J Anesth (2012) 26:456–459
DOI 10.1007/s00540-012-1340-7

CLINICAL REPORT

Stridor due to an innominate artery compression and posterior mediastinal mass in a pediatric patient

Edwin Abraham · Tariq Parray · Kim Poteet-Schwartz

Received: 17 August 2010 / Accepted: 15 January 2012 / Published online: 14 February 2012

Abstract There are many causes for stridor in a pediatric patient. We present an interesting case of a pediatric patient who had stridor due to an innominate artery compression and posterior mediastinal mass. We discuss the anesthetic complication and management of patients with stridor.

Keywords Stridor · Posterior mediastinal mass · Innominate artery syndrome

Introduction

Stridor is a harsh vibratory sound of variable pitch caused by partial obstruction of the respiratory passages. This results in turbulent airflow through the airway. Although stridor can be a sign of a benign process, it may be the first sign of a serious and even life-threatening process. The causes of stridor can be categorized by location: supraglottic, glottic, and subglottic obstruction. We present a unique case of a child with stridor due to innominate artery compression syndrome (IACS) and a posterior mediastinal mass. This case highlights some of the complications in managing pediatric patients with stridor.

Case report

A fourteen month old boy presented to the Ambulatory Surgery Center for sedation for a computed tomography (CT) scan. Medical history revealed a 3 month history of worsening stridor, cough, dysphagia, and respiratory distress as well as a recent emergency department (ED) visit several weeks prior to this visit. While in the ED he had an abnormal chest X-ray read as anterior tracheal compression from the innominate artery, and received a recommendation for a follow-up evaluation by CT.

The mother described the stridor as having becoming worse over the past 3 months to the point that the child would fall asleep sitting up with his neck hyperextended, with nasal flaring and suprasternal retractions. The stridor did not improve in any body position (supine vs. prone vs. lateral), and worsened while he was asleep. His dysphagia was also pronounced; he would attempt to swallow food, vomit, and reingest the food in smaller pieces.

Physical examination while asleep on the mother's lap revealed a physically healthy appearing child in mild respiratory distress. He had mild nasal flaring and suprasternal retractions with biphasic stridor. Expiratory stridor was greater than inspiratory stridor, with a prolonged expiratory phase and coarse breath sounds throughout. Cardiac examination was normal without murmurs. Upon awakening, his respiratory status improved, stridor decreased, and his oxygen saturation (SaO_2) was 98% on room air.

General anesthesia with inhalational induction was performed with the child in the lateral position for the CT scan. A 4.0 cuffed ETT was used, and the intubation was easy with only propofol 20 milligram (mg). There was initial difficulty in ventilation which improved after the ETT tube was advanced from 11 cm to 14 cm at the lip. The child was extubated awake following the CT but before the CT scan was read. He was desaturating into the 80s with moderate cyanosis which responded to deep suctioning and a racemic epinephrine nebulizer treatment.

E. Abraham (✉) · T. Parray · K. Poteet-Schwartz
Department of Pediatric Anesthesiology and Pain Medicine, Arkansas Children's Hospital, 1 Children's Way, Slot 203, Little Rock, AR 72202, USA
e-mail: abrahamedwinj@uams.edu

