

Abnormal mucociliary action in asthma and bronchiectasis

A. A. AWOTEDU*, O. O. BABALOLA, E. O. LAWANI AND P. D. HART

Department of Medicine, University College Hospital, Ibadan, Nigeria

Summary

Nasal mucociliary clearance (NMCC) time was measured in four groups of patients: asthmatics with allergic rhinitis, asthmatics without rhinitis, bronchiectatics and normal subjects. The saccharin method was used for the study. The NMCC time was prolonged significantly in the asthmatic groups and group with bronchiectasis when compared with control subjects ($P < 0.001$). It is likely that the impaired mucociliary clearance is due to a combination of mucus abnormality and ciliary malfunction.

Résumé

Le temps d'élimination muco-ciliaire nasal (TEMCN) était calculé dans quatre groupes des malades: les asthmatiques avec rhinites allergique, les asthmatiques sans rhinites; les bronchiectatiques et les sujets normaux. Pour arriver à les résultats, la méthode saccharine a été utilisé. Le TEMCN était significativement prolongé dans les bronchiectatiques et les asthmatiques que chez les normaux ($P < 0.001$). C'est possible que cet affaiblissement d'élimination muco-ciliaire est causé par un combinaison d'un anomalie mucueuse et d'un dérèglement ciliaire.

Introduction

The respiratory tract from the nose to the bronchioles is lined with cilia. The main function of cilia in the respiratory tract is to move mucus to the oropharynx where it is expectorated. Cilia beat in transportation of mucus has been likened to an escalator mechanism, while electron microscopy has shown that there are

hooks at the tips of respiratory cilia which help in the propagation of sputum [1]. It can therefore be seen that when mucociliary clearance is impaired, the problem could be with either the mucus or cilia.

Mucociliary clearance (MCC) in the respiratory tract is an important defence mechanism and has been observed to be impaired in some respiratory diseases such as asthma [2,3], bronchiectasis [4], and chronic obstructive pulmonary diseases [5].

Primary ciliary dyskinesia is a well recognized feature of Kartagener's syndrome and it is believed that poor MCC in this syndrome leads to chronic sinusitis and bronchiectasis [6].

Various methods have been used to measure MCC. These include measurement of the speed of removal of radiolabelled particles [7], a barium sulphate technique [8], and the saccharin technique [9,10].

This study was designed to evaluate the integrity of the mucociliary action in our patients with asthma and bronchiectasis.

Subjects and methods

Four groups of patients were recruited into the study.

- Group A — patients with asthma and associated allergic rhinitis — 20 patients (10 males, 10 females, age 32 ± 10.5 years).
- Group B — patients with asthma but no rhinitis — 20 patients (10 males, 10 females, age 34 ± 11.8 years).
- Group C — patients with bronchiectasis — 6 patients (4 females, 2 males, age 38 ± 6.2 years).
- Group D — normal controls — 22 patients (12 males, 10 females, age 30 ± 9.8 years).

*To whom correspondence should be addressed.

The patients with asthma satisfied the American Thoracic Society Definition of asthma [1]. They were in steady state, not having exacerbation of their asthma or rhinitis and were not on any medication 12 h before the examination. The patients with bronchiectasis had classical symptoms and radiological confirmation of the disease. All the controls did not have respiratory disease and were non-smokers.

Saccharin test

The saccharin test used was that of Andersen *et al.* [9]. A 1-mm diameter particle of saccharin was placed in the inferior nasal turbinate. The patient was instructed not to sneeze, sniff, smoke, eat or drink during the test. The time from the placement of the particle to the patient's first experiencing a sweet taste was recorded. Patients who failed to experience a sweet taste after 60 min were deemed to have grossly prolonged nasal MCC. Their ability to taste was determined by placing saccharin on the tongue.

