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Ultrastructural Defects of the Ciliary Epithelium in a Child with Kartagener's Syndrome

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SUMMARY **Kartagener's syndrome is a well known classical triad of presentations consisting of bronchiectasis, sinusitis and situs inversus. It is now recognized that the syndrome is an extreme presentation of primary ciliary dyskinesia, a large group of conditions with ultrastructural ciliary defects, leading to poor ciliary motility in various organ systems. A case of Kartagener's syndrome is presented in an eight year old Thai boy in whom the ultrastructural ciliary defects have been examined and described in detail for the first time in Thailand. Incomplete lack of dynein arms was recognized. In addition, disorientation of ciliary axis was noticed. Due to severe bronchiectatic changes of the right lower lobe and right lingular lobe which did not improve despite adequate antibiotics, these lobes were surgically removed. The child has done well since, but still suffers occasional and recurrent bouts of sinusitis.**

Aside from cellular and humoral immunity, mechanical clearance of mucous secretion by ciliated epithelium from the respiratory system plays a major role in the immune defense of the lungs. In 1933, Kartagener[1] described an unusual patient with chronic infections of the respiratory system (viz. bronchiectasis, sinusitis) who presented along with situs inversus totalis. The pathogenesis of this association had remained elusive until in 1976 when morphologic and functional abnormalities of ciliary microtubules in these patients were examined.[2] Common ciliary ultrastructural defects among these patients were the lack of the ATPase-containing dynein arms of peripheral microtubules within the cilia.[2,3] These abnormalities results in the lack of effective recovery strokes of the cilia, leading to accumulation of secretions within the respiratory tract and frequent infections of the respiratory system. In addition, most men with this syndrome were found to be sterile because of similar microtubular defects found within spermatozoa tails. Several other ciliary defects have since then been described and it is now known that patients afflicted with this condition represent a spectrum of presentations ranging from those with only mild to moderate respiratory infections who manifest their problems very early in life, to those with the complete syndrome as originally described by Kartagener.[4]

In Thailand, cases of Kartagener's syndrome are not uncommon and have been previously reported.[5,6] However, these cases were diagnosed on the basis of their clinical findings only. The purpose of this report is to describe a child with a complete constellation of the syndrome in whom the ultrastructure of ciliary axonemes have been thoroughly studied and elaborated.

MATERIALS AND METHODS

Case Report

P.B. is a 9 year old Thai boy who was referred to Siriraj Hospital from the Nakhon Pratom Provincial hospital for a further work up and treatment of his chronic cough. He had had frequent episodes of cough

