Mechanical Cause for Acute Left Lung Atelectasis after Neonatal Aortic Arch Repair with Arterial Switch Operation: Conservative Management

Abstract

Respiratory complications due to mechanical obstruction of the airways can occur following pediatric cardiac surgery. Clinically significant intrathoracic vascular compression of the airway can occur when extensive dissection and mobilization of arch and neck vessels is involved as in repair of interrupted aortic arch. This case report describes a neonate who underwent interrupted aortic arch repair along with an arterial switch operation and developed a left lung collapse immediately after tracheal extubation. Fiber-optic bronchoscopy revealed vascular compression as the real culprit. The child was successfully managed conservatively.

Keywords: *Aorta, airway obstruction/etiology, congenital/surgery, heart defects, postoperative complications/surgery, thoracic/surgery*

Introduction

Respiratory complications due to extrinsic vascular compression of the airways can occur following repair of interrupted aortic arch.[1] Compression of the left bronchus by the descending aorta can result in a dynamic intrathoracic airway obstruction as against extrathoracic obstruction caused by problems related to the vocal cords. This case report describes a neonate who underwent interrupted aortic arch repair along with an arterial switch operation and developed a left lung collapse immediately after tracheal extubation. Fiber-optic bronchoscopy revealed an extrinsic vascular compression as the likely cause for the left lung collapse. The important role that fiber-optic bronchoscopy could play as an initial diagnostic tool for identifying the cause for bronchial compression is highlighted. The child was managed successfully by conservative measures. Approval from the Institutional Medical Ethics and Scientific Research Committee and written consent from the parents for publication were obtained.

Case Report

A 20‑day‑old girl baby (weight: 3 kg; length: 48 cm) with D-transposition of great arteries with a large perimembranous ventricular septal defect (8 mm) and Type A

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aortic interruption underwent single‑stage aortic arch repair with arterial switch operation and closure of the ventricular septal defect through a median sternotomy. Tracheal extubation was done after 36 h of mechanical ventilation. Before tracheal extubation, the baby was comfortable, generating about 5 ml/kg tidal volume on synchronized intermittent mandatory ventilation with a backup respiratory rate of 10 breaths/min, positive end‑expiratory pressure of 5 $\text{cm}H_2O$, fraction of inspired oxygen of 0.3, and a pressure support of 8 cm H₂O. As the arterial blood gas reports were satisfactory on these settings, tracheal extubation was done, and within a short span of time, the child had complete left lung collapse. This did not improve despite administration of high-flow humidified oxygen‑enriched air through a nasal cannula that was delivered at a rate of 2 L/kg. This flow through the nasal cannula was to generate a continuous positive airway pressure (CPAP) of about $5-6$ cmH₂O. The lung collapse was attributed to paresis of the left dome of the diaphragm as ultrasound of the chest suggested decreased diaphragmatic movements although they were not paradoxical.

Following tracheal reintubation, fiber-optic bronchoscopy was performed, which showed left bronchial obstruction due to external compression [Figure 1 and Video 1].

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The fiber-optic bronchoscope was advanced beyond the obstruction, and it was found that the bronchial lumen was patent with no significant secretions or mucus plugging. On withdrawal of the bronchoscope, posterior indentation of the left main bronchus was seen in the form of transmitted pulsations from the aorta [Video 2]. This was due to the reconstructed descending aorta that had been pulled up and anastomosed to the distal ascending aorta, causing a pulsatile mass effect on the bronchus. A diagnosis of left lung collapse due to left main bronchial obstruction secondary to compression by the descending thoracic aorta was made. As a reoperation for surgical mobilization of the vessels was not easy, a trial at conservative management was planned. The steps in conservative management envisaged were: (a) conversion from invasive mechanical ventilation to noninvasive ventilation in the form of application of CPAP, (b) gradual weaning from CPAP support to high-flow oxygen-enriched air through nasal prongs and later to face mask oxygenation, (c) control of blood pressure and heart rate to reduce the mass effect of the aorta on the bronchus, (d) constant observation of the child's spontaneous ventilation capabilities, and serial blood gas analysis and urgent chest radiographs when needed. It was decided that any deterioration in the child's ventilation mechanics would warrant some kind of "release" surgery.

After 3 h of mechanical ventilation, tracheal extubation was performed. The ventilator settings were similar to those that were used at the time of the first tracheal extubation. Chest radiographs before and after tracheal extubation showed complete reexpansion of the left lung [Figure 2]. As planned, noninvasive ventilation in the form of CPAP (about 9 cmH₂O) was applied by nasal prongs with the help of an infant Flow SiPAP system (VIASYS Healthcare, Yorba Linda, CA 92887, USA) [Figure 3]. Baby improved over a period of 6 days on nasal CPAP and was subsequently transferred to high-flow oxygen-enriched air through nasal prongs [Figure 3]. The left lung

remained completely expanded even after removal of CPAP [Figure 2]. The blood pressure and heart rate were controlled to age appropriate levels (systolic/diastolic blood pressure: 75–80/45–50 mmHg [approximately] and heart rate: 120–140 bpm) with oral propranolol (0.5 mg TID) and captopril (0.5 mg OD). Computerized tomography (CT) performed on the 10th day showed left bronchial narrowing with segmental collapse of the left upper lobe [Figure 4]. The lung collapse was not identified on the chest radiograph which continued to appear normal. As the baby was not exhibiting any features of respiratory distress and was clinically normal, the child was discharged home. During the 3‑month follow‑up, the left lung stayed fully expanded, and no further intervention was deemed necessary.

