Case Report

Neonatal and Pediatric Airway Team: A unique multidisciplinary concept in a childrens hospital for management of complex airway and pulmonary issues

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ABSTRACT

Concept of neonatal and pediatric airway team is somewhat new in India. In western countries this concept is also recent and limited to children's hospitals. We at Rainbow Childrens hospital, Delhi have put together first dedicated neonatal and pediatric airway team for comprehensive medical and surgical management of all neonatal and Pediatric patients with congenital or acquired lesions affecting the airway. We report a case of 15 month old girl with critical life threatening severe subglottic stenosis with pulmonary issues and failure to thrive who was managed in a comprehensive manner with the help of neonatal and Pediatric airway team of experts at our institution.

Keywords: Sub glottic stenosis, post intubation injury, Stridor, Pediatrics, Neonate, Airway team, Difficult airway, negative pressure pulmonary edema.

Case Report

A 15 month old female presented with, fever for 2 days, breathing difficulty for 2 days, decreased oral intake for 1 day before admission to a peripheral hospital. Child was apparently well 2 days ago when she developed fever which was moderate to high grade, not associated with chills or rigors, relieved on taking medicines. Child also had breathing difficulty in with rapid breathing associated with noise by history as given by mother both during inspiration and expiration, with chest and abdominal retractions. With these complaints child was taken to a hospital, where on examination child had low oxygen saturation levels, which improved on oxygen support, documented as biphasic stridor. A flexible fiberoptic bronchoscopy was attempted but could not be done as child developed life threatening hypoxemia associated with bradycardia. Hence the procedure was abandoned and child was referred to our hospital for stabilization and further management.

Birth history was uneventful. She was delivered by vaginal route, cried immediately after birth. There was no history of stay in the neonatal intensive care unit . Birth weight was 1.8 kg. She started to roll over at the age of 7 months. She had partial neck holding, had started to sit with support, was able to speak

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monosyllables, and had a unidexterous approach, not able to sit or stand with support.

Child was admitted to a small nursing home on 2 previous occasions with similar complaints. Upon 3rd admission 3 weeks later she had fever and was intubated and started on mechanical ventilation for approximately 7 days. Child was admitted at the hospital for total of 20 days. After discharge mother noticed some base line distress at all times. 2 days ago distress and noisy breathing seemed to have gotten worse and she was taken to peripheral hospital where bronchoscopy attempt was unsuccessful.

Upon arrival to our hospital in the emergency room examination revealed a 15 month old girl in respiratory distress refusing feeds, who was thin built with thin brittle hair. Weight was 5 kg. She had severe stridor that was biphasic, nasal flaring was present, with intercostal and marked subcostal retractions. Heart rate was 178/min, peripheries were warm to touch and peripheral pulses were well palpable, respiratory rate(RR) was 46/min, BP 101/64 Temp 99.6 °F, SpO_2 76-77% on room air, 92-93% on O2 at 2 lit/min. Bilateral air entry was equal. She was alert, making eye contact moving all 4 limbs. Muscle Tone was normal, DTR (Deep Tendon Reflexes) were present. She was admitted to the Pediatric intensive care unit(PICU)

A venous blood gas (VBG) upon admission to pediatric intensive care unit revealed pH 7.22, pCO2 55.5 and HCO3 22.7, CBC was 12000/cmm with unremarkable liver function tests, kidney function test, CRP (C- Reactive Protein) was within normal reference range. X-ray chest with neck AP view and lateral view are as shown (Fig 1(a) (b)). No major abnormality was identified except ballooning of hypopharynx seen in the lateral airway radiographs suggestive of upper airway obstruction and slight narrowing of the subglottic tracheal area at the vertebral level of cervical vertebra at C3-4.

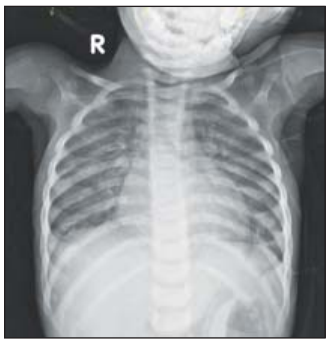

Figure 1(a): AP Neck and Chest radiograph (suggestive of narrowing of airway at C3-4 level though not very clearly seen)

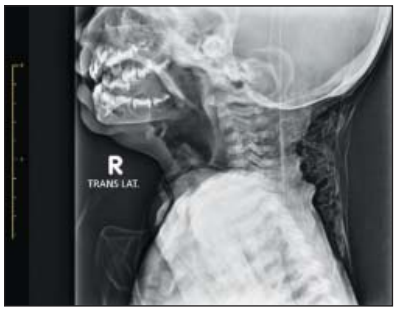

Figure 1(b): Lateral neck radiograph revealing ballooning of hypopharynx

Initial Management

Child was started on low flow oxygen at 2 litre/min and the SpO2 levels were ranging from 89-91%. She was then placed on HFNC (high flow nasal cannula) with FiO2 60% and flow rate of 10 lit/min. SpO2 improved to 95-96%. Child was started on IV Piperacillin, Tazobactam and oral Azithromycin. Nebulizations with Adrenaline and Levolin were started. IV Fluids were administered. There was no relief in the biphasic stridor over the next two hours.

