Case Report

Acute baroreflex-mediated hemodynamic instability during thyroid surgery: A case report

ABSTRACT

During thyroid surgery, intraoperative hemodynamic instability can be attributed to episodic surges of thyroid hormones. Thyroid storms are emergency situations characterized by persistent hypertension, tachycardia, hyperthermia, and end-organ damage. However, baroreflex-mediated neurogenic phenomena due to surgical procedures near the carotid sinus are a likely cause of acute hemodynamic changes during thyroid surgery. We describe a case of sudden hemodynamic instability occurring after thyroid manipulation during thyroidectomy in a 61-year-old man who presented with a 6-cm-sized thyroid mass in a euthyroid state.

Key words: Acute; baroreflex; thyroid surgery

Introduction

Airway management is important during thyroid surgery because the trachea can be deviated or compressed due to large goiters or thyroid masses.[1] More attention is also needed in relation to hemodynamic instability caused by thyroid hormone–secreting tumors or hyperthyroidism. A thyroid storm is an acute, severe hypermetabolic state caused by an excessive release of thyroid hormones or increased adrenal activities.[2] However, we present a case of unexpected hemodynamic instability during thyroid manipulation in a patient in a euthyroid state.

Case Report

A 61-year-old man (172 cm, 64 kg) with no underlying disease was scheduled to undergo right thyroidectomy for a 6-cm-sized thyroid mass. This mass was a nonfunctional tumor, so no medication was needed. On preoperative evaluation, an electrocardiogram (ECG) indicated normal sinus rhythm, and a laboratory evaluation including a thyroid function test (TFT) revealed normal findings. On chest X-ray, the lungs were essentially clear, but there was significant left-sided displacement of the trachea at level T1–2 due to a large thyroid mass.

In an operating room, after placement of routine monitoring, he was sedated with propofol (70 mg) and infusion of remifentanil at a rate of 0.15 μg/kg/min. Rocuronium (50 mg) was administered to achieve muscle relaxation for intubation. On laryngoscopic view, the vocal cord was deviated to the left side, as confirmed by chest X-ray at preanesthetic evaluation. The trachea was deviated, but not narrowed, so there was no difficulty during intubation when using a 7.5-mm endotracheal tube. Anesthesia was

Access this article online	
Website: www.saudija.org	Quick Response Code
DOI: 10.4103/sja.SJA_76_19	

How to cite this article: Choi EK, Kwon H, Park S. Acute baroreflex-mediated hemodynamic instability during thyroid surgery: A case report. Saudi J Anaesth 2019;13:368-70.

Eun Kyung Choi, Hyojin Kwon, Suyong Park

Department of Anesthesiology and Pain Medicine, Yeungnam University College of Medicine, Daegu, Korea

Address for correspondence: Dr. Eun Kyung Choi, Department of Anesthesiology and Pain Medicine, Yeungnam University College of Medicine, 170, Hyeonchung-ro, Nam-gu, Daegu, Republic of Korea. E-mail: zzini0527@naver.com

maintained with sevoflurane and an oxygen–air mixture. Fifteen minutes after initiating the surgery, his blood pressure suddenly decreased to 70/50 mmHg. Intravenous ephedrine and phenylephrine were administered, but no effect was observed. The same doses of vasoactive drugs were injected, and his blood pressure only improved to 90/60 mmHg and then decreased again. Repeated doses of ephedrine and phenylephrine showed no effect, so we asked the surgeon to discontinue the surgery, and then initiated an infusion of dopamine at a rate of 5 μg/kg/min and phenylephrine at 0.5 μg/kg/min. An arterial cannulation was placed in the left radial artery and a 16-gauge peripheral line was added in the right foot. His blood pressure gradually increased to 150/80 mmHg. There was no sign of multiorgan failure. The saturation was 100%, and there was no change in the ECG. After stabilization of blood pressure, the surgery was resumed. The surgeon manipulated the superior pole of the thyroid gland, and the blood pressure dropped again, while the heart rate increased. After removing the thyroid gland, the patient's vital signs stabilized during the surgery, and we could complete the surgery without encountering any other problems.

He remained hemodynamically stable in the postanesthesia care unit. Postoperative laboratory findings including troponin I and TFT results were within normal limits. On the day after surgery, a T-wave abnormality was newly found on the ECG, but the echocardiogram was normal with an ejection fraction of 56% and no regional wall motion abnormality. Five days after the surgery, the patient was discharged without any other problems. One month later, a myocardial perfusion imaging test showed no evidence of ischemia. Histological diagnosis revealed a thyroid mass as a follicular carcinoma, but no evidence of catecholamine-secreting tissues.

Discussion

This patient showed intraoperative hemodynamic instability during manipulation of the thyroid mass, which is not normally associated with thyroidectomy, except in cases of thyroid storms. These sudden hemodynamic changes during thyroid manipulation can be explained by baroreflex of carotid baroreceptors due to the large size of the thyroid mass.

Thyroid surgery is performed for thyroid cancer, solitary thyroid nodules, and autoimmune thyroid disease. Anesthesia for thyroid surgery may be complicated by difficult airways due to an enlarged thyroid mass, hemodynamic fluctuations due to thyroid hormone secretion, and recurrent laryngeal nerve injury followed by vocal cord dysfunction.[3] Possible complications after thyroid surgery are hematoma, tracheomalacia, laryngeal edema, hypocalcemia, postoperative pain, and postoperative nausea and vomiting.[3] However, in general, thyroid surgery is considered safe and is performed successfully with no major problems.