Results

The nasal mucociliary clearance (NMCC) obtained for the four categories is shown in Fig. 1. Normal subjects (Group D) had a NMCC time of 11.9 ± 6.2 min while the values obtained for asthmatic patients with and without rhinitis were prolonged (22.8 ± 2.9 and 21.1 ± 2.8 min, respectively). The differences between each of these two groups and the control was statistically significant ($P < 0.001$). However, the difference between the asthmatic subjects with rhinitis (Group A) and those without rhinitis (Group B) was not statistically significant ($P > 0.05$). The six patients with bronchiectasis (Group C) also had a prolonged NMCC of 24.2 ± 3.5 min which was statistically significant when compared with the control group ($P < 0.001$). There were no statistically significant differences between Groups C and A ($P > 0.05$), and Groups C and B ($P > 0.05$). Two patients had a NMCC greater than 60 min, 1 patient had a NMCC of 75 min (Group A) while the other had 68 min (Group B). These patients did not have any nasal septal deformity

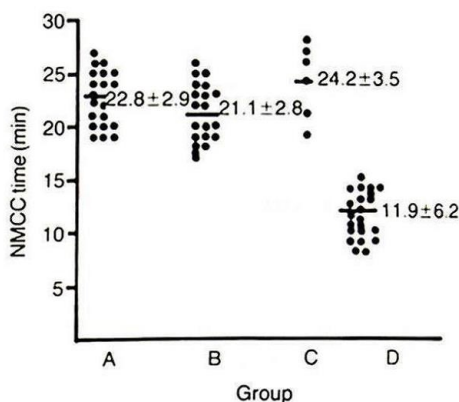


Fig. 1. Scatter diagram showing nasal mucociliary clearance times in patients with asthma, bronchiectasis and normal controls. Figures represent means \pm s.d.

and were able to taste the saccharin placed on the tongue. These patients were excluded from the analysis.

Discussion

Mucociliary clearance is an important defence mechanism of the respiratory tract. It depends on effective functioning of the mucus and cilia. Mucus is produced by goblet cells which are found lining the tracheo-bronchial tree. The mucociliary clearance time effectively assesses the function of both the mucus and cilia, while ciliary beat frequency and ciliary ultrastructure specifically assess ciliary function.

In our study, the NMCC time was prolonged in our asthmatic patients. This is consistent with findings from other studies [3,12,13]. However, Hady *et al.* [8] observed a rapid transit time; the reason adduced for their observation was that the nasal secretion of their patients was alkaline and that ciliary action is increased under such conditions [8]. Although there were differences between the patients with associated allergic rhinitis and those without, the difference was not statistically significant, which is similar to the observation of Stanley *et al.* [12]. A plausible explanation for this is that the associated rhinorrhoea in the rhinitis group produced inefficient mucociliary transport.

In our patients with bronchiectasis the mucociliary clearance was considerably impaired, similar to the observation of other workers [12,14-18]. This is due to the presence of irreversible bronchial damage and production of purulent sputum. The presence of abnormal glycoproteins in bronchiectasis results in mucus with sub-optimal visco-elastic properties while ciliary beat frequency is slower in pus than in clear secretion [15,16]. Bronchiectasis is a prominent feature of Kartagener's syndrome in which primary ciliary dyskinesia has been reported [6]. The ciliary abnormality is not limited to the bronchial epithelium but affects cilia in all organs; this has been termed immotile-cilia syndrome [6].

Although ciliary ultrastructure was not established in this study, it is unlikely that our patients have ciliary dyskinesia. There was no evidence to suggest Kartagener's syndrome as none of the patients had sinusitis, situs inversus or infertility.

There were two patients with abnormally prolonged NMCC similar to that reported by other workers [12]. Such abnormally prolonged clearance time has been associated with nasal septal deformities [17] which neither of these two patients had.

The contribution of abnormal mucociliary action in respiratory diseases is gradually unfolding. Attempts are being made to enhance ciliary action in patients with primary ciliary dyskinesia. Most of the time therapy had been directed at altering the visco-elasticity of mucus. Attempts are, however, being made to enhance ciliary action in patients with primary ciliary dyskinesia.