Child continued to have respiratory distress and in view of life threatening critical airway obstruction, ENT opinion was sought and an emergent tracheostomy was performed in operating room with 3.5 uncuffed tracheostomy tube. A detailed upper and lower airway evaluation and bronchoscopy was deferred to a later date after achieving adequate

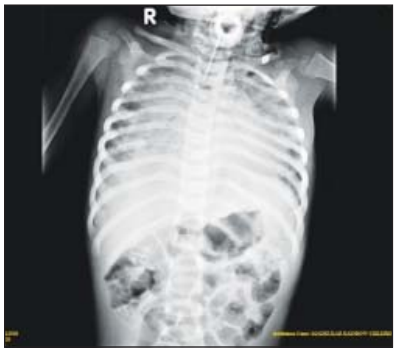

Figure 2: Post-tracheostomy chest X-ray showing pulmonary edema

airway with tracheostomy . Child continued to have distress and had oxygen needs postoperatively. Chest X-ray done post operatively (Fig 2) revealed evidence of pulmonary edema, most likely negative pressure pulmonary edema.Child was kept on mechanical ventilation overnight. In the next 24hrs there was marked improvement in respiratory status with tracheostomy in situ.

Negative pressure pulmonary edema (NPPE) was first described by Moore in 1927, essentially of two types[1]

1. **Type 1 NPPE** is seen with post extubation laryngospasm, epiglottitis, croup, choking/ foreign body (FB), strangulation, hanging. It occurs following acute obstruction of upper airway due to negative pressure generated in chest while struggling to move air into the lungs. This negative pressure leads to flooding of alveoli due to hydrostatic pressure of pulmonary capillary circulation exceeding the alveolar pressure.
2. **Type 2 NPPE** seen after relieving of chronic upper airway obstruction. Due to chronic obstruction there is generation of auto PEEP, with an increase of end expiratory lung volume. When this obstruction is relieved auto PEEP disappears, lung volume and pressure returns to normal, resulting in transudation of fluid in the lung interstitium and alveoli.

Case Progression

Child was continued on same treatment with intravenous antibiotics, oxygen support and close monitoring in PICU. Gradually her condition improved, she was weaned off to room air. She failed to gain weight while in the hospital, stool examination showed cysts of Giardia, She was started on oral metronidazole and a detailed diet chart was also prepared in view of poor weight gain to maximize her daily caloric and protein intake. Her echocardiogram (ECHO) revealed no cardiac abnormality or any suggestive finding regarding a vascular ring or extrinsic compression of the upper airway.

There were persistent bilateral infiltrates suggestive of lower respiratory tract infection (Fig 3 and 4), Venous blood gas done showed compensated respiratory acidosis: pH 7.38 pCO2 56 HCO3 38. While during stay in the PICU on day7, the child developed new fever, her tracheal secretions were thick and purulent, and she was having tachypnea. A hospital acquired superinfection was suspected on top of interstitial pulmonary involvement. She was started empirically on Amoxicillin with clavulanic acid; CRP was185, CBC revealed a white count of 9.500/cmm, Tracheal aspirate cultures showed Acinetobacter sensitive to Colistin only. She was started on colistin nebulization.

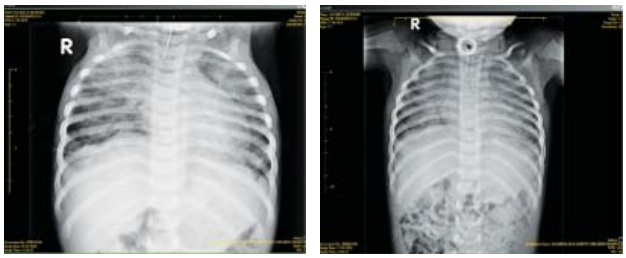

Figure 3 and 4: Chest radiograph revealing bilateral infiltrates

Fever subsided however tracheal secretions were remained thick and purulent and there was not much clearance on Chest radiograph. Child was started on IV colistin. A CECT (Contrast Enhanced CT) chest and neck was performed (Fig 5 (a) (b)) preceded with with a flexible fiberoptic bronchoscopy for evaluation of the upper airways as well as airway beyond the region of stenosis and the status of lungs, to obtain a bronchoalveolar lavage. Bronchoscopy (size 2.8 mm outer diameter) revealed normal nasal airway and vocal chord movements, depicting a pin hole appearance at the laryngeal inlet just below the level of vocal chords (Fig 6a-b). Bronchoscope was then also passed via tracheostomy and revealed evidence of tracheal inflammation, with normal anatomy of distal airways and purulent secretions suggestive of lower respiratory infection. BAL was negative for AFB and fungal cultures. Baby's HIV status was negative. Child was weaned off oxyegen with good spontaneous respiratory effort.

Her tracheal secretions decreased and the child began to gain weight.

