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VOCAL CORD PARALYSIS FOLLOWING ENDOTRACHEAL INTUBATION

Pages with reference to book, From 294 To 295

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INTRODUCTION

Endotracheal intubation has many complications. We describe a case of bilateral vocal cord paralysis following endotracheal intubation which is rare but a known complication. The possible factors contributing to this complication and measures which can be taken to prevent it have also been discussed.

CASE REPORT

A 60 year old lady presented to the emergency room with 2 days history of abdominal distention, colicky pain and constipation. Her chest x-ray showed pneumoperitoneum. She underwent exploratory laparotomy the same day. She was found to have a tumour of the sigmoid colon alongwith caecal perforation. A subtotal colonic resection with ileocolic anastomosis was performed. Her preoperative assessment was ASA III and she was intubated with a red rubber tube with an 8mm cuff It was intubation grade I. The cuff was inflated with room air. During the procedure O₂ was kept at 1.5 L/min and N₂O 2L/min. The procedure took 3 hours after which she was extubated. Her post-anaesthetic recovery was unremarkable. On 2nd postoperation day the patient developed inspiratory stridor. She also became agitated and breathless. The following day she was referred to the ENT department. On fiberoptic laryngoscopy examination both vocal cords were in the paramedian position with slight mobility. She was put under strict observation to evaluate for a possible tracheostomy but on 4th postoperation day her condition started to improve progressively and on the 8th postoperation day another fiberoptic laryngoscopy showed complete left vocal cord paralysis while the right vocal cord had normal mobility. Patient was discharged and repeat fiberoptic laryngoscopy two weeks later found both vocal cords to be mobile and normal.

DISCUSSION

Complications resulting from endotracheal intubation have been well reviewed¹. Fortunately, serious problems are rare but a great many people after intubation complain of pain in the throat. Hoarseness, which is a less common problem can occur due to nodules, granulomas and other consequences of the injury to the mucosae. But sometimes hoarseness is due to paralysis of the vocal cords which if bilateral can even cause stridor and respiratory obstruction². Kambic and Radsel³ examined 1000 people after extubation and detected severe lesions of larynx in 62 patients but they did not mention vocal cord paralysis due to recurrent laryngeal nerve palsy. Searching published work Cavo⁴ in 1985 found 36 cases which were clearly the outcome of endotracheal intubation. Following this there have been a few reports of this complication in literature^{5,6}. Most of these cases when followed recovered in days to months. The inference that many of these cases were avoidable led to the study of the anatomy of the recurrent laryngeal nerve and pressure within the endotracheal cuff during anaesthesia. Ellis and Palister⁷ after tracing the recurrent laryngeal nerve in the larynx described its two branches. An anterior and a posterior one. They proposed that it was the anterior branch which was compressed between the

cuff of the endotracheal tube and the lamina of the thyroid cartilage. Cavo's⁴ dissection of recurrent laryngeal nerve largely confirmed its anatomy. He found that the anterior branch of the nerve, as it ascends on the medial side of the rim of the thyroid cartilage, comes to lie on the top of the lateral cricothyroid muscle. At this level it lies close to the mucosal surface beneath the body of arytenoid about 6-10 mm below the end of true vocal cord. Here it is vulnerable to compression between an expanded cuff and the overlying thyroid cartilage. The pressure changes occurring in the endotracheal cuff while the subject is under anaesthesia have also been analysed⁸. It has been found that a diffusion of gas occurs across the semipermeable membrane of the endotracheal tube; nitrous oxide particularly has been found to diffuse rapidly across the membrane from an area of high concentration to an area of relatively low concentration thus causing an increase in the cuff pressure after some time. In our case also the endotracheal cuff was inflated with (room) air which might have resulted in diffusion of N₂O into the tube and thus increasing endotracheal cuff pressure resulting in pressure neuropraxia of the anterior branch of the recurrent laryngeal nerve and bilateral vocal cord paralysis. As in our case nearly all patients with this rare complication recover spontaneously, usually within six months⁹. The incidence of vocal cord paralysis might be reduced almost to nil if anaesthetists make a routine of marking endotracheal tubes 1.5 cm above the upper level of the cuff to facilitate accurate placement below the susceptible area and to use nitrous oxide or normal saline to inflate the cuff and periodic evacuation and reinflation of the cuff⁴⁻¹⁰.

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