Vasovagal syncope during spirometric exercise

Sir,

A 48-year-old male patient operated for aortic valve replacement was extubated uneventfully. Patient was off inotropic and vasoconstrictor support. During the recovery period, patient was advised to do spirometric breathing exercises. While doing spirometric breathing, patient suddenly had a sudden loss of consciousness with bradycardia and hypotension. Immediately the patient was intubated, and atrial pacing was started with epicardial pacing. As patient recovered consciousness and hemodynamic stability, he was extubated.

The reflex syncopes are because of a sudden failure of the autonomic nervous system to maintain adequate vascular tone during orthostatic stress, resulting in hypotension with bradycardia resulting in cerebral hypoperfusion and loss of consciousness.[1] Reflex syncope is (1) neurocardiogenic (vasovagal) syncope and (2) carotid sinus syndrome. It is commonly seen in younger patients and presents with a prodromal phase (nausea, lightheadedness, visual changes or diaphoresis) followed by a sudden loss of consciousness. Recovery is generally rapid and uneventful. The reasons for neurocardiogenic syncope are unclear. Predisposing factors can be pain, emotional distress, or prolonged standing. Pathophysiology of vasovagal syncope is often related to orthostatic stress.[2] Increased amount of peripheral venous pooling decreases venous return to the heart so precipitously that a significant rise in ventricular inotropy occurs. This hypercontractile state stimulates mechanoreceptors that would normally discharge impulses only during stretch.[3] The sudden increase in neural signal to the medulla causes "paradoxic" decline in sympathetic activity that results in bradycardia, hypotension and syncope.[4,5] Spirometric breathing exercises are routine practices after cardiac surgery to improve pulmonary function. In the present case, forceful and repeated blowing while performing spirometry breathing exercises might have caused sudden decrease in venous return to the heart and precipitated vasovagal syncope. Slow and steady breathing exercise can be helpful in preventing such a complication.

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References

1. Grubb BP. Neurocardiogenic syncope. In: Grubb BP, Olshansky B, editors. Syncope: Mechanisms and Management. Malden, MA: Blackwell-Futura; 2005. p. 47-71.
2. Mosqueda-Garcia R, Furlan R, Tank J, Fernandez-Violante R. The elusive pathophysiology of neurally mediated syncope. Circulation 2000;102:2898-906.
3. Kosinski D, Grubb BP, Temesy-Armos P. Pathophysiological aspects of neurocardiogenic syncope: Current concepts and new perspectives. Pacing Clin Electrophysiol 1995;18:716-24.
4. Morillo CA, Ellenbogen A, Pava LF. Pathophysiologic basis for vasodepressor syncope. In: Klein G, editor. Syncope: Cardiology Clinics of North America. Philadelphia, PA: Saunders; 1997. p. 233-50.
5. Grubb BP. Neurocardiogenic syncope and related disorders of orthostatic intolerance. Circulation 2005;111:2997-3006.

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Acute epiglottitis from corrosive ingestion

Sir,

Acute epiglottitis (AE) constitutes a medical emergency and most often results from infection with *Haemophilus influenzae*. Corrosive ingestion is a rare and hence less appreciated cause of AE.

A 40-year-old female was brought to the emergency department following suicidal ingestion of about 100 ml of liquid toilet cleaner HARPIC™, containing 10.5% hydrochloric acid. She complained of dyspnea, dysphagia, odynophgia, difficulty in speaking and drooling. She was noted to have hoarseness of voice, respiratory distress with stridor and cyanosis. Examination revealed oral burns, cyanosis and crackles at both lung bases. A lateral soft tissue X-ray of the neck showed "thumb sign" [Figure 1], suggesting AE. She was kept nil per oral and managed with supplemental oxygen, nebulized budesonide and adrenaline, intravenous fluids and antibiotics. Orotracheal intubation was required after 48 h for worsening stridor and hypoxemic respiratory failure, and mechanical ventilation was initiated. Enlarged and inflamed epiglottis was confirmed by direct

