Bronchial Transection: Delayed Diagnosis and Successful Repair

J.K. Mahajan
Prema Menon
K.L.N. Rao
B.R. Mittal*

Traumatic bronchial transection is usually recognized and repaired immediately after injury. Bronchial transection has a variety of clinical presentations due to air leak into the pleural cavity and it is very rare to have total absence of air leak from the transected bronchus at presentation. We present one such case of right main bronchus injury with total absence of initial clinical signs and symptoms, leading to a delay in the diagnosis. However, the surgical repair eight months after injury showed excellent recovery of the chronically collapsed lung.

Key words: Bronchial transection, Chest, Tracheobronchial disruption, Trauma

Introduction

Tracheo-bronchial rupture, due to blunt trauma in children, is an uncommon injury with a variety of clinical presentations(1-3). The clinical features even with the major disruptions can be minimal, delaying the diagnosis in 25 to 68% of the cases and hence, high index of suspicion is required for diagnosis(1,4). However, a total absence of initial clinical signs and symptoms is unusual. We present a case of right main bronchus injury with absence of initial clinical features resulting in a delayed diagnosis.

Case Report

An 8-year-old, 13 kg boy sustained blunt trauma with an iron door falling on his chest and pinning him to the ground. There was no history of head injury, respiratory distress, subcutaneous emphysema or hemodynamic instability following trauma and the chest radiograph did not reveal pneumothorax or pneumomediastinum. He was given pain relief and did not require hospitalization. The child remained well for 3 months before he started having recurrent cough and occasional difficulty in breathing on exertion. The patient was treated symptomatically for another 5 months in a peripheral hospital before referral to our center. The clinical examination revealed a stable child with no respiratory distress. The chest wall on the right side was slightly depressed. The trachea and heart were shifted to the right and the air entry was poor in the right lower half of the chest. An X-ray of the chest revealed collapsed right lung near the right dome of the diaphragm and hyper-inflation of the left lung. The underlying bony cage was normal. An ultrasonography of the chest showed minimal fluid in right thoracic cavity. The echocardiography was normal. The CT scan of the chest showed collapsed right lung lying in the right para-vertebral gutter and hyper-inflated left lung with herniation of the apex of left lung to the opposite side. The right main bronchus...
showed abrupt cut-off with no evidence of extrinsic compression or endo-bronchial lesion including foreign body thereby suggesting a bronchial injury (Fig. 1). Bronchoscopy revealed normal trachea and bronchial take-off on both the sides but the right main bronchus was completely occluded at 1.5 cm from the carina. The origin of upper lobe bronchus was not seen. A perfusion scan of the lung showed negligible perfusion (only background activity) to the collapsed right lung (Fig. 2).

The endotracheal tube for general anesthesia was manipulated into the left main bronchus. The patient underwent right postero-lateral thoracotomy. The right main bronchus was found to be transected at about 1.5 cm from the carina, and the other end was somewhat buried into the collapsed lung near its hilum. The gap between the two ends was approximately 4 cm. The distal end was dissected out from the hilar structures and opened. After suction of the mucus, the collapsed lung could be inflated by inserting a separate endotracheal tube into the distal bronchial end. An end-to-end anastomosis was performed with interrupted sutures of 5-0 Vicryl in a single layer. Post-operatively, the patient recovered well and did not require ventilation. A perfusion scan four months later showed near-normal expanded lung with increased perfusion (Fig. 2). The chest radiograph and the CT scan showed near normal lung expansion and the patient had developed normal exercise tolerance.

Discussion

The patients with traumatic rupture of tracheo-bronchial tree exhibit well-recognized signs and symptoms of bronchial transection such as shortness of breath, mediastinal and subcutaneous emphysema, hemoptysis, pneumothorax, atelectasis, persistent air leak

Fig. 1. CT scan of chest showing herniation of hyperinflated left lung and atelectatic right lung in right paravertebral gutter.

Fig. 2. Radionucleide perfusion scan showing majority of the perfusion going to the left lung preoperatively (upper scan) and near equal perfusion of both the lungs 4 months after repair (lower scan).
and failure to expand the lung with thoracostomy tube drainage. The lung drops below the level of the carina if the bronchus is completely transected and it is a pathognomonic radiological sign(1,5,6). Various mechanisms of injury are compression of tracheobronchial tree between the sternum and the vertebral column resulting in distraction of the carina, shearing of bronchus by rapid deceleration and rapid increase in tracheobronchial pressure as a result of crush injury with a closed glottis(1,5).