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and dyspnea since age 3. Cough generally preceded the development of fever and dyspnea which responded slowly to antibiotic administration. Productive cough occurred in the morning, upon changing of position and during the night-time; color of sputum ranged from white or yellow to greenish-yellow. One episode of otorrhea occurred at 4 years of age and resolved with medical therapy. No history of similar or unusual illnesses were reported from among immediate and remote family members. On physical examination, he was afebrile with vital signs as follows: body temperature 37.2°C, pulse rate 112/min, respiratory rate 26/min and blood pressure 100/70 mmHg. The body weight was 18.5 kg (10th percentile) and the height was 120 cm (25th percentile). No cyanosis, tachypnea, nor digital clubbing was noted. Central perforation of right tympanic membrane was observed with the ear canal filled with greenish-yellow discharge which grew *Citrobacter freundii* and *Pseudomonas aeruginosa* upon culturing. There was some hyperexpansion of the thoracic cage upon inspection and coarse crepitations with medium to coarse rhonchi was heard over both lung fields, particularly over the right lower lobe area. The cardiac apex was noted to be at the right 5th intercostal space over the midclavicular line, with all heart sounds heard more prominently over the right anterior chest wall. The rest of the physical examinations were within normal limits. Complete blood count revealed Hct 37%, white blood cell count of 17,500 cells/mm.^{3}with 70% polymorphonuclear leukocytes, 2% eosinophils, 7% monocytes, 31% lymphocytes and with adequate platelets. A chest radiograph revealed dextrocardia and bronchiectatic changes of both lower lobes. Paranasal sinus films showed mucosal thickening of both ethmoid air cells and maxillary sinuses with fluid collection in the left maxillary antrum (Fig. 1a). Tuberculin test was negative (with BCG scar present). Spirometry showed both restrictive and obstructive changes with minimal improvement after bronchodilator administration. A radioisotopic technecium-99 lung scan revealed 41% clearance at 1 hour post-inhalation from the left lung and 50% from the right lung. Immunoglobulins, total T cell counts (and CD4, CD8 enumerations), B cells, NK cells, NBT and Rebuck skin window were all within normal limits. The patient underwent a bronchoscopic examination which revealed inflammatory changes of both major bronchi with profuse amount of purulent material which grew *Streptococcus pneumoniae*. Biopsies from the inferior turbinate and the carina were obtained and were submitted for electron microscopic examination with results indicated below. A subsequent bronchography revealed saccular bronchiectatic change of right lower lobe and of what appeared to be right lingular lobe (Fig. 1b). The bronchiectatic involvement of the left lower lobe was of cylindrical type. He was treated over 3 weeks with appropriate parenteral antibiotics without any significant improvement (no reduction of the amount of sputum which ranged from 5 to 80 ml/day or 30-100 g/day). A decision was then made to proceed with the removal of both right lower and right lingular lobes. The histologic examination of the removed lobes was compatible with bronchiectatic changes. The amount of sputum decreased significantly after the operation, however the patient was never free of cough. The family was informed of his diagnosis and the child was discharged with adequate instruction for chest physiotherapy, effective voluntary cough and with prophylactic antibiotics. Two years after discharge, the patient continued to have episodes of sinusitis despite prophylaxis with cotrimoxazole. In spite of his continuing problems, his growth parameters were unaffected and proceeded well among the 10th percentile for his age.

Ultrastructural examination of the ciiliated epithelium

Method

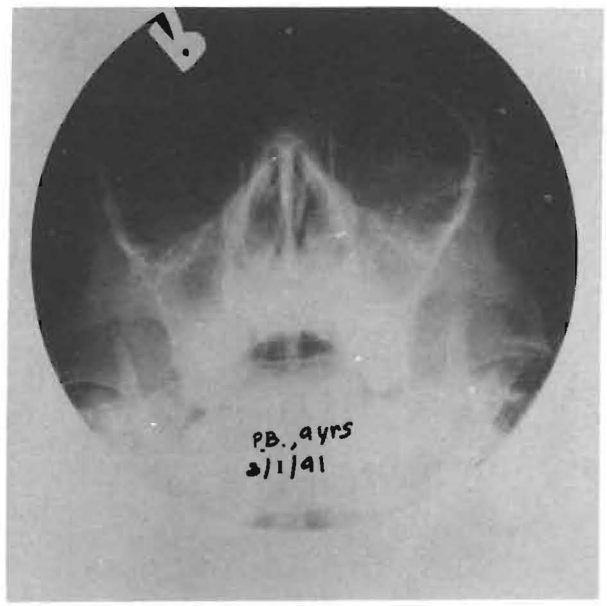
The biopsy specimens were obtained from the carina and from the posterior portion of the inferior turbinate. The tissues were fixed immediately in 4% glutaraldehyde using Millonig's phosphate buffer pH 7.4 as diluent, and post-fixed in 2% buffered osmium tetroxide. After dehydrating in ascending concentration of ethanol, the tissue was embedded in Epon. Areas of ciliated epithelium were selected for the study by examing 1 μm thick toluidine blue stained sections. Ultrathin sections were also cut on an LKB ultratome/ultramicrotome and stained with uranyl acetate and lead citrate. The sections were examined under the Jeol JEM-100SX electron microscope at 80 kV.

Only transversely sectioned cilia, in which the microtubular arrangement was clearly seen, were examined. In addition, three plastic blocks were selected for ultra-thin sectioning. The number of axonemes counted in each specimen were at least fifty axonemes.