A detailed discussion with family was held by the

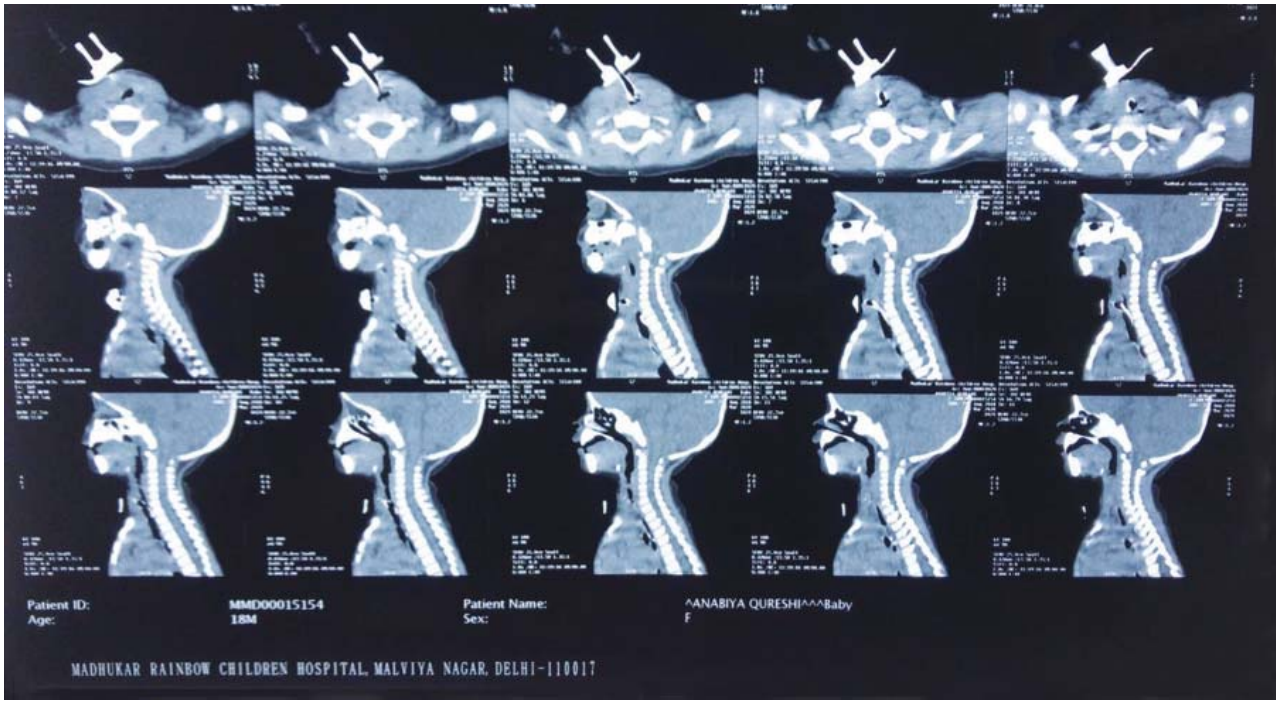

Figure 5 (a): Contrast enhanced CT neck and chest: Diffuse lung disease mostly ground glass type suggesting partial alveolar filling and greater interstitial involvement, with small subglottic stenotic segment with pinhole stenosis of airway.

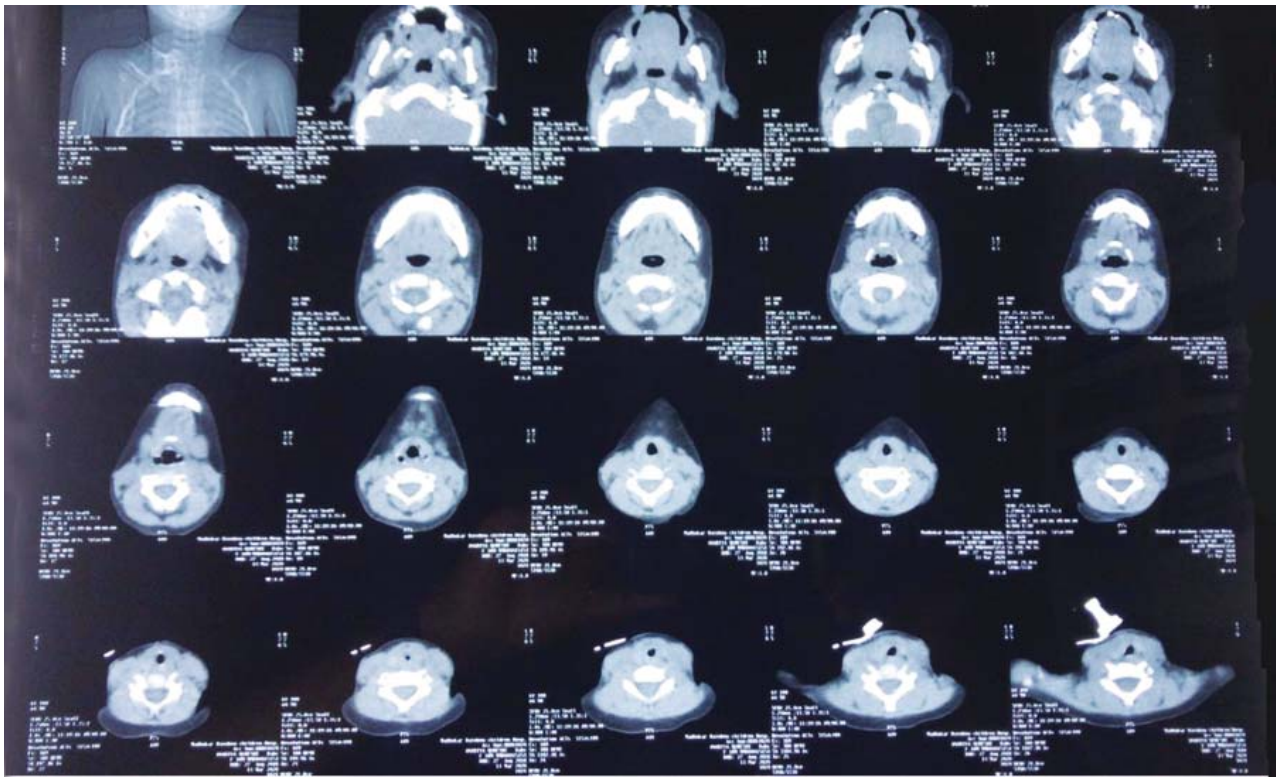

Figure 5(b): CE CT Neck Airways (Transverse views) post tracheostomy revealing pin hole subglottic lumen

