Lesson of the Week

Asthma presenting as cor pulmonale

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Carbon dioxide retention in patients with asthma is usually seen only in a severe attack and is generally regarded as a poor prognostic feature. We describe three relatively young patients who presented with severe hypoxaemia, hypercapnia, fluid retention, and polycythaemia and who after investigation and treatment clearly had asthma. All caused diagnostic difficulty and were initially referred to cardiologists with suspected right to left shunts or unexplained pulmonary hypertension.

Any patient presenting with suspected right heart failure or unexplained pulmonary hypertension should undergo careful respiratory assessment for asthma

Case reports

Case 1—A 29 year old lawn mower engineer who did not smoke presented with dyspnoea for five months and ankle oedema for three months. He had had intermittent wheezing since the age of 5 but had never taken regular treatment. On examination there was central cyanosis, raised jugular venous pressure, oedema of both ankles, a loud pulmonary component of the second heart sound, and widespread wheezing. Chest *x* ray examination showed cardiomegaly with prominent pulmonary arteries and an electrocardiogram showed right atrial and right ventricular hypertrophy. There was pronounced polycythaemia; respiratory function tests showed severe airways obstruction and moderate hypercapnia and hypoxaemia (see table I).

TABLE I—*Results of spirometry, haemoglobin concentrations, and arterial blood gas values before and after treatment*

Case No		FEV_1 (l)	Vital capacity (l)	Haemoglobin (g/dl)	Pao_2 (kPa)	$Paco_2$ (kPa)	Venous HCO_3^- (mmol/l)
1	Before treatment	0·85	3·85	22·7	7·1	7·2	33
	Best value after treatment	2·00	4·80	17·7	10·9	4·7	
2	Before treatment	0·80	2·40	18·2	*	7·6	32
	Best value after treatment	1·70	3·50	15·3	9·6	6·0	
3	Before treatment	0·95	3·50	17·7	5·9	7·5	38
	Best value after treatment	3·50	5·50	16·8	8·1	5·6	

*Not available.
Conversion: SI to traditional units—Pao_2, $Paco_2$: 1 kPa ≈ 7·5 mm Hg. HCO_3^-: 1 mmol/l ≈ 1 mEq/l.

Case 2—A 42 year old weighing machine engineer, also a non-smoker, with a long history of intermittent wheezing presented with increasing dyspnoea for four months and ankle swelling for one month. He had been diagnosed as asthmatic at the age of 18 months but had never taken regular bronchodilators. Examination showed central cyanosis and signs of airflow obstruction, pulmonary hypertension, and right heart failure similar to those in case 1. Chest radiography showed cardiomegaly, and an electrocardiogram showed right axis deviation with clockwise rotation and inverted T waves in leads V1-V4. There was polycythaemia, and spirometry confirmed severe airways obstruction with a small acute response to bronchodilator and hypercapnia (see table I).

Case 3—A 50 year old groundsman presented initially with a three month history of hoarseness, and laryngoscopy showed paralysis of the left vocal cord. In addition, he complained of increasing shortness of breath over five months and ankle swelling for two months. On direct questioning he admitted to intermittent cough and wheeze for 10 years; he had stopped smoking five years previously. Examination showed central cyanosis and signs of airflow obstruction, pulmonary hypertension, and right heart failure as in cases 1 and 2. Chest radiography showed cardiomegaly and prominent pulmonary arteries, and an electrocardiogram showed right axis deviation, incomplete right bundle branch block, and right ventricular hypertrophy. There was mild polycythaemia and severe airways obstruction but after two puffs of salbutamol the forced expiratory volume in one second (FEV_1) doubled from 0·95 to 1·80 l. Blood gas values indicated hypercapnic respiratory failure (see table I).

PROGRESS

The first two patients had clearly had asthma since childhood and it seemed probable that the third patient had developed asthma some 10 years before presentation. All were treated initially in hospital with controlled oxygen, diuretics, nebulised bronchodilator, and oral steroids and all showed a dramatic improvement within one to two weeks. The best subsequent measurements of respiratory function and haemoglobin concentration (table I) showed considerable improvement but all three patients had some persisting airflow obstruction. Maintenance treatment consisted of inhaled salbutamol and beclomethasone with, in one case, oral theophylline; no patient remained on long term oral steroids. All made an excellent subjective improvement and independently said that they had not appreciated how restricted their activities had been before regular treatment.

In each case the physical, radiographic, and electrocardiographic evidence of pulmonary hypertension and cor pulmonale regressed; the figure shows the evolution of the electrocardiograms.

Table II gives the results of further investigations. Maximum respiratory pressures were measured to exclude respiratory muscle weakness, and these were normal. The ventilatory responses to carbon dioxide measured during rebreathing[1] were reduced; the measurements were made when the patients were in a stable state after a period of intensive treatment and when airway function was optimal. To assess ventilatory control further, measurements were also made of the mouth occlusion pressure response to carbon dioxide,[2] and the results were again below normal (table II).

