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Original article

Influence of migration on characteristics of type 2 diabetes in sub-Saharan Africans

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Abstract

Aim. – This study compared the clinical and biochemical characteristics and microvascular complications found in three groups of type 2 diabetes (T2D) patients: Africans living in Africa; African immigrants living in France; and Caucasians living in France.

Methods. – Diagnosed T2D Africans living in Cameroon ($n = 100$) were compared with 98 African migrants diagnosed with T2D after having moved to France, and a group of 199 T2D Caucasian patients living in France. All underwent clinical and biochemical evaluations, and all were assessed for microvascular complications.

Results. – The median duration of stay of the migrants in France was 15 years before being diagnosed with diabetes. Despite similar durations of diagnosis, they were 8.9 years younger at the time of diagnosis than Africans living in Cameroon ($P < 0.001$). Caucasians and African immigrants in France had lower HbA_{1c} values than Africans in Cameroon ($P < 0.001$); they were also more aggressively treated for hypertension and dyslipidaemia and, therefore, had significantly lower blood pressure levels and better lipid profiles. Diabetic nephropathy and retinopathy rates were higher in Cameroon than in the two other groups. After adjusting for age, diabetes duration, HbA_{1c}, hypertension and other covariates, only the prevalence of diabetic nephropathy (OR: 5.61, 95% CI: 2.32–13.53; $P < 0.0001$) was higher in Cameroon compared with those living in France.

Conclusion. – Our results suggest that Africans who emigrate to France may develop diabetes earlier than those staying in their home country. However, the latter may be a reflection of late diagnosis of diabetes. Also, the less adequate diabetes and hypertension control in the latter would explain their higher rates of nephropathy. Large-scale cohorts are now warranted to substantiate these observations.

Keywords: Type 2 diabetes; Migration; Complications; Africans; Caucasians

1. Introduction

Populations of sub-Saharan African origin are considered at high risk of developing diabetes [1]. The incidence of type 2 diabetes (T2D) reported in the Atherosclerosis Risk in Communities (ARIC) Study carried out in the US was 1.5- to 2.4-fold greater in African Americans compared with their white American counterparts [2]. In the third National Health and Nutrition Examination Survey (NHANES III), it was also reported that black Americans were diagnosed with T2D at a significantly younger age compared with whites living in the same environment [3]. In Africa, the prevalence of T2D varies widely depending on the degree of urbanization of the geographical location and time spent in that particular environment [4,5]. Accordingly, many reports have found that the prevalence of T2D shows a rising gradient from Africans living in Africa to

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Afro-Caribbeans, African Americans and African immigrants living in other developed countries. Mbanya et al. [4] reported T2D prevalence of 0.8% and 2% in rural and urban Cameroon, respectively, of 8.5% in Jamaica and of 14.6% among blacks living in Manchester, UK. Likewise, Cooper et al. [5] reported prevalence of 2% in Nigeria, 9% in the Caribbean, and 11% among African Americans in the US and African immigrants in the UK. This disease pattern has been attributed to the effects of environmental factors, especially lifestyle changes. Indeed, epidemiological transition, urbanization and emigration to a more developed geographical environment can often lead to rapid changes in lifestyle that together with genetic background, increase the propensity to develop non-communicable diseases [6,7].

The link between lifestyle changes and the development of T2D is alterations in insulin sensitivity and secretion, the two underlying mechanisms of the disease, as shown by metabolic studies. Osei et al. [8] demonstrated differences in insulin secretion and sensitivity between African Americans and Africans residing in Africa, with the former having greater insulin secretion, but also more insulin resistance. However, no difference was found on comparing African Americans with African immigrants who had settled as adults in the US one to 18 years prior to the study, suggesting that the metabolic changes leading to T2D may be arising soon after their emigration [9].

In the absence of immigrant cohorts, our present study aimed to assess the impact of migration on the characteristics of T2D by comparing three diabetic populations: Africans living in sub-Saharan Africa; Africans who emigrated to France before being diagnosed with the disease; and a Caucasian population living in France serving as a reference population.

2. Patients and methods

2.1. Patients

This cross-sectional study included and compared three groups of people with T2D. The first was from a population of around 250 T2D patients of West and Central African origin attending the department of diabetes and endocrinology at the Saint-Louis Hospital in Paris (a referral centre for Africans with T2D in Paris and its suburbs) [10–13]: those who had emigrated from Africa to France as adults (age $\geq$ 18 years) and been diagnosed with T2D within at least 1 year of their arrival were consecutively selected over a 1-year period (from November 2005 to October 2006). This yielded a sample population of 98 patients, all of whom agreed to participate in our study. The second group comprised Cameroonian adults who were consecutively enrolled at the outpatients department of the Yaoundé Central Hospital in the capital city of Cameroon during the same time period. Details of the study setting have been described elsewhere [14]. The third group included 199 Caucasian patients consecutively enrolled during the same period from the outpatients department of diabetes and endocrinology at the Saint-Louis Hospital, all of whom were receiving chronic diabetes care at the department.

All patients gave their informed consent to participate in the study, and the procedures used at both sites (Yaoundé and Paris) were standardized to allow comparison. The diagnosis of diabetes was based on 1998 World Health Organization (WHO) criteria [15] and included non-ketotic diabetes, which does not require insulin within the first 2 years of diagnosis.

2.2. Clinical examination

Age, gender, history of hypertension, smoking and year of diabetes diagnosis were recorded for each patient using a structured questionnaire. Also, anthropometric parameters were measured using validated methods, and included weight to the nearest 0.5 kg using a scale, height to the nearest 0.5 cm using a wall-mounted stadiometer and body mass index (BMI) calculated as weight divided by the square of height (kg/m^2). Overweight was defined as a BMI score $\geq$ 25 kg/m^2 and < 30 kg/m^2, and obesity as a BMI $\geq$ 30 kg/m^2. Blood pressure (BP) was measured on the right arm as per the British Hypertension Society guidelines [16], using an automatic BP monitor (HEM-711, Omron Healthcare, Inc., Lake Forest, IL, USA). The average of two readings (first and second) taken 5 min apart was calculated. Hypertension was defined as a self-reported history of hypertension diagnosis and/or the use of antihypertensive medications, or an average systolic BP $\geq$ 140 mmHg and/or diastolic BP $\geq$ 90 mmHg. The number and class of antihypertensive drugs used were also recorded.

2.3. Assessment of microvascular complications

The fundus of the eye was examined by retinal photography, and fluorescein angiography when necessary, performed by a specialized ophthalmologist. The same ophthalmologist, who was experienced in diabetic retinopathy, was also in charge of reading the results from each study centre. Examination of sensory function of the lower limbs, including Semmes–Weinstein monofilament (10 g) and tendon reflex testing, was performed to assess the presence of peripheral neuropathy. Diabetic neuropathy was defined as the presence of any sensory symptoms or signs, or abnormal monofilament perception with or without abnormal tendon reflexes. All patients underwent urinary albumin excretion (UAE) and serum creatinine measurements, and diabetic nephropathy was defined as UAE $\geq$ 20 mg/L or $\geq$ 30 mg/24 h as confirmed by a second sample, with or without an increase in serum creatinine levels. Patients with kidney disease without abnormal albumin excretion or known to be due to a cause other than diabetes were classified as having non-diabetic kidney disease (NDKD). The participants were further divided into two categories – one with and the other without complications – with no further subclassifications. Because it was not possible to thoroughly assess the presence or absence of macrovascular complications in all patients in Cameroon (ankle–brachial index, stress electrocardiography testing, cardiac scintigraphy), these tests were excluded from the scope of our study.