

Lesson of the Week

Variable intrathoracic airways obstruction masquerading as asthma

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Chronic upper airways obstruction may be unrecognised or misdiagnosed as asthma or chronic obstructive bronchitis. Clinical features, such as stridor, or the results of pulmonary function tests may lead to a diagnosis.¹ Indeed, there is usually no difficulty in the diagnosis of patients with extrathoracic airway obstruction, when the forced inspired volume in one second (FIV₁), peak inspiratory flow, or maximum mid-inspiratory flow recorded from a flow volume curve is reduced.² Conversely such clues do not exist to aid recognition of variable intrathoracic obstruction of the trachea, carina, or main bronchi, when the flow volume curve simply shows expiratory airflow obstruction and the physical signs are similar to those of asthma or chronic obstructive bronchitis—namely, an expiratory wheeze. We report on such a patient in whom asthma was diagnosed erroneously and as a result the definitive investigations and treatment were delayed.

Case report

A 36 year old man developed cough, chest pain, and a wheeze after a holiday in Crete. He had no personal or family history of atopy. Despite treatment with aerosols of salbutamol and beclomethasone dipropionate and a course of oral prednisolone (20 mg/day for three weeks), his symptoms persisted for two months and he was referred to hospital. He had an expiratory wheeze and FEV₁ of 1.2 (predicted 4.5), together with 3 mm wheals to cat fur and grass pollen on prick testing. His chest radiograph (both posteroanterior and lateral views) showed clear lungs and no mediastinal abnormality. A further course of prednisolone 30 mg per day resulted in a rise of his peak expiratory flow rate from 140 l/min to 220 l/min in four days but his symptoms were unchanged.

He was admitted to hospital, where treatment with salbutamol through a nebuliser failed to produce an improvement. A flow volume curve at this time showed an expiratory airflow obstruction not inconsistent with asthma (fig 1). He deteriorated acutely, becoming more breathless at rest, and then collapsed, requiring resuscitation and assisted ventilation. Despite a further 36 hours of corticosteroid treatment and assisted ventilation, great difficulty in lung inflation was experienced: peak inspiratory pressures exceeding 40 cm of water were required for a tidal volume of 600 ml. Rigid bronchoscopy was undertaken, at which the distal third of the trachea was found to be occluded by tumour like tissue (fig 2). This tumour was resected as an emergency through a right posterolateral thoracotomy, the lower trachea and origins of both main bronchi being excised and a gap of 12 cm closed

Failure of recognised treatment for asthma over a reasonable period in a suspected asthmatic is an indication not only for radiographic definition of the lower central airways but also endoscopic examination

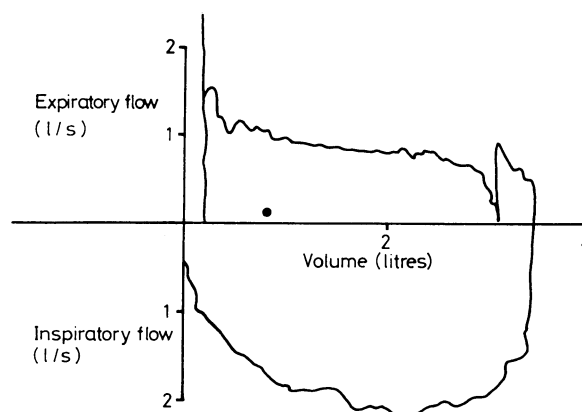


FIG 1—Flow volume curve is performed by recording flow and volume change simultaneously; the patient inhales to total lung capacity then forcibly exhales into the spirometer to residual volume and then rapidly inhales again from the spirometer to total lung capacity. Our patient's flow volume curve is shown, expiratory loop upwards and inspiratory loop downwards, expiratory flow being more limited than inspiratory flow.

by end to end suture of the left main bronchus to the lower trachea. It was necessary to perform a right upper lobectomy and anastomose the right intermediate bronchus end to side on to the left main bronchus. The patient was extubated after closure of the chest and required suction bronchoscopy to remove slough on the first and second days after operation. He went home three weeks after operation with a paralysed right vocal cord, which was treated by an injection of Teflon paste as an outpatient. Histological examination of the excised specimen showed a malignant carcinoid tumour which had invaded the main bronchial resection margin. Repeat bronchoscopy at five weeks showed moderate stenosis at the anastomosis, the histology of which was granulation tissue only.

Comment

This man presented in his 30s with airflow obstruction which was progressive despite adequate medical treatment for asthma. It is salutary to look again at the general practitioner's referral letter: "I am concerned about what his diagnosis might be since I would have expected asthma of a simple nature to have respon-

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ded to the above [Prednisolone 20 mg per day plus aerosols] treatment." The failure of recognised treatment for asthma over three months was an important clue that the diagnosis was in-