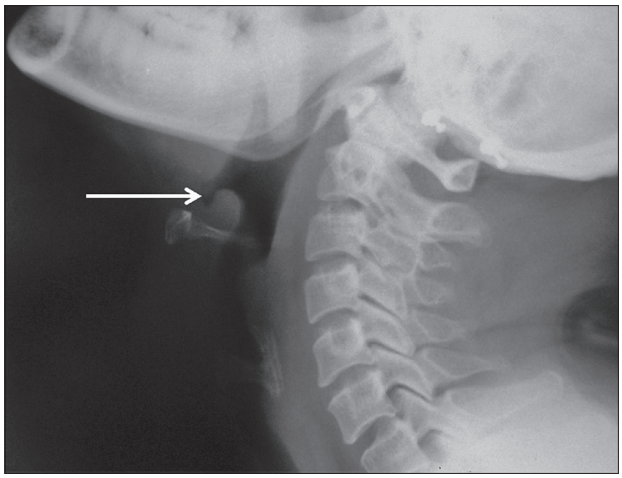

Figure 1: Lateral soft tissue radiograph of the neck showing "thumb sign" (arrow), suggesting a severely inflamed, oedematous and enlarged epiglottis

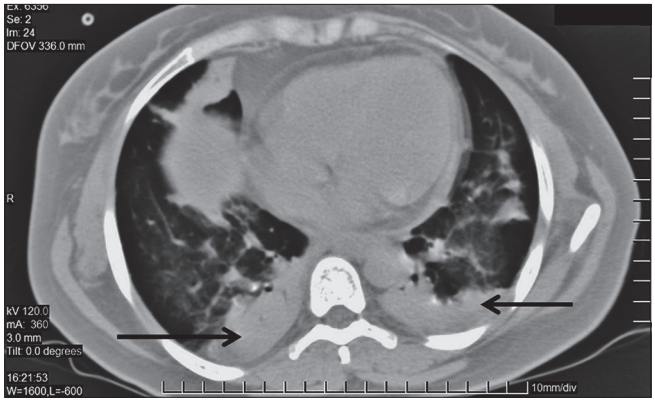

Figure 2: Contrast-enhanced computed tomography of the chest (lung window), showing areas of consolidation (black arrows) in both lungs, suggesting aspiration pneumonia

laryngoscopy during intubation. Contrast-enhanced computed tomography of the chest showed areas of consolidation in the right middle and lower lung lobes and left lower lobe [Figure 2], suggesting aspiration pneumonia. She required mechanical ventilation for a week and was subsequently extubated.

"Thumb sign" in lateral soft tissue radiograph of the neck indicates a severely inflamed, edematous and enlarged epiglottis, resulting most commonly from infections with *H. influenza* - type b and Group-A β-hemolytic Streptococci.[1] However, corrosive injury is a rare cause of AE.[1] Along with epiglottis, rest of the supraglottic area including the vallecula, aryepiglottic folds and arytenoids are also inflamed and edematous and hence life-threatening acute upper-airway obstruction may develop rapidly.[2] Thus, AE constitutes a medical emergency. In a study involving AE patients,[3] positive "thumb sign" on lateral neck radiograph and stridor were significant predictors of requirement for airway intervention and hence their presence should warn of imminent upper-airway occlusion. Timely fiberoptic nasotracheal or less preferably orotracheal intubation by a skilled anesthetist or tracheostomy may be required for securing the airway. Adjunctive measures to reduce upper airway edema in AE include intravenous dexamethasone, nebulized budesonide and intravenous antibiotics.[1]

Aspiration pneumonia and acute respiratory distress syndrome are known complications of corrosive ingestion. In a study of 273 adult patients of caustic ingestion reported from Taiwan,[4] 11% had aspiration pneumonia and 8% had respiratory failure. In corrosive ingestion, presence of upper airway compromise with stridor, along with dysphagia, odynophagia, voice change and drooling should raise suspicion of AE. Timely recognition of this complication and meticulous upper airway management may be lifesaving. Absence of radiological "thumb sign" should not rule out AE.[5] In a clinically unstable patient with suspected AE, clinical stabilization and airway securement should get priority over X-ray imaging of the neck.

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References

1. Abdallah C. Acute epiglottitis: Trends, diagnosis and management. Saudi J Anaesth 2012;6:279-81.
2. Sack JL, Brock CD. Identifying acute epiglottitis in adults. High degree of awareness, close monitoring are key. Postgrad Med 2002;112:81-2, 85.
3. Chan KO, Pang YT, Tan KK. Acute epiglottitis in the tropics: Is it an adult disease? J Laryngol Otol 2001;115:715-8.
4. Cheng HT, Cheng CL, Lin CH, Tang JH, Chu YY, Liu NJ, *et al.* Caustic ingestion in adults: The role of endoscopic classification in predicting outcome. BMC Gastroenterol 2008;8:31.
5. Stankiewicz JA, Bowes AK. Croup and epiglottitis: A radiologic study. Laryngoscope 1985;95:1159-60.

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