The bronchial injury may be sealed by the peribronchial tissues and the patient survives the initial damage but absence of air leak at presentation is unusual and rare(6,7). However if the tear is in the mediastinal part of the main bronchus or trachea, pneumothorax may not occur and only mediastinal air will be seen(3,5). In cases, which have no pleural communication, ventilation may proceed through the torn area and the diagnosis is frequently delayed(5). Bronchoscopy is mandatory to make the diagnosis of bronchial transection when such a lesion is suspected even with minimal clinical and radiological symptoms(1-3,6). Granulation tissue invades the area over next 1 to 3 weeks after injury. Incomplete obstruction will lead to restriction of normal removal of the secretions and infection. However, if the bronchial communication snaps suddenly and completely, there is no aeration, which protects the lung from the air-borne sepsis and sterile mucus collects inside the atelectatic lung(5). The possibility of bronchial repair should be entertained even in the cases recognized long after injury(6). The prognosis depends upon the time interval between the diagnosis and treatment, associated vessel injury and the condition of the distal transected lung(6). Expansion and deflation of the lung on the operation table, before repair, demonstrates compliance and elasticity of the atelectatic lung and the likelihood of regaining a good lung growth in the postoperative period. When repair is impractical, resection is indicated to avoid infection and pulmonary vascular shunt(1).

The vascularity of the chronic atelectatic lung is a concern especially in the cases, which are diagnosed late(8). The radionuclide perfusion scan is a simple, efficient, non-invasive modality to evaluate the circulation of chronically collapsed lung and can be used repeatedly to assess the immediate and delayed response to the surgical repair(4,5).

In our case, the initial symptoms were subtle and the child did not show the air leak either clinically or radiologically, to suggest the presence of tracheo-bronchial disruption. Such near total absence of clinical symptoms has been unusual in the pediatric population. The diagnosis is usually delayed if air ceases to leak from the chest drainage tube, which our patient did not require. However, persistent atelectasis of the lung in our patient aroused the suspicion of a bronchial injury and bronchoscopy confirmed it. Hence, significant bronchial injuries may occur in the absence of usual initial symptoms. Therefore, the patients of obvious chest trauma should be on follow up in the immediate post-injury period for detecting these lesions to avoid unnecessary morbidity and possible mortality.

Contributors: Study concept and design JKM, KLN, PM, BRM. Analysis and interpretation of data: JKM, KLN, PM, BRM. Drafting of manuscript: JKM, PM, KLN. Guarantor of the manuscript: KLN.

Funding: None.

Competing interests: None stated.

REFERENCES
1. Slimane MAA, Becmeur F, Aubert D, Bachy B, Varlet F, Chavrier Y et al. Tracheobronchial...
CASE REPORTS


From the Department of Pediatrics and Dermatology*, Karadeniz Technical University, Faculty of Medicine, Trabzon, Turkey.

Correspondence to: Murat Çakir, KTÜ Tip Fakültesi, Pediatri Anabilim Dali, 61080 Trabzon/Turkey. E-mail: muratcak@hotmail.com

Manuscript received: July 1, 2003; Initial review completed: August 8, 2003; Revision accepted: September 25, 2003.

We report a case of toxic epidermal necrolysis who was successfully treated with intravenous immunoglobulin and granulocyte colony-stimulating factor. He had poor prognostic factors such as extensive epidermal loss, neutropenia, acute respiratory distress syndrome and Candida sepsis, but nonetheless made a complete recovery.

Key words: G-CSF, Immunoglobulin, Toxic epidermal necrolysis.

Introduction

Toxic epidermal necrolysis (TEN) is a severe drug-induced life-threatening disease and characterized by fulminant and widespread blisters responsible for epidermal sloughing(1). It is associated with high mortality and the majority of the patients die from complications of infection(1). Supportive therapies and antiseptics are used in patients with TEN. Different drugs such as cyclophosphamide, pentoxifylline, thalidomide, cyclosporine and plasmapheresis have been reported to be useful in single case observations(1). Recently, a few cases have been treated with human intravenous immunoglobulin (IVIG)(2-4). Here, we report a case of TEN treated with IVIG and granulo-