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EMERGING PICTURE OF MULTIPLE SCLEROSIS IN KENYA
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ABSTRACT

Objective: **To report on the occurrence of clinical multiple sclerosis among indigenous Bantu African Kenyans who have never been out of the country.**
Design: **A retrospective study.**
Setting: **A private neurology and clinical electrophysiology clinic.**
Subjects: **All the patients referred to the clinic by neurologists and other specialists for electrophysiological tests with diverse neurological complaints. The patients examined and diagnosed as having multiple sclerosis on clinical grounds and established criteria are reported.**
Results: **Out of 2831 patients referred for electrophysiological tests over a ten year period, nine patients were diagnosed as having definite multiple sclerosis on clinical grounds. Four of these had supporting laboratory findings (MRI scans, CSF studies and visual evoked responses). The presenting symptoms were predominantly visual disturbances and somatic sensorimotor disturbances which were seen in all the patients. Cerebellar dysfunction was observed less frequently, in less than half of the patients while sphincter disturbances were conspicuously rare. The sex distribution was overwhelmingly in favour of the female at a ratio of 7:2. Apart from two patients of Indo-asian ethnicity, all the others were indigenous Bantu Africans who had never travelled outside their country before the onset of the illness.**
Conclusions: **Multiple sclerosis occurs amongst Bantu Africans and may not be as rare as previously suggested and its prevalence is certainly on the increase. The development of higher incidence rates in communities where the illness was previously unknown may present opportune settings for the study of aetiological factors of this illness as it emerges. There is a need therefore for proper epidemiological studies to evaluate these factors, especially environmental factors, as the new disease continues to appear.**

INTRODUCTION

Multiple sclerosis (MS) is a chronic neurological disorder characterised by patches of inflammation and demyelination in the brain and spinal cord. These lesions are multiple and spread out both in space and time. The clinical manifestations present as variable neurological deficits of the central nervous system (CNS) that cannot be explained by a single anatomical lesion which appear and then fully or partially resolve only to re-appear later. These CNS lesions (sclerotic plaques) occur unpredictably in the central white matter but the cervical cord, optic nerve, brain stem and peri-ventricular regions are affected more often. The disease typically has a variable clinical course with partial or complete remissions and exacerbations occurring after hours, days or weeks. Fresh lesions appear even as old ones resolve, while a steadily mounting functional deficit occurs over the years.

The specific aetiology or trigger of MS remains unclear but it is suspected to be multi-factorial with a poorly understood interplay of genetic and environmental factors. A look at the global distribution of MS reveals that there exists in more or less clearly defined geographical areas of high, moderate and low risk(1). This high risk areas with a prevalence of 30 to 80 per 100,000 population are generally associated high latitude(2), but as has been pointed out, latitude and the environment alone do not adequately explain the discrepant distribution(3). For example, at latitude 40° to 45° north, multiple sclerosis prevalence is high in America, moderate in Europe (5 to 30 per 100,000 population) and low in Asia (Japan with < 5 per 100,000 population). This suggests that other environmental and genetic factors that may or may not be related to latitude are involved.

Although there is no specific aetiological environmental factor that has been identified, the reported clustering into small epidemics(4) and migration studies offer circumstantial evidence that such an agent or agents exists. It has been proposed that the agent may be a virus or viruses(5). It is probable that a slow virus that infects exposed and susceptible persons at a vulnerable unprotected period in their life (thought to be the peripubertal age) results in the development

of MS later in life. It is also suggested that the virus may lie quiescent in the dorsal root ganglia of spinal and cranial nerves while awaiting activation by other viruses, or other events in the person (including endocrine changes at puberty) such as happens with the Herpes zoster virus(6). In addition, aberrant and dysregulated immunity most certainly has a significant role in the pathogenesis. It is not clear whether the immune system malfunction is a cause or a consequence of the disease.

It has further been postulated that the timing of the immune stimulation by the presumed infective agent might be crucial to the development of MS(7). While childhood infections occurring later in life (during adolescence for example) may render the immune system more vulnerable, the same infections occurring during infancy and early childhood (like they are likely to do in the developing countries with low multiple sclerosis prevalence) appear to be protective (5).