Discussion

A conservative management regimen for vascular compression of the left bronchus that was initially diagnosed by fiber-optic bronchoscopy and subsequently confirmed by CT is described.

Upper airway obstructions could occur after cardiac surgery, and they exhibit characteristic features when flow-volume loops are constructed. There are three classic patterns of flow-volume loop contours in patients with upper airway obstructions depending on the location and the type of obstruction, i.e., fixed or variable. Fixed obstruction, whether extrathoracic or intrathoracic, causes a decrease in inspiratory and expiratory flows which is evident as flattening of both inspiratory and expiratory portions of the flow‑volume loop. Fixed obstruction is seen in cases of postextubation tracheal strictures. Lesions that produce variable obstruction behave differently during inhalation and exhalation depending on their location, i.e., extrathoracic or intrathoracic. Variable extrathoracic obstruction (e.g., caused by vocal cord paralysis or marked

Figure 1: Fiber-optic bronchoscopy image of the left main bronchus showing external compression

Figure 2: Anteroposterior chest radiographs showing completely expanded left lung following tracheal reintubation and on mechanical ventilation (left image), following tracheal extubation with continuous positive airway pressure (middle image) and on nasal prongs (right image)

Figure 3: The child on continuous positive airway pressure on the left and on nasal prongs on the right

pharyngeal muscle weakness) is not affected by changes in the intrathoracic pressures during the respiratory cycle. During inspiration, there is an acceleration of air flow from the atmosphere into the lungs, and the intraluminal pressure decreases compared to atmospheric pressure due to a Bernoulli's effect. This effect results in a limitation of the inspiratory flow which is seen as a flattening of the inspiratory limb of the flow-volume loop. The air is forced out of the lungs through a potentially expandable extrathoracic airway, and hence, the maximal expiratory flow-volume curve appears normal. On the other hand, with variable intrathoracic obstruction due to extrinsic vascular compression as in the present case, the airway narrowing increases during forced expiration which is seen as a normal looking inspiratory limb and a flattened expiratory limb on the flow-volume loop. We did not record flow‑volume loops in this baby.

There could be a query if the bronchial narrowing caused by the descending aorta was the actual culprit for the left lung collapse in this child. As stated by the Poiseuille's law, the inverse relationship between the fourth power of radius to the airway resistance could exaggerate the clinical impact of even a small reduction in the radius of the airway lumen resulting in life-threatening airway compromise.^[2] In the postoperative period when the aortic pressure was uncontrolled, the airway obstruction might have been more prominent. Under controlled conditions of blood pressure and heart rate, CT pictures confirmed narrowing as already mentioned. This baby exhibited an intrathoracic, extraluminal airway obstruction that was more dependent on the stress in the descending aorta. Once the stress in the aorta was reduced by the control of heart rate and blood pressure, the obstruction probably was gradually relieved. Simultaneous application of positive airway pressure helped in keeping the airway open by producing a stenting effect. Hence, it is proposed that the stretched descending aorta produced the extraluminal obstruction and the lung collapse.

Airway compromise after repair of interrupted aortic arch due to external vascular compression could be a vexing

Figure 4: Computed tomography images in transverse and sagittal planes at the level of the left main bronchus and the descending thoracic aorta, respectively, demonstrating left bronchial compression by the descending thoracic aorta

postoperative problem.[1] Aortopexy, arch remodeling, and transverse aortic arch extension using pulmonary artery autograft along with left bronchial sleeve resection are some of the surgical procedures adopted to manage left bronchial compression following interrupted aortic arch repairs.[3‑5] Conservative management with prolonged mechanical ventilation, tracheostomy, and/or bronchial stenting has also been advocated to manage airway obstruction after interrupted aortic arch repair.[6] In the current patient, a trial at conservative management was adopted as the obstruction was not total, and surgical mobilization of the mediastinal and hilar structures would be difficult and probably be of dubious benefit.

The significance of the location of the left bronchus that could contribute to it getting "trapped" in the relatively fixed and narrow space under the curve of the aorta and the left pulmonary artery has been suggested earlier.^[7] The space becomes narrower both anteroposteriorly and superoinferiorly when the curve of the arch of aorta that was originally resembling a Roman curved arch is replaced with a pointed Gothic arch formation following anastomosis of the descending aorta to the ascending aorta.[8] The mechanism suggested for the left mainstem bronchial compression after interrupted aortic arch repairs is the anterior and upward displacement of the descending thoracic aorta.[5] During an arterial switch operation, the space under the arch becomes narrow after the Lecompte maneuver (although the pulmonary artery exits the space). The space is further narrowed by the neoaortic anastomosis as the ascending aorta is displaced posteriorly and caudally.

It has been suggested that noninvasive maneuvers such as regular chest physiotherapy and splinting of the malacic segment with either CPAP or bilevel positive airway pressure can overcome bronchial narrowing.[9] In addition, tracheomalacia is considered to be self‑limiting with a resolution of symptoms by 2 years of age.^[10] This baby was managed conservatively, and it is expected that with growth and maturation of the neonatal airways and growth of the aortic arch, the space under the aortic arch would widen, and the bronchial compression would resolve spontaneously. Children with similar airway problems should be followed up at regular intervals and repeat

fiber-optic bronchoscopy and CT would give an insight to the status of the airway and could determine the future course of action.

Intraoperative fiber–optic bronchoscopy could play an important role in identifying larger airway compression during aortic arch repair.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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