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CASE REPORT

An unexpected functional cause of upper airway obstruction

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Summary

A case of acute respiratory obstruction in the immediate postoperative period is described in a young woman who emerged from general anaesthesia after a Caesarean section for fetal distress. She had a pregnancy complicated by disabling polyhydramnios and anxiously anticipated the birth of a child with a diaphragmatic hernia, diagnosed antenatally. The cause of the airway obstruction was functional in nature as confirmed by flexible fibreoptic laryngoscopy. The diagnosis, paradoxical vocal cord motion, has to be considered as an infrequent cause of postoperative airway obstruction; its recognition and treatment are discussed. The patient did not have a history which might have indicated its possible occurrence. It is suggested that paradoxical vocal cord movement in a more mild form may be overlooked as cause for postoperative stridor and airway obstruction.

Key words

Complications: respiratory obstruction.

Acute upper airway obstruction is a serious medical condition which requires prompt diagnosis and treatment. There is usually an organic cause for the obstruction, but an increasing number of reports have documented cases without a definite organic aetiology.[1–8] Paradoxical vocal cord motion, an abnormal motion of the vocal cords characterised by their adduction during inspiration and abduction with exhalation, has been found in several of these patients.[2,5,6] The only reported case in the anaesthetic literature concerned a patient with a longstanding history of respiratory symptomatology and previously documented episodes of paradoxical vocal cord movement who had persistent postoperative stridor which required acute intervention and prolonged intensive care.[8] We now present a case of acute respiratory obstruction due to the condition after tracheal anaesthesia in a previously healthy patient without a history of any respiratory illnesses.

Case history

A 26-year-old white female was transferred to the University Women's Hospital during her 35th week of pregnancy with the diagnosis of incapacitating polyhydramnios. Her medical history was significant for one normal vaginal delivery and a dilatation and curettage performed under general anaesthesia. Both procedures were achieved without complications. She did not have a history of asthma, upper or lower respiratory tract infections or allergies. Except for pregnancy and polyhydramnios, her physical examination was unremarkable, in particular with respect to her head and neck, upper airway and pulmonary status. She was normotensive without oedema or other signs or symptoms of pre-eclampsia. Her prepregnant weight was 68 kg while her weight at admission was 85 kg, at a height of 1.60 m.

An abdominal ultrasound confirmed the polyhydramnios, and in addition, revealed the presence of a left diaphragmatic hernia in an approximately 36-week-old fetus. An amniocentesis demonstrated an immature lecithin sphingomyelin (L/S) ratio. She was kept in hospital and the delivery was delayed until extracorporeal membrane oxygenator support was available and the fetal lungs had matured. A mature L/S ratio was present one week later and an amniotic catheter was sited. Amniotic fluid was drained slowly over a period of 8 hours. The next day the patient had a spontaneous rupture of membranes and labour started. Internal uterine and fetal heart rate monitors were placed. The labour became irregular and dysfunctional at which time an intravenous infusion of oxytocin was started. Shortly after, late decelerations were noted on the fetal heart rate tracing. The oxytocin was discontinued and the patient was put in the left lateral decubitus position. Oxygen was given by nasal cannula. A fetal scalp pH was 7.32. The patient was managed expectantly for the next 15

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minutes and the decelerations diminished. A repeat fetal scalp pH was 7.28; fetal distress was diagnosed and an urgent Caesarean section was undertaken.

The anxious patient was given sodium citrate 30 ml by mouth and 100% oxygen by mask. General anaesthesia was induced in a 'rapid sequence' fashion with thiamylal 300 mg and suxamethonium 100 mg intravenously while cricoid pressure was maintained. The trachea was easily intubated with a 7.0 mm internal diameter, cuffed tracheal tube and anaesthesia was maintained with 50% nitrous oxide in oxygen and 0.5% halothane. The baby was delivered 5 minutes after induction; he appeared cyanotic, hypotonic, was not irritable and did not cry, but improved promptly after resuscitative measures. His Apgar scores were two and seven at 1 and 5 minutes respectively; the umbilical cord's venous blood pH was 7.33. The mother was given morphine 10 mg intravenously, and vecuronium 4 mg. The surgery proceeded uneventfully and finished 25 minutes later. General anaesthesia was discontinued and the muscle relaxant reversed with neostigmine 2.5 mg and atropine 1.2 mg.