There is currently no single or unifying explanation for abnormal mucociliary action and there is most likely to be an interplay of factors. The clinical importance of this study and others will become apparent when the effect of drugs affecting ciliary action is properly studied.

References

- Jeffrey PK, Reid LM. The respiratory mucous membrane. In: Brain JD, Proctor DF, Reid LM, eds. *Respiratory Defence Mechanisms*, Vol. 1. New York: Marcel Dekker, 1977:192-5.
- Dunhill MS. The pathology of asthma with special references to changes in the bronchial mucus. *J Clin Path* 1960;13:27-33.
- Pavia D, Bateman JMR, Clarke SW. Deposition and clearance of inhaled particles. *Bull Eur Physiopath Resp* 1980;16:335-66.
- Lourenco RV, Loddenkemper R, Carton RW. Patterns of distribution and clearance of aerosols in patients with bronchiectasis. *Am Rev Respir Dis* 1972;106:856-66.
- Camner P, Mossberg B, Philipson K. Tracheo-bronchia clearance and chronic obstructive lung disease. *Scand J Respir Dis* 1973;54:272-81.
- Eliasson E, Mossberg B, Camner PK, Afzelius BA. The immotile cilia syndrome. A congenital ciliary abnormality as an etiological factor in chronic air way infections and male sterility. *New Engl J Med* 1977;297:1-6.
- Quinlan MF, Salzman SD, Swift DL, Wagner HN, Proctor DF. Measurement of mucociliary function in man. *Am Rev Respir Dis* 1969;99:13-23.
- Hady MRA, Shehata O, Hassan R. Nasal mucociliary function in different diseases of the nose. *J Oto Laryngol* 1983;97:497-502.
- Anderson I, Camner P, Jensen PL, Philipson K, Proctor DF. Nasal clearance in monozygotic twins. *Am Rev Respir Dis* 1974;110:301-5.
- Rutland J, Cole PJ. Non-invasive sampling of nasal cilia for measurement of beat frequency and study of ultra-structure. *Lancet* 1980;ii:564-5.
- Statement of the American Thoracic Society Committee on diagnostic standards for non-tuberculous respiratory diseases. *Arch Environ Health* 1962;5:375-82.
- Stanley PJ, Wilson R, Greenstone MA, Mackay IS, Cole PJ. Abnormal nasal mucociliary clearance in patients with rhinitis and its relationship to concomitant chest disease. *Br J Chest Dis* 1985;79:77-82.
- Mezey RJ, Cohn MA, Fernandez RJ, Jamsz-kiewicz J, Wanner A. Mucociliary transport in allergic patients with antigen induced bronchospasm. *Am Rev Respir Dis* 1978;118:677-84.
- Rutland J, Cole PJ. Nasal mucociliary clearance and ciliary beat frequency in cystic fibrosis compared with sinusitis and bronchiectasis. *Thorax* 1981;36:654-8.
- Wood RE, Boat TF, Doershuk CP. Cystic fibrosis. *Am Rev Respir Dis* 1976;113:833-78.
- Friedman M, Stott FD, Poole DO, Dougherty R, Chapman GA, Watson M, Sackner MA. A new roentgenographic method for estimating mucus velocity in airways. *Am Rev Respir Dis* 1977;115:67-72.
- Yergin BM, Saketkhou K, Kichaelson ED, Serafini SM, Koritz K, Sackner MA. A roentgenographic method for measuring nasal mucus

- velocity. *J Appl Physiol Resp Environ Exercise Physiol* 1978;44:964-8.
18. Currie DC, Pavia D, Agnew JE, Lopez-Vidriero MJ, Diamond PD, Cole PJ, Clarke SW. Impaired tracheobronchial clearance in bronchiectasis. *Thorax* 1987;42:126-30.

(Accepted 5 December 1988)