Flexible fiberoptic bronchoscopy reveals evidence of Subglottic stenosis and fibrosis

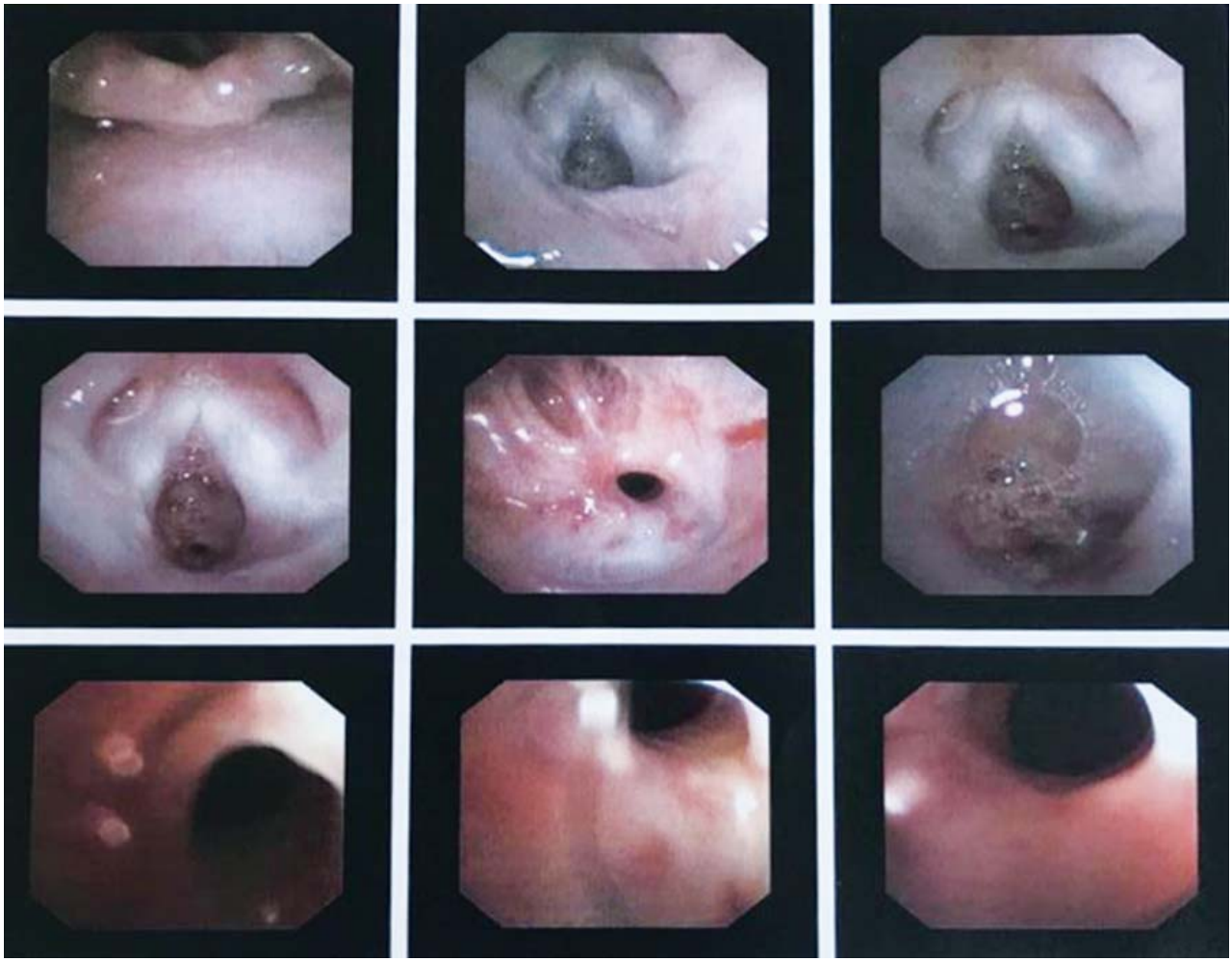

Figure 6(a): Bronchoscopy report images revealing clear evidence of Subglottic stenosis

airway team (Pediatric intensivist, ENT specialist, Pediatric surgeon and Anesthesiologist) and the plan for further management of subglottic stenosis was discussed. A combination of rigid and flexible bronchoscopy for upper airway evaluation was planned to be undertaken in the operating room with a plan to intervene surgically as required by ENT and pediatric surgeon.

All the possible options including dilatation of the stenotic segment with or without surgical intervention were discussed and risks and benefits informed to the parents . In view of fibrosis in the subglottic space a decision was taken to repair it surgically with cautery or laser and Mitomycin C and follow it up with balloon dilatation of subglottic space.

