Vocal cord dysfunction after cardiac surgery: an overlooked complication

H. Shafei*, A. El-Kholy, S. Azmy, M. Ebrahim, K. Al-Ebrahim

Al-Hada Armed Forces Hospital, P.O. Box 1347, Taif, Saudi Arabia

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Abstract

Objective: To evaluate the complication of vocal cord dysfunction following open heart surgery and its short- and long-term effects. Materials and methods: Five cases (1.9%) of vocal cord dysfunction which were diagnosed among 270 cases of adult open heart surgery performed at this centre between February 1993 and May 1995. Results: All five patients developed respiratory insufficiency following tracheal extubation. They required reintubation and ventilation. Diagnosis was delayed in three cases because of unawareness of the problem. Tracheostomy was performed in three cases and minitracheostomy in two. In one case, shortness of breath on exertion persisted due to partial laryngeal obstruction from bilateral cord paresis and required arytenoidectomy 11 months postoperatively. Conclusions: Vocal cord dysfunction can be an easily overlooked complication after open heart surgery. It can be the cause of respiratory insufficiency following tracheal extubation and may lead to reintubation and reventilation. The cause of the problem cannot always be traced but it may be due to direct trauma of the vocal cords during tracheal intubation, or trauma of the recurrent laryngeal nerve from the cuff of the endotracheal tube. A less likely possibility is that it may result from nerve injury due to central venous cannulation, or from cold. The condition may resolve within months, but, in rare cases, may lead to permanent morbidity. © 1997 Elsevier Science B.V.

Keywords: Intermittent positive pressure ventilation; Open heart surgery; Postoperative complications; Vocal cord dysfunction

1. Introduction

Laryngeal examination is rarely performed in the cardiac surgical patient who presents with respiratory insufficiency postoperatively. We found that an overlooked cause of this problem was vocal cord dysfunction. Our experience suggests that more attention should be given to this possibility in patients who otherwise have no apparent cause of inadequate spontaneous breathing after tracheal extubation following cardiac surgery.

2. Patients and methods

A retrospective study to evaluate the problem of vocal cord dysfunction was carried out. All adult patients who underwent open heart surgery in this centre had standard anaesthetic technique, with central venous cannulation, peripheral arterial cannulation, urinary catheterization, nasogastric intubation and Swan-Ganz catheterization in patients with an indication, e.g. poor left ventricular function. Endotracheal intubation is performed using high-volume low-pressure cuff disposable endotracheal tubes (Mallinckrodt Laboratories, Athlone, Ireland) made of PVC. The size of the tube is in the range 8–8.5 mm in males and 7.5–8 mm in females. All our patients had routine cardiopulmonary
bypass with ascending aortic arterial cannulation and either one right atrial or two caval cannulae for venous return. Systemic hypothermia was induced to variable degrees, depending on the expected time of the procedure, and lowest core temperature reached was 25°C.

Myocardial protection during the ischaemic period is achieved by antegrade intermittent cold blood cardioplegia and topical cooling with slushed ice. We do not use pads for protection of the phrenic nerves. In the postoperative period, all patients are electively ventilated for variable periods depending on several factors, but the majority are ventilated till the morning following the day of surgery. The diagnosis of vocal cord dysfunction was confirmed by fibreoptic laryngeal examination performed by the otolaryngologist after extubation.

3. Results

Five cases out of 270 adult cases who underwent open heart surgery (1.9%), were diagnosed to have unilateral or bilateral vocal cord palsy in the postoperative period. Cases 1, 2 and 4 had endotracheal tube size 8.5 mm. Cases 3 and 5 had tube size 7.5 mm. None of the cases had difficulty in intubation. Table 1 shows the clinical and postoperative findings in the five patients. All these cases had initial satisfactory weaning criteria from artificial ventilation when they were still intubated. Several hours after extubation, they developed an increasing work of breathing and tiredness. It was obvious that they had weak and inefficient cough, with accumulation of secretions. All of them had persistent hoarseness of voice. Gradually, over a period of several hours to 2 days, all of them developed respiratory insufficiency and required reventilation. This pattern was repeated after tracheal extubation, more than once. Minitracheostomy (Mini-Trach II, Portex, Hythe Kent, UK) was performed in two cases and formal tracheostomy in three cases. One case (case 4) died suddenly prior to discharge from hospital and no post-mortem examination was performed. His death, from the available information, cannot be related to vocal cord dysfunction. Another patient (case 1), with bilateral cord palsy, complained of shortness of breath and hoarseness of voice for several months postoperatively. He underwent arytenoidectomy to increase the laryngeal aperture 11 months postoperatively. The other three cases recovered gradually and no significant residual effect was present at the 3 month follow-up examination.