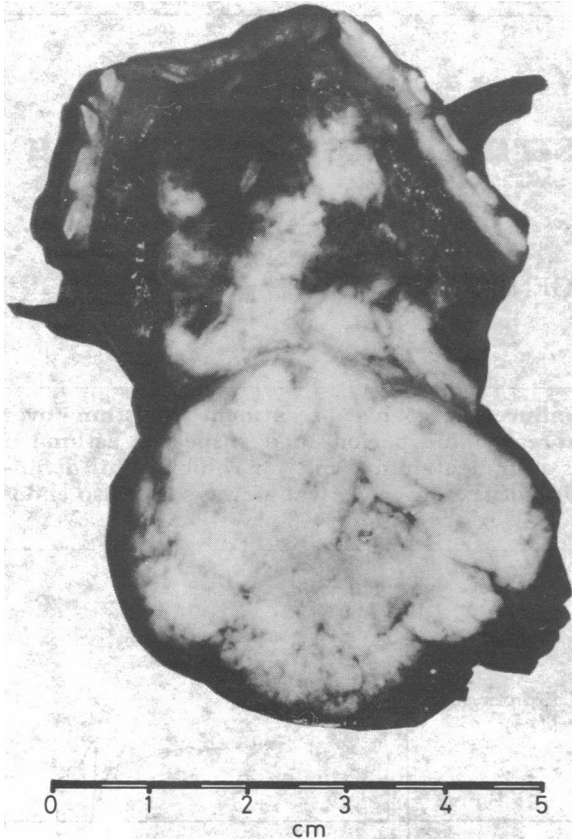


FIG 2—Excised tumour invading subcarinal node and extending into the carina.

correct. Recognition of a variable central but intrathoracic airway obstruction may be delayed if physical signs and lung function tests alone are relied on, because a pliable lesion of the central intrathoracic airways is compressed during expiration in a similar way to the more peripheral obstructed airways in asthma or chronic obstructive bronchitis. Both the flow volume curve abnormalities and the wheeze were predominantly expiratory.

In such patients radiographic definition of the central airways is a vital investigation. Unfortunately routine standard x ray films usually fail to define the lower trachea and carina. If facilities for high kilovoltage radiography are available we would suggest that right and left posterior oblique views of the chest should be performed. They are easy to obtain and contrast well the air-containing central airways from the other mediastinal structures. Conventional and computed tomography provide excellent definition of the major airways but may be difficult to perform and hazardous if the patient is severely dyspnoeic. Nevertheless, as shown in our patient, the definitive investigation is endoscopic examination of the airways.

For central primary malignant tumours of the trachea and carina operative excision remains the treatment of choice.³ As reconstruction of the bronchi and carina is required such treatment should be planned, with adequate preparation for intraoperative ventilation via bronchial cannulas. Preoperative histology from bronchoscopic biopsy may not be obtainable as difficulty in ventilation can result if bleeding occurs in already narrowed central airways.

We thank Mr B B Milstein and Mr T A H English for permission to report this case and Dr C R Flower for his helpful advice.

References

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Is it wise to give penicillin injections to patients with asthma or with other features of atopy? Would a penicillin sensitivity test be of value?

It is often stated that patients with a history of atopic disease (asthma, hay fever, etc) are at increased risk of allergic reactions to penicillins but the evidence for this is conflicting.¹ Early studies found an increased incidence of anaphylaxis to penicillin in atopic individuals but more recent studies have not confirmed this. For example, two large studies found no correlation between penicillin sensitivity and a history of atopy in over 4000 patients.² On this evidence there seems no good reason to deny penicillin to patients with asthma or other features of atopy. Some authorities, however, recommend restricting the use of penicillin in such patients to cases which cannot be equally well treated with other antibiotics.³ Sensitivity testing may be useful in patients who give a history of penicillin allergy and who need penicillin treatment for an infection for which other antibiotics are less effective. Skin tests are more useful than in vitro tests for predicting the patient who may develop anaphylaxis, but both false negatives and false positives occur even in expert hands. It is important to test with both multivalent penicilloyl haptens such as penicilloyl-polylysine (PPL) and with a mixture of the "minor determinants" thought to be primarily responsible for anaphylaxis.¹ ³—LINDA BEELEY, consultant clinical pharmacologist, Birmingham.

¹ Erffmeyer JE. Adverse reactions to penicillin. *Ann Allergy* 1981;47:288-300.

² Horowitz L. Atopy as a factor in penicillin reactions. *N Engl J Med* 1975;292:1243-4.

³ Assem E-SK. Drug allergy. In: Davies DM, ed. *Textbook of adverse drug reactions*. 2nd ed. London: Oxford University Press, 1981:534-68.

A man in his middle 70s suffers from what I believe is called a stammering bladder. He has difficulty in starting to micturate when away from home. A successful prostatectomy three years ago has not affected the complaint. Can you suggest any treatment?

Some men are unable to initiate the act of micturition in the presence of others in a public lavatory.¹ This has been termed the "anxious bladder" and occurs in a younger age group than the patient in question. A "successful" prostatectomy whether undertaken for prostatism or for urinary retention will almost always cure the patient of hesitancy even in the presence of considerable detrusor decompensation or failure. It is implied that the patient in question does not have hesitancy when in familiar surroundings and has no complaint regarding the force of his urinary stream. Does he take any medication that may adversely affect detrusor function when he travels away from home? This man could usefully complete a frequency/volume chart over a period of one to two weeks and indicate the occasions when he has experienced difficulty. The problem may, for instance, be related to attempts to void relatively small volumes of urine "prophylactically" before attempting a return journey by car or coach. A detailed and careful history could well provide the vital clue to this man's problem, but if that is not the case then he should be referred for a full urodynamic assessment, preferably combined with video cine cystourethrography.—J C GINGELL, consultant urologist/lecturer in urology, Bristol.

¹ George NJR, Slade N. Hesitancy and poor stream in younger men without outflow tract obstruction—the anxious bladder. *Br J Urol* 1979;51:506-10.