

Postoperative psychogenic respiratory distress mimicking laryngospasm

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Postoperative respiratory distress with stridor may be due to psychogenic aetiology. Suspicion of psychogenic respiratory distress in a patient not responding to conventional treatment for laryngospasm and recognition of psychogenic aetiology can prevent unnecessary invasive investigations and interventions.

Keywords: Postoperative laryngospasm; psychogenic respiratory distress; postoperative stridor, psychogenic stridor; functional airway obstruction.

Introduction

Laryngospasm, causing air way obstruction, is a common complication of anaesthetic practice¹ and may present atypically. If inadequately managed, it can cause morbidity and mortality such as severe hypoxaemia, pulmonary aspiration, and post-obstructive pulmonary oedema.

Psychogenic respiratory distress could mimic postoperative laryngospasm, indicating airway obstruction. It may not be responding to conventional interventions terminating laryngospasm, and can be a dilemma to the anaesthetist if the psychogenic nature of the condition is not recognised. Here we present a case report of a lady who developed psychogenic respiratory distress postoperatively following general anaesthesia for laparoscopy and dye test. Early recognition and intervention could prevent unnecessary intubation and critical care unit admission reducing morbidity and mortality.

Case report

A 26 year old lady presented for laparoscopy and dye test for subfertility. She had a history of bronchial asthma and had undergone general anaesthesia for evacuation of retained products of conception six months ago, without any complications.

She also had a history of allergy for an antibiotic

given for childhood chest infection but no records are available regarding the name of the antibiotic or the nature of the reaction.

Her weight and height were, 57kg and 150cm respectively, and she was a non-smoker, and a teetotaler. During pre-operative assessment anxiety about undergoing surgery and tachycardia were noted. Intravenous propofol 200mg, morphine 9mg, atracurium 30mg, cefuroxime 750mg and inhalational agents, halothane (1%) and nitrous oxide (50%), were administered during anaesthesia. The surgery was uneventful and she was stable during surgery. Muscle relaxation was successfully reversed with neostigmine 2.5mg and atropine 1.2mg intravenously and she was extubated when her airway was clear of secretions and she was awake.

About 20 minutes after the extubation she complained of shortness of breath. She had an inspiratory noise, which was thought to be stridor, tracheal tug and chest recession during inspiration. She was flexing her neck attempting to breathe in. She was alert, fully awake and her 'stridor' and laboured breathing worsened progressively. However, her airway was clear, there were no rhonchi on auscultation of chest, and no desaturation observed. She was taken back to the theatre and her ventilation was assisted with a tight mask with continuous positive airway pressure (CPAP), airway manoeuvres and 100% oxygen. There was no difficulty in ventilating her with tight mask and airway manoeuvres despite her 'laryngospasms'. Dexamethasone 8mg i.v. and adrenaline nebulisations (1:1000, 1ml) were also administered, but she showed no improvement.

Then she was given propofol 50mg i.v. and anaesthesia was maintained with 1% halothane in

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99% oxygen. The laryngospasm completely stopped, movement of vocal cords were normal under anaesthesia when laryngoscopy was performed, and after about 3 minutes of anaesthesia she woke up without stridor. As she became more awake, she developed stridor and laboured breathing again, which again needed general anaesthesia. Once more, intravenous propofol 50mg was given with fentanyl 25mcg and succinylcholine 20mg with 1% halothane in 99% oxygen; stridor ceased and she continued to have spontaneous respiration. Once again when she was fully conscious, she developed stridor.

Meanwhile, when her husband was updated about her condition, he mentioned that she had been very anxious and requested him to be in the theatre during the surgery. Due to unusual presentation of her symptoms psychogenic aetiology was suspected, and it was decided to allow her husband to the theatre to comfort her. She was reassured and instructed to breath in a slow pattern; subsequently her laryngospasm gradually disappeared. She was then kept under observation for 12 hours and her stridor never returned. Although suggested, she was reluctant to consult a psychiatrist for follow up.

Discussion

Laryngospasm, sustained closure of the vocal cords resulting in the partial or complete loss of the patient's airway², is a common complication of airway management in anaesthetic practice.^{1,3} The presentation with difficulty in breathing, inspiratory noise, tracheal tug, tachycardia, and chest recession during inspiration, suggested that this lady was having upper airway obstruction, most probably caused by post anaesthesia laryngospasm. Thus conventional treatment for post-operative laryngospasm was instituted: removing any triggering stimulation such as undue suctioning, ensuring a clear larynx of blood or stomach contents, relieving any possible supra-glottic component to the airway obstruction by applying airway manoeuvres (head tilt, chin lift and jaw thrust), and application of CPAP with 100% oxygen.² Intravenous dexamethasone 8mg and adrenaline nebulisations (1:1000, 1ml) were initially given as her airway obstruction could have been secondary to laryngeal oedema.^{4,5}

Since, laryngospasm was not rapidly settling, anaesthesia was given and deepened with intravenous boluses of propofol (0.5 mg/kg

increments), and total of 50mg was given initially.² Halothane was used to maintain anaesthesia as it was acceptable to be used in laryngospasm.³ Since, 'stridor' recurred after successful abolition, patient was given anaesthesia again with muscle relaxant, intravenous succinylcholine (dose range from 0.1 to 2 mg /kg).^{2,3} Even after succinylcholine, 'stridor' ceased only to return when she was fully awake and the magnitude of distress always progressively increased. Thus, her respiratory distress had unusual clinical presentation and course, with failure to respond to conventional treatments, suggestive of a psychogenic aetiology.

Psychogenic respiratory distress presents with signs of respiratory distress suggestive of airway obstruction or asthma.⁶ A number of psychiatric disturbances are related to psychogenic respiratory distress, including conversion and anxiety disorders. This condition is non organic and is associated with severe psychosocial stress and difficulties with modulation of intense emotional states.⁶

Psychogenic causes are supported by normal blood gas values despite marked symptoms of respiratory distress, neck flexion (in contrast to the extension often encountered in asthma), when airway difficulties are precipitated by suggestion, when patients demonstrate exacerbations related to stress, and symptoms that abate with distraction, support, and reassurance or placebo use.⁶ Although blood gas analysis was not done, despite apparently severe respiratory distress she did not demonstrate desaturation on pulse oximetry. She was flexing her neck during attacks and her condition spontaneously resolved by distraction, support and reassurance by her husband and the medical team. The failure to respond favourably to conventional treatments, also suggests a functional aetiology.⁶ Psychogenic stridor decreases and may cease with relaxation, distraction, reassurance, sleep, anaesthesia, breathing through nose, placebo, sedation, coughing, and speech.^{5,7,8} This type of stridor may actually decrease when not under direct observation.⁷

The treatment for psychogenic respiratory distress includes consultation with psychiatrist and speech pathologist.⁶

Conclusion

As an anaesthetist, it is vital to recognise psychogenic nature of respiratory distress, as post anaesthesia laryngospasm not responding to conventional treatment could be a very confusing and a distressing situation, often leading to unnecessary invasive interventions such as intubation and tracheostomy, and unnecessary ICU admissions. Symptoms may be abolished by distraction, support, and reassurance or placebo use; however, psychiatric consultation is recommended for follow up.

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