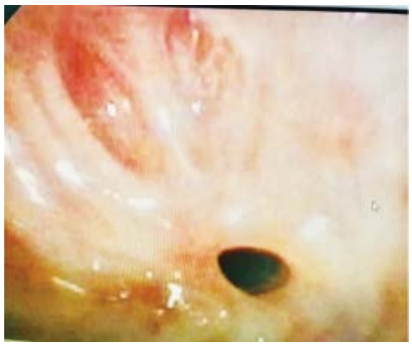

Figure 6 (b): Bronchoscopic view revealing pin hole stenosis with fibrosis

The procedure was jointly performed by ENT and Pediatric surgeon. Resection with cautery and use of Mitomycin C to widen the airway was undertaken

via a rigid bronchoscope while anesthesia was administered via tracheostomy tube with adequate airway control.This procedure was followed by a check bronchoscopy performed by a flexible fiberoptic bronchoscope (Fig 7)

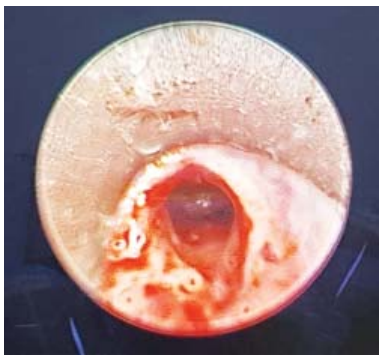

Figure 7: Subglottic repair (view via a rigid bronchoscope) revealing wider subglottic lumen

Cauterization was used to resect subglottic fibrous membrane. Mitomycin C (MMC)was used in the subglottic area locally to help with inhibition of fibroblast proliferation and possible reduction of further fibrosis. After the procedure child was discharged home in stable condition with tracheostomy in situ. Weight at the time of discharge was 6.1 kg. Plan was to evaluate upper airway in 3-4 weeks with possible balloon dilatation of subglottic stenotic segment if necessary. There was a delay on part of the family for regular evaluation as the child was doing well at home.

2nd Admission

4 months later child was admitted with complaints of fever and poor oral intake to the pediatric intensive care. On examination: weight was 6.4 kg, tracheostomy was intact. Child had tachypnea, increased work of breathing. There were subcostal and intercostal retractions. She was febrile (temperature 103□), tracheal secretions were thick. Heart rate: 178/min, peripheries were warm, SpO2 86% on room air and 97% on O2 at flow rate of 2 lit/min. Upon auscultation of lungs, crepitations were present on the both sides in the infrascapular and interscapular region.

Child was started on ceftriaxone, and admitted for close observation and further progression. Initial WBC count was 17560/cu mm with CRP of 47, saturations were maintained on $2LO_2$ via tracheostomy.

Bilateral haziness with diffuse inhomogeneous opacities were noted on chest X-Ray. Initial admitting diagnoses was interstitial pneumonitis with superadded right pneumonia. A chest X ray revealed right parenchymal and perihilar infiltrate (Fig 8).

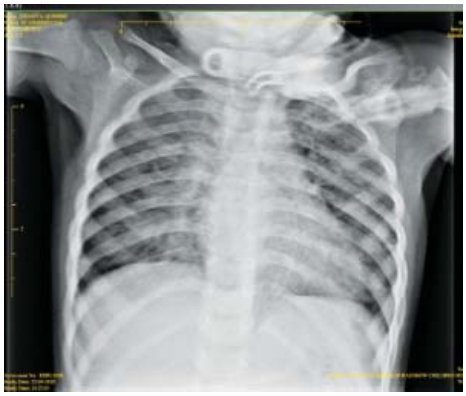

Figure 8: Chest Radiograph on second admission revealing diffuse infiltrates on right side

Tracheal aspirate culture showed heavy growth of pseudomonas aeruginosa. Antibiotics were upgraded to Piperacillin Tazobactam. Child developed generalized edema, received albumin infusion. Gradually the general condition improved and fever subsided. Oxygen support was tapered and stopped.

An expert review by pediatric airway team was called in regarding management plan for tracheostomy. A flexible fiberoptic bronchoscopy was planned to evaluate the staus of sbglottic area and plan the way forward to aim towards decannulation.

Repeat bronchoscopy showed an improving subglottic stenosis and flexible bronchoscope was

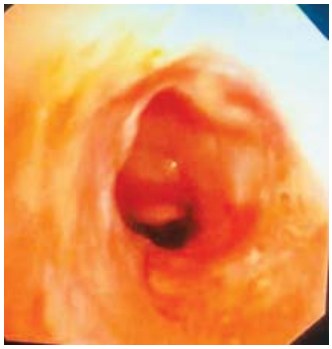

Figure 9(a): Subglottic tracheal bronchoscopic view after repair

passed to the level of tracheostomy and beyond with normal distal trachea (Figure 9 a-b).