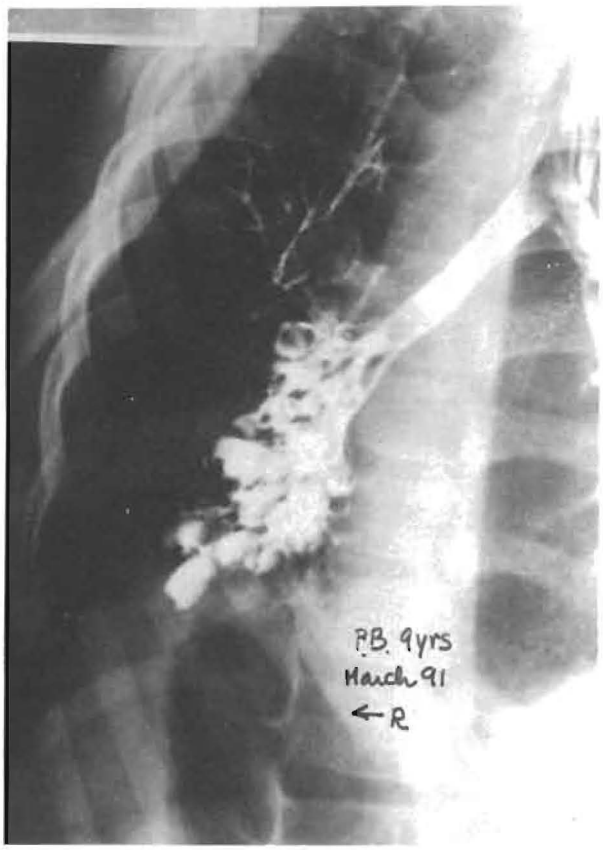
RESULTS

Normal cilia

The ultrastructure of the normal axoneme has the classical 9+2 microtubular pattern as described by Fawcett and Porter[7] as schematically illustrated (Fig. 2). The central pairs consist of two single microtubules (singlets). Those in the peripheral nine microtubules are doublets consisting of subunit A and subunit B which share a common wall. Two short diverging arms (outer and inner dynein arms) project clockwise from subunit A of each doublet toward the next doublet. Subunit A is also connected by a radial spoke to a



a



b

Fig. 1 Roentgenograms of the patient; **Fig. 1a** is the patient's sinus X-ray revealing total opacification of the left maxillary sinus with thickening of mucosa on the right side. **Fig. 1b**, the bronchography of the right lower lobe and the right lingular lobe showing extensive saccular bronchiectatic changes.

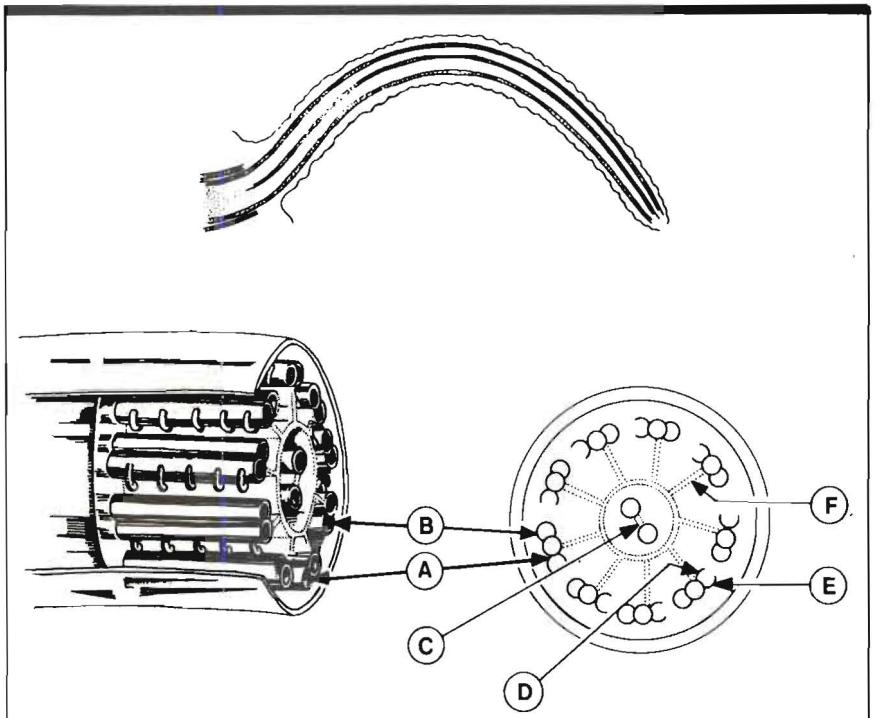
central sheath surrounding the central singlets. Strands interconnecting peripheral microtubules are termed the nexin-link.