Her trachea was extubated when she was fully awake and her muscular strength had returned as determined by the presence of an adequate tidal volume, extremity movement, and head lift. She was transferred to a stretcher while breathing well. Upon turning her to the lateral decubitus position she developed stridor followed shortly by the cessation of ventilation and signs of upper airway obstruction. The awake patient appeared in obvious distress and grasped her throat with her hands. Neck extension, jaw lifting, the insertion of a nasopharyngeal airway, and attempted positive pressure ventilation applied by way of a mask failed to relieve the obstruction and establish ventilation; her lips and nail beds became cyanotic. Laryngospasm was suspected and she was given thiamylal 300 mg and suxamethonium 100 mg, after which ventilation by mask was easy, while cricoid pressure was applied; her trachea was again intubated without difficulty with a 7.0-mm cuffed tracheal tube. No anatomical abnormalities, foreign bodies, or oedema were noted during direct laryngoscopy. Ventilation was unimpeded with bilaterally clear breath sounds; her oxygen saturation, measured by a pulse oximeter, was 99%. She received 50% nitrous oxide in oxygen to keep her sedated until a transcutaneous nerve stimulator demonstrated the spontaneous, complete return of the train-of-four and a sustained tetanus at 100 Hz.

The nitrous oxide was discontinued, and when she was awake and responding to commands with adequate muscle strength as determined by hand grip and neck lift, she was again extubated. She developed stridor immediately, which persisted despite manoeuvres including neck extension, jaw lift and positive airway pressure by mask. The patient sat up and improved only slightly. She alternated between severe stridor and mild to moderate dyspnoea over the next 15 minutes. Her oxygen saturation remained at 98–99% on room air, however; she was able to speak intermittently in a weak hoarse voice, and affirmed to difficulty with breathing and to a 'foreign body' feeling in her throat. She was unable to cough. Lignocaine 100 mg and hydrocortisone 100 mg were given intravenously without any apparent effect. The otolaryngologists were consulted urgently and fibreoptic nasopharyngeal laryngoscopy was performed. This revealed paradoxical movement of her vocal cords with intermittent episodes of normal function. No foreign body, oedema, bleeding or other evidence of trauma was present. No attempt was made to pass the fibrescope beyond her vocal cords to avoid further compromise of her airway. The patient's varying degree of respiratory difficulty, especially upon assuming the recumbent position, the inability to rule out subglottic abnormality, and her own request led to her re-intubation and sedation overnight. Ventilation was unassisted throughout the night. A chest X ray demonstrated the tracheal tube to be in a satisfactory position; no pulmonary abnormalities were noted.

A direct laryngoscopy and a bronchoscopy were performed under general anaesthesia the next day. No anatomical abnormalities, evidence of trauma, oedema, or infections were found. The patient breathed well after extubation while sedated; however, once she was fully awake in the recovery room, she developed a mild stridor, which disappeared after the patient was given reassurance. An interview with her husband revealed that the patient had been very tense and anxious in the weeks preceding her operation because of difficulties experienced during pregnancy. There was no history of psychiatric problems or of previous episodes that would suggest a conversion disorder. The patient returned to the obstetric service and was discharged in good condition 4 days later.