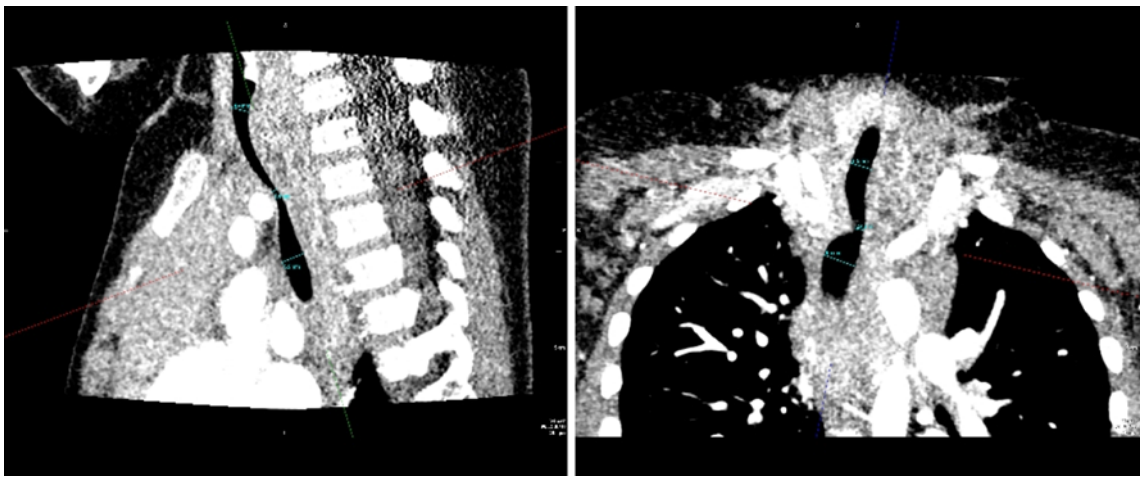

Fig. 1 CT scan showing tracheal stenosis from posterior mediastinal mass and innominate artery syndrome

His chest CT showed severe airway obstruction with an intubated tracheal diameter measuring 3.5 mm × 1.7 mm and anterior compression by the brachiocephalic artery crossing anterior to the trachea (Fig. 1). A posterior mediastinal mass was seen on the CT which was anterior to the spine and dorsal to the airway. The child was admitted to the Pediatric Intensive Care Unit (PICU) post scan.

The following day, the child underwent a general endotracheal anesthetic with an uneventful gentle mask induction for a thoracoscopy and biopsy of the posterior mediastinal mass. The patient received only fentanyl 40 micrograms (μg), glycopyrulate 80 μg, and was maintained on 50% oxygen throughout the case. Postoperatively the patient remained intubated and returned to the PICU due to concern over the mediastinal mass. Preliminary biopsy results were reported as neuroblastoma, but final frozen section results showed no malignant cells, only reactive lymphadenopathy. He had started all the pre-chemotherapy diagnostic testing, including CT of the head/abdomen/pelvis, bone marrow aspirate, labs, and cardiac echocardiography; all were negative. Five days after the initial thoracoscopy, the child returned to the operating room for a second thoracoscopy with rigid bronchoscopy and for retrieval of additional tissue from the posterior mediastinal mass.

Pre-operative anesthetic evaluation prior to second thoracoscopy was conducted. The patient's vital signs, while stable, revealed a sinus bradycardia in the low 80s and a low-grade fever of 38°C. Also noted was a weight increase from admission (4 days prior) of a kilogram. On physical examination, his lung sounds were coarse with rhonchi throughout and his cardiac exam was normal. Laboratory tests showed a hemoglobin of 8.5 g/dl and hematocrit of 28.1 g/dl. Oxygenation saturation was 98% on 40% FiO_2 with pressure support of 15 mm of mercury. Chest X-ray was read as atelectasis versus developing pneumonia. The general surgeons had requested this thoracoscopy with biopsy followed by rigid bronchoscopy. ENT was called and made aware of the possible need for emergency back-up for this case.

After transporting him to the operating room, the patient was connected to the anesthesia ventilator and maintained at 50% FiO_2. The patient was given propofol 30 mg, fentanyl 50 μg, glycopyrrolate 100 μg and maintained on the morphine and versed drips from the PICU. Sevoflurane was used during the procedure to maintain anesthesia. Initially the patient was kept on spontaneous respiration, but rocuronium 6 mg was given in the middle of the procedure by the request of the surgeons when they were dissecting to the mass.

Throughout the case, the patient required intermittent manual ventilation because of desaturation that responded to increased tidal volume and 100% FiO_2. Approximately 1 h into the procedure, the surgeons requested the placement of a radiopaque esophageal dilator to direct dissection around the esophagus and biopsy of the mass. The dilator was placed by the attending anesthesiologist with protection of the endotracheal tube on insertion. Following the dilator placement, ventilation deteriorated almost immediately. Attempts to suction and pass a fiberoptic bronchoscope down the ETT were unsuccessful. The position of the ETT was checked and the surgeon was informed that it had not changed. The dilator was removed while the patient was in the lateral decubitus position without any improvement in ventilation; he was then turned supine.

