SHORT REPORTS

"Transferable deaths": their epidemiological importance

Until April 1981 the Office of Population Censuses and Surveys sent area health authorities data from death certificates of residents who died outside the authorities' boundaries (transferable deaths). Not only did this provide a complete local record for epidemiological purposes but the prompt notification allowed the names of these people to be removed from health service recall systems. The increase in clerical work that would have been entailed if this procedure had been continued after the 1982 reorganisation of the National Health Service from 98 area health authorities to 201 district health authorities combined with a backlog that had built up during industrial action taken by local registrars of births and deaths led the Office of Population Censuses and Surveys suddenly to stop this service. It has just decided to reinstate it. Permanent loss of this information would have had several implications locally.

Methods and results

A mortality register for City and Hackney Health District was set up on a microcomputer. Details from death returns on residents of the district who died during 1979-81 were entered. The information on deaths registered locally was routinely supplied, as was information on transferable deaths from the Office of Population Censuses and Surveys until April 1981. Details of transferable deaths that occurred after this date were specially obtained. The number of transferable deaths from January 1979 to April 1981 was verified to ensure a complete data set, and any missing data were obtained. The Office of Population Censuses and Surveys trained a research assistant in coding deaths according to the International Classification of Diseases; she coded all the information. Information from 6862 death certificates was coded; the items included the date and place of death, sex, date of birth, postcode and electoral ward of the last residence, all causes of death, and the certifier.

The table shows where deaths outside the district took place, most being in hospitals in neighbouring district health authorities or postgraduate hospitals. Further analysis showed that 67% (93/138) of infant deaths and 56% (28/50) of deaths of children aged 1-16 took place outside the district during 1979-81. This was expected and due to the local pattern of paediatric and neonatal care.

Place of death of residents who died outside City and Hackney Health District (transferable deaths)

	1979	1980	1981
Hospitals in neighbouring district health authorities	258	251	243
Postgraduate hospitals	73	51	77
Other hospitals	125	100	98
Private address outside district	39	29	25
Other (dead on arrival, etc)	37	45	26
Total (%)	532 (22)	476 (21)	469 (21)

The underlying cause of death was compared for deaths occurring within and outside the district. Causes were grouped into four categories (neoplasms, cardiovascular disease, external causes, and other). The three years were analysed together. The distribution of cause of death between deaths occurring inside and outside the district was different ($\chi^2=39.47$, $df=3$, $p<0.001$): an excess of deaths due to neoplasms and external causes occurred outside the district.

Comment

To be of use, mortality data need to be accurate,^{1 2} complete, and up to date. Analysis of deaths in City and Hackney Health District showed that a similar pattern occurred in each of the three years examined, with one fifth of deaths occurring outside the district. This is a high proportion compared with results of a study in Newcastle, where local records provided details on 95% of residents' deaths and it was concluded that the loss of information on transferable deaths would have only a small effect on district statistics.³

The data presented here reflect the pattern of health care in London and may apply in other areas where patients are referred for treatment outside their district of residence. For these district

health authorities the loss of information on transferable deaths would have serious implications for service planning, operation, and evaluation. In particular, the distribution of deaths from certain causes such as neoplasms or in age groups such as children, which may be of special value locally, would be considerably distorted without information on transferable deaths. It is concluded that a system of notifying districts promptly of deaths occurring elsewhere is essential. The recent decision taken by the Office of Population Censuses and Surveys to resume the service is therefore welcomed.

The mortality register was developed with a grant from the North East Thames Regional Health Authority fund for locally organised research.

¹ Royal College of Physicians and Royal College of Pathologists. Medical aspects of death certification. *J R Coll Physicians Lond* 1982;16:205-18.

² Alderson MR, Bayliss RI, Clarke CA, Whitfield AGW. Death certification. *Br Med J* 1983;287:444-5.

³ Jessop EC. Deaths occurring outside the district of residence. *Community Med* 1983;5:224-6.

(Accepted 19 January 1984)

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Eosinophilic leukaemia presenting with intolerance of alcohol

We report on a patient with eosinophilic leukaemia¹ whose initial symptom was intolerance of alcohol.

Case report

A 54 year old man presented with a nine month history of night sweats and a three month history of malaise, anorexia, and weight loss. He had abstained from alcohol for the previous three years because soon after drinking beer or spirits he would experience lower abdominal pain accompanied by lightheadedness, generalised weakness, and nausea.

