

Case series: two cases of life threatening dynamic airway obstruction from thyroid mass

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Abstract

We present 2 cases of dynamic airway obstruction with respiratory failure leading to cardiac arrest. Both have significant aetiology of cystic thyroid mass with sudden haemorrhagic changes leading to airway obstruction. We discuss the plausible pathophysiology leading to cardiopul-

monary compromise where tracheobronchomalacia (TBM) and excessive dynamic airway collapse (EDAC) play a significant role. We note that this is under recognised and can be present as a life-threatening event. Recognition of these pathophysiology process facilitate surgical management of thyroid obstructive airway disease.

Key words: Dynamic, airway, compression, collapse, thyroid, tracheobronchomalacia, excessive dynamic airway collapse.

Case 1

A 68-year-old Chinese woman with a history of thyroid mass was brought in by ambulance after sustaining out-of-hospital cardiac arrest. She had been having an asymptomatic thyroid mass for the last one year and had the thyroid mass investigated with ultrasound the same day. A few hours after ultrasound, she felt asphyxiated and lost consciousness within 3 minutes whilst holding her neck between thumb and index fingers. After that, she went into cardiac arrest. CPR was commenced by a bystander and laryngeal mask airway (LMA) was inserted on arrival of paramedics. Return of spontaneous circulation (ROSC) was achieved approximately after 8 minutes of cardiac arrest. Subsequently she was transferred to emergency department (ED).

She was intubated in ED and transferred to Intensive Care Unit (ICU) for further management. Chest X-ray (**Figure 1**) showed pulmonary oedema with unremarkable electrocardiogram (ECG) and bedside echocardiography findings. Blood gas showed normoxia and normocarbida with mildly elevated

lactate consistent with post cardiac arrest picture. There was a suspicion of airway obstruction leading to a computed tomography (CT) of neck and larynx (**Figure 2**). A thyroid cystic structure (2.5x5.5x5 cm³) with suspicious haemorrhagic content was discovered, however there was no retrosternal extension or airway compression as suspected clinically. She recovered without any neurological sequelae and underwent a trial of extubation under close monitoring with otolaryngologist standby in view of the thyroid mass. The otolaryngology team performed naso-endoscopy post-extubation demonstrating a patent airway with mobile bilateral vocal cords. Within 5 minutes of extubation in ICU, she developed significant tracheal retraction with audible stridor and desaturation, leading to re-intubation.

Patient subsequently had a total thyroidectomy performed in view of life threatening airway obstruction. Operative findings showed tracheomalacia with enlarged thyroid mass with haemorrhagic cystic changes. Patient had a total thyroidectomy with tracheostomy and recovered well post operatively.

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Case 2

A 70-year-old Chinese gentleman with history of hypertension and bilateral glaucoma presented acutely shortness of breath to ED 6 hours after he had his dinner. On presentation he was diaphoretic, dyspnoeic, and hypertensive. He was able to speak in short phrases but quickly become drowsy and desaturated afterwards. Blood pressure was 230/148, respiratory rate was 40, and he has desaturation to 64% on non-rebreather mask pre-intubation. His arterial blood gas showed severe hypercapnic respira-

tory failure (pCO₂ 90 mmHg) and he was promptly intubated in ED with rapid sequence induction, averting a cardiac arrest.

After intubation, patient was sedated and ventilated with stable hemodynamics. His chest X-ray (**Figure 3**) showed bilateral infiltrates likely secondary to negative pressure pulmonary oedema. His ECG showed sinus tachycardia, left ventricular hypertrophy with no ischaemic changes. A palpable thyroid mass prompted a CT neck, which showed a large cystic mass (9.2x4.4x6.4 cm) replacing the left lobe of thyroid, causing deviation and partial effacement of the trachea (**Figures 4, 5 and 6**). He had a fine needle aspiration of his thyroid mass which showed fluid containing red blood cells confirming a cystic thyroid mass which has ruptured. He was transferred to cardiothoracic service where a left hemithyroidectomy was performed. On histopathology the thyroid mass was observed to be cystic in nature and had hyperplastic nodules with no evidence of malignancy. He recovered well after that.

Discussion

We present two cases of thyroid cystic nodule haemorrhage leading to airway compression and significant enough to cause dynamic airway obstruction. We have made deduction of pathophysiology leading to cardio-respiratory compromise.

