

Acute airway obstruction due to spontaneous intrathyroid hemorrhage precipitated by anticoagulation therapy

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Abstract

Acute airway compromise due to hemorrhage in of thyroid gland is a rare life-threatening condition. The increasing use of anticoagulants for various reasons is likely increased the occurrence of this this complication. We describe an elderly patient on anticoagulation for atrial fibrillation, which developed swelling on the right side of neck causing acute airway obstruction requiring emergency intubation for airway protection. Computed tomographic scan showed massive intrathyroid hemorrhage along with substernal extension. She had supratherapeutic INR which was appropriately corrected emergently. She underwent resection of the thyroid gland which showed multinodular goiter without any evidence of malignancy. Our case illustrates the rare but lethal bleeding complication of anticoagulants in critical anatomical area and we request physicians should be wary of similar conditions.

Keywords: Airway obstruction, anticoagulation, hyperthyroidism, substernal goitre, thyroid hemorrhage

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Introduction

Bleeding in vital areas of the body such as the neck can be life-threatening due to a sudden compromise in breathing. Despite being highly vascularized, thyroid hemorrhage leading to airway compromise is uncommon and occurs following trauma, fine-needle aspiration and malignancy.^[1] Spontaneous bleeding due to concomitant use of anticoagulants is very rare, and only few case reports have been published. Preexisting benign conditions such as goiter, which occurs in 4% of adults will lead to increase vascularity of the gland and can increase the risk of bleeding. Patients on anticoagulants therapy have been increasing, and approximately 4.2 million Americans are on these medications currently.^[2] The risk of bleeding due to goiter induced increased capillarity precipitated by increased use of anticoagulants can cause massive hemorrhage and

sudden unexpected airway compromise. We present a case of spontaneous thyroid hemorrhage that was timely recognized and treated.

Case Report

The patient is a 73-year-old white female with a history of atrial fibrillation on warfarin therapy presented with complaints of fever, burning micturition, and abdominal pain. She was admitted at a community hospital for the treatment of complicated urinary tract infection. She was started on levofloxacin, intravenous fluids and continued on warfarin. After 3 days, she started having difficulty in breathing. Chest X-ray showed prominent pulmonary vasculature, and she was treated for congestive heart failure. The next day, she developed some difficulty in swallowing and underwent bedside swallow study, which showed oropharyngeal dysphagia. By the evening, she developed severe neck pain and noticed sudden swelling on the right side of her neck. In view of worsening respiratory distress, urgent computed tomographic of the neck was done, which showed hemorrhage in the thyroid gland with retrosternal extension along with significant deviation and compression of the trachea [Figure 1]. Emergent

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intubation with Fr 6 endotracheal tube was done and was transferred to our institute for further management.

On the presentation, her INR had increased to three, immediately corrected with four units of fresh frozen plasma and was continued on mechanical ventilation with sedation protocol. She was seen by ENT services and underwent biopsy of the thyroid gland to rule out any underlying malignancy. Even with anticoagulant therapy, spontaneous hemorrhage is considered rare, and the presence of underlying malignancy would definitely change her management. Her biopsy specimen showed nodular goiter with bleeding and no evidence of malignancy. Later, she underwent median sternotomy and excision of retrosternal goiter with combined collaboration of ENT and thoracic surgeons. During surgery, the thyroid gland was observed as extending up to the level of the innominate vein.

She continued to be febrile, tachycardic, and diaphoretic with elevated T4 levels, which was indicative of hyperthyroidism [Table 1]. Because she was scheduled for elective major surgery, antithyroid medications were initiated for control of increase sympathetic activity. As expected, following surgery she developed hypothyroidism and was started on thyroid supplements. She was discharged in a stable condition to the sub-acute rehabilitation facility and is presently doing well at the time of this writing.

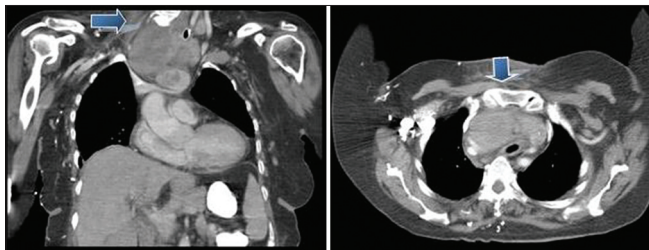


Figure 1: Axial and coronal computed tomographic scan image showing intrathyroidal hemorrhage causing compression and deviation of the trachea (blue arrows)

Table 1: Levels of thyroid hormones before and after the surgery showing features of hyper and hypo thyroidism respectively

| Hormones | Serum levels | Before surgery | After surgery |
|---------------|------------------|----------------|---------------|
| TSH | 0.34-5.60 uIU/ml | 0.43 | 21.79 |
| T3 | 0.7-2.0 ng/ml | 1.2 | 0.6 |
| Free T4 | 0.58-1.64 ng/dl | 3.21 | 0.62 |
| Free T3 | 2.5-3.9 pg/ml | 3.3 | - |
| Thyroglobulin | 1.6-50.0 ng/ml | 3135.9 | 0.8 |

TSH: Thyroid stimulating hormone

Discussion

Thyroid is one of the most highly vascularized organs of the body; however, spontaneous thyroid hemorrhage has been rarely reported only as case reports following trauma or fine-needle aspiration cytology and spontaneous bleeding with or without the use of antihemostatic agents.^[1,3-12] Pre-existing conditions like thyroid malignancy or colloid goiter can increase the risk of bleeding due to increased and altered vascularity. The source of bleeding is usually venous due to rupture of vessels with deficient adventitia, musculature, and elastic tissue.^[13] The capsule is highly vascular and has many anastomotic channels with the penetrating vessels supplying core of the nodule. Physical exertion, coughing, straining, or any valsalva maneuver that increases venous pressure can cause rupture of these vessels leading to spontaneous bleeding.^[13]

Although the use of iodized salt has reduced the incidence of goiter in the United States, up to 25% of the population continues to develop thyroid enlargement.^[14] With the increase in the population size of the elderly, the incidence of atrial fibrillation and the use of anticoagulants are rising, and at present, 4.2 million individuals are on anticoagulation medications.^[2,15] In large clinical trials, bleeding rates due to the use of warfarin are 18.15%, 14.5%, and 25.8%, with major bleeding constituting 3.36%, 3.4%, and 3.4%, respectively.^[16-18] In the real world, however the incidence of bleeding is lower, at approximately 9.6% of the total bleeding rate, which is also supported by postmarketing analysis of these major trials.^[19] As thyroid hemorrhage is not captured in any of these trials, the true incidence of thyroid hemorrhage is not known.^[20] To date, only two cases of thyroid hemorrhage due to warfarin have been described in the literature.^[1,8] With more than 5% of the population with goiter on warfarin, these two cases, along with our case, might only be representing the tip of the iceberg.

Hemorrhage to the thyroid gland can cause sudden compression of the trachea and if more than two-third of the lumen is compressed than it can lead to sudden upper airway obstruction warranting emergency intubation. Furthermore, if the bleeding is not controlled; the patient will need emergency thyroidectomy.^[21] Bleeding into the gland can cause a sudden release of preformed thyroid hormones leading to acute hyperthyroidism.^[9,22-24, 25] This rare cause of destructive hyperthyroidism is usually self-limiting and does not require any treatment. However, most patients require emergency thyroidectomy and eventually develop hypothyroidism.

Our report describes the third case of thyroid hemorrhage in the literature and reiterates the risk of spontaneous intrathyroid hemorrhage and the potential life-threatening complication due to airway compromise in patients taking warfarin. Early recognition and timely intervention could be lifesaving.

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