4. Discussion

We have identified here a complication with a characteristic pattern that, to our knowledge, has not been addressed before in the context of cardiac surgery. Although there are several general, respiratory and cardiac causes of respiratory insufficiency after cardiac surgery, we found that vocal cord palsy is an easily overlooked cause. The condition typically appears in a patient after tracheal extubation who seemed to be progressing normally when he was intubated. It is noticeable that such patients can be breathing spontaneously in a satisfactory way, with the tracheal tube still in place. After its removal, and to a variable extent, gradual respiratory insufficiency develops. In general, it is well known that vocal cord paralysis can present with hoarseness, stridor, poor cough, aspiration or dysphagia [11]. There is accumulation of secretions, increasing work of breathing with increasing exhaustion and respiratory insufficiency which often requires reventilation. Diagnosis can be made with certainty from fibreoptic laryngoscopy.

It is possible that the true incidence of vocal cord dysfunction may be different, possibly higher, as this study mainly detected patients with severe symptoms. However, it remains very useful to highlight the significance of the problem in the cardiac surgical patient. The incidence of vocal cord dysfunction after general surgical operations, which require the same degree of monitoring and postoperative ventilation as cardiac surgery, is expected to be similar. Although this complication of tracheal intubation has been well documented in the literature [1], to our knowledge, the incidence of vocal cord dysfunction in short-term tracheal intubation of anaesthesia has not been reported. There are well-identified factors which determine the severity of intubation trauma. These include intubation difficulty,
duration of intubation, movement of the tube, the presence of nasogastric tube, and tube characteristics such as the external diameter, the shape, composition and cuff characteristics, in addition to other factors [1].

The aetiology of the dysfunction of the vocal cord following open heart surgery remains unclear. Traumatic endotracheal intubation can cause direct cord damage and palsy [6,10]. It has been observed that unilateral vocal cord paralysis may follow endotracheal intubation without any local lesion [8]. Cadaveric dissection showed that the inflated cuff of an endotracheal tube can compress the anterior branch of the anterior recurrent laryngeal nerve against the thyroid cartilage and cause paralysis [5]. Since nerve injury has been reported to result from penetrating neck injury, it has to be assumed that recurrent laryngeal nerve injury can be caused by traumatic injury from central venous line insertion [10]. The chances of direct surgical injury through a sternotomy should be remote, although injury of the nerve has been reported following surgical procedures such as mediastinoscopy, oesophageal resection and others [10]. Cold injury from local cooling of the heart may be a possible aetiology, but is more frequently associated with phrenic nerve palsy [9]. It has been stated that when the paralysis is idiopathic, the chance of spontaneous recovery before 6 months is 85% [7]. In our experience, the duration of the dysfunction varies and spontaneous recovery may take place within weeks. If the condition does not resolve by then, it is likely that the damage will remain permanent.

The significance of recurrent laryngeal nerve palsy after cardiac surgery has not been emphasized. Although phrenic nerve palsy is a well-known complication of open heart surgery [4,9], recurrent laryngeal nerve palsy is less recognized. The purpose of this report is to alert practitioners to the fact that it can be an overlooked cause of respiratory insufficiency as well as a cause of significant morbidity following open heart surgery. In a patient who develops respiratory insufficiency after extubation, with inefficient cough and voice hoarseness, in the absence of cardiac, respiratory or general cause, this complication should be seriously suspected. After diagnosis has been made by fibreoptic laryngoscopy, if conservative measures, including mini-tracheostomy, fail to avoid reventilation, early tracheostomy should be considered. Lack of awareness of the diagnosis, with subsequent several tracheal reintubations, usually exposes the patients to severe vocal cord oedema, ulceration and more damage. Long-term management in cases with residual damage may require arytenoidectomy to increase the size of the laryngeal aperture, or vocal cord injection with Teflon [3] or autogenous fat [2] to increase its tension.

References