Ultrastructure of the patient's cilia

The electron microscopic examination of the tissue obtained from this patient revealed remarkable changes of axonemes. Although the arrangement of central and peripheral microtubules appeared to be of 9+2 patterns as seen with normal axonemes, deficiencies of both outer and inner dynein arms, with special attention to the outer ones, were clearly observed (Fig. 3a). These deficiencies, however, appeared to be incomplete, i.e. in some sections, the axonemes still retained some remnants of the dynein arms appearing as short projections from the A subunits (Fig. 3a). Other structures of the axonemes i.e. the radial spokes, the central sheaths, and the nexin-links were well preserved. It could also clearly be appreciated that there was a random orientation of the axis of the central microtubules (the central axis is the line which is drawn through the centre of both central microtubules). There was no parallelism of these lines among neighbouring axonemes as can be seen in Fig. 3b. Some nonspecific changes of the axonemes, commonly found associated with chronic infections[8], such as fusions of cilia (megacilia) and supernumerary microtubules were occasionally observed (not shown in the figures).

DISCUSSION

The ultrastructural description of ciliary defects in our patient is the first to be described from a Thai patient with Kartagener's syndrome. The lack of outer dynein arms were the initial ciliary defect observed[2,8] and has since been the most commonly observed ultrastructural findings with cases of immotile cilia syndrome.[4] Since dynein arms are believed to contain ATPase,[9]



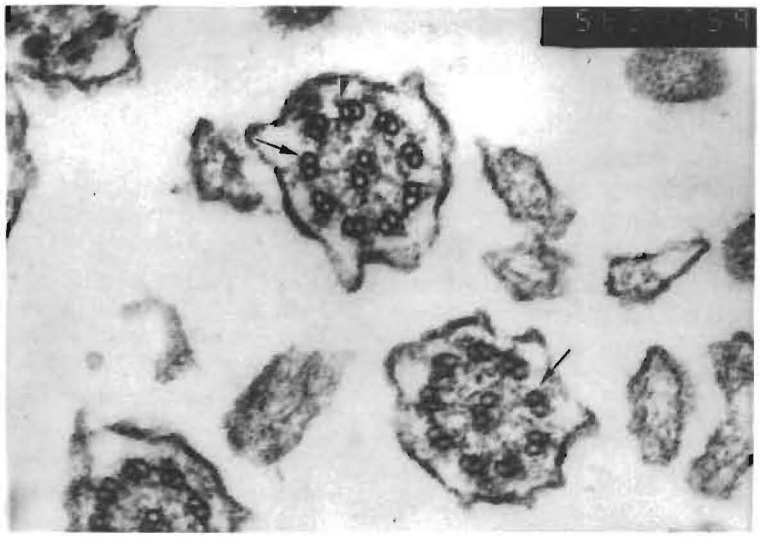
(AB) PERIPHERAL DOUBLETS
(C) CENTRAL MICROTUBULES
(D) INNER DYNEIN
(E) OUTER DYNEIN
(F) RADIAL SPOKE

Fig. 2 Illustrations of normal cilias on longitudinal section, on imaginary lateral profile, and on cross section. The cross-sectioned drawing shows essential parts of the ciliary axoneme, namely, microtubules, dyneins (outer and inner) radial spokes, and central sheath.

