CASE REPORT

Alarming Reactions to Ketamine in Patients Taking Thyroid Medication—Treatment with Propranolol

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The administration of ketamine to patients may increase blood pressure and pulse rate. In some patients the increases are barely perceptible, while in others they can be alarming.[1] The mechanism producing the hypertension and tachycardia is unclear. There have been no reports suggesting a relationship between the level of thyroid activity and the cardiovascular response to ketamine. The following two cases illustrate striking increases of blood pressure and pulse rate after administration of ketamine to patients taking thyroid replacement medication. Treatment with a beta-adrenergic receptor blocker is described.

CASE REPORTS

Patient 1. A 33-year-old woman was admitted for removal of a cyst of the scalp. She had had a subtotal thyroidectomy for a "toxic goiter" six months previously. Since then, medications had been levothyroxine, 0.1 mg, and iothiouracil, 300 mg, daily. Physical examination revealed a clinically euthyroid patient weighing 135 pounds, blood pressure 125/85 mm Hg, pulse 85 beats/min, and normal heart and lungs. Preoperative laboratory values were: hemoglobin 14 gm/100 ml, normal leukocyte count, basal metabolic rate +6 per cent, T_4 level = 12 μg/100 ml (normal 5–13), and normal EKG.

The patient was premedicated with morphine, 10 mg, and atropine, 0.5 mg, intramuscularly an hour preoperatively. On arrival in the operating room, the patient was anxious, with blood pressure 140/80 mm Hg and pulse 110 beats/min. Anesthesia was induced with ketamine, 125 mg, given slowly intravenously. During the next five minutes, the blood pressure rose rapidly to 240/140 mm Hg and the pulse rate to 190 beats/min. The EKG showed a supraventricular tachycardia. Nasopharyngeal temperature remained 36.0 C. The patient was treated with 100 per cent oxygen and carotid sinus massage, which were unsuccessful in terminating the arrhythmia. Propranolol, 1 mg, was administered slowly intravenously. During the next five minutes, the blood pressure returned to 140/80 mm Hg and the pulse rate to 100 beats/min, and both remained at these levels. Twenty minutes after the initial dose of ketamine, the patient became inadequately anesthetized, and a second dose of ketamine, 25 mg, was given intravenously. This dose had no effect on the blood pressure or pulse, but did give adequate anesthesia. The procedure was completed uneventfully; there were no postoperative complications.

Patient 2. A 47-year-old woman was admitted for removal of a lipoma. In the past she had been hypothyroid, and she had been treated with levothyroxine, 0.2 mg, *t.i.d.*, for two and a half years. Thyroid function studies in the previous year were normal. The patient was also taking diphenylhydantoin and phenobarbital for a seizure disorder. Physical examination showed a clinically euthyroid individual weighing 140 pounds, with blood pressure 110/80 mm Hg and pulse 110 beats/min. The thyroid was of normal size; the skin was dry; no tremor was present; heart sounds were normal without murmurs or gallops; lungs were clear. Hemoglobin was 14.7 gm per cent, and EKG and chest x-ray were normal.

The patient received morphine, 10 mg, secobarbital, 150 mg, and atropine, 0.4 mg, intramuscularly, an hour preoperatively. On arrival in the operating room she was alert, with blood pressure 130/80 mm Hg and pulse 90 beats/min. Anesthesia was induced with ketamine, 150 mg, given slowly intravenously. Immediately, the blood pressure rose to 210/130 mm Hg and the pulse to 150 beats/min. The EKG showed supraventricular tachycardia. Propranolol, 1 mg, was administered slowly intravenously. The blood pressure decreased to 170/110 mm Hg and the pulse to 120 beats/min. Repeated doses of ketamine, 50 mg, intravenously did not change the blood pressure or heart rate. The procedure was completed uneventfully, and there were no postoperative complications.

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Discussion

Ketamine's side effects, hypertension and tachycardia, may be advantageous, but unfortunately their degree is not predictable. Our patients developed alarming reactions. The stress to the heart of this degree of hypertension and tachycardia must be considerable. These two patients were healthy and able to tolerate the extreme hypertension and tachycardia for short periods of time. Had they been patients with intrinsic heart disease, the outcome might not have been so favorable.

The mechanism leading to the hypertension and tachycardia has been studied in dogs by Traber and colleagues, using various blocking agents as premedicants.[2-5] They were able to 1) eliminate the hypertension and tachycardia with hexamethonium; 2) reduce the hypertension and eliminate the tachycardia with atropine; 3) eliminate the hypertension with phentolamine. However, they found that propranolol and practolol had no effect on the hypertension and tachycardia, and concluded that ketamine's effects were related to vagal blockade and alpha-adrenergic receptor stimulation. In contrast, Chen [6] showed that pretreatment of dogs with the beta-adrenergic blocker, pronethalol, partially and sometimes completely suppressed hypertension due to ketamine. Our case reports suggest that an interaction between thyroid hormones and ketamine may in part produce the marked hypertension and tachycardia. We selected propranolol since it has been recommended for the treatment of supraventricular tachycardias [7] and hyperthyroidism.[8]

Summary and Conclusions

Two cases of patients on thyroid replacement therapy who developed severe hypertension and tachycardia following the administration of ketamine are reported. Both patients were successfully treated with propranolol. We believe that ketamine should be used with caution in patients who are either hyperthyroid or taking thyroid-replacement medication. If an adverse reaction should occur, beta-adrenergic blockade may prove helpful.

References

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Paraphernalia

PARADOXICAL CATHETER EMBOLISM This is a case report of a patient in whom a portion of an inlying catheter was sheared off in the femoral vein. X-rays failed to show the fragment in the groin or the chest. The patient died of a burn injury, with pulmonary edema. Autopsy disclosed a recent pyramid-shaped infarct in the kidney, secondary to an embolus in a branch of the renal artery which contained a 1.5-cm segment of catheter. There had been no hematuria or alteration of BUN or creatinine. Examination of the heart revealed a patent foramen ovale. Since 20 per cent of patients have a patent foramen ovale, a catheter fragment may cause a paradoxical embolism to the arterial side of the circulation. (*Nash, G., and Moylan, J. S.: Paradoxical Catheter Embolism, Arch. Surg. 102: 213 (Mar.) 1971.*) Editor's comment: A possible stroke should be of greater concern than the renal infarct.