The surgeon placed chest tubes which were connected to suction; however, ventilation and oxygenation progressively worsened. The child's SaO_2 was in the mid-80s and decreasing. An emergency was declared; the OR staff set up for a rigid bronchoscopy and ENT was called into the room. At this point, the SaO_2 was in the mid-70s and the EKG showed inverted T-waves. The OR table was turned

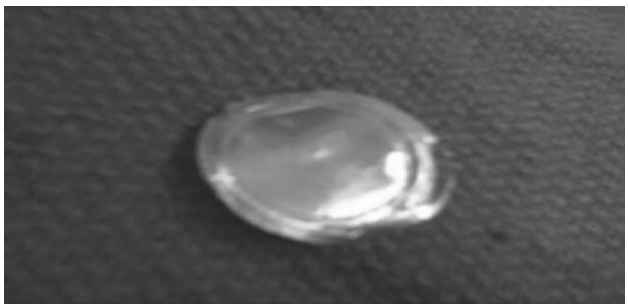

Fig. 2 Foreign body found in the esophagus

with the ETT still in place, and the rigid bronchoscope was placed. The patient was extubated and ventilation was accomplished via the side port of the bronchoscope. The ETT lumen was filled with solidified secretions, explaining the inability to pass the suction catheter or fiberoptic bronchoscope. Ventilation and SaO_2 improved, but the ST segments did not return to baseline until about 10 min into the bronchoscopy. Copious, tenacious secretions were repeatedly suctioned from the bronchi.

Following the bronchoscopy, the patient was re-intubated with a 4.0 uncuffed ETT and an esophagoscopy was performed. The patient was connected to the ventilator and there was great improvement in ventilating the patient. The FiO_2 was able to be titrated down to 50% and the patient had adequate tidal volumes on the ventilator. During the esophagoscopy, a thin round foreign body was extracted from the esophagus. The foreign body was determined to be a 3 cm piece of paper from the top of a juice bottle (Fig. 2).

The child remained intubated and on the ventilator for an additional 4 days and was started on a course of antibiotics for bilateral pneumonia. Repeat CT scan results showed complete resolution of the soft tissue mass in the posterior mediastinum. The child's pneumonia had improved and the patient was taking good tidal volumes on minimal pressure support and weaned to room air. The child was extubated following the results of the CT and transferred to the floor for completion of his course of antibiotics. The dysphagia had progressively improved and the patient was feeding normally by a few days after extubation. He was discharged home without any further airway events and his stridor completely resolved.

Discussion

IACS is a type of vascular ring, but not a true complete anatomical ring that can cause tracheal compression. IACS is caused when the innominate artery branches off the aortic arch distally and posteriorly, compressing the trachea anteriorly in the thoracic inlet as it passes from the mediastinum to the right arm [1]. Symptoms present in the first year of life in 50–70% of patients; the most common presenting symptom is stridor [2]. Most children with IACS are asymptomatic or have mild symptoms during infancy and the compression decreases with age. As the child grows and the tracheal cartilage matures, the patient's respiratory symptoms will lessen and disappear [3].

The etiology of this patient's stridor and tracheal compression was both innominate artery compression anteriorly and compression posteriorly by a mediastinal reactive lymphatic mass secondary to the esophageal foreign body. Most masses in the posterior mediastinum are neurogenic in nature. These can arise from the sympathetic ganglia (neuroblastoma) or from the nerve roots (schwannoma or neurofibroma). It is important not to forget lymphadenopathy and the vertebrae and descending thoracic aorta as sources of a mass in this space, as seen with this patient.

Spontaneous ventilation should be maintained, avoiding muscle relaxants in patients with tracheal compression and unexplained stridor. There could be a risk of tracheal compression after paralysis, but this is more likely with anterior mediastinal masses. Preparation for airway emergencies and, in some cases, back-up extracorporeal membrane oxygenation (ECMO) may be necessary. It is important to anticipate problems when dealing with patients with mediastinal masses, and help should be readily available. In the second surgery we had ECMO on backup, and for the third surgery we had ENT nearby in case the airway was lost during bronchoscopy.

Esophageal foreign body (EFB) ingestions are a common cause of acute stridor in the 1–2 year old age group. In a retrospective review by Miller et al. [4] of 522 children with EFB, 76% presented with respiratory symptoms (cough, wheezing, stridor, respiratory distress), while 22% had GI symptoms (nausea, vomiting, and/or dysphagia). Coins were the most common ingested object, and the children that presented with EFB >24 h old had an increased risk of complications. A thorough history, physical exam, and radiographic examinations are the key to diagnosis. In addition to microlaryngoscopy and bronchoscopy, an esophagoscopy is essential when the ingestion was not witnessed. Unfortunately, in this case the dysphagia was ignored initially, and all the attention was focused on the stridor and posterior mediastinal mass. Early esophagoscopy needs to be considered if there is any suspicion of foreign body ingestion.

Conflict of interest None identified.

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