PAPERS AND ORIGINALS

Asthma deaths in Cardiff 1963-74: 53 deaths in hospital

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Summary

In a study of factors associated with death from bronchial asthma in hospital 53 patients were investigated. Typically the fatal attack persisted for several days before admission to hospital and normally occurred in patients with a long history of asthma. The patient or doctor often underestimated the severity of the attack. On admission most patients were severely ill, and over a third died within 24 hours. Peak flow rate and blood gases were rarely measured. Corticosteroid treatment was often underused, and patients rarely received assisted ventilation before death. Infection played a part in 14 deaths, five of them associated with assisted ventilation.

Admitting asthmatics to a special respiratory ward with facilities for standardised assessment and treatment and introducing a self-admission service may help to prevent some of these deaths.

Introduction

Every year between 150 and 200 patients are admitted to the Cardiff hospitals with severe exacerbations of asthma, and four to five of these die. We analysed deaths occurring in asthmatics in this area between 1963 and 1974 to attempt to identify areas where improved management might have prevented the deaths.

Methods

Data on every death in hospital in the Cardiff area mentioning "asthma" were collected shortly after the death as detailed previously.[1] Criteria for inclusion were death inside hospital with necropsy evidence of asthma or a convincing clinical history.

In 40 patients necropsy showed hyperinflated lungs that did not collapse on opening the thoracic cavity and a bronchial tree extensively plugged with sticky mucus. Ten of the patients who died had attended the asthma clinic and had been shown to have reversible airways obstruction, and three others had classical histories of asthma. Patients in whom asthma was not the sole cause of death or with less convincing evidence for asthma were eliminated from the study.

Analysis of deaths

GENERAL FACTORS

The number of hospital deaths in Cardiff in 1963-74 are shown in the table. Two-thirds of the deaths occurred in April to September. Twelve of the 24 patients tested had at least one strongly positive skin test, though atopic status was not correlated with the season of death.

Seventeen patients were male and 36 female. Age at death ranged from 10 to 82 years with a mean of 46 years for males and 60 years for females.

Asthma deaths in hospital in Cardiff 1963-74

Year:	1963	1964	1965	1966	1967	1968	1969	1970	1971	1972	1973	1974
	13	9	4	3	2	3	5	3	3	6	2	0

SPECIFIC FACTORS

Severity of asthma—The age of onset was known in 51 patients and ranged from 1 to 72 years. Fifteen (29%) of these patients had had their first attack by the age of 20 and in 24 (47%) attacks, started after age 40. Length of history ranged from six months to 63 years, with a mean of 20 years. Only two patients died within five years of onset and most were chronic asthmatics. The history of previous hospital admissions with severe asthma was known in all 53 subjects. Thirteen patients (25%) had had no previous admissions, while 16 patients (30%) had had three or more. In the year before death 27 patients (51%) had been admitted, and 13 of these had been admitted more than once. Only four patients had been discharged less than six weeks before their final admission. Of the 41 patients with adequate records 16 (39%) had never been referred to a clinic. Nineteen patients (46%) were seen in the month before admission and seven were admitted directly from the clinic.

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Drug treatment—Immediately before final admission 34 patients were taking oral steroids and one was on ACTH. Ten patients were taking prednisone 5 mg/day, 12 on 10 mg/day, and 10 on at least 15 mg/day. Steroid dosage was not recorded in two patients. Thirteen patients were taking antibiotics before admission.

FINAL EPISODE

Length of attack—The duration of the episode of severe wheezing before the final admission was known in all 53 subjects. The mean duration was 11 days. No attack had lasted less than two hours before admission but 10 attacks lasted less than one day. Fourteen episodes had lasted between one and four days. Twenty-eight attacks had persisted for at least four days, and 15 of these lasted over 14 days. One final attack started during an admission for anaemia secondary to hiatus hernia.

Severity of admission—With the classification suggested by Jones[2] to assess severity, 13 patients were still able to carry out their jobs with great difficulty before admission, 13 were confined to bed but could get up with difficulty, and 15 were completely confined to bed. Twelve patients were moribund. On admission 29 patients were alert, 11 drowsy, and 13 unconscious. Sixteen patients had extreme or severe wheezing and in 25 subjects respiratory effort was severe or maximal. Thirty-two patients were noted to be cyanosed on admission. Mean pulse rate was 135/min. Only two patients had pulse rates under 100/min and 12 pulse rates were over 160/min. Most patients were normotensive.

Peak flow rate was measured on admission in 17 patients. No peak flow exceeded 95 l/min, and in nine subjects it was unrecordable. Arterial P_{CO_2} was recorded in 19 severely ill patients. Mean P_{CO_2} was 10 kPa (75 mm Hg) and was below 6·6 kPa (50 mm Hg) in only three patients. Mean arterial P_{O_2} was 5·6 kPa (42 mm Hg) in five patients. Plasma electrolyte measurements were uninformative.

