# Congenital bilateral abductor vocal cord palsy in previous cesarean section in pregnancy - a case report

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#### **ABSTRACT**

A 34 year old G<sub>2</sub>P<sub>1</sub>L<sub>1</sub> at 35 weeks 6 days previous LSCS with bilateral congenital vocal cord palsy was referred to our tertiary care hospital with worsening of congenital stridor. On examination her respiratory rate was 32/min, heart rate was 132/min and temperature of 96.4°F. Obstetric examination revealed a gravid uterus of 36 weeks size, cephalic presentation with good fetal heart sound, not in labour. On further evaluation, ABG suggested worsening respiratory acidosis and hence taken up for emergency LSCS under SA and delivered a live baby girl of 2.5kg with good APGAR. She was intubated during immediate postoperative period due to acute respiratory acidosis. She was extubated after 24 hours once her arterial blood gas analysis (ABG) showed improvement. Postnatal period was uneventful. Otorhinolaryngologist planned for cordectomy after 6 weeks. Patient improved symptomatically and discharged on 7<sup>th</sup> postoperative day.

**Keywords:** Vocal cord, palsy, stridor, ventilatory support.

Congenital laryngeal lesion is although rare, may result in life threatening respiratory distress during pregnancy. Stridor and respiratory distress are seen in bilateral vocal cord paralysis <sup>2</sup>.

Due to physiological changes that occur during pregnancy, the larynx becomes more anterior and cephalad with oedema and is friable. Narrowed upper airway occurs due to splinting of the diaphragm by the gravid uterus. The rate of failed obstetric intubations is eight times higher than normal. Prolonged valsalva maneuver during delivery, even with tracheostomy in situ can result in subcutaneous emphysema<sup>3</sup>. Here we see a rare case scenario of a pregnant woman with acute stridor.

A 34 years old unbooked  $G_2P_1L_1$  with previous caesarean at 36 weeks gestation with known case of congenital bilateral vocal cord palsy, presented with worsening of congenital stridor and shortness of breath for the past 10 days to our high dependency unit (HDU). On general examination, she was conscious, oriented, afebrile, no pallor, tachypneic and tachycardic with loud breathing, high pitched whistling sound. Her blood pressure was 140/80 mmHg, respiratory rate was 42/min and heart rate was 123/min. Her cardiovascular system examination was normal and lungs were bilaterally clear on auscultation but conducted sound were present due to stridor. On obstetric examination, uterus was corresponding to the period of gestation, relaxed, fetal

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heart rate was 142/min and there was no scar tenderness. Ultrasound showed single live intra uterine fetus of 36 weeks gestation with normal umbilical artery doppler study. ABG (arterial blood gas) analysis suggestive of respiratory acidosis. Other routine investigations were normal. We proceeded with emergency LSCS in view of worsening stridor and delivered a baby girl weighing 2.5 kg with birth APGAR score of 8/10, 9/10 after 1<sup>st</sup> and 5<sup>th</sup> minutes respectively. Post operatively patient required ventilatory support in view of worsening of stridor and respiratory acidosis. Repeat ABG after 6 hours showed improvement. Extubation was done, 24 hours later after correcting the acidosis. Patient improved symptomatically and discharged on 7th postoperative day. She was followed up, two weeks after delivery and was found symptomatically better with reduced stridor. Her Otorhinolaryngologist had planned cordectomy after 6 weeks.

#### Discussion

Vocal cord paralysis poses obstetric challenge during labour due to unavailability of recommendations for the



Figure 1: Fiberoptic bronchoscopy image showing narrowed vocal cord with glotic stenosis

exact mode of delivery. The detoriation of the respiratory function is aggravated during the third trimester of pregnancy <sup>4</sup>. Emergency tracheostomy and elective caesrean section at 38 weeks of gestation is considered if the patient is clinically stable <sup>5</sup>, however there are some patients who delivered spontaneously at term.

Larygomalacia is the most common cause of congenital vocal cord paralysis. It can be acquired but most commonly idiopathic. Other causes are neoplasm of the lung, oesophagus, thyroid and larynx. Infections such

as Reye's syndrome, poliomyelitis, diphtheria, rabies, tetanus, syphillis, genetic X linked autosomal recessive disorder. The characteristic features of vocal cord paralysis are normal or near normal phonation with inspiratory stridor leading to complete respiratory obstruction. Symptoms are due to flaccid midline position of the vocal cords. Flexible laryngoscopy is the diagnostic tool for vocal cord paralysis as shown in the figure 1. **Conclusion** 

Congenital bilateral abductor vocal cord paralysis (CBAVP) is very rare and may result in aggravated life threating respiratory failure in pregnancy. Regular antenatal visits, early fetal surveillance, in hospital care from third trimester, elective caesarean is preferred for better maternal and fetal outcome.

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