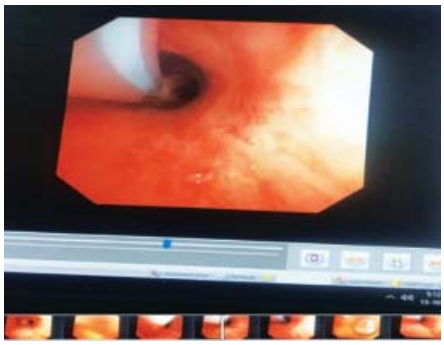

Figure 9(b): Distal trachea intact with adequate space around tracheostomy allowing partial occlusion of Tracheostomy and subsequent decannulation

Tracheostomy was partially occluded by tape in a gradual way starting with occlusion of 1/3rd of lumen for 6 hours followed by 50% occlusion at the proximal end of tracheostomy for 24 hours followed by complete occlusion for another 12 hours under close monitoring of respiratory status. The child did seem to tolerate occlusion of tracheostomy without desaturations or CO2 retention. After 72 hours of gradual increments in degrees of occlusion, child was finally decannulated and she maintained her respiratory effort and saturations well on room air. A chest X-ray done immediately after decannulation of tracheostomy tube revealed good expansion of both lungs. (Fig 10)

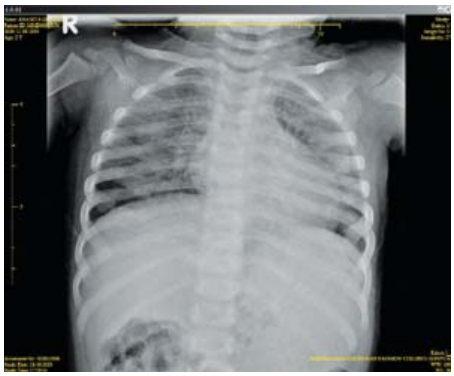

Figure 10: Chest Radiograph after decannulation of tracheostomy

Discussion

This case demonstrates the multidisciplinary approach to a child failure to thrive severe lower respiratory tract infection requiring mechanical ventilation possibly leading to subglottic stenosis progressing to critical life threatening upper airway obstruction. Following discussion will include all the issues related to neonatal and pediatric airways as they were managed in a comprehensive way by the multidisciplinary approach with the help of neonatal and Pediatric airway team.

The Infant Airway is different (Fig 11) when compared to adult airway[2,3].

Compared to adults, in infants the airway structures are smaller, softer, collapsible and anteriorly placed. Epiglottis is floppier, tongue is large. Head is large compared to body. Airway is easy to obstruct and even a blocked nostril can cause breathing issues as children are obligate nose breathers while adults breathe by mouth as well as nose.

Infants and younger children predominantly use diaphragm to breathe, as the capacity for chest expansion is not adequate. Ongoing development, size and curvature of the ribs with resultant mechanical efficiency of bucket handle movement at costovertebral joints is less as compared to older patients. Infant in respiratory distress, therefore tends to have tachypnea, rather than increased depth of breathing. The closing volume is greater than the functional residual capacity (FRC) until 6-8 years due to the poor elastic properties of infant lungs, so airways closure occurs during normal tidal ventilation. Functional residual capacity is relatively low (an indicator of lung reserve/store for oxygen in an apneic patient). Diaphragmatic muscle fibers distribution is such that majority of fibers have a high mitochondrial content, therefore consuming higher oxygen (6-7ml/kg/min) compared to adults (3-4 ml/kg/min) , causing further stress upon cardiopulmonary reserves to meet the higher oxygen demand whenever there is airway (upper or lower) or lung parenchymal pathology impinging upon oxygenation status. Due to above mentioned differences in anatomy and physiology of airway in children when compared to adults ,infants pose a greater challenge due to frequent acute life threatening episodes(ALTE) without giving adequate time to families or a general pediatrician to get expert specialist attention of a pulmonologist or an ENT specialist. Due to lesser reserves (relatively

smaller functional residual capacity) and high vagal tone and easier tendency for bradycardia secondary to mild hypoxemia ALTEs tend to be more common. This has also been incriminated as one of the many possible factors contributing to Sudden infant death syndrome(SIDS) in infants. Stridor (or noisy breathing), respiratory distress, and poor feeding with or without cyanosis may be the only signs that bring the patient to medical attention(as was seen in our patient).

The subglottic area around the level of cricoid ring is the narrowest part of the airway that is only 4–5 mm wide when compared to the glottis which is the narrowest part in adults.

Subglottic stenosis (SGS) can be congenital (quite rare) or acquired. The most common cause of acquired SGS is prolonged intubation. The narrowest and most susceptible area of the subglottic trachea is the portion circumscribed by the cricoid cartilage as opposed to other portions where the cartilage rings are incomplete and the dimensions are wider. Therefore, the presence of an endotracheal tube in this area, along with other factors, may cause edema, ulceration and necrosis of subglottic structures and may lead to the development of stenosis. Clinical presentation is characterized by the onset of varying degrees of dyspnea and stridor in the post-extubation period which can progress in severity.

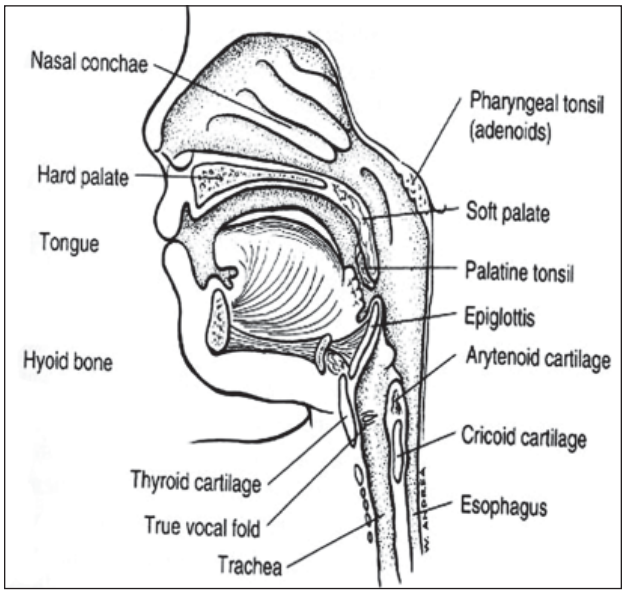

Figure 11: Infant Airway

Incidence of subglottic stenosis in children (median age 3.1 months; range 0.3–59.5) after prolonged intubation is 11.3%.

