SHORT REPORTS

Acute myocardial infarction in Asians and whites in Birmingham

There is evidence that immigrant Asians suffer worse coronary artery disease than Asians remaining in their own countries and the indigenous population of the country of adoption.¹⁻³ Asians may make poor use of National Health Service provisions.⁴ This study compares Asians and indigenous whites admitted to our coronary care unit from the casualty department with a history of acute chest pain due to suspected myocardial infarction.

Methods and results

The records of 3208 admissions to the coronary care unit from June 1976 to December 1981 were examined and 169 Asians were identified by their names. Their original case notes were retrieved and scrutinised; 27 Asians had not been admitted to the unit directly from the casualty department, and these were excluded from analysis. A further 14 sets of records were untraceable or inadequate. The remaining 128 Asian patients were then matched for age, sex, month of referral, and time of arrival in the casualty department (morning, afternoon, or evening) with indigenous whites. It proved impossible to match four Asians, who were exceptionally young (18 to 22 years), leaving 124 matched pairs for the final analysis. Sixty per cent of the Asians had been born in Pakistan, the remainder coming from east Africa, Bangladesh, and India. With few exceptions these patients were Moslem and at least half spoke no English. Differences between the two groups were assessed by Student's *t* test for paired data; where subgroup comparisons were made, the χ^2 test was used.

Of 124 Asian patients, 52 had been seen by their own general practitioners and sent into hospital; the remainder had been sent by a deputising doctor or had dialled 999. Sixty four of the whites had been seen by their own general practitioner, the remainder being admitted either by a deputising service or a 999 call. The patients were well matched for age (Asians 52.6 (SD 7.8) years, whites 52.9 (7.9) years). There were 111 men and 13 women in each group of 124 patients. The mean time from the onset of chest pain to arrival in the casualty department was 301 (30) minutes for Asians and 320 (33) minutes for whites, but the mean time from arrival in the casualty department to arrival in the coronary care unit was significantly longer in Asians (117 (94) v 84 (50) minutes; $p < 0.001$). The main comparisons concerning the diagnosis and complications of myocardial infarction and site of infarction are listed in the table.

Comment

Asian patients who developed chest pain did not differ in their use of emergency medical services when compared with whites suffering the same symptoms. An equal proportion of both groups was seen either by their own general practitioner or by the deputising service, or made use of the 999 call. Delay between the onset of symptoms

and arrival in the casualty department was similar in both groups. Most Asians had Asian general practitioners but many were seen by the deputising service outside normal hours; in Birmingham the general practitioner deputising service employs many Asian doctors and thus our Asian patients probably had less of a language problem with doctors than after their admission to hospital.

Our results indicated that Asians admitted to our coronary care unit from the casualty department with acute chest pain differed little from matched whites with regard to the incidence and complications of myocardial infarction. There were two exceptions: the incidence of ventricular arrhythmias was greater in the Asians, and the length of time elapsing between arriving in the casualty department and being admitted to the coronary care unit was also longer. Few of our Asian patients spoke fluent English and at least half spoke no English at all. We believe that difficulty in communication between patient and doctor probably accounted for the longer delay that Asian patients experienced. A direct correlation between the delay and the increased incidence of ventricular arrhythmias is impossible to prove from this study. Neither of these complications, however, adversely affected the mortality in the Asian group.

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Pancreatitis in sarcoidosis

We studied two patients with acute episodes of pancreatitis caused by sarcoidosis, an association not previously recorded. Prompt resolution of the pancreatitis occurred after treatment with steroids, which are usually contraindicated for the more usual forms of pancreatitis.

Case 1

A 47 year old woman was admitted to hospital with a history of headache and vomiting. Computed tomography of the brain showed an infarct of the left occipital lobe. Sarcoidosis had been diagnosed 19 years earlier when she had presented with erythema nodosum and was found to have non-caseating granulomas on a cervical node biopsy. One year before this admission she suffered a cerebellar infarction and had an attack of unilateral parotitis. She also suffered from hypertension which was treated with bendrofluazide 2.5 mg daily.

Three days after admission she developed severe abdominal pain. Serum amylase activity was 3885 IU (normal 70-300) and mainly pancreatic in origin.¹ Serum calcium and lipid concentrations, ultrasonic appearances of the biliary tree, computed tomograms of the pancreas, and stool fat estimations were all normal. Active pulmonary sarcoidosis was diagnosed on the basis of chest radiographs, gallium scan, bronchoalveolar lavage findings, and raised serum angiotensin converting enzyme activity. Both skin and transbronchial biopsies showed granulomas.