In terms of acute hemodynamic alterations during thyroid surgery, careful attention should be paid to thyroid storms by anesthesiologists. These can occur in patients with uncontrolled hyperthyroidism by a trigger such as surgery. They show features of tachycardia, hypertension, palpitation, and hyperthermia.[4] In particular, an acute thyroid crisis at the time of anesthetic induction may be mistakenly diagnosed as malignant hyperthermia. In both cases (thyroid storm and malignant hyperthermia), an increase in blood pressure is the main phenomenon, but in this case, the intraoperative blood pressure suddenly dropped with no response to inotrophic (ephedrine) or vasoactive (phenylephrine) agents. There was no evidence to indicate an endocrine surge because the patient was in a euthyroid state, and there was no evidence of a tumor that secreted catecholamines histologically.

There are several possible causes of intraoperative hemodynamic instability, except thyroid hormone surges such as thyroid storms. The first is supposedly baroreflex-mediated hemodynamic instability caused by surgical manipulation during thyroidectomy. In this case, the mass was too large, and the carotid sinus was depressed and stimulated when the surgeon touched the superior pole of the thyroid gland. Therefore, we asked the surgeon to discontinue the surgery; a few minutes later, the blood pressure returned to within a normal range. The second cause is supposedly acute heart failure or thromboembolism. In thyrotoxicosis, catastrophic events such as dysrhythmia, thromboembolism, and heart failure can occur.[4] However, these events rarely occur in a euthyroid state.[5] Although there was a low risk of thyrotoxicosis in this case, a transesophageal echocardiogram (TEE) was prepared in case of emergency. However, we did not perform a TEE at this time point because the hemodynamic instability was resolved over a short timescale of minutes after temporary discontinuation of thyroid manipulation, and probe insertion of TEE was difficult due to hyperextension of the neck for surgery.

The baroreflex is a short-term mechanism to maintain adequate blood pressure by regulating the sympathetic vasomotor tone. It controls the heart rate, contractility, and peripheral resistance in response to nerve impulses entering the baroreceptors.[6] Baroreceptors are mechanoreceptor

sensory neurons located in the aortic arch and carotid sinus.[7] They sense a stretch in the arterial wall when the pressure changes.[8] The signals from the baroreceptors will eventually reach the vasomotor center in the brain. The vasomotor center will modulate blood pressure by suppressing sympathetic excitement and increasing the vagal tone on the sinoatrial node of the heart. In this case, the temporary pressure increase on the carotid sinus by surgical manipulation may act on the baroreceptor reflex, which appears to acutely lower blood pressure. In contrast, there was a case of a huge thyroid goiter that chronically depressed the carotid sinus.[2] In that case, the baroreflex adapted to chronic changes by changing the sensitivity with intrinsically normal baroreceptors and set point, and surgical decompression of the carotid sinus led to acute rebound baroreceptor dysfunction, which showed a great increase in blood pressure.[2]

Before thyroid surgery, evaluation of the size and location of the thyroid mass as well as evaluation of thyroid function are important. If surgical procedures near the carotid sinus due to a large thyroid mass and contiguity of the carotid sinus are expected, intraoperative hemodynamic fluctuations caused by the baroreflex should be anticipated, and prophylactic ipsilateral carotid sinus block may be necessary in cases such as those of carotid endarterectomy or neck dissection surgery.[9] In addition, the administration of prophylactic atropine or glycopyrrolate decreases the incidence of intraoperative bradycardia and hypotension.

In conclusion, not only preoperative assessment of thyroid masses but also prompt intraoperative detection and management of hemodynamic instability by the carotid baroreflex should receive particular care in patients who have undergone thyroid surgery.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Alfonso AE, Christoudias G, Amaruddin Q, Herbsmans H, Gardner B. Tracheal or esophageal compression due to benign thyroid disease. Am J Surg 1981;142:350-4.
2. Nouraei SA, Davies MR, Obholzer R, Sandhu GS, Porter F. Haemodynamic instability during thyroid surgery: A baroreflex-mediated neurogenic phenomenon? Anaesthesia 2006;61:282-4.
3. Bacuzzi A, Dionigi G, Del Bosco A, Cantone G, Sansone T, Di Losa E, *et al*. Anaesthesia for thyroid surgery: Perioperative management. Int J Surg 2008;6:S82-5.
4. Idrose AM. Acute and emergency care for thyrotoxicosis and thyroid storm. Acute Med Surg 2015;2:147-57.
5. Osman F, Franklyn JA, Holder RL, Sheppard MC, Gammage MD. Cardiovascular manifestations of hyperthyroidism before and after antithyroid therapy: A matched case–Control study. J Am Coll Cardiol 2007;49:71-81.
6. Eckberg DL, Sleight P. Human Baroreflexes in Health and Disease. Oxford: Clarendon Press; 1992. p. 327-45.
7. Barazanji MW, Cornish KG. Carotid and aortic baroreceptor control of heart rate in conscious monkey. Am J Physiol 1987;253:811-7.
8. Cowley AW, Liard JF, Guyton AC. Role of the baroreceptor reflex in daily control of arterial blood pressure and other variables in dogs. Circ Res 1973;32:564-76.
9. Al-Rawi PG, Sigaudo-Roussel D, Gaunt ME. Effect of lignocaine injection in carotid sinus on baroreceptor sensitivity during carotid endarterectomy. J Vasc Surg 2004;39:1288-94.