they appear to be involved in the generation of energy required in the sliding motion of the microtubule A over microtubule B of the peripheral doublet and thus along with othe ciliary structures of the axoneme transforming this motion into the active bending of the cilia. Since these findings could be observed in patients with and without a complete syndrome of Kartagener's the term "immotile cilia syndrome" was initially proposed for this group of patients.[10] However, it is to be observed that in most cases in the literature, the lack of dynein arms was usually not complete ie usually absent in about 70% from the total number of cilia[8,11] and some remnants of this structure could frequently be observed in some axonemes as in our case. As a result, not all the cilia are inactive. In fact, with the recent discovery of other ultrastructural defects such as the absence of the radial spokes,[12] transposition of the microtubules[13-15] and the random orientation of the central tubules[16,17] in which ciliary motion are not completely lacking, the term "ciliary dyskinesia" has been proposed to replace the former nomenclature.[18] The clinical manifestations of such patients could ranged from mild infections to those with very severe diffuse bronchiolitis[19,20] with onset of the disease occurring at a very young age.[4]

Besides the lack of the dynein arms, a lack of central orientation of the central microtubules[21] has also been observed in our patient. The active plane of ciliary beating is believed to occur perpendicular to the axis drawn through the central microtubules. The random orientation of microtubules therefore leads to chaotic and uncoordinated beats of cilia and thus leading to ineffective mucous clearance. This random orientation could be responsible for frequent infections observed in patients with Kartagener's syndrome who have been found to have normal ultrastructural findings of the cilia.[22,23] Although situs inversus found in about one-third to one-half of the patients with this disorder[4,24,25] was initially proposed to be the result of the ineffectual beating of the embryogenic cilia responsible for random lateralization of the viscera,[2,26] it is currently believed to be a facultative phenomenon. Other associated abnormalities include cardiac problems,[27] hydrocephalus[28] and retinitis pigmentosa,[29,30] all of which could be explained by the central theory of ciliary abnormality within the components of these systems.

The delay in mucociliary transport can be assessed with inhalation of radiolabelled technicium-99.[2,3,31] Some clearance of the isotope observed in our patient could be due to coughing which occurred during the study. Other diagnostic tests to exclude the presence of ciliary dyskinesia would include ciliary motion analysis, the method of which is cumbersome, expensive and limited only to highly sophisticated research centres.[32] The screening "saccharin test"[33,34] measures the time it takes for the perception of



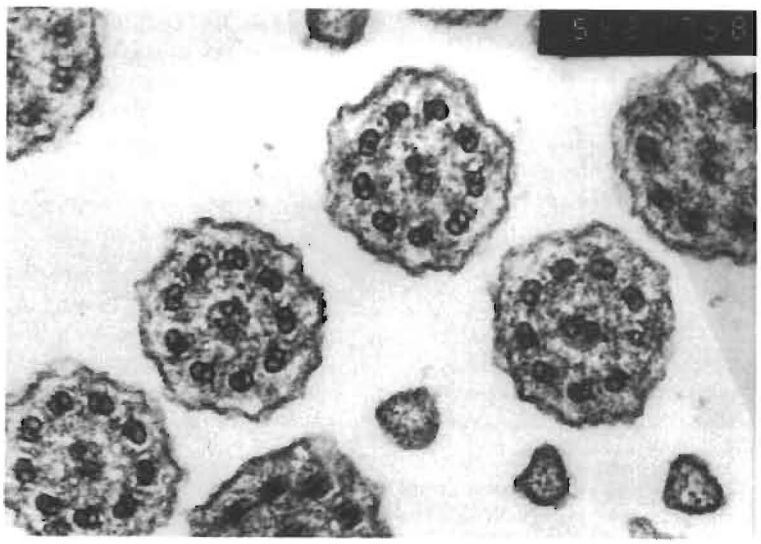


Fig. 3 Electron micrograph of cross-sectioned cilias from this patient. **Fig. 3a** shows the absence of outer dynein arms (two complete arrows). The upper arrow head demonstrates a remnant of the arm attaching to the A-tubule. **Fig. 3b** illustrates the lack of parallelism of central tubular axes among neighbouring axonemes.

sweetness after placement of saccharin particle on the anterior end of the inferior turbinate (average = 19 minutes) can be used as an initial screening test.

