ATELECTASIS IN CHILDREN

T.S. Raghu Raman, Sunil Mathew, Ravikumar and P.S. Garcha

From the Departments of Pediatrics, Otorhinolaryngology and Anesthesia, Armed Forces Medical College, Punc-411 040.

Reprint requests: Wg. Cdr. T.S. Raghu Rarnan, Associate Professor, Department of Pediatrics, AFMC, Pune 411 040.

Manuscript received: July 1,1997; Initial review completed: August 27,1997; Revision accepted: November 27,1997.

Background: In children with lower respiratory tract symptoms, the elicited signs are not enough to distinguish common diagnosis like pneumonic consolidation, foreign body aspiration and atelectasis. Radiology and bronchoscopy would identify the true nature of the etiology. Design: Prospective study. Subjects: Thirty five children with both acute and chronic lower respiratory tract symptomatology, were analyzed for clinical and radiological signs of atelectasis. ***Results:*** *There were 23 cases in the acute group and 12 in chronic group. Acute group included cases of pneumonia, foreign body aspiration and mucus plug syndrome. Chronic group included cases of congenital heart disease, endobronchial tuberculosis and bronchial stenosis. Clinical recognition of atelectasis on the basis of localized loss of breath sounds and mediastinal shift was seen only in a minority of cases (8/35). The presence of atelectasis in children with pneumonia, missed clinically were diagnosed by the presence of tracheal shift, elevated hemidiaphragm and silhouette sign. In 21 cases, silhouette sign was positive making it an important radiological sign. Twenty one children underwent either diagnostic or/and therapeutic bronchoscopy. Findings included foreign bodies (n = 5), mucus plugs (n = 4), narrowing of main bronchus (n = 4) and inflammatory mucoid secretions and narrowing of lumen (n = 8). There were no major complications.* ***Conclusion:*** *The diagnosis of atelectasis in children may pose difficulties and there is a need to have a high index of suspicion to exclude atelectasis in children with either acute or chronic respiratory tract symptomatology.*

Key words: *Atelectasis, Bronchoscopy, Radiology.*

A TELECTASIS is a collapsed and air- less peripheral gas exchange region of the lung. It is an abnormal condition associated with a variety of pulmonary disorders and represents a manifestation of underlying pulmonary pathology rather than a disease entity *per se.* Clinical identification of atelectasis in children remains difficult in view of non specific symptomatology. Hence many a time the presence of atelectasis is missed. The clinician who identifies atelectasis in a child must determine the mechanisms by which atelectasis developed, the functional significance of the collapsed lung, and the etiology responsible for its presence. Airway size constraints and physiologic considerations have placed restrictions on the technique employed to derive information about the airways of pediatric patients. This presentation highlights the clinical profile and endoscopic experience in children with atelectasis at our center.

Subjects and Methods

This prospective study was based on

children admitted with symptoms suggestive of lower respiratory disorders. The period of the study was from January 1991 to May 1997. A detailed history in every child including the onset of illness, symptoms like sudden onset of dyspnea, choking episodes, fever, cough and retractions of chest was recorded. Children with chronic symptoms of recurrent bouts of fever and cough were also profiled in detail. In addition to detailed general examination, an effort was made in each child to identify and locate the region of atelectasis. An effort to classify the etiology of atelectasis clinically was also included. Laboratory investigations included complete blood counts, blood cultures, radiological assessment, ultrasonography (USG) chest, bronchoscopy and culture studies of secretions from the airways. Other investigations were done on specific indications. Atelectasis was diagnosed on clinical and radiological criteria. The radiological signs looked for included opacification, shift of mediastinum, displacement of interlobar fissure, elevated diaphragm, shifting of interlobar fissure and silhouette sign. Silhouette sign refers to the finding that the borders of contiguous structures of similar radiodensity are obscured by one another. Applying this concept to a disease process that produces a water density when it is in contact with structures of similar density such as the heart, aortic arch, and superior surface of the diaphragm, silhouette sign is present when the contiguous borders of these structures are lost. This sign is used most frequently for localization of disease to the right middle lobe and lingula, in which the right and left borders, respectively are obscured. If disease is localized in the basilar segments of the lower lobes, the adjacent diaphragmatic surfaces are obliterated.

