Management Of Repeated Postextubation Laryngospasm: A Case Report
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Citation

Abstract
Laryngospasm is one of the common causes of airway obstruction in the postextubation period. Though definitive predisposing or causative factors can usually be identified, it is also known to occur without any apparent provocation.[1] Absence of a specific etiology makes treatment difficult in such cases. We describe our management of a patient who had repeated episodes of unanticipated postextubation laryngospasm. The various treatment options that can be used for this recurring condition are described.

CASE REPORT
A 50 year old, 60 kg, ASA grade I, nonsmoker male patient was scheduled for a decompressive laminectomy and discectomy for degenerative lumbar spine disease. Preanaesthetic evaluation including the airway assessment was within normal limits. General anaesthesia included glycopyrrolate, midazolam, fentanyl, thiopentone, vecuronium, isoflurane and oxygen (O\textsubscript{2}) - nitrous oxide(N\textsubscript{2}O) mixture. The patient was intubated and put on mechanical ventilation. Surgery was done in the prone position and lasted for 110 minutes.

At the end of the surgery, neuromuscular blockade was reversed with neostigmine and glycopyrrolate. The patient was extubated when he was awake and following commands; breathing adequately, had a good cough reflex and muscle tone and was maintaining 100% oxygen saturation (SaO\textsubscript{2}@). However, suddenly he complained of difficulty in breathing and vocalising. Visible inspiratory stridor and paradoxical respiration soon followed and his SaO\textsubscript{2}@ decreased to 92%. Inspiratory spasm was detected on chest auscultation. Pharyngeal suctioning did not reveal any secretions and a jaw thrust applied to clear the airway failed to improve his condition; his SaO\textsubscript{2}@ further decreased to 85%. The stridor responded to intravenous (I.V) propofol (0.25mg/kg and positive pressure ventilation with 100% O\textsubscript{2}@. The patient was stable with a SaO\textsubscript{2}@ of 98%, but shortly thereafter had a repeat episode of laryngospasm. More propofol (2.0 mg/kg, I.V) was administered, laryngoscopy was done to rule out any obstruction and the vocal cords were sprayed with lignocaine (2ml of 2% solution). The laryngospasm resolved with no further recurrence and the patient was transferred to the recovery room with a SaO\textsubscript{2}@ of 98% on O\textsubscript{2}@ (50%) via a face mask.

DISCUSSION
Postextubation laryngospasm most often occurs due to presence of secretions or blood in the throat and extubation or airway stimulation in a light plane of anaesthesia.[3] The risk is higher in smokers and obese patients, in the presence of underlying airway disease and following a difficult intubation.[2,3]. None of these factors were identified in our patient, and yet laryngospasm occurred. The Australian Incident Monitoring Study (AIMS) reports an incidence of 22% of laryngospasm without an attributable cause.[2] Transient laryngeal hyper excitability observed during emergence from anaesthesia, could increase the vulnerability to laryngospasm in such cases.[4] Immediate treatment of laryngospasm is essential to avert serious complications like severe hypoxia and hypercarbia, pulmonary aspiration, pulmonary edema and even cardiac arrest.[2] However repeated laryngospasm may not respond to standard initial maneuvers.[3] In our patient, we had to use propofol to deepen the level of anaesthesia, together with lignocaine to suppress the laryngeal reflexes. Usually, a sub hypnotic dose of propofol (0.25mg/kg) suffices,[,] but we had to resort to an anaesthetic dose (2.0mg/kg) to terminate the recurring episodes. We used lignocaine topically, but it can also be administered IV (2mg/kg) or as an aerosol (10ml. of 1%solution) to abolish postextubation laryngospasm.[4]
Other drugs which can be used for repeated laryngospasm include nitroglycerine, diazepam and doxapram.\cite{6,7,8}

Nitroglycerin (4 µg/kg IV bolus) acts by relaxing the airway smooth muscle via the nitric oxide guanylate cyclase pathway.\cite{6} Diazepam (1mg I.V increments; up to 3mg) acts via a centrally mediated relaxation of the laryngeal muscles in addition to its anxiolytic effect.\cite{7} Relief of laryngospasm by doxapram is related to its stimulant effect on the respiratory centre.\cite{8}

Complete laryngospasm is a life threatening emergency that may necessitate the use of suxamethonium in sub paralytic doses (0.1mg/kg I.V). Emergency intubation may be required as a last resort.\cite{2}

Thus, absence of an attributable cause, does not preclude the development of postextubation laryngospasm. Repeated laryngospasm responds better to multimodal therapy with drugs having different mechanisms of action rather than a single line of treatment.

References
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