Degree of subglottic obstruction has been divided into 4 grades[4] in order of severity of obstruction

12.5% are grade I (<50%obstruction);

31.25% grade II (51%–70%obstruction);

37.5% grade III (71%–99%obstruction); and

18.75% grade IV (>99%obstruction)

For every five intubation days, there is 50.3% increase in the risk of developing SGS[5]

How can Intubation Cause Subglottic Stenosis?

The cause of subglottic stenosis suggested by many authors is a tracheal localized ischemic necrosis and an excessive granulation tissue formation with a process of scar contracture.

Need of multiple sedatives, presumably for agitation with resultant rubbing between the endotracheal tube (ETT) and the tracheal mucosa, correlates with increased risk. Whether the ETT had a cuff did not influence the incidence of SGS. Neonates have a soft cricoid cartilage and are less susceptible, with an estimated incidence between 0-2%.

Some authors also suggest that trauma during intubation itself can be a cause of subglottic stenosis, which explains development of subglottic stenosis after even a short duration anesthesia.

Prolonged Intubation

Definition of prolonged intubation has always been a matter of debate. Some authors have defined prolonged intubation as intubation in excess of seven days[6]. Others defined prolonged intubation as longer than three days of endotracheal intubation. No matter what the definition, it is quite apparent that extended periods of endotracheal intubation can cause serious complications. Astrachan[7] observed that complications were four times more likely to occur following endotracheal intubation than following tracheostomy. Among these complications of endotracheal intubation most common complication was subglottic stenosis. As the length of the period

of intubation increased, the incidence of subglottic stenosis also increased.

Cuffed vs Uncuffed Tube

The experience of Black et al. on 2953 pediatric patients intubated by uncuffed tubes in intensive care unit reports that none of the patients had clinical symptoms of acquired subglottic stenosis[8].

Other authors showed that the use of cuffed endotracheal tubes in pediatric patients was not associated with an increased risk of subglottic stenosis and it avoided the unwanted aspiration of gastric contents[9].

Other Factors Leading to Subglottic Stenosis[10]

The mean age of the patients with SGS detected by bronchoscopy was 12.6 months. The female/male ratio was 0.9. The mean duration of intubation was 27.5 days in the patients with SGS, whereas it was 9.9 days in the patients without SGS. In the SGS group, the mean leukocyte count was 16988/mm^3 (min-max: 5600 37800/mm^3) and the mean CRP value was 15.1 (min-max: 0.02-70), whereas in the patients without SGS had a mean leukocyte count of 13558/mm^3 (min-max: 2400-36120/mm^3) and a mean CRP value of 7.3 (min-max: 0.02-50). An intake of oxygen at a concentration of 30% and over during intubation was required in 31 patients with SGS (62%) and in 14 patients without SGS (26%) to maintain the oxygen saturation level at 95% and above[11].

The duration of intubation, increased levels of leukocytes and CRP, and hypoxia were important factors for the development of SGS in intubated patients.[12]

Management of Subglottic Stenosis

Bronchoscopy is the mainstay of diagnosis of post-intubation tracheal stenosis. Computerized tomography (CT) will reveal the exact location and extent of the stenosis. Chest X-ray may be performed but rarely detects stenosis due to smaller diameter of the neonatal and pediatric airways. Virtual bronchoscopy done with reconstructed CT images is available to measure the length of stenotic segment and any extra luminal obstruction, however it tends to miss dynamic obstruction related measurements and precise nature of intraluminal obstruction with respect to specific mucosal changes in various pathological diagnoses.

With help of flexible fiberoptic bronchoscopy the degree and extent of obstruction can also be evaluated and this helps in classifying the degree of stenosis. This is important as it determines the type of treatment options available and its outcome. For example; for a type 4 stenosis, treatment options may include tracheal resection and reconstruction and stent implantation. As a word of Caution; as per American thoracic society tracheal stents are not recommended in Pediatric age group due to chances of dislodgement ,however with more and more clinical reports getting published as well as the availability of biodegradable stents this controversy may be resolved sooner.

There are many more options for the treatment of tracheal stenosis following prolonged endotracheal intubation.

In a case series 32 patients with post-intubation tracheal stenosis were treated with rigid bronchoscopy with laser resection or stent implantation as the management of choice. The authors reported that laser resection cured 66% of tracheal stenosis patients, while stents were inserted for the remaining patients[12]. Mitomycin C (MM) was used in our patient as a local application in the resected raw subglottic area to help with inhibition of fibroblast proliferation and possible reduction of further fibrosis. MMC application has been found to be beneficial in few studies published on surgery for laryngeal stenosis but long term effects on reduction of incidence of restenosis has not been studied[13,14]. Another method of managing tracheal stenosis is balloon bronchoplasty[15]. This can be done either with rigid or flexible bronchoscopy. The efficacy and safety of balloon bronchoplasty was evaluated in 26 patients in a 2007 study conducted by Freitag et al[10]. They reported a success rate of 100% when balloon dilation was used as part of a multimodal approach to the management of tracheal stenosis. As there were no adverse events reported, they concluded that balloon

bronchoscopy is an effective and safe way to manage tracheal stenosis. Bougienage was carried out at our center instead because balloon bronchoplasty was not available.

