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SHORT REPORTS

Prevalence of known diabetes in an urban Indian environment: the Darya Ganj diabetes survey

A high prevalence of diabetes among migrant Indian Asians has been reported from several places, most recently from Southall, London.¹ The prevalence in India has not been found to be unduly high,² implying that the high prevalence in migrants may relate to newly encountered environmental factors. No large studies of prevalence have been performed in India in recent years. We therefore undertook a survey in New Delhi, using a protocol closely similar to that used in the Southall diabetes survey.

Methods and results

A house to house inquiry was made at all households within a defined area in Darya Ganj, a fairly affluent suburb of New Delhi. Residents were asked whether anyone living there was known to be diabetic. If the answer was yes a questionnaire, formulated as for the Southall diabetes survey, was completed.

Information was obtained on 6878 residents (3643 (53%) men and 3235 (47%) women). A total of 213 people were reported to be diabetic, giving an overall crude prevalence of 3.1%. The table shows the age distribution of the diabetics and the age specific prevalence of the disease. None of the diabetics reported were

aged under 30; above this age the prevalence rose sharply to a peak of 16.9% in the age group 60-64. There were almost twice as many men (138) as women (75) with diabetes (crude prevalences of 3.8% and 2.3% respectively), and the age specific prevalence in men exceeded that in women at virtually all ages. All patients had been aged over 21 when diabetes was diagnosed, and 159 (75%) had been aged 30-54. Thirty five (16%) were treated with insulin and a further 150 (70%) received oral hypoglycaemic drugs. The mean known duration of diabetes was 8.4 years. Altogether 209 of the diabetics (98%) were Hindu and 154 (72%) vegetarian. One hundred and seventeen (85%) of the men with diabetes were businessmen and 97 (70%) graduates; the mean income of all the men with diabetes was 2500 rupees a month.

Comment

This survey showed an unexpectedly high prevalence of known diabetes in a fairly affluent population in New Delhi. The prevalence is much higher than that found in previous Indian surveys, although direct comparison is difficult owing to different methods of ascertainment. In a large study performed by the Indian Council of Medical Research in 1975 the total prevalence (of known and newly ascertained diabetes) in subjects aged over 15 in six urban centres ranged from 0.9% (Delhi) to 3.7% (Ahmedabad), with a mean of 2.1%.³ About one half of the subjects were previously known diabetics. In Ahmedabad the prevalence of known diabetes was 1.65%,³ compared with 4.1% in the same age group in this study.

The high prevalence in Darya Ganj may relate to socioeconomic state, improved relative survival of patients with diabetes, more intensive screening of the population, or all of these factors. Most subjects came from business or professional families, and the mean monthly income was considerably above the national average. Whatever the explanation, the reported prevalence was much higher than we had expected. Moreover, there were probably other people in the community with undiagnosed diabetes. The true prevalence can be ascertained only by systematically applying standard diagnostic techniques.⁴ The age specific prevalences reported are strikingly similar to those found in Asians in Southall and at least five times as high as those in Europeans in Southall aged 40-64.¹ More complete diagnostic ascertainment would be unlikely to negate these major differences. Although Asians in Southall comprise mainly Punjabi Sikhs, the economic state of the Southall and Delhi samples is probably comparable. A high prevalence may therefore occur within India as well as in migrants elsewhere if the population is exposed to appropriate environmental factors. Indian people would seem to rank high in terms of ethnic susceptibility to diabetes.

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Prevalence of diabetes by age

Age (years)	Total population	No (%) with diabetes		
		Both sexes	Men	Women
30-34	555	7 (1.3)	5 (1.6)	2 (0.8)
35-39	546	10 (1.8)	5 (1.9)	5 (1.8)
40-44	437	18 (4.1)	9 (3.8)	9 (4.5)
45-49	415	32 (7.7)	25 (10.7)	7 (3.8)
50-54	374	32 (8.6)	20 (9.8)	12 (7.1)
55-59	300	27 (9.0)	16 (10.8)	11 (7.2)
60-64	266	45 (16.9)	30 (20.7)	15 (12.4)
65-69	154	18 (11.7)	10 (11.9)	8 (11.4)
70-74	109	14 (12.8)	10 (14.1)	4 (10.5)
75-79	47	7 (14.9)	5 (16.7)	2 (11.8)
80-84	28	2 (7.1)	2 (10.5)	
85+	22	1 (4.5)	1 (7.1)	

*No cases of diabetes were found in people aged under 30.

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Acute encephalopathy associated with campylobacter enteritis

We report a case of acute encephalopathy accompanied by increased intracranial pressure in a child with campylobacter enteritis. This complication has not to our knowledge been previously reported.

Case report

A previously healthy 6 year old boy was admitted to the paediatric intensive care unit, confused after a history of being unable to speak for two days. Five days before admission he suffered from abdominal pain and diarrhoea followed by a headache and temperature of 39°C. On admission he was afebrile and well nourished. His pulse was 50 beats/minute, sinus rhythm, blood pressure 100/60 mm Hg, and respiratory rate 20 breaths/minute without distress. There were no signs of meningeal irritation. On neurological examination the child looked drowsy but was arousable with bursts of irritability. He could follow only simple orders with oriented motor responses. He cried on painful stimuli but was unable to speak. His cranial nerves were intact, and sensory and cerebellar examination findings were normal. His deep tendon reflexes were hyperactive, and bilateral Babinski's sign was elicited. Funduscopic examination showed no papilloedema.