Classically, thyroid mass is known to cause intra and extra-thoracic obstruction flow pattern (**Figures 7A and B**). These flow-volume loops may be over simplistic when assessing patients with compression from significant thyroid mass. A large thyroid mass can traverse the thoracic inlet, leading to variable intra-extra-thoracic compression of trachea, causing mixed inspiratory-expiratory flow restriction on volume loop (**Figure 8**). Coupled with element of dynamic airway obstruction, the air flow may approach zero.

In case 1, surgical finding of tracheomalacia points toward a predominant inspiratory flow restriction pathology. In case 2 the retrosternal extension of haemorrhagic thyroid cyst was likely to cause extra-thoracic and intra-thoracic airway compression likely leading to both expiratory and inspiratory flow limitation. The evidence of negative pressure pulmonary oedema strongly suggests likelihood of inspiratory flow limitation.

Both cases responded and recovered quickly post intubation with positive pressure ventilation. We postulate two mechanisms which has ameliorated airway collapse after intubation:

1. Endotracheal tube bypasses the section of the airway which suffered from external compression and excessive dynamic airway collapse

(EDAC).

2. Continuous positive airway pressure acts as a pneumatic stent and keeps the affected airway open.

In case 1 dynamic airway obstruction quickly manifested after extubation. We believe tracheobronchomalacia (TBM) and EDAC were the mechanisms contributing to dynamic airway obstruction. TBM and EDAC are both dynamic forms of large airway obstruction characterized by a decrease of $\geq 50\%$ in the cross-sectional area of the tracheobronchial lumen. (1,2) While TBM is defined as weakening of the tracheobronchial cartilage (**Figures 9C and 9D**), EDAC occurs when the posterior membrane bulges into the airway lumen during expiration, as demonstrated in **Figure 9B**. (3,4)

These two entities are common and has been described in various literatures. (4-7) The preferred mode of diagnosing EDAC and TBM are bronchoscopy (gold standard) and dynamic CT thorax. (5,7) Tracheal collapsibility does vary widely among patients with unknown significance. Bruno and Mariotta (2014) have described up to 23% of patients who underwent bronchoscopy for various reason had TBM or EDAC; however, majority of these patients remain asymptomatic. (1,8) Nevertheless, thyroid mass rupture or haemorrhage leading to airway obstruction has been reported in some case reports and similar mechanism may have led to dynamic obstruction. (9,10) Besides thyroid mass compression, there are various other causes of TBM and EDAC, described comprehensively by Kalra et al (2011). (4)

In both cases, our patients have remained asymptomatic despite having thyroid masses before the cysts haemorrhaged. Chronic compression may have contributed to weakening of airway structures without significant airway compromise. When haemorrhage occurred and compressed on trachea, the lumen collapsed each time during inspiration due to presence of high airflow causing Bernoulli effect. (1) This eventually leads to asphyxiation, respiratory fatigue and finally respiratory arrest. Despite the severity of presentation, the histopathology of both thyroid specimens were benign cysts with haemorrhage.

Conclusion

Thyroid mass with haemorrhage poses significant risk of upper airway compromise despite not histologically malignant in nature. Often there is an interplay of acute airway narrowing, TBM and EDAC. Diagnosis of TBM or EDAC can be pursued with bronchoscopy or multiple detector CT thorax. Timely recognition of these entities will ensure prompt management and survival.

Figure 1. CXR - post intubation

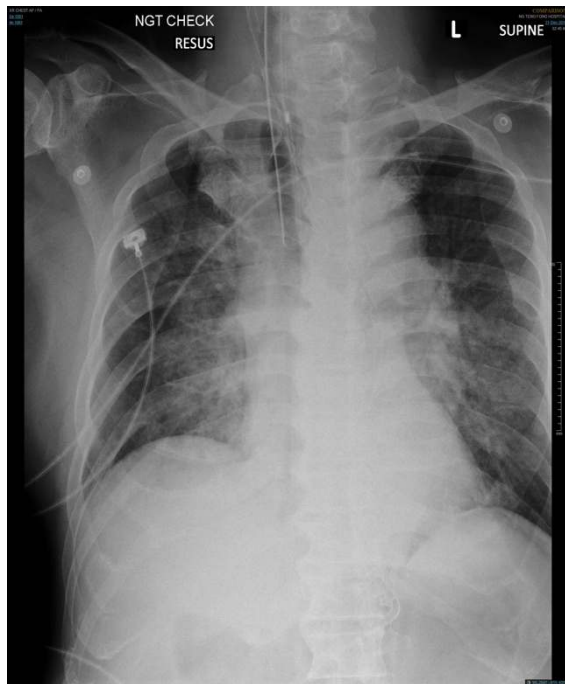


Legend: CXR=chest X-ray.