Those requiring bronchoscopy either for diagnostic or therapeutic purposes were subjected to the procedure. The findings so recorded were correlated with clinical findings. All cases of atelectasis did not require bronchoscopy. The selection of cases for bronchoscopy was determined by the need for diagnostic or therapeutic purpose. For endoscopic examination and intervention we utilized Karl Storz rigid ventilating bronchoscope with fibreoptic distal lighting, size 3, 4 and 5. The sizes represent inner diameter of the bronchoscope in mm. General anesthesia was used in all cases. Induction of anesthesia was done by inhalation of oxygen and halothane or either through mask. After achieving a suitable plane of anesthesia the rigid bronchoscope was passed beyond the glottis. Subsequent maintenance of anesthesia was by inhalational anesthesia given through the side arm (anesthetics channel) of the bronchoscope. Bronchoscopic lavage was done in those cases where mucopurulent secretions were seen. The material was aspirated and sent for culture studies. Other treatment modalities included antibiotics, chest physiotherapy, humidification and hydration. Follow up of those children who were on long term therapy, i.e., cases of tuberculosis, congenital heart disease and bronchiectasis was done in the Outpatient Clinic.

Results

During the study period, a total of thirty five children presented with features of atelectasis. There were nineteen males and sixteen females. For clinical simplicity, cases with duration of illness less than three weeks were classified as acute (Group A) and cases with duration of illness longer than three weeks as chronic (Group B). The distribution of cases in the two groups and the mean age were: Group A- n = 23; age range : 6 months to 5 years; mean age : 3.5 years and Group - B n = 12; age range : 2 to 8 years; mean age : 5.5 years. The historical

and clinical data is reflected in *Table I. A* few important observations drawn from the data are: *(a)* Cough and fever were universally present; *(b)* History of choking episode and foreign body aspiration was elicited only in 5 (20%) and 9 (38%) cases in Group A, respectively; (c) Clinical recognition of atelectasis on the basis of localized loss of breath sounds and mediastinal shift was seen only in a minority of cases (8/35); (d) Universally all children with atelectasis presented with cough, fever, retractions, crepitations and rhonchi. The radiological findings in the present study are highlighted in *Table II.* The presence of atelectasis in children with pneumonia, missed clinically was diagnosed by the presence of tracheal shift, elevated hemidiaphragm and silhouette sign. Children with mucus plug syndrome (four cases)

TABLE I *Historical and Clinical Data (n = 35)*

Data	Group A (n = 23)	Group B (n = 12)
Historical		
Fever	100	40
Cough	100	100
Choking episode	20	Nil
Dyspnea	75	45
Foreign body aspiration	38	Nil
Cyanosis	Nil	Nil
Chest retractions	100	45
Failure to thrive	Nil	100
Repeated LRTI	Nil	100
Wheezing	36	42
Clinical		
Reduced air entry	52	75
Bronchial breath sounds	45	Nil
Mediastinal shift	20	53
Crepitations	100	100
Rhonchi	75	34

LRTI-Low respiratory tract infection

presented with acute respiratory distress, fever, cough and non specific clinical signs (opacification, elevation of hemidiaphragm and silhoute sign in all; air bronchogram in 2; emphysema in 1) which were non contributory in confirming the diagnosis. In Group B there were a total of 12 children with atelectasis. The three cardiac cases were congenital acyanotic heart disease with ventricular septal defect in two and patent ductus arteriosus in the third with pulmonary hypertension. All three cases presented with recurrent pneumonia and atelectasis of left lower lobe. The eight cases of tuberculosis presented with recurrent episodes of lower respiratory tract infection, failure to thrive, chronic cough, and signs of collapse consolidation clinically. In addition to radiological findings of atelectasis, hilar and paratracheal adenopathy was also seen. Bronchoscopy with rigid bronchoscope was done in 21 cases with atelectasis. In addition to removal of foreign bodies and mucus plug, bronchial lavage and culture studies were done. On bronchoscopy in Group A, the five foreign bodies removed were bits of groundnuts, custard apple seed, multiple stones (twice bronchoscopied) and an unidentified foreign body. Four children had presence of mucus plug obstructing the left main bronchus.

In Group B, three cases of congenital heart disease had narrowing of left main bronchus with visible pulsation, six children with chronic respiratory symptoms had narrowing of right middle bronchus with mucoid to muco - purulent secretions, two had similar findings on left main bronchus and one case had extreme narrowing of the left main bronchus. Culture results of the secretions removed during bronchoscopy were not of significant value except in three cases in whom acid fast bacilli (AFB) was isolated. No major complica-

