Reminder of important clinical lesson

Vocal cord paralysis following central line insertion in a neonate: a review of the literature

Faisal A Arshad, Chee Yean Eng, William Daw, Mahilravi S Thevasagayem, Neil Bateman

ENT, Royal Hallamshire Hospital, Sheffield, UK

Correspondence to Faisal A Arshad, fais121@hotmail.com

Summary

Acute onset stridor in a neonate following general anaesthesia is commonly attributed to laryngeal oedema secondary to anaesthetic airway trauma. The authors present a case of recurrent laryngeal nerve injury resulting in vocal cord paralysis causing stridor following central venous catheterisation under general anaesthesia in a neonate. This was managed expectantly and resolved spontaneously over a period of 6 months.

BACKGROUND

Central venous access is often necessary in neonates who require prolonged venous access for parenteral feeding, monitoring, drug administration and repeated blood sampling. Central venous catheters can be introduced peripherally (peripherally introduced central venous catheter (PICC)) or surgically placed in major neck or subclavian veins and tunnelled.^{1–4} In both techniques, placement could be percutaneous or open to expose the vessels. Given the proximity of great vessels, trachea, oesophagus, lungs and neural structures in the neck, the risk of injuries to these structures exist during catheter placement. Complications associated with central venous catheters placements are well documented.^{2 3 5–6} Stridor from neural complications associated with percutaneous Seldinger technique neck central venous catheter placements has been reported in adults,⁷ but has yet to be reported on open technique in neonates or children.

We present a case where a neonate developed acute inspiratory stridor following right-sided open neck tunnelled Broviac line insertion.

CASE PRESENTATION

We report on a child who was born via normal vaginal delivery at 35 weeks gestation (5 weeks premature) with a birth weight of 2.66 kg (50th centile) and with no antenatal problem or perinatal complications. He was discharged home the following day. There was no history of congenital stridor or intubation.

He presented 5 days later to Accident and Emergency with diarrhoea, severe dehydration, acute renal failure and metabolic acidosis. He underwent immediate endotracheal intubation and had mechanical ventilation initiated along with fluid resuscitation and commencement of intravenous antibiotics. He was successfully extubated in less than 24 h. There was no stridor or respiratory difficulty following this. He was subsequently diagnosed with microvillus inclusion disease, which necessitated complete dependence on total parenteral nutrition until his bowel transplantation. Central venous access was therefore indicated.

Under general anaesthesia, a PICC line was attempted but failed due to difficult peripheral access. He then proceeded to have open right Broviac line insertion (Broviac 4.2 Fr single lumen central venous catheter, cut down tray with SureCuff Tissue Ingrowth Cuff) into the right internal jugular vein. The skin incision was placed just above the right clavicle and the line was tunnelled subcutaneously. The operation was uneventful and he was intubated for just over 2 h with a 3.5 mm endotracheal tube.

The child developed inspiratory stridor with subcostal retraction immediately following extubation after the above central catheter placement. The initial working diagnoses were laryngeal oedema secondary to postintubation trauma or laryngospasm. He was treated accordingly with nebulised epinephrine, nebulised budesonide and oxygen for 48 h. There was minimal response. Despite his increased work of breathing with marked subcostal recession, he had normal respiratory rate and vital signs. After his stridor had persisted for more than 48 h, a paediatric otolaryngology opinion was obtained. Awake flexible fiberoptic laryngoscopy and upper airway endoscopy under general anaesthetic confirmed a paralysed unilateral right-sided vocal cord in the paramedian position. The cord was mobile to endoscopic palpation. The remaining supraglottis, glottis, subglottis and trachea appeared normal.

Given that he was able to maintain his oxygen saturation above 96% on room air comfortably, he was treated expectantly. A repeat awake fiberoptic laryngoscopy 6 weeks later showed return of right arytenoid movement. However, the right true cord was still fixed in the paramedian position. The child's stridor continued to improve and was only audible when he was excited. His subcostal and sternal retraction has also improved and is not present at rest.

It was felt that the unilateral vocal cord paralysis on this occasion was caused by injury (most likely neuropraxia) of the right recurrent laryngeal nerve given its close proximity to the inferior pole of the thyroid gland and the lower end of the internal jugular vein. In subsequent further follow-up at 6 months, the vocal cord paralysis showed spontaneous resolution and the child was asymptomatic.

DIFFERENTIAL DIAGNOSIS

- ▶ Laryngeal oedema
- ▶ Laryngospasm
- ▶ Pressure neuropraxia secondary to endotracheal intubation
- ▶ Direct trauma to the vocal cord from endotracheal intubation
- ▶ Vocal cord paralysis from neural complications arising from additional procedures such as central venous catheterisation
- ▶ Respiratory distress due to aspiration or infection.

OUTCOME AND FOLLOW-UP

A complete resolution of right vocal cord paralysis was observed. The patient was asymptomatic at 6 month follow-up.

DISCUSSION

Acute respiratory stridor or distress following general anaesthesia for a surgical procedure including central venous catheterisation is often attributed to laryngeal oedema or intubation trauma. The above case highlights that this is not always true and neural complications associated with central venous catheter placement should always be considered.

Pressure neuropraxia secondary to endotracheal intubation has been reported in the literature in the adult population.⁷ Nevertheless, existing literature failed to document whether central venous catheterisation was also performed as part of the surgical procedure.⁷ However, this mechanism of injury was unlikely in our case given that an uncuffed and correct-sized endotracheal tube was used. Also, the anaesthetist confirmed that the endotracheal intubation and extubation were atraumatic.

Injury to the vagus nerve was also considered given its close proximity to the internal jugular vein. Injury to the vagus nerve would give rise to the cord paralysed in the abducted position.⁸ Injury to the main recurrent laryngeal nerve (before branching) would result in the paralysis of the vocal cord in the paramedian position. Therefore, this child had neural complication to the recurrent laryngeal nerve as opposed to the vagus nerve given the paramedian position of the paralysed vocal cord.

Bilateral recurrent laryngeal nerve injury from thyroid surgery would usually result in bilateral vocal cord paralysis in the paramedian position and may condemn the patient to a permanent tracheostomy. Given that a neonate has a relatively smaller laryngeal airway, a tracheostomy should be considered if a baby failed to cope with a unilateral

vocal cord paralysis in the paramedian position narrowing the glottic airway. Fortunately, the neonate in our study did not require this.

The incidence of this complication could be underestimated given that some children are intubated for a prolonged period of time following central line placements and any temporary paralysis due to neuropraxia could have resolved spontaneously following extubation.

Treatment of unilateral vocal cord paralysis secondary to recurrent laryngeal nerve injury should initially be expectant if tolerated given that spontaneous recovery has been reported for up to 1 year.⁸ Corrective surgery should therefore be delayed during this time.

Learning points

- ▶ Not all acute onset stridor after general anaesthesia is due to intubation trauma, especially, when the duration of intubation is short and the anaesthetist can confirm an uncomplicated intubation or extubation course.
- ▶ Central venous catheterisation can cause recurrent laryngeal nerve injury resulting in vocal cord paralysis and stridor.
- ▶ Spontaneous recovery of neuropraxia injury can occur up to 1 year and therefore could initially be managed expectantly if well tolerated.

Competing interests None.

Patient consent Obtained.

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