Figure 2. Thyroid mass is shown abutting trachea with endotracheal tube in-situ without retrosternal extension



Figure 3. CXR - post intubation



Legend: CXR=chest X-ray.

Figure 4. Crossectional view of ruptured thyroid cyst

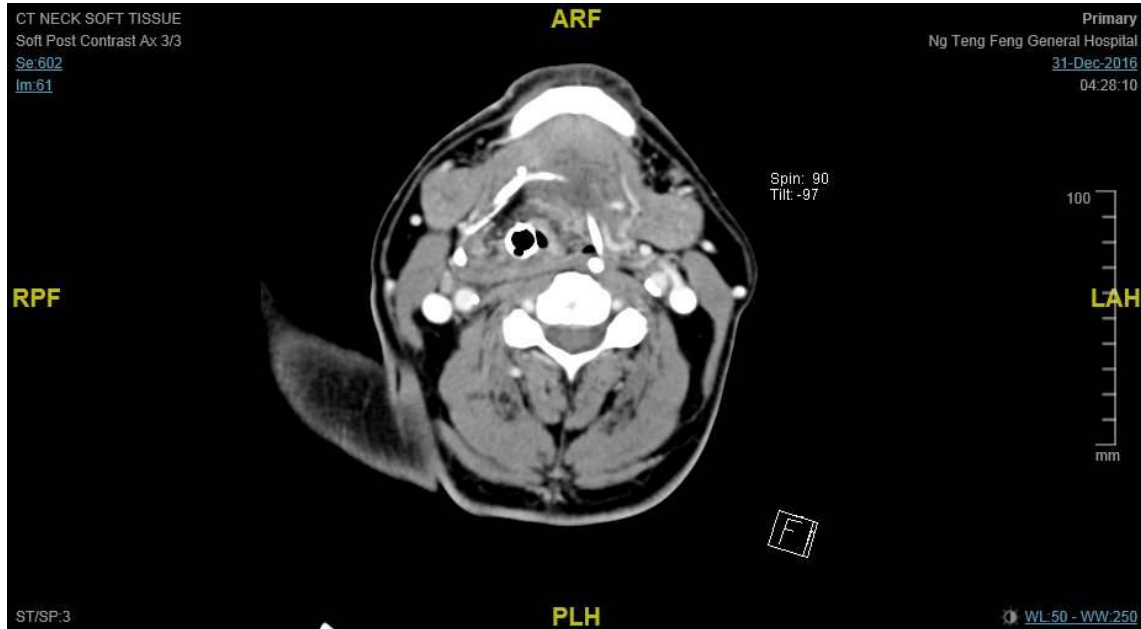


Figure 5. Sagittal view of huge cystic lesion compressing on trachea

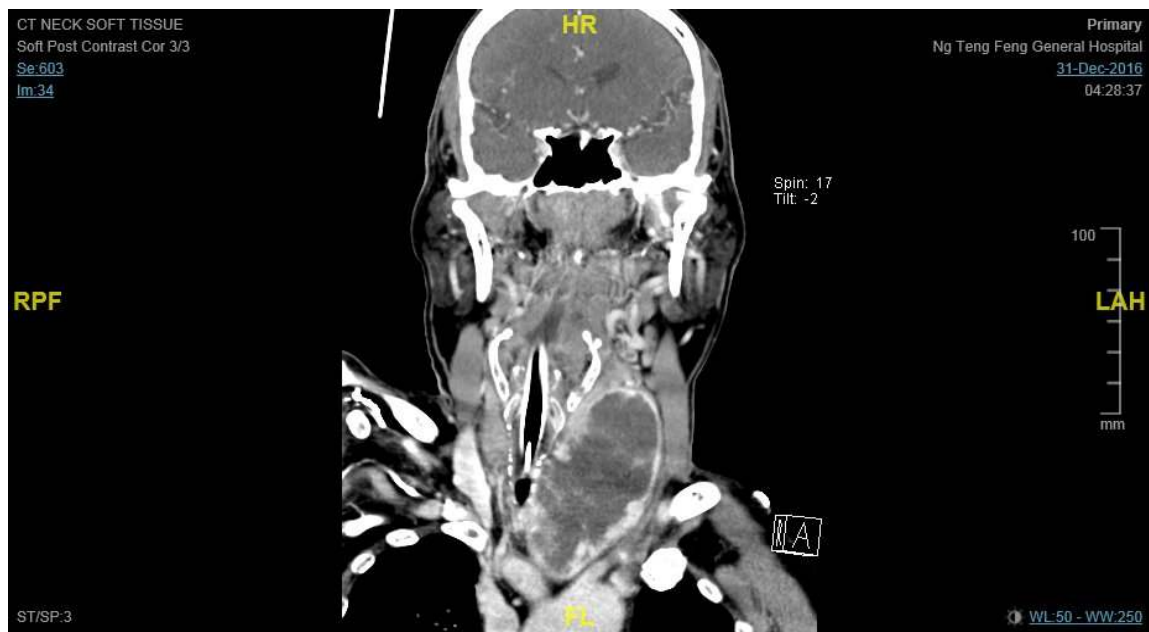


Figure 6. Cross sectional view of thyroid cyst compressing trachea

