Occasional Review

West Indian diabetic population of a large inner city diabetic clinic

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Abstract

West Indians form a sizable minority of diabetics attending many inner city diabetic clinics. There are 554 diabetics of West Indian origin on our computer files—7% of the total recorded clinic population. Of these 554 patients (56% female, 44% male), 70% have been diagnosed within the past five years; and only 9% have had diabetes for over 10 years; in only five (1%) was diabetes diagnosed before the age of 20. Sixteen per cent were taking insulin, but only 4% of the total West Indian population were truly insulin dependent. Of 65 patients admitted in hyperglycaemic coma or precoma over the past three years, 10 were of West Indian origin; eight of these 10 had hyperosmolar coma compared with only six of the remaining 55.

We conclude that diabetics of West Indian origin attending our clinic show differences in the distribution of age and duration of diabetes from the caucasian population. Most are non-insulin dependent, and the frequency of hyperosmolar coma is higher than that of ketoacidosis. Diabetics of West Indian origin may have a different pattern of disease from the rest of the clinic population.

Introduction

West Indians may form a sizable minority of diabetics attending many inner city diabetic clinics. Despite this, no large-scale studies of diabetes in patients of West Indian origin have been reported in Britain. The many such diabetics attending the King's College Hospital diabetic clinic and the availability of computer records have enabled us to perform a retrospective study of the composition of this population.

Patients and methods

All the essential clinical details of our diabetic patients are recorded on a computer file.¹ The computer was used to provide details of patients of negroid origin, and those of West Indian origin were hand picked from that group. The notes of every patient were examined as well as the computer records, and the following details were recorded: age, duration of diabetes, sex, treatment, and type of diabetes.

When considering the type of diabetes, our criteria used to establish true insulin dependence were that the patient had been treated with insulin from the first few weeks of diagnosis or that he was prone to ketosis.

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All patients who had developed diabetic coma or precoma in the years 1978-80 were further considered from the point of view of clinical and biochemical presentation, outcome of treatment, and long-term management.

FIG 1—Age distribution of diabetics of West Indian origin attending King's College Hospital diabetic clinic. Only a small proportion (0.9%) of the total are under 20 and peak frequency of diabetes occurs in age group 45-54 years.

Results

Of a total recorded clinic population of 8241, 626 patients (7.6%) were of negroid origin. Of these, 554 (7%) were of West Indian origin and were selected for study (243 (44%) male, 311 (56%) female). Figure 1 shows the age of the patients at diagnosis.

In most patients (390) diabetes had been diagnosed within the past five years, and only 54 had had diabetes for over 10 years (fig 2).

Of 554 patients, 209 (38%) were being treated with diet alone, 232 (42%) with diet plus oral hypoglycaemics, and 81 (15%) with insulin. Insufficient details of treatment were available on the remaining 32 (6%).

Of the 81 insulin-treated subjects, 24 (4% of the total clinic population of West Indians) were truly insulin dependent. The rest were taking insulin because of persistent poor control or diabetic symptoms. The mean $\pm$ SEM age at diagnosis of those diabetics who were insulin dependent was 40 ± 2.0 years, duration 13 ± 1.4 years, compared with 47 ± 0.8 and 13 ± 0.7 years respectively for those who were taking insulin but were not truly insulin dependent.

The five children (4 boys, 1 girl) (age range at diagnosis 4-10 years, duration 1-8 years) were truly insulin dependent. Three of these presented in frank ketoacidosis and two with diabetic symptoms, weight loss, and heavy ketonuria. All have pursued a typical insulin-dependent course. Four of these children are at present well over the 50th centile for both height and weight, two of these over the 90th centile.

Full records were available in 65 patients in diabetic coma who had been treated by us over the past three years. Ten patients were West Indian and eight showed a hyperosmolar type picture, compared

FIG 2—Duration of diabetes distribution of patients of West Indian origin attending King's College Hospital diabetic clinic. Most cases are of short duration, 70.4% having been diagnosed within past five years and only 9.7% have a duration greater than 10 years.