Examination showed gross splenomegaly, enlarged axillary nodes, a palpable liver, and a loud pansystolic murmur at the cardiac apex. Haemoglobin concentration was 9.5 g/dl, white cell count $18.8 \times 10^9/l$ (42% eosinophils, 32% neutrophils, 14% lymphocytes, 6% monocytes, 1% myelocytes), and platelet count normal. Marrow aspirate was hypercellular with an appreciable excess of eosinophils and eosinophil precursors but no increase in blast cells. Liver biopsy showed numerous eosinophils invading the portal spaces and parenchyma. A chest radiograph showed bilateral pleural effusions, and an echocardiogram thickening of the left ventricular endocardium. Neutrophil alkaline phosphatase activity, antinuclear factor and serum immunoglobulin concentrations were normal. Examination of stools for parasites yielded negative results.

To confirm the effect of alcohol the patient, after giving his consent, drank 20 ml beer. About five minutes later he developed severe lower abdominal pain radiating to both groins, malaise, nausea, sweating, and diarrhoea. His symptoms abated over the next 45 minutes.

He was given a blood transfusion, and treatment was started with prednisolone 40 mg daily, hydroxyurea 1 g daily, and vincristine 2 mg at fortnightly intervals. Over the ensuing three months his general health improved; his spleen reduced in size, and the eosinophil count fell to normal. Three months later, however, he relapsed: his constitutional symptoms returned; haemoglobin concentration was 10.9 g/dl; white cell count $52.9 \times 10^9/l$ (52% eosinophils, 23% blasts); and platelet count $37 \times 10^9/l$. Biopsy of an axillary lymph node showed extensive infiltration by mature and immature eosinophils, and blast cells of the myeloid series were shown by staining with chloroacetate esterase. Chromosome studies showed C group trisomy. Chemotherapy was changed: he received a single dose of daunorubicin 70 mg and then cytarabine 240 mg and thioguanine 240 mg daily for five days. Prednisolone was continued at 30 mg daily. The eosinophil count fell, but he became increasingly confused and incontinent of urine. A further relapse was heralded by diffuse pulmonary infiltration and recurrent pleural effusions, and five months after presentation he died. Permission for necropsy was refused.

Comment

Although best known as a symptom of Hodgkin's lymphoma, intolerance of alcohol occurs in other types of malignancy, such as carcinoma of the cervix,² and occasionally in association with benign disease.³ Its underlying mechanism is not clear. Localised pain induced by alcohol often corresponds to the site of disease and may be a result of sudden vasodilation and congestion in neoplastic tissue. This, however, does not account for the systemic symptoms that often accompany pain induced by alcohol, as in our patient. These effects appear to be mediated by an unidentified substance released into the circulation.³ Among patients with Hodgkin's disease more extensive infiltration of nodes with eosinophils was found in those who experienced pain induced by alcohol.⁴ We, however, know of only one other patient with the hypereosinophilic syndrome and intolerance of alcohol; he experienced abdominal pain and diarrhoea after drinking alcohol (D J Weatherall, personal communication).

There was a long interval between the onset of symptoms in our patient and the final stage of his illness. Intolerance of alcohol antedating a clinical diagnosis has been well documented; of 54 patients with carcinoma of the cervix and alcohol intolerance, 20 had had symptoms for at least three years before carcinoma was confirmed.²

Intolerance of alcohol merits serious attention, and eosinophilic leukaemia should be considered in the differential diagnosis.

We thank Dr B J Boughton of Queen Elizabeth Hospital, Birmingham, who helped in the management of this case.

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³ Brewin TB. Alcohol intolerance in neoplastic disease. *Br Med J* 1966;**ii**:437-41.

⁴ James AH. Hodgkin's disease with and without alcohol-induced pain. *Q J Med* 1960;**29**:47-66.

(Accepted 19 January 1984)

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Unilateral myopathy: an unusual presentation of thyrotoxicosis

Proximal muscle weakness, sometimes associated with atrophy and usually with electromyographic abnormalities, often occurs in patients with thyrotoxicosis.^{1,2} It affects men more commonly than women and may occasionally overshadow the other manifestations of thyrotoxicosis. It is usually bilateral.