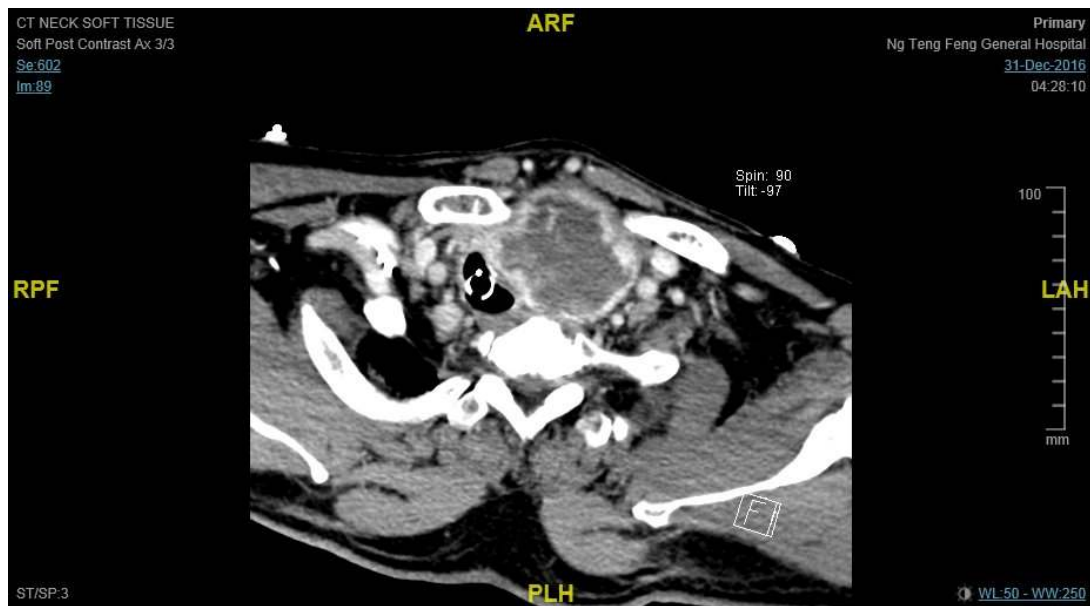


Figure 7. Fixed extra and intra-thoracic obstruction flow-volume loops

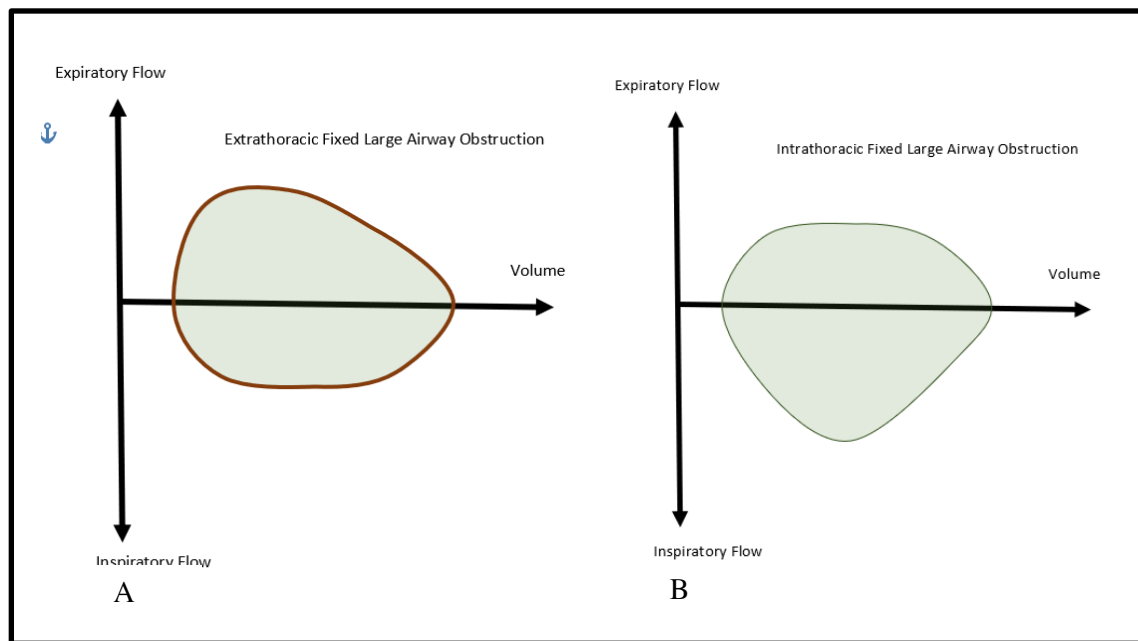
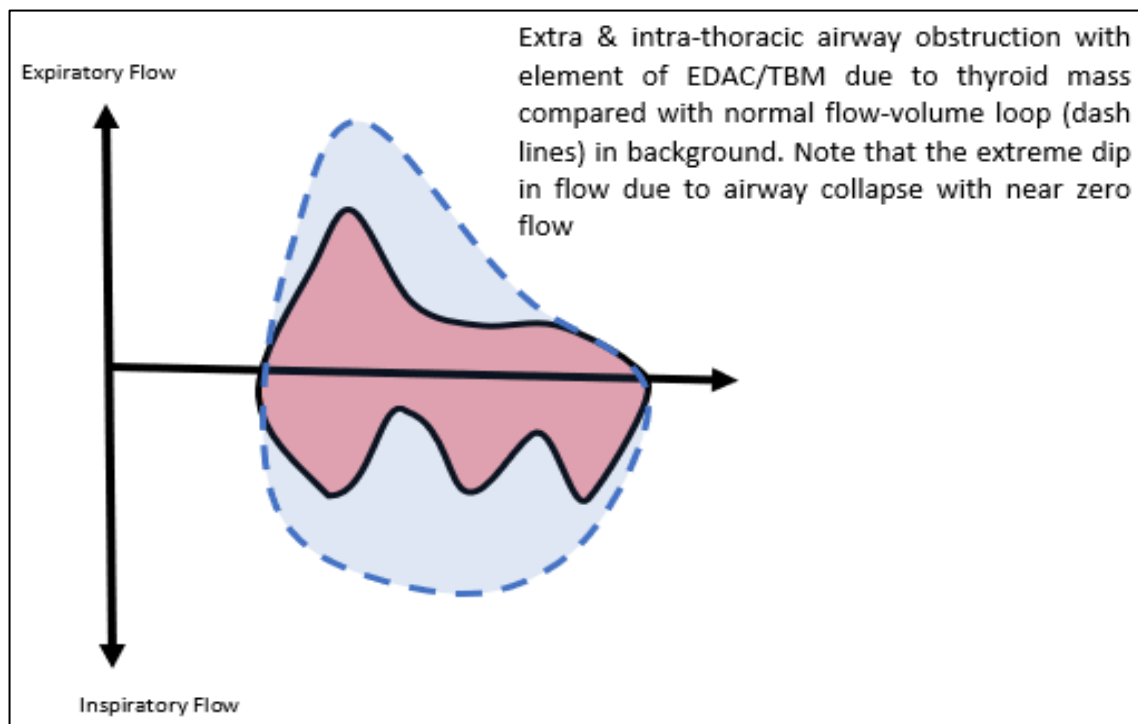


Figure 8. Flow-volume loop with variable obstruction due to EDAC/TBM superimposed on a normal flow-volume loop



Legend: EDAC=excessive dynamic airway collapse; TBM=tracheobronchomalacia.

Figure 9. Cross sectional view of trachea (adapted from Kalra, et al 2011)

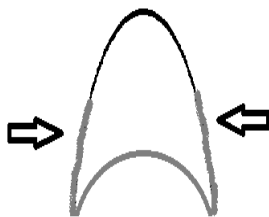
A. Normal tracheal shape



B. Trachea with significant posterior wall bulging with EDAC



C. TBM with lateral cartilage weakening



D. TBM with weak cartilaginous structure with flattening of anterior walls of trachea



Legend: EDAC=excessive dynamic airway collapse; TBM=tracheobronchomalacia.

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