Concept of Neonatal and Pediatric Airway Team (Fig 12)

Availability and organization of airway team for neonates and older children is a unique concept[16]. However at childrens hospitals such a team should not be difficult to organize in view of availability of various pediatric specific superspecialists such as Pediatric intensivists, neonatologists, pulmonologists, pediatric anesthesiologists, pediatric cardiologists, pediatric radiologists, pediatric otolaryngologists, pediatric gastroenterologists and pediatric surgeons (general, plastic, thoracic and vascular surgeons) in addition to pediatric critical care nurses, trained neonatal and pediatric technical staff for bronchoscopy as well as ready availability of all sizes of neonatal and pediatric airway equipment. A group of skilled individuals with experience in dealing with critically ill meomates and children is the key to success of such a team.

To our knowledge this the first ever organized neonatal and pediatric airway team in India. We have found it useful to manage cases of difficult airways as well as complex patients with syndromes associated with multiple congenital anomalies who need expertise of various other medical services such as genetics, metabolic, endocrinology, neurologist, developmental or specific surgical services such as pediatric neurosurgery, ophthalmology or orthopedics. All this is possible at a dedicated hospital or health care facility specifically for children.

Figure 12: Neonatal and Pediatric Airway team

(Pediatric Anesthesthesiologist, Pediatric intensivist, Pediatric pulmonologist, Neonatologist, Pediatric Otolaryngologist, Pediatric surgeon, Pediatric and neonatal nursing and technical team)

Neonatal and Pediatric Airway Team Equipment (Fig 13-17)

1. Flexible neonatal and Pediatric bronchoscopes
2. Video laryngoscopes
3. Rigid bronchoscopes
4. Pediatric endoscopes
5. Echocardiography
6. Pediatric Radiology with facilities such as

CECT with airway reconstruction and Fluoroscopy

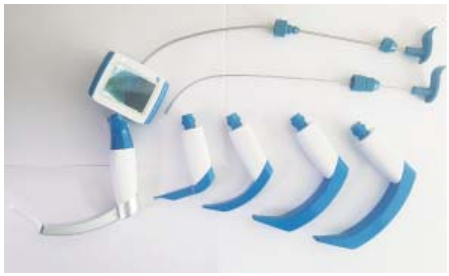

Figure 13: Video laryngoscopes: Neonatal to Pediatric sizes

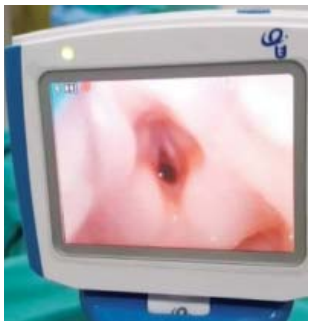

Figure 14: Video laryngoscopic view of pediatric airway (laryngeal inlet)

Figure 15: I-gel Equipment for Neonatal and Pediatric airway

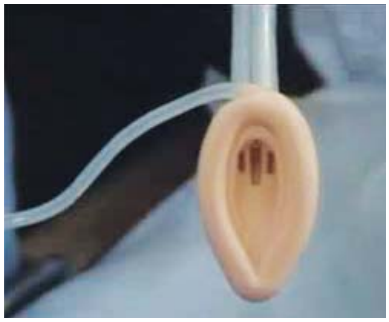

Figure 16: Pediatric Laryngeal Mask Airway

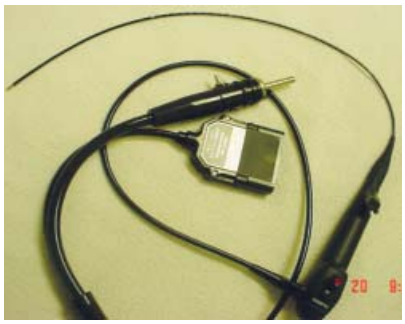

Figure 17: Neonatal and Pediatric ultrathin bronchoscopes (Size 1.8 mm-2.8 mm -3.5 mm)

Conclusion

As this case report (amongst many other children with difficult airways treated at our hospital) demonstrates that airway issues such as subglottic stenosis are common in neonates and pediatric age group. Due to differences in anatomy and physiology of airway in children when compared to adults , younger children pose a greater challenge due to frequent acute life threatening episodes(ALTE) without giving adequate time to a general pediatrician or families to get expert medical attention. Due to lesser reserves and high vagal tone and easier tendency for bradycardia secondary to even a mild degree of hypoxemia ;ALTEs tend to be more common. Stridor (or noisy breathing) , respiratory distress , and poor feeding with or without cyanosis may be the only signs that bring these children to medical attention. Diagnosis and management of subglottic stenosis or other airway lesions is complex and multidisciplinary in nature in many infants. This is often achievable at a dedicated childrens hospital to be able to deliver a comprehensive holistic care to the child. Concept of having a dedicated neonatal and Pediatric airway team to consult and intervene in a timely fashion can be life saving and achieves the best results for the specific airway problem and impact favourably on the outcomes directly related to long term growth and development of the child

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