We report on a patient with thyrotoxicosis who presented with unilateral myopathy affecting the left shoulder girdle.

Case report

A 46 year old man presented with stiffness and weakness of his left shoulder muscles of 12 months' duration. His disability did not appear to be progressive, and there were no other neurological features. In the past 12 months he had lost 12.7 kg in weight despite having had a normal appetite, and he had had occasional diarrhoea.

On examination his pulse rate was 72 beats/minute and his blood pressure 176/94 mm Hg. He had drooping of the left shoulder with wasting of the left pectoralis major, the left supraspinatus, and to a lesser extent the left deltoid. Weakness of these muscles was apparent, with slight winging of the left scapula. Reflexes were normal, and there was no loss of sensation. Electromyography showed action potentials of abnormally short duration and increased polyphasic units. The thyroid gland was not enlarged, and

there was no bruit. There was no proptosis or ophthalmoplegia, or other eye signs.

Basal serum thyroid hormone concentrations were measured on two occasions by radioimmunoassay (Amersham), and stimulation tests with thyrotrophin releasing hormone were performed. The serum thyroxine and triiodothyronine concentrations were variable, and the serum free thyroxine concentration was on the borderline of being high, but the response of thyroid stimulating hormone to intravenous administration of 200 µg thyrotrophin releasing hormone was consistent with thyrotoxicosis on both occasions. Immunological studies showed neither thyroglobulin antibody nor thyroid microsomal antibody. Serum creatine kinase activity was normal.

On the basis of the flat response of thyroid stimulating hormone to thyrotrophin releasing hormone he was treated with carbimazole 10 mg three times a day and was assessed clinically and biochemically every four weeks. The table shows his serum thyroid hormone concentrations before and during treatment. After eight weeks some response of thyroid stimulating

Serum thyroid hormone concentrations before and during treatment with carbimazole 10 mg three times daily

Time	Thyroxine (nmol/l)	Triiodo- thyronine (nmol/l)	Free thyroxine (pmol/l)	TSH response (mU/l) to TRH test after:		
				0 min	20 min	60 min
Normal range	54-142	0.8-2.5	8.8-23.2		up to 6	
Before treatment	163 101	3.4 2.2	23.9	1.5 1.5	1.5 1.5	1.5 1.5
During treatment:						
4 weeks	95	2.7	13.8	1.7	1.3	1.5
8 weeks	92	1.6	12.6	2.2	7.6	6.2
12 weeks	72	1.5	18.0	1.9	6.9	6.4
16 weeks	113	1.8	15.0	3.5	3.4	12.7
20 weeks				4.0	17.0	15.2

TSH = Thyroid stimulating hormone. TRH = Thyrotrophin releasing hormone.

Conversion: SI to traditional units—Thyroxine: 1 nmol/l ≈ 77.7 ng/100 ml. Triiodothyronine: 1 nmol/l ≈ 65 ng/100 ml. Free thyroxine: 1 pmol/l ≈ 77.7 pg/100 ml.

hormone to thyrotrophin releasing hormone was seen, and by 20 weeks the response was normal. After 12 weeks' treatment he had gained 6 kg in weight and the weakness had improved slightly. The improvement was maintained, and by 40 weeks his muscle power was clinically normal, although some muscle wasting persisted. He was still receiving carbimazole 10 mg twice a day.

Comment

Muscular weakness in hyperthyroidism is well recognised. It may be the presenting feature and may precede all other manifestations of the disease. Recent studies concluded that the clinical findings of muscle weakness and atrophy cannot be ascribed to defects in motor neuron activity or to changes in neuromuscular transmission. Metabolic and contractile function also seem insufficiently altered to explain the muscle weakness.³ The development of myopathy is probably a direct effect of the increased concentration of circulating free thyroid hormones and is therefore usually bilateral. In this patient the myopathy was unilateral, affecting the left shoulder girdle muscles. The only consistent biochemical finding was the flat response of thyroid stimulating hormone to thyrotrophin releasing hormone.

We thank Dr I F Pye, consultant neurologist at Leicester Royal Infirmary, for permission to report on a patient admitted under his care and for his valuable comment on the manuscript.

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² Ramsay I. *Thyroid disease and muscle dysfunction*. 1st ed. London: Heinemann, 1974:208.

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(Accepted 25 January 1984)

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