TABLE II—*Radiological Signs (n = 35)*

Rdiological Sign	Group A (n = 23)			Group B (n = 12)		
	Pneumo-nia (n = 14)	Foreign-body (n = 5)	Mucus Plug (n = 4)	Cardiac (n = 3)	Endo-bronchial TB (n = 8)	Bronchial stenosis (n = 1)
Opacification						
Right upper lobe	3	0	0	0	0	0
Right middle lobe	3	0	0	0	6	0
Right lower lobe	4	5	0	0	0	0
Left lower lobe	4	0	4	3	2	1
Tracheal shift	1	0	0	3	1	0
Mediastinal shift	0	1	0	3	1	0
Diaphragm elevation	9	5	4	3	1	4
Silhoutte sign	10	5	4	3	1	8
Rib crowding	0	0	0	2	1	0
Fissure shift	2	0	0	0	0	2

tions were noted with the bronchoscopy procedures. One patient had delayed recovery from anesthesia (ether). Patients given ether had excessive salivation, nausea and vomiting for 6 to 12 hours. They are controlled with metoclopramide. The 8 cases of endobronchial tuberculosis were confirmed on the basis of narrowing of lumen and presence of mucoid secretions, positive AFB (3 cases), positive Mantoux test (3/8) and BCG diagnostic test (5/8). USG chest was useful in identifying minimal synpneumonic pleural effusion not apparent in routine X-ray chest PA view in both Group A (pneumonia) and Group B (tuberculosis). Complete blood count and culture studies were not of significant help in establishing the etiology.

The final outcome of cases were: (i) Atelectasis associated with pneumonia was transient and complete resolution was seen in all 14 cases within three weeks on follow up X-rays; *(ii)* Cases with mucus plugs and foreign bodies showed dramatic clinical and radiological resolution during immediate postoperative period; *(Hi)* Out of the three cases of congenital acyanotic heart disease, one child died of infective endocarditis, other two due to want of surgical correction continued to have persistent atelectasis of the left lower lobe; *(iv)* the case of congenital bronchial stenosis is awaiting specific therapy; and *(v)* Out of the eight cases of endobronchial tuberculosis, resolution was seen only in four cases despite adequate antitubercular treatment with steroids. The other four during follow up developed signs of bronchiectasis, *i.e.,* persistent and localized crepitations and radiological signs localized to lower lobe segments. Bronchography was not done in any of them. All four are on regular physiotherapy and follow up.

Discussion

Atelectasis occurs in three ways: *(a)* increased surface tesion in small airways and alveoli; *(b)* compression of pulmonary

parenchyma by intrathoracic chest wall, and extrathoracic processes; and (c) obstruction of airways. Diseases producing airway obstruction account for most of the atelectasis encountered in children. Processes that produce obstruction involve the airway lumen directly, the airway wall, or the tissue surrounding the airway. The incidence of atelectasis accompanying several respiratory disorders in childhood is reported to range as: meconium aspiration 40 to 55%, post extubation collapse in infants - 35%, bronchopulmonary dysplasia - 46 to 50%, bronchiolitis -12 to 24%, pneumonia - 23 to 25%, asthma - 4 to 19%, foreign body aspiration - 10 to 20% and tuberculosis - 8%(1).

History and clinical examination are insensitive means of detecting the presence of atelectasis. The clinical manifestations of atelectasis depend on the underlying cause as well as the degree of volume loss within the lung. When the atelectasis is relatively small and associated with little physiological impairment, there may be no signs or symptoms. The most specific sign on physical examination is a localized loss of breath sounds. When unilateral atelectasis is massive, tracheal deviation and shift of heart sounds towards the atelectic side may occur. However, the majority of children with atelectasis will present with cough, tachypnea, rales, rhonchi, a history of chest pain, or fever and less often dyspnea or cyanosis. Further, atelectasis that occurs during the course of tuberculosis, asthma, or infections such as bronchiolitis, bronchitis, and pneumonia produces no change in clinical picture unless the obstructed area is large(2). In the present study also, children in Group A with acute atelectasis due to pneumonia, foreign body aspiration and mucus plugs, did not clinically demonstrate specific signs of atelectasis.

In contrast, the chest radiograph is the only clinical means of accurately documenting the presence, extent, and distribution of atelectasis. The most direct and reliable sign is the displacement of an interlobar fissure. Other signs of volume loss, such as elevation of hemidiaphragm and mediastinal shift, are maximal nearest to point of volume loss and accompanied by an increase in focal density(3). However, in the present study, shifting of interlobar fissure could be demonstrated only in 4/35 cases.

In the lower lobes and middle lobe collapse, silhouetting of diaphragm and left and right cardiac border was an important sign in recognizing volume loss apart from the presence of elevated hemidiapgragm and density increase (31/35). The important contributing sign was elevation of hemidiaphragm (26/35) in atelectasis of lower lobes. Crowding of ribs and mediastinal shift was seen in chronic cases and wherein more than one lobe was involved. Generally the distribution of atelectasis does not help identify a specific underlying etiology in an individual patient. However, observations from some studies indicate that atelectasis in the right lung occurs in more than 70% children with asthma, cystic fibrosis, and tuberculosis. In contrast, the left lower lobe is the most common location for atelectasis associated with acute upper or lower respiratory tract infection(4). Our observations were similar. When all causes for obstruction of the airway are considered, the right lower and left lower lobes are most frequently collapsed. Further differentiation based on bronchial anatomical considerations is possible by the fact that mucus plugs cause more often left lower lobe collapse and foreign bodies more often right lower lobe collapse(5). In the present study, there were five children with foreign body aspiration and atelectasis of right lower lobe and four with atelectasis of left lower lobe as a result of mucus plugs. Right

