Case Report

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Intractable Asthma and Mitral Stenosis

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Key Words

Reversible airway obstruction
Bronchial hyperresponsiveness
Left ventricular dysfunction

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Clinically the presentation of asthma with airway obstruction is easily distinguished from pulmonary vascular congestion and oedema [1]. However, on occasions the clinical features of the two can overlap and pose both a diagnostic and management dilemma unless the co-existence of the two conditions is recognized.

We report 2 such cases and briefly review some of the current thoughts on the subject.

Case Reports

Case 1

A 29-year-old woman of Asian origin had suffered from asthma since childhood. She had a family history of asthma and was a non-smoker. Her condition was well controlled on terbutaline and budesonide inhalers, however she reported increasing shortness of breath at rest over the previous 6 months. In particular she complained of a troublesome nocturnal cough, with wheeze. During this time she had two admissions to this hospital, acutely short of breath, on both occasions she responded well to standard acute asthmatic therapy. On her follow-up in the out-patient clinic she was started on oral aminophylline and inhaled salmeterol with poor symptom control.

Relevant past medical history included the finding of mild hypothyroidism 6 years previously, treated with thyroxine 50 µg daily. At this time she was noted to have the pansystolic murmur of mitral regurgitation, this was confirmed on echocardiography, which showed it to be mild, with evidence of rheumatic involvement of the mitral valve. There was good left ventricular function and a normal-sized left atrium.

On examination, at this presentation, she had a malar flush, was in sinus rhythm, at a rate of 100 beats per min, with no murmur audible

on auscultation. A loud pulmonary second sound was noted. On chest examination she had marked bilateral wheeze and a peak expiratory flow rate (PEFR) of 110 l/min.

Chest X-ray showed cardiomegaly (cardiothoracic ratio; 15/25), with an enlarged left atrial appendage and prominent upper lobe blood diversion. An echocardiogram at this time showed tight mitral stenosis, with a valve area of 0.8 cm². There was moderate tricuspid

regurgitation and also pulmonary hypertension (80 mm Hg + right atrial pressure). Pulmonary function tests after stabilisation on medical therapy showed a marked obstructive defect, with a substantial reduction from predicted values in forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁) and PEFR. The actual values were 1.13 l (predicted value 3.05 l), 1.00 l (2.64 l) and 192 l/min (380 l/min), respectively.

Case 2

A 61-year-old woman had a diagnosis of late-onset asthma. She was an ex-smoker, however there was a significant reversible component to her pulmonary disease, with a 21% increase in PEFR after an inhaled B₂ agonist.

She was well controlled on terbutaline and beclomethasone inhalers, but had noticed a gradual deterioration in her breathing over the 4 years prior to this admission. She was known to have long-standing mitral stenosis, and gave a history of childhood rheumatic fever. She underwent open mitral valvotomy 8 years previously, but subsequent serial echocardiography noted gradual restenosis of the valve.

On presentation she reported a 2-day history of severe breathlessness, only partially alleviated by inhaled B₂ agonists, but no paroxysmal nocturnal dyspnoea, orthopnoea or swelling of the ankles. Her medication on admission included digoxin and diuretics.

On examination she was dyspnoeic at rest, had a malar flush and was mildly centrally cyanosed. She was in atrial fibrillation at a rate of 86 beats per min, and had the murmurs of both mitral stenosis and

mitral regurgitation on auscultation. On examination of her chest she had bilateral basal crackles and wheeze, and a PEFR of 100 l/min.

Chest X-ray showed cardiomegaly (cardiothoracic ratio; 17/26), an enlarged left atrial appendage prominent and upper lobe vessels. An echocardiogram showed severe mitral stenosis with a valve area of 0.9 cm². There was good left ventricular function, but an enlarged left atrium (5.6 cm internal diameter).

Pulmonary function tests showed a marked obstructive defect, with much reduction from predicted values in FVC 1.74 l (2.48 l), FEV₁ 0.68 l (2.08 l) and PEFR 146 l/min (342 l/min).

Case 1 is at present waiting for mitral valvotomy. Case 2 declined further operative intervention, for the present time, and was stabilized on medical therapy. She is now being followed up regularly in the out-patient clinic.

Discussion

It is well known that there is reversible airway obstruction in bronchial asthma. Reversible obstruction has also been demonstrated in both mitral stenosis and left ventricular failure of any cause [2]. This airway narrowing is also associated with hyperresponsiveness, (i.e. to methacholine challenge) [3].

Studies have shown that approximately 80% of patients with severe mitral stenosis have a history of severe bouts of wheezing and cough [4]. Overall airway resistance is approximately doubled in mitral stenosis [5] and patients are up to five times more sensitive than age-matched controls to bronchoconstrictor stimuli such as methacholine challenge [6]. It can be seen that mitral stenosis alone can produce a marked respiratory deficit, but, as shown in these cases, when combined with bronchial asthma causes severe respiratory

dysfunction. The degree of airway obstruction and hyperresponsiveness directly attributable to the mitral stenosis is uncertain.

Mitral stenosis is now rare in the Western world, however left ventricular dysfunction is increasingly common. It should be remembered that the latter can produce significant respiratory compromises, with signs and symptoms similar to airway obstruction. If this is not looked for, and recognised, patients will receive sub-optimal therapy.

These cases suggest that in patients with bronchial asthma, who have symptoms poorly responsive to standard therapy, occult valvular heart disease or left ventricular dysfunction should be highly suspected.

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