with only six of the other 55. Five of the ten cases of West Indian origin were a first presentation of diabetes. The mean $\pm$ SEM age of the eight patients (4 men, 4 women) in hyperosmolar coma was 55 ± 2.5 years (range 47-65 years). All were dehydrated with grossly raised blood glucose concentrations (mean $\pm$ SEM 50 ± 4.0 , range 38-92 mmol/l (685-1658 mg/100 ml)), and blood urea concentrations (mean $\pm$ SEM 14 ± 2 , range 7-19 mmol/l (42-114 mg/100 ml)), but no signs of pronounced acidosis (mean $\pm$ SEM blood bicarbonate concentration, 22 ± 3 , range 15-32 mmol(mEq)/l). All required large quantities of isotonic or half-normal saline to correct dehydration. The remaining two patients had ketoacidosis, and one developed disseminated intravascular coagulation, which was successfully treated with fresh frozen plasma.² Four of the eight patients who developed hyperosmolar coma stopped taking insulin within three months of the episode and have been treated by diet and oral hypoglycaemic treatment ever since. The patient who developed disseminated intravascular coagulation is well controlled on diet alone.

Discussion

In those studies described from the West Indies³⁻⁵ the many combinations of methods of detection—different diagnostic criteria, laboratory methods, and presentation of data—make the value of comparing prevalence rates with other populations doubtful. It does appear, however, that among adult West Indians of negroid origin the prevalence of glycosuria is between 1 and 2%.⁶

Interest in the features of diabetes in Jamaica was stimulated by the description of an insulin-resistant, non-ketotic type of diabetes occurring in young, thin Jamaicans—J type or Jamaican-diabetes.³ Follow-up of these cases, however, showed that many became ketosis-prone.⁴ Moreover, some of these patients were later shown not to have been truly insulin dependent and were in fact typical non-insulin-dependent diabetics.

There has been no survey of diabetics of West Indian origin living in Britain, although differences in presentation of hyperglycaemic coma have been reported.⁷ Our study was retrospective and can give no information about the prevalence of diabetes in the people of West Indian origin living in Britain. These subjects do, however, make up about 7% of our total clinic population and a larger percentage still of new referrals (19% of all new diabetic patients over the past two years). The higher frequency of women to men in this study confirms previous

work,⁴ and the peak frequency of diabetes occurs in the age group 45-54 years, which is below that reported from Jamaica with a peak of 55-64 years.⁶ This discrepancy may be explained by immigration biasing the age ranges to a lower level in Britain.

Most of our patients of West Indian origin (70%) have been diagnosed only within the past five years. Short of suggesting that there is an epidemic of diabetes in the West Indian community in Britain the most likely explanation lies again in the nature of the population we are dealing with—the population is only now reaching the age where the peak incidence of diabetes occurs. Another possible explanation may be the effect of the Western-style diet. It has been suggested that a threshold value for consumption of sugar of more than 30 kg a year must be exceeded before a high prevalence of diabetes is found in a population, and that on emigration to Britain this value is exceeded by the West Indian population, which previously consumed only an average of 16.8 kg sugar a year.⁴ This suggestion that diet may have an important effect on the prevalence of diabetes has, however, been challenged.⁸

Only 4% of the total clinic population of diabetics of West Indian origin were truly insulin dependent. This is much less than an expected frequency of about 20%⁹ in the caucasian population. This might support the J-type theory already mentioned, but the fact that all the West Indian children in our clinic are truly insulin dependent points to immigration as an explanation of the discrepancy. Perhaps in the future we will see a more even distribution of insulin-dependent and non-insulin dependent-diabetes among the people of West Indian origin living in Britain.

The high frequency of hyperosmolar coma when compared with ketoacidosis raises the possibility that this population has a higher genetic predisposition for non-insulin-dependent diabetes, which may be triggered by Western diet or some other environmental factor. Even if the hyperosmolar character of the hyperglycaemic coma is explained on the basis that 96% of the West Indians under study are non-insulin dependent this still does not explain why 554 West Indians contributed eight cases of hyperosmolar coma over three years compared with only five occurring in the rest of the clinic population.

In conclusion, patients of West Indian origin form a sizable minority of our diabetic clinic. They show differences in age and duration of diabetes distribution from the indigenous diabetic population, most having only recently been diagnosed. The great majority are non-insulin dependent, but the fact that all the children are insulin dependent suggests that some of this bias may be accounted for by immigration. This does not, however, explain the high frequency of hyperosmolar coma as distinct from ketoacidosis in this group when compared with the caucasian population. Possibly diabetics of West Indian origin have a different pattern of disease from the rest of the clinic population.

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