Length of survival—Mean survival time after admission was 4·6 days in 53 patients. Five patients died within two hours of admission and 20 patients died within 24 hours. A further 12 died between one and three days and nine patients survived at least 10 days before death. The hour of death was known in 41 of the 53 subjects and showed no significant trend.

Treatment—Detailed treatment was analysed in 50 patients. In the first two hours after admission 31 of the 45 surviving patients were given steroids. Parenteral hydrocortisone was given to 23 patients: 11 received 100 mg and five received over 300 mg. Twenty-five patients were given oral prednisone, although only seven received more than 10 mg. A mean dose of 60 units of ACTH was given to 15 patients. Thirty-four patients received a mean dose of 400 mg aminophylline intravenously. An intravenous infusion was set up in 10 patients. Antibiotics were given to 25 patients and respiratory stimulants to five. By 24 hours after admission 25 of the 30 surviving patients had received steroids. Eighteen had been given a mean dose of 950 mg hydrocortisone; only seven patients had received less than 400 mg. A mean dose of 35 mg oral prednisone had been given to 18 patients, of whom eight received 20 mg or less. Eleven patients had received a mean of 120 units ACTH. An average dose of 650 mg aminophylline had been given to 28 patients, although nine of these patients had 250 mg or less. Two patients had received no treatment at all. Patients dying within the first 24 hours had normally been given considerably less initial treatment than survivors.

Progress—In the first 24 hours 17 patients improved, six substantially and 11 slightly. Five patients showed no improvement, 11 patients deteriorated, and 20 patients died. The clinical course during admission was a rapid deterioration to death in 20 patients and a slower inexorable decline to death in 11 patients. Seven patients showed an initial improvement, then declined gradually to death. Nine patients showed a prolonged improvement, then died after a clinically satisfactory recovery. Twenty-one patients therefore died despite a good initial clinical response. Seventeen of these 21 patients had pulse rates persistently raised above 120/min, and respiratory rates continued above 30/min in nine patients. Pulse or respiratory rates showed an inadequate response in 19 out of 21 patients who subsequently relapsed. Steroid dosage had been rapidly reduced in only two of these 21 patients. Only five patients had repeated measurements of ventilatory function, and arterial blood gases were measured more than once in only seven.

Assisted ventilation—Seven of the 12 patients given assisted ventilation were ventilated within 24 hours of admission. Before ventilation 10 patients were unconscious. Mean arterial P_{O_2} was 4·4 kPa (33 mm Hg) in eight patients. The mean arterial P_{CO_2} of the 12 patients was 12·5 kPa (94 mm Hg) and mean pulse 154/min. Five patients died while being put on the ventilator, one of aspiration of gastric contents. Four died with pulmonary infections: two with penicillin-sensitive coagulase positive staphylococci, one with pneumococci, and one with bronchopneumonia and lung abscesses caused by an undiscovered organism. One died as a result of cerebral softening related to prolonged hypoxia before ventilation, one of asthma complicated by a pseudomonas infection after coming off the respirator, and one of irreversible bronchial obstruction after three weeks on the ventilator. Bronchial lavage was used in this and one other patient without effect.

Infection—In addition to the five patients in whom infection played a part in causing death during or after assisted ventilation, the asthma in a further nine patients was complicated by chest infection sufficiently serious to contribute to death, while one other patient died of aspiration of stomach contents. Five of these were shown to have an acute purulent tracheobronchitis in addition to their typical asthmatic plugs at necropsy; in one a coagulase-positive *Staphylococcus* was cultured from sputum. One other patient's sputum contained this organism, while sputum from two others showed *Klebsiella* and *Proteus*. In one patient there was clinical and radiographic evidence of bronchopneumonia, but no organism was cultured.

Sedation—In five patients mild sedation seemed to produce no problems, but four patients died within one hour of parenteral sedation.

Discussion

In contrast to our findings in patients who died at home,[1] those who died in hospital had suffered a much more prolonged illness, and at the time of admission the danger signs of tachycardia, cyanosis, and exhaustion were often pronounced. Most patients died within the first three days, particularly on the first day. The initial treatment administered to these patients was generally less intensive than that used nowadays, with relatively small doses of bronchodilators and corticosteroids. The few patients in whom arterial gases were measured had often reached the stage of hypercapnia, where assisted ventilation becomes almost inevitable in asthmatic adults. This contrasts with the management of patients with chronic bronchitis in respiratory failure, in whom hypercapnia is often tolerated until high levels are reached, whereas in severe asthma hypercapnia is a terminal event. Clearly, assisted ventilation would probably have saved those who died too quickly to obtain full benefit from steroid treatment. When assisted ventilation was used, however, it was introduced late in the course of the illness, in most cases when the patient was already unconscious.

A small but important group of patients showed an initial clinical improvement only to deteriorate again after several days. Despite their symptoms being eased, their pulse rates remained raised, and tachypnoea often persisted. Measuring peak flow and blood gas levels would have alerted the clinicians to approaching danger in these patients, but these measurements were not made. In general few measurements other than pulse rate were made after the initial assessment, and even on admission peak flow and blood gases were measured in only a few patients. Our findings show clearly that most patients were severely ill when admitted to hospital and many were past the stage of responding to conventional treatment other than assisted respiration.

