permitted to return home (3 hr after the episode).

DISCUSSION

Atrial fibrillation is a common disorder but of uncertain etiology. Both paroxysmal and persistent forms are recognized, the latter generally occurring in patients with

Figure 1. Atrial fibrillation during tooth extraction.



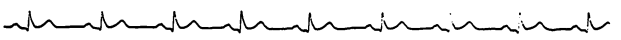


Figure 2. Electrocardiogram after spontaneous resolution of atrial fibrillation.

cardiovascular disease: congenital and acquired valvular disorders, hypertensive cardiovascular disease, atrial septal defects, various cardiomyopathies, congestive heart failure, and other disorders. Atrial fibrillation may also be a result of chronic lung disease and thyrotoxicosis. Enlarged atria are prone to fibrillation, which is why conditions resulting in cardiac hypertrophy and enlargement are commonly associated with the arrhythmia. Factors that can precipitate atrial fibrillation, even in patients without any recognizable form of heart disease, include emotional stress, heavy exertion, acute hypoxia or hypercarbia, metabolic derangements, drug overdose (eg, digitalis or alcohol), and fever.

As shown in Figure 1, atrial fibrillation is characterized by an irregularly irregular heart beat without discernible P waves. Although individuals with relatively healthy myocardiums often tolerate atrial fibrillation well, even on a chronic basis, patients with impaired cardiac function who require the assist of organized atrial beats to maintain adequate cardiac output may quickly decompensate, especially if an altered ventricular rate contributes to the problem. Immediate synchronized DC countershock is indicated in such cases. When cardioversion is not immediately necessary, other interventions may be attempted to control atrial fibrillation of sudden onset. Removal of an inciting stimulus, as discussed below, often proves effective. Initial pharmacotherapy involves agents that control the ventricular tachycardia often associated with the fibrillation. Digitalis glycosides (eg, digoxin), calcium-channel blockers (eg, diltiazem or verapamil), and β-adrenergic blockers (eg, esmolol or propranolol) may depress transmission of impulses through the atrioventricular node sufficiently to control ventricular tachycardia. Subsequent pharmacologic conversion to normal sinus rhythm may be attempted with type IA (eg, quinidine or IC (eg, flecainide) antiarrhythmic agents. In the uncommon patient who cannot be converted to a normal rhythm and who cannot tolerate chronic atrial fibrillation, ablation of the atrioventricular junction is performed, and a permanent ventricular pacemaker is implanted.

The causes or promotors of arrhythmia in dental treatment may be categorized as follows: nerve reflex,[3,5] psychosomatic stress from pain, anxiety, and fear,[9] vasoconstrictors contained in local anesthetics,[9,10] hypoxia, hypercapnia, and cardiac ischemia. In our patient, it is likely that several factors contributed to the atrial fibrillation: the pain of the tooth extraction, the epinephrine in the local anesthetic, the decreased BP, and the presence of cardiovascular disease.

Psychosomatic stress can produce effects on the heart

at least as severe as those from the epinephrine in local anesthetics. Pain, anxiety, and fear activate the sympathoadrenal system, leading to release of the catecholamines epinephrine and norepinephrine in amounts that enhance the excitability of the heart. In this case, insufficient analgesia is considered as one of causes of atrial fibrillation. Emotional stress can be excluded, however, because adequate sedation was obtained during treatment.

Vasoconstrictors contained in local anesthetics can generate arrhythmias by enhancing the excitability of the heart. Both endogenous and exogenous epinephrine exert potent β effects on the heart. These increase both the force and rate of contraction of the heart and also enhance the irritability of the cardiac muscle.[10]

Previous reports[11–15] have documented that the plasma epinephrine concentration is elevated to a peak of 2.4 to 15 times the baseline value at 3 to 8 min after the injection of local anesthetics containing epinephrine. In the present case, tooth extraction began 5 min after the injection of the local anesthetic. At this stage, the irritability of the cardiac muscle was probably enhanced by the exogenous catecholamine in the local anesthetic. Under situations such as this, it is likely that the additional release of endogenous catecholamine as a result of pain could participate in the development of atrial fibrillation. However, the measured hemodynamic changes were modest in our patient. The BP decreased from 120 to 110 mm Hg, while the HR increased only 12 beats/min (84 to 96) before atrial fibrillation occurred. This minimal hemodynamic effect may have been due to the sedation provided by the midazolam. Chernow et al,[12] Tolas et al,[13] and Gioffi et al,[15] reported that the plasma epinephrine concentration does not necessarily correlate with changes in BP or HR. Accordingly, we could not estimate the plasma epinephrine concentration nor predict the development of atrial fibrillation in this patient from the hemodynamic parameters studied.

A decreased coronary blood flow also could have contributed to the development of atrial fibrillation. Even a slightly decreased BP tends to reduce coronary blood flow in elderly patients with atherosclerotic cardiovascular disease.

Since patients with cardiovascular diseases such as hypertension, aneurysm, ischemic heart disease, valvular heart disease, arrhythmia, abnormalities of cardiac conduction, or cardiomyopathy have generally poor tolerance to catecholamines and psychosomatic stress, these patients are more prone to arrhythmia by the injection of a local anesthetic with vasoconstrictor[9,10] or by intraoperative stress. Our patient's health was not only complicated with aneurysm of the thoracic aorta, hypertension, and cerebrovascular disease, but he was also aged. Accordingly, we believe that he was especially prone to arrhythmia by local anesthesia and enhanced pain. Hypoxia and hypercapnia as causes of the atrial fibrillation could be excluded, since the SpO_2 and RR were within the normal range during treatment.

With regard to the use of epinephrine as a vasoconstrictor, some reports[16,17] have documented that epinephrine is safe in patients with cardiovascular disease if it is administered carefully. On the other hand, epinephrine possibly can induce dangerous arrhythmia, ST depression, and ventricular fibrillation in patients with severe hypertension, unstable arrhythmia, and advanced ischemic heart disease.[10,18] Hughes[18] found that older patients with cardiovascular disease had a greater incidence of arrhythmia than those of the same age with clinically normal cardiovascular systems when a 2% solution of lidocaine with 1:100,000 epinephrine was used.

In this case, 2% lidocaine with 1:200,000 epinephrine was given for the extraction of four teeth with long roots in order to provide a more potent anesthetic effect than could be obtained with prilocaine and felypressin. But even when a low concentration of epinephrine is employed, caution should be paid to development of unexpected cardiovascular reactions in elderly patients with severe cardiovascular disease.

From these findings, we conclude that the ECG, arterial BP, and oxygen saturation should be monitored in order to prevent and detect abnormal cardiovascular reactions in elderly patients with severe cardiovascular disease, even if dental treatment is scheduled under local anesthesia.

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