Comment

These three patients had insidiously developed severe airways obstruction; in case 3 the acute bronchodilator response

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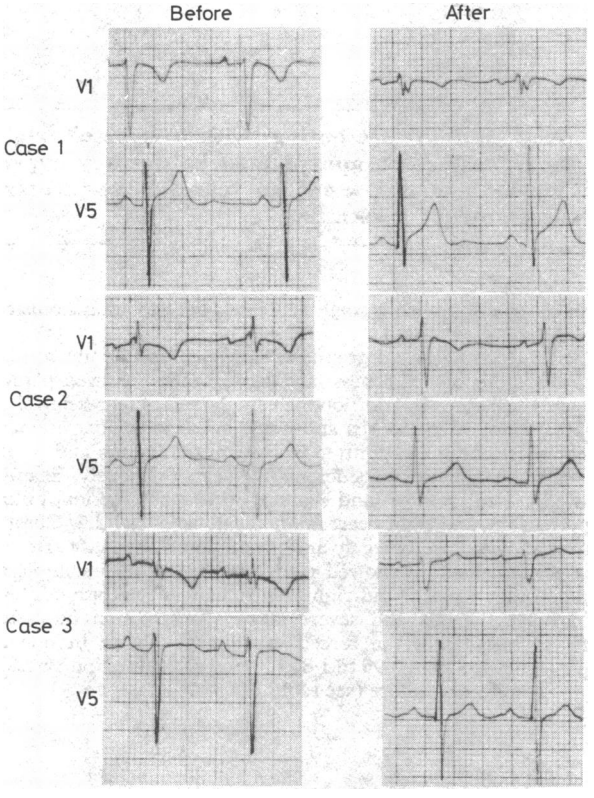
TABLE II—*Ventilatory responses and maximum respiratory pressures*

	Ventilatory responses		Maximum respiratory pressures	
	$V/PACO_2$ (l/min/mm Hg)*	$P_{0.1}/PACO_2$ (cm H_2O/mm Hg)*	Inspiratory (cm H_2O)	Expiratory (cm H_2O)
Normal value	1-5	0·37 (SD 0·17)	< − 75	> + 75
Case 1	0·23	0·14	− 73	+ 140
Case 2	0·50	0·12	− 150	+ 150
Case 3	0·71	0·09	− 100	+ 170

V = Expired ventilation. $PACO_2$ = Pressure of alveolar carbon dioxide.
*1 mm Hg ≈ 0·13 kPa.



Evolution of leads V1 and V5 of electrocardiogram of each of three patients before and after regular treatment. Note dominant S waves in lead V5 in each patient before treatment and regression after treatment, consistent with improvement in right ventricular hypertrophy.

was diagnostic of asthma but this was not confirmed in the other two patients until steroids had been given. Clinical features of right heart failure developed over a few months, presumably because of severe pulmonary hypertension, consequent on hypoxaemia and hypercapnia. The regression of the clinical radiographic, and electrocardiographic abnormalities with improvement of airflow obstruction and normalisation of blood gas values supports this suggested sequence of events. All three patients had a raised venous bicarbonate (HCO_3^-) concentration on presentation (table I), suggesting chronic respiratory acidosis. Two of the patients had been referred to cardiologists for investigation of possible right to left shunts, and an anatomical shunt was excluded by finding a normal arterial oxygen pressure (Pao_2 >67 kPa; >500 mm Hg) after breathing 100% oxygen. The left recurrent laryngeal nerve palsy in case 3 was attributed to stretching of the nerve by the dilated pulmonary artery.

Rebuck and Read[3] showed that the patients who tended to develop carbon dioxide retention during an acute severe asthmatic attack were those who appeared to have constitutionally low chemosensitivity, as they had low ventilatory responses to carbon dioxide in remission. Probably similar mechanisms operated in our patients. Since the ventilatory response to carbon dioxide is difficult to interpret in the presence of airways obstruction, the mouth occlusion pressure ($P_{0.1}$) response has been suggested as an alternative which avoids the problems of impaired mechanical performance of the respiratory system.[2] The $P_{0.1}$ response in our three patients was also reduced, and the normal maximum inspiratory pressures (table II) suggested that impairment of respiratory muscle function was unlikely to have contributed to the reduced carbon dioxide responses.

It was noteworthy that the patients had similar phlegmatic personalities; mixed venous Pco_2 in patients with airflow obstruction is higher in introverts than in extraverts[4] and a relation between extraversion and chemoreceptor sensitivity has been shown in healthy young women.[5] We conclude that our patients probably had a constitutionally low sensitivity to carbon dioxide and that this permitted the development of hypercapnia and its consequences more readily than usual in the face of gradually worsening airflow obstruction. This is an unusual occurrence in patients with asthma but the three patients described here were seen over only two years in a regional referral centre. The dramatic improvement after appropriate treatment emphasises the need for a careful respiratory assessment in any patient presenting with right heart failure or pulmonary hypertension and for intensive treatment with bronchodilators and a trial of corticosteroids in those with airways obstruction.

We thank Professor D G Julian and Dr C B Henderson for referring patients originally under their care.

References

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Clinical curio: treatment of sea urchin stings

Some years ago in Bora Bora I was confronted with a patient with 20-25 long black sea urchin spines embedded deep in the plantar surface of the foot. Rather than immediately embark on what looked like a formidable surgical procedure I chose to consult the local French speaking medical auxiliary. He informed me that the widely used local treatment was soaking the foot in a bucket of freshly voided urine. Being somewhat incredulous (and not entirely sure of my translation), I pursued the matter further with him and was told that there was usually sufficient acetone in urine to dissolve the organic matrix of the spines. As acetone (used as paint thinner on our boat) was available I started acetone soaks, which caused the rapid resolution of the pain and the complete disappearance of the spines in a couple of days without surgical intervention. A painless blue tattoo marking the site of each puncture gradually faded over a few weeks.

Although this method of treatment has been practised traditionally in several areas of the Pacific, I have never seen any reference to it in standard textbooks or review articles on venomous stings; likewise various standard manuals on scuba diving and marine first aid mention only surgical removal of deeply embedded spines. I suggest urine or acetone soaks be tried rather than surgery.—JOHN S MILLAR, medical officer, Manus Province, New Guinea.