This state of affairs is analagous to that in the home deaths group and reflects the tendency of asthmatics and their attendants to wait until death is close before treating an attack with the respect it deserves. Analysis of our results has shown that the introduction of an Edinburgh-type self-admission service[3] together with better education of our patients along the lines used by Jones[2] would have allowed 28 of the 53 to have been admitted earlier and with a greater chance of survival. The patients in this study were not admitted to a special unit but to the general medical and geriatric wards of the Cardiff hospitals. Ready access to facilities for assisted ventilation may well have been a problem while the necessity of treating a wide range of medical problems may have obscured the importance of continuing assessment of the asthmatics. In the Cardiff area blood gas analysis was introduced as a laboratory service and lung function tests were rarely carried out on inpatients before

the late 1960s. An assisted ventilation unit was opened in 1967. Since 1974 most patients with severe attacks of asthma in Cardiff have been admitted to a special respiratory unit in keeping with the recommendations of Jones[2] and Cochrane and Clark.[4] This has allowed standardisation of treatment and assessment and has paved the way for the introduction of a service for earlier admission of patients. We recommend that such units be introduced more widely by physicians interested in asthma.

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Prospective comparison of double-contrast barium meal examination and fibreoptic endoscopy in acute upper gastrointestinal haemorrhage

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Summary

Fibre-optic endoscopy was compared prospectively with double-contrast radiology in 53 consecutive patients admitted with acute gastrointestinal haemorrhage. The bleeding site was correctly identified by endoscopy in 94% of patients and the final diagnosis was correctly given in 89%. The corresponding figures with radiology were 83% and 74%. Among the 50 patients with a final diagnosis of a bleeding site in the upper gastrointestinal tract endoscopy indicated the site of bleeding in all and radiology indicated it in 88%. Both investigations were well tolerated by patients. Endoscopy is the investigation of choice, but when it is not available double-contrast radiology will show the site of bleeding in 80-90% of patients.

Introduction

Until the advent of fibre-optic endoscopy the barium meal was the principal tool for investigating patients with acute upper gastrointestinal haemorrhage. A cause for bleeding could be found in 76% of patients using the Hampton technique,[1] and the acute ward barium-meal examination had an accuracy of 83%.[2] Workers using fibre-optic endoscopy have shown, however, that false-positive radiological diagnoses are not uncommon,[3] that multiple lesions are present in 15% of patients, and that 26% of patients with duodenal ulcers bleed from a different source. Lesions were present in 26 out of 34 patients with a negative barium-meal result.[4] The diagnostic accuracy of endoscopy in the early investigation of acute upper gastrointestinal haemorrhage varies from 86% to 97%,[3 5-8] but the accuracy of radiology has fallen since 1952 to 37% to 51%,[3 5 7] although accuracy rose to 59% when possible diagnoses were included[5] and 65% when the emergency films were reviewed.[7]

Many recent studies can be criticised because they have compared endoscopy with a retrospective review of routine radiology reports. A retrospective review of radiology and endoscopy over four years showed a bleeding-site detection rate of 61·5% by radiology and 57% by endoscopy.[9] Another retrospective review of radiology and endoscopy also failed to show a significant difference between the two techniques.[10] Not all hospitals are equipped with fibreoptic endoscopes and in those that are standards must vary. Double-contrast radiography of the stomach is more accurate than the conventional techniques, reducing the error rate from around 22% to 6% when compared with endoscopy,[11] and has been strongly advocated in acute upper gastrointestinal bleeding.[12] We carried out a prospective comparative study of endoscopy and double-contrast barium-meal examination in patients with acute upper gastrointestinal haemorrhage.

Methods

All patients admitted to the medical wards with a diagnosis of haematemesis or melaena underwent both endoscopy and a barium-meal examination, with the order of investigation determined by random allocation. When possible the first investigation was carried out within 24 hours and the second the next day. The endoscopies were performed by one author (GWS) and the double-contrast barium meals by two (RRC and GWS). The second investigation was always performed with full knowledge of the clinical findings and the results of the previous investigation. Endoscopes used were the forward-viewing Olympus GIFP and ACMI F8 and the side-viewing Olympus JFB2 and GFB2. Barium meal examinations were performed using an under-couch tube with a 2·0-mm focal spot, barium sulphate (Baritop), effervescent powder, and an anti-foaming agent. Some patients were given Metoclopramide 30 minutes before the examination, and most had either intravenous glucagon 0·2 mg or hyoscine butylbromide (Buscopan) 20 mg.

Results

Sixty-six patients entered the trial. Two-thirds were examined within 24 hours of admission and the average delay was 1·8 days for the first examination and 3·7 days for the second (range 2 hours to 17 days and 6 hours to 8 days respectively). Fifty-three patients underwent both endoscopy and double-contrast barium meal examination, endoscopy being the first examination in 25 and the second in 28. The

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