middle lobe atelectasis is relatively common in children and has been the source of considerable interest in the literature. It is most vulnerable when there is enlargement of hilar lymph node and is often referred to as right middle lobe syndrome and the usual causes are tuberculosis and asthma(6,7). Out of nine cases with middle lobe atelectasis, six were due to tuberculosis presenting with chronic recurrent pneumonia and hilar and parateracheal lymph nodes in the present study. Pneumonic consolidation and atelectasis is frequently associated with loss of volume. The subtle signs of elevated hemidiaphragm, fissure position and silhouette sign are important to recognize in this setting(l). However, atelectasis of pulmonary segments occurs less often because collapse is prevented by collateral air drift(8). CT scan and high resolution computerized tomography (HRCT) are other modes of identifying structural lesions of parenchyma of lung. HRCT is a valuable method of studying the lung parenchyma which combines very thin slices with mathematical filtering of the computer data to produce increased edge definition in the final image. HRCT images closely resemble macroscopic appearances of pathological specimens of inflated lung and this has led to attempts to describe HRCT patterns of disease in anatomical terms rather than the sometimes vague terminology used for the description of diffuse shadowing on chest radiography. There is no doubt that the fine morphological detail available from HRCT images is responsible for the increased confidence with which specific histopathological diagnosis can be made(9).

The experience of bronchoscopy in this study has been very rewarding both as a diagnostic tool and has a therapeutic value. The beneficial aspect of an early and if required repeated bronchoscopic examination has been well illustrated by many studies(10-13). Our study has reinforced the point that correct diagnosis of airway obstruction in infants and young children is limited by the mimicking nature of symptoms and signs and difficult to demonstrate radiological signs. Equally important is early recognition of atelectasis, determining the etiology and instituting appropriate therapy to ensure a better prognosis.

REFERENCES

1. Redding GJ. Atelectasis in childhood. Pediatr Clin North Am 1984; 31: 891-905.
2. Hazinski TA. Atelectasis. *In:* Disorders of the Respiratory Tract in Children, 5th edn. Ed. Cherniv V. Philadelphia, W.B. Saunders Company, 1990; pp 509-516.
3. Proto AV, Tocino I. Radiographic manifestations of lobar collapse. Semin Roentgenol 1980; 15:117-173.
4. Gillies LD, Reed MH, Simons FER. Radiographic findings in acute childhood asthma. J Assoc Can Radiol 1978; 29: 28-33.
5. Holinger LD. Foreign bodies of the lar ynx, trachea and bronchi. *In:* Pediatric Otolaryngology, 2nd edn. Ed. Philadelphia, W.B. Saunders Company, 1990; pp 1206-1214.
6. Livingston GL, Holinger LD, Luck SR. Right middle lobe syndrome in children. Int J Pediatr Otorhinolaryngol 1987; 13: 11-23.
7. Miller FJW. Intrathoracic tuberculosis and miliary spread. *In:* Tuberculosis in Children, 1st edn. Edinburgh, Churchill Livingstone, 1982; p 105-136.
8. Miller TW. Radiographic evaluation of the chest. *In:* Pulmonary Diseases and Disorders 2nd end. Ed. Fishman AP. New York, Mcgraw - Hill, 1988; pp 479-528.
9. Sostman HD, Matthay RA. Chest imaging. *In:* Chest Medicine; 3rd edn. Eds. George RB, Light RW, Matthay MA, Matthay RA. Philadelphia, Williams and Wilkins, 1995; pp 110-131.

10. Casasnovas BA, Martinez EE, Sanchez BR, Perez RE, Gancedo CR, Cives VR. Pediatric fibre bronchoscopy. Ann Esp Pediatr 1993; 39: 313-316.

11. Ledesma - Albarran JM, Perez - Ruiz E, Fernandex V, Martinez GB. Endoscopic evaluation of endobronchial tuberculosis in children. Arch Broncopneumol 1996; 32:183-186.

12. Wong KS, Lin TY, Lan RS. Evaluation of chronic atelectasis in children using chest computed tomography and flexible bronchoscopy. Acta Pediatr - Sin 1996; 37: 193-196.

13. Burton EM, Brick WG, Hall JD, Riggs W, Houston CS. Tracheobronchial foreign body aspiration in children. South Med J 1996; 82:195-198.