The thiazide diuretic was stopped and the pancreatitis treated conservatively. As the pancreatitis failed to resolve after six weeks, oral prednisone 40 mg daily was given, producing a prompt clinical improvement; the serum amylase returned to normal. After being discharged she was

Details of myocardial infarct and complications, history, and previous treatment in 124 Asians and 124 whites admitted to coronary care unit with suspected myocardial infarction. Unless otherwise shown results are numbers of patients

	Asian	White
Myocardial infarct	87	89
Anterior	41 (47%)	51 (57%)
Inferior	37 (42%)	27 (30%)
Other sites	9	11
Peak mean (SD) serum aspartate aminotransferase (IU/l)	156.6 (144.6)	188.8 (203.0)
Mean (SD) Norris index ²	6.08 (3.11)	6.27 (3.07)
Cardiac arrest	9	13
Death	6	12
Heart failure	26	29
Supraventricular arrhythmias	27	21
Ventricular arrhythmias (excluding cardiac arrest and death)	30	18*
Conduction disturbance	22	22
Temporary cardiac pacing	5	8
Smoking	58	81
Hypertension	26	22
Diabetes	12	6
Previous cardiovascular disease	10	11
Previous coronary heart disease	63	53
Previous arrhythmias	12	15
Diuretics	18	15
β blockers	27	26
Calcium antagonists	8	12
Other antiarrhythmics		

* $p < 0.05$.

inadvertently treated with bendrofluazide for two months. Despite this she had no recurrence of pancreatitis over a year although she was maintained on a small dose of steroids.

Case 2

In 1982 an 18 year old man was found to have sarcoidosis on the basis of bilateral hilar adenopathy and a mediastinal node biopsy containing non-caseating granulomas. He was given oral steroids.

During each of the following three summers he was admitted with hypercalcaemia and acute pancreatitis after exposure to strong sunlight. He also had a history of high alcohol intake. On admission in 1982 the serum calcium concentration was 3.7 mmol/l (14.8 mg/100 ml) (normal 2.2-2.6 mmol/l (8.8-10.4 mg/100 ml)) with a serum amylase value of 4440 IU/l. The admission figures in 1983 and 1984 were: serum calcium 2.9 and 3.22 mmol/l (11.6 and 12.9 mg/100 ml) respectively and serum amylase 4280 and 2480 IU/l respectively. Primary hyperparathyroidism was ruled out. Ultrasonography of the biliary tree and serum lipid and 25-hydroxyvitamin D determinations produced normal results. Serum angiotensin converting enzyme activity was raised. Prompt resolution of the clinical and biochemical abnormalities followed an increase in the dose of steroids on each occasion. The accompanying figure shows the course of the first and most dramatic attack of pancreatitis. Between each of these attacks the serum amylase and calcium concentrations were normal.

Case 2. First episode of acute pancreatitis. Maintenance dose of prednisone was 40 mg on alternate days, both before admission and after discharge.

Comment

The pathogenesis in both patients was different. In the first granulomatous infiltration of the pancreas probably occurred; in the second the pancreatitis was caused by hypercalcaemia induced by sarcoidosis.

Sarcoidosis of the pancreas has rarely been described in reported series of necropsies.² Pancreatic disease with sarcoidosis has been diagnosed during life in only seven patients;^{3, 4} none of whom had clinically acute pancreatitis. All were diagnosed from the biopsy specimens taken at exploratory laparotomy in the absence of a clinical diagnosis of pancreatitis. One patient had a mildly raised serum amylase value,⁴ but in all the others the serum amylase activity was either normal or not recorded.

The prompt response of pancreatitis to steroids in our patients can be attributed to suppression of active granulomatous pancreatitis in the first case and suppression of hypercalcaemia in the second.

As steroids are generally contraindicated for most other forms of pancreatitis, these findings are of considerable therapeutic importance.

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Cutaneous myiasis caused by larvae of *Cordylobia anthropophaga* acquired in Europe

Cutaneous myiasis caused by the larva of the tumbu fly *Cordylobia anthropophaga* occurs sporadically in Britain as an imported infection contracted in tropical Africa.^{1, 2} We report two cases in which the disease was acquired in northern Europe.

Case reports

Two brothers, aged 8 and 5 years, of Indian extraction but resident in London were recently seen at this hospital. They had visited India six months previously but had never been to Africa. During a brief visit to Holland a furuncular lesion developed on the neck of the elder boy. A few days later his younger brother developed a similar lesion on his right upper arm. These two lesions continued to develop over three weeks until the mother, attempting to express pus, extracted an insect larva from the centre of each of them. The next day she brought both the larvae and the children, to be examined at the Hospital for Tropical Diseases. The boils were 2 cm in diameter and unremarkable except for a distinct 2 mm central aperture. They healed uneventfully over the next 10 days. Microscopic examination of the posterior spiracles showed the larvae to be third stage larvae of *Cordylobia anthropophaga*.

Comment

The tumbu fly is believed to be restricted to Africa, south of the Sahara. The flies lay their eggs on dry shaded ground and sometimes

Characteristic posterior spiracles of the larva of *Cordylobia anthropophaga* ($\times 400$).