Discussion

Upper airway obstruction from paradoxical vocal cord movement may resemble airway obstruction from other causes; it must be differentiated from laryngospasm, foreign body aspiration, vocal cord paralysis, allergic reactions, trauma, infection, oedema, and tumours. Patients with the condition have been treated for uncontrollable asthma, anaphylaxis, laryngospasm, and vocal cord paralysis. Thus it is not surprising that several of the patients with this functional disorder have received aggressive therapy which included high dose steroids, tracheal intubation, and tracheostomy.[4,5,7,8] The diagnosis is made by evaluation of vocal cord motion during an attack. Appropriate therapeutic manoeuvres may be applied before having to resort to drastic treatment once the correct diagnosis has been reached. Most of the cases of paradoxical vocal cord movement, confirmed by laryngoscopy, have responded to verbal support, placebo treatment, phonation therapy, or psychological support.[5] Focused professional attention on the seriousness of the respiratory symptomatology of paradoxical vocal cord movement may magnify the symptoms as it reinforces the conversion reaction.

There appear to be several elements common to patients with this diagnosis. The majority have been women in the 18–50 age range who develop stridor or signs of upper airway obstruction, become tachypnoeic, contrary to what would be expected with an inspiratory obstruction, have a weak or absent voice, and are unable to cough.[3,5] Arterial blood gases have been largely normal during attacks.[3,7] The patients improved with the treatment described above, and psychological evaluation revealed a variety of psychiatric disorders with conversion elements. The patients are not consciously aware of the problem, and typically, cannot simulate the condition voluntarily.[5,7] The episode in some of them had been preceded by an upper respiratory tract infection or a surgical procedure with general anaesthesia. This led Kellman and Leopold to suggest that the upper

respiratory tract infection or tracheal intubation may direct a patient's attention to the larynx.[5]

The case presented here has several of the common elements described previously, including age, sex, characteristics of the obstruction, laryngeal findings, and the response to verbal support. She experienced psychological stress in the immediate peripartum period, but she did not have a history of conversion reactions, psychiatric disorders, respiratory disease or paradoxical vocal cord movement, unlike a previous case described in conjunction with general anaesthesia.[8] She had been under a significant amount of stress and it is indeed possible that the presence of a tracheal tube directed her attention to the larynx when she woke up after surgery. A complete psychological evaluation was not carried out. We were concerned about the potential for aspiration so the tracheal tube was kept in place until she was fully awake. The symptoms became apparent only after extubation. Sedative therapy with benzodiazepines or narcotics might have been a therapeutic alternative at that time, but sedation has had variable success and may fail and compromise the patient in the postoperative period.[7,8] Intubation and extubation at a deep level of anaesthesia has been advised in patients who have a history of the condition.[8] However, once the patient emerges from general anaesthesia, it may again present itself. Furthermore, as exemplified in this report, extubation at a deep level of anaesthesia may not be advisable. We elected to re-intubate the patient as a temporary measure to allow for postoperative stabilisation and further evaluation.

Recently, we cared for a 31-year-old woman who presented for a diagnostic laparoscopy. She had a history of stridor which developed after a previous laparoscopy and subsided with oxygen delivery by mask, and verbal reassurance. The patient underwent an uneventful surgical procedure under general tracheal anaesthesia and was extubated at the end of the case. She developed stridor and became anxious and tachypneic upon arrival in the recovery room, but remained well oxygenated. She was able to speak in a weak voice. Paradoxical vocal cord movement was suspected, but fibreoptic laryngoscopy was not available to confirm the diagnosis. She was given verbal support with instructions to concentrate on the expiratory phase of her respiratory cycle. The stridor disappeared with these measures. It recurred 20 minutes later, and again responded to verbal support. The patient was discharged home the same day in good condition. We suspect that this case was also caused by paradoxical vocal cord movement, even though we were unable to confirm it.

In milder forms the problem may be overlooked and be more common than generally acknowledged. If this is true, anaesthetists are likely to see patients with paradoxical vocal cord movement. Thus it should be considered in the differential diagnosis of postoperative stridor and respiratory obstruction; an increased awareness of this disorder should help to treat it correctly. However, the diagnosis should only be made after other causes of airway obstruction have been ruled out and the characteristic findings identified at laryngoscopy. We suggest further that if verbal reassurance and placebo intramuscular injections fail to relieve the symptoms, the administration of antipsychotic medications (haloperidol, droperidol) may also be considered in the acute phase as a temporary measure.

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