Infections of the respiratory system are the major morbidity suffered by these patients. Infection usually begins in childhood[19] and those without proper diagnosis and without proper planned care usually end up with destructive bronchiectatic lesions such as were encountered in our patient. It is to be observed that respiratory infection, by itself, could also cause marked cytopathic effects and abnormal microtubular changes to the ciliated epithelium. The common findings were often associated with central microtubular elements of the cilia while involvement of the dyneins, radial-spoke deficiencies and transpositional phenomena were found to be rare.[35] Normal epithelial organization and ciliary structure appeared to be reestablished during the convalescent period within 2 to 20 weeks after the infections.[35] The normal Rebuck skin window in our patient is in agreement with the finding of others that chemotaxis and chemokinesis of polymorphonuclear leukocytes is usually not impaired in patients with this syndrome[36-38] although previous investigations had indicated otherwise.[39,40] Other mucociliary clearance mechanisms such as postural drainage and effective cough can be instituted as substitute measures to reduce mucous burden. Recurrent atelectasis[25] is often encountered both due to mucous plugging and loss of elastic recoil within the ectatic bronchi.

Radiological findings can range from bronchial wall thickening, hyperinflations, recurrent atelectasis and finally bronchiectasis with predominant involvement of the middle lobe.[25] The role of prophylactic antibiotics is at current controversial. Some patients may require in-hospital "intensive tune up" such as the regimen frequently employed for patients with cystic fibrosis. Decision to remove the bronchiectatic lobes in our patient was reached after an adequate length of antibiotic administration and postural drainage had been instituted along with the saccular appearance of bronchiectasis indicating an advanced stage of bronchial involvement. This is to be carefully considered because the chance for reoccurrence of bronchiectasis to the remaining bronchi is high and, thus, one should be absolutely certain to remove the worst lobe involved.

Otitis media is a common finding and perforation of the tympanic membrane is not uncommonly encountered[19,41] since the clearance of the mucous through the eustachian tube is impeded by the poor ciliary motility leading to the increase in the pressure within middle ear cavity. Patients stand the chance of reperforating the membrane should tympanoplasty be contemplated. Pulmonary function often reveals a combined pattern of restrictive and obstructive changes.[24] Response to bronchodilators is variable although some younger patients with this disorder may primarily present with wheezing indistinguishable from those found in asthmatic patients with some demonstrable increase in the bronchial hypereactivity.[42]

Since the immotile cilia syndrome or ciliary dyskinesis tends to cluster within a family, genetic predisposition has been suspected. No family members of our patient suffered similar infectious episodes although we did not seek to study cilia in these members. Mossberg[43] as well as Sturgess[44] extensively reviewed the existence of cases with Kartagener's syndrome within extended family members of propositii and concluded that the transmission of the syndrome follows an autosomal recessive mode of inheritance. Since structures of the sperm tails are of the axomenal pattern as with cilia elsewhere in the body, sperm from men with this syndrome are usually immotile leading to infertility.

Despite what appears to be relentless infections in these patients, most patients survive into middle age and to late adulthood.[32] Our patient exhibited normal growth after the extensive plan to cope with his pulmonary infections had been instituted and frequent follow-ups were ensured. No deterioration of the lung function was observed during the two years after his initial work up although infiltrates of the in situ left lower lobe appeared denser. In general, the outlook of these patients appeared not to be as dismal as would be expected. It cannot be overemphasized that the work up for patients with frequent upper and lower respiratory tract infections, although manifested at a very young age[16] should include screening tests to exclude primary ciliary dyskinesia which includes ultrastructural examination of the ciliated epithelium. With adequate follow up plan and timely intervention, growth and lung functions among these patients should proceed to reasonable levels.

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