

Iatrogenic Vocal Cord Paralysis after Cardiac Surgery: Evocative Note for Surgeon and Anesthesiologist

The Editor,

A 45-year-old female patient presented with the shortness of breath on mild exertion and palpitation for 6 months. Patient's echocardiographic examination revealed severe mitral stenosis with enlarged left atrium. The patient was also found to have severe tricuspid regurgitation with moderate pulmonary hypertension. There was no history suggestive of voice change or hoarseness in the past. The patient was scheduled for mitral valve replacement and tricuspid annuloplasty with maze procedure for chronic atrial fibrillation (AF). Anesthesia was induced without any incident. Laryngoscopic examination showed Cormack and Lehane grade 1 glottic view. Trachea was intubated with 7.5 mm endotracheal tube with cuff (Portex® Profile Soft Seal® Cuff Tracheal Tubes from Smiths Medical). Intubation was smooth, atraumatic and in single attempt. Hypothermic cardiopulmonary bypass was established using aortic and bicaval cannulation. Heart was arrested using antegrade cold blood cardioplegia. Mitral valve was replaced with 27/29 mm. On-X mitral valve prosthesis along with tricuspid ring annuloplasty was performed using 26 mm Edwards MC3 tricuspid annuloplasty ring and pulmonary vein isolation on both left and right sides with bipolar radiofrequency ablation was done. Total surgical duration was 220 min and cardiopulmonary bypass time was 152 min. Patient was smoothly weaned off cardiopulmonary bypass and was shifted to Intensive Care Unit in stable condition. After 23 h of mechanical ventilation, trachea was extubated. Hoarseness was noticed with low pitch voice following tracheal extubation. Patient's respiration was comfortable and her pulse oximetry saturation on room air was 97%. Patient was started with routine steam inhalation and inhaled budesonide nebulisation. Patient's weak voice persisted on the 2nd postoperative day also. Videolaryngoscopic examination revealed decreased movement of the left vocal cord suggestive of left vocal cord palsy. Patient's nebulisation was continued. On postoperative day 10, patient's voice became better with no hoarseness.

In 1897, Ortner syndrome was first described as left recurrent laryngeal nerve paralysis caused by compressive effect by cardiovascular conditions resulting in hoarseness.^[1] Common cardiovascular conditions causing hoarseness are aortic diseases, left atrial disorders followed by congenital heart disease (e.g., total anomalous pulmonary venous connection, atrial septal defect, patent ductus arteriosus [PDA], idiopathic pulmonary hypertension, etc.).^[1] Course of the left recurrent laryngeal nerve between the aorta and pulmonary artery makes it

vulnerable to compressive injury. Various cardiovascular interventions can damage recurrent laryngeal nerve resulting in Iatrogenic cardiovocal syndrome.^[2] Intraoperative systemic and local hypothermia during cardiac surgery can potentially cause dysfunction of recurrent laryngeal nerve. PDA ligation, thoracic aortic surgery, left pulmonary artery stenting, and transcatheter ablation of AF have been reported to cause left vocal cord palsy.^[1,3]

Traumatic or prolonged intubation can cause laryngeal joint dysfunction and hoarseness. Inflated cuff of the endotracheal tube within distance of 15 mm below the vocal cords can cause compression of anterior branch of recurrent laryngeal nerve.^[4] High intracuff pressure can damage the mucous membrane of trachea and recurrent laryngeal nerve. Inadvertent traumatic injury to recurrent laryngeal nerve injury can occur during central venous cannulation.^[5] Lateral flexion and hyperextension of the neck during sternotomy can compress the crossing point of the vagal and hypoglossal nerve on transverse process of C1 vertebra.^[6] The resultant injury of hypoglossal and the vagal nerves is described as Tapias syndrome which presents as ipsilateral paralysis of the vocal cord and tongue. Fiberoptic laryngoscopy can easily diagnose the condition. Recently introduced, new noninvasive technique transcutaneous laryngeal ultrasonography can be used to examine perioperative vocal cord function.^[7]

In the present case, there were multiple contributory factors to affect vocal cord function like prolonged surgical and intubation duration, intraoperative hypothermia, AF ablation procedure. Fortunately, vocal cord function improved within a few days. Vocal cord palsy or hoarseness after cardiovascular surgery generally recovers spontaneously within weeks; however, permanent vocal cord paralysis was observed in 3.5%–5.3% of patients.^[1] This underlines the significance of written informed consent and explaining the possible surgical risks to the patients. Consultation and documentation by laryngeal specialists in postcardiac surgery vocal cord dysfunction is important. Such steps can potentially reduce the chance of medicolegal litigation in case of unfortunate event. Knowledge of possible etiological factors by anesthesiologists and surgeons is essential to prevent such complications.

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Conflicts of interest

There are no conflicts of interest.

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