Complete blood count showed 9000 white blood cells/mm³ with normal differential count, haemoglobin concentration 110 g/l, and 200 000 platelets/mm³. Biochemical values were all within the normal range. Tests on blood gases, coagulation studies, and urine analysis yielded normal results. The cerebrospinal fluid was initially clear with a pressure of 260 mm Hg, but a brisk movement of the child during lumbar puncture caused it to become macroscopically bloody; it contained 70 white blood cells/mm³, of which 80% were monocytic and 20% polymorphonuclear. Glucose concentration was 5.0 mmol/l (90 mg/100 ml); concomitant blood glucose concentration was 6.1 mmol/l (110 mg/100 ml). Results of all tests and cultures for bacteria and viruses were negative, except for repeated stool cultures, which grew *Campylobacter fetus*, confirmed by serotyping as *C fetus* subspecies *jejuni* serotype 1,18.

An electroencephalogram showed no α rhythms but instead high voltage diffuse δ activity 2-3 H admixed with a few θ waves, consistent with acute encephalopathy. Computed tomography of the brain showed oedema with small compressed ventricles.

He was treated with dexamethasone and mannitol but no antibiotics. After three days in hospital his condition had greatly improved and he could utter a few words. After eight days he resumed his normal behaviour and speech, and neurological examination was normal. Repeated computed tomography of the brain on the fifth day after admission showed normal ventricle size and no oedema. The electroencephalogram returned to normal three weeks after his illness began, and stool cultures contained no campylobacters.

Comment

Over the past two decades *Campylobacter* has been recognised as a major cause of diarrhoeal disease in children.^{1,2} Extraintestinal manifestations of campylobacter enteritis, most commonly presenting as bacteraemia without localised infection, occur mainly in early infancy and compromised adults with underlying systemic diseases.³ In a review of 247 cases Schmidt *et al* found that the central nervous system was affected in 12% of cases of extraintestinal disease.³

Besides seizures, a well documented neurological complication of campylobacter enteritis,⁴ effects on the central nervous system include meningitis, meningoencephalitis, stroke, subarachnoid haemorrhage, subdural em-

pyema, and the Guillain-Barré syndrome,³ all of which are associated with invasion of the central nervous system by the bacteria themselves or with localised neurological disease. The pathogenesis of systemic or extraintestinal campylobacteriosis is unclear. Neurological expression has been attributed to a neurotoxin produced by the bacteria, a process similar to that which causes shigella gastroenteritis. This theory is still controversial and has to be proved.⁴

Most extraintestinal neurological manifestations of campylobacter infection are associated with the subspecies *intestinalis*, whereas in our case *C fetus* subspecies *jejuni* was isolated. It has been recently reported that this subspecies can also affect the brain, and our report supports this observation.⁵ Infection with *Campylobacter* should be considered when neurological symptoms such as seizures or increased intracranial pressure complicate gastrointestinal disease.

We express our appreciation to Dr P Lerman for interpretation and follow up of the electroencephalogram, and to the staff of the Campylobacter Reference Laboratory, Israeli Ministry of Health, for confirmation of serotype.

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Lack of antibody to HTLV-I and HIV in patients with multiple sclerosis from France and French West Indies

Recent data have suggested that human retroviruses have an aetiological role in some acute and chronic neurological diseases. Human immunodeficiency virus (HIV; formerly known as lymphadenopathy associated virus or human T cell lymphotropic virus type III) was detected in brain tissue and isolated from cerebrospinal fluid of patients with encephalopathy related to the acquired immune deficiency syndrome.¹ We observed a high prevalence (60%) of antibodies to human T cell lymphotropic virus type I (HTLV-I) in serum from patients with tropical spastic paraparesis, a neuromyelopathy common in tropical areas where HTLV-I is endemic.² These findings were confirmed in both serum and cerebrospinal fluid from patients with tropical spastic paraparesis from Jamaica and Columbia.³ Recently, Koprowski *et al* reported that American and Swedish patients with multiple sclerosis had antibodies that cross reacted with HTLV-I or HIV polypeptides and that cells from the cerebrospinal fluid of four out of eight patients contained sequences related to HTLV-I.⁴ We report the results obtained when we tested serum from 55 patients with multiple sclerosis from two other geographical areas.

Patients, methods, and results

We studied 96 patients with neurological disease from Paris hospitals (48 with multiple sclerosis, 10 with amyotrophic lateral sclerosis, and 38 with other neurological diseases) and seven black patients with multiple sclerosis from Martinique (French West Indies). All serum samples were tested by enzyme linked immunosorbent assay for HTLV-I (Biotech, using disrupted virions as antigen) and for HIV (Elavia-Pasteur). None of the samples was positive for antibodies to HTLV-I (estimated by the ratio in the assay and the baseline value) or to HIV. Furthermore, the mean reactivity levels of HTLV-I in multiple sclerosis, amyotrophic lateral sclerosis, and the other neurological diseases did not differ significantly when tested by one way analysis of variance.