Numerous studies have attempted to link MS to many of the common environmental factors such as the high consumption of dairy products and animal fats(8) a high calorie consumption(9), the lack of iodine and selenium in the diet(10) and exposure to solar radiation.

There is a case for genetic factors being major contributors in the aetiology of MS. It has been observed that multiple sclerosis is a disease of people of Western European stock and that the disease seems to follow their migration patterns. Thus, people living in regions near the equator and who have no genetic connections with the Western Europeans have very low prevalence rates of MS(1). Indeed, it has been observed that MS does not occur among the Bantus in Africa. Recently however, MS has been regularly diagnosed amongst indigenous Kenyan Africans who have never been outside their country with some regularity (4,11).

Table 1

Clinical diagnostic criteria for multiple sclerosis (Poser)

Criteria for diagnosis	Diagnostic categories
History of two attacks with positive oligoclonal bands or increased IgG in CSF; no clinical or para clinical evidence of a disease	Probable MS with laboratory support
History of two attacks without laboratory abnormalities	Clinically probable MS
History of two attacks with clinical and para clinical evidence of one lesion; Oligoclonal bands or increased IgG present in CSF	Laboratory-supported definite MS
History of at least two attacks; Clinical evidence of at least one lesion and clinical or para clinical evidence of another lesion	Clinically-definite MS

Clinical presentation of MS is varied. In the majority of cases, the onset is insidious with vague symptoms of fatigue, excess tiredness, and mood changes antedating definite objective neurological deficit. More definite neurological symptoms consisting of double or blurred vision, numbness, weakness in one or two extremities, instability in walking, tremors and problems with bladder control, heat intolerance may then appear representing lesions in the visual, somatic sensory, motor and cerebellar pathways. The time course may run over a few hours, days or weeks during which the deficits may worsen or improve while new lesions appear. To make a diagnosis of MS one needs to identify evidence of more than one lesion anatomically and/or in time as shown in Table 1 delineating the criteria by Poser(12).

MATERIALS AND METHODS

Over a period of ten years, between April 1989 and March 1999, an average of 250 to 300 per year patients are seen in the electrophysiology clinic. Out of the 2831 patients seen in the ten year period, nine cases of clinically definite MS according to the above criteria are presented. The patients were identified retrospectively by perusing through the records of our clinic.

All the patients had been seen by the author and were diagnosed exclusively on clinical findings. However, patients 2,3,5 and 7 had additional collaborating Magnetic Resonance Imaging (MRI) scans. Visual evoked responses (VER) and somatosensory evoked potentials had also been done in patient 2. Patients who had a monophasic and simultaneous involvement of the optic tracts and the spinal cord (neuromyelitis optica) or transverse myelitis were excluded.

RESULTS

The ethnicity of the nine patients is shown in Table 2. Apart from two patients, both of Indian descent, all the other patients were black indigenous Africans of Bantu stock. The majority were Kikuyus (five out of seven) who were residing in and around Nairobi. The occupations of the patients were also quite diverse with students and teachers being the majority (five out of seven). They all presented at early adulthood and their ages ranged from 17 years to 39 years. Estimated mean age of onset was 23.6 years. They had had the illness on an average of 4.1 (range: 1 - 7 years) years when seen in our clinic. A preponderance towards female affliction was also observed (female: male ratio = 7:2). None of the black Africans had travelled outside their country prior to the onset of their illnesses. However, five of them did travel to the US, India and Britain later for purposes of diagnosis - specifically for MRI scanning. The pattern of presentation was similar in all nine patients with sensory symptoms predominating at the onset of the disease. Eventually however, all the other symptoms and signs developed during the course of the illness as shown in Table 3. Sphincter disturbances (retention) were present in only one patient whose disease had a relatively rapid course. Cerebrospinal fluid protein electrophoresis was done in one of the patients (patient 2). The same patient was the only one who had VER and somatosensory evoked potentials (SEP) and they were abnormal showing a delay in the P_{100} on the right visual pathway and a delayed N_{14} on the median SEP.

Table 2

Demography of patients diagnosed with MS

Patients	Residence	Age	Sex	Ethnicity	Occupation	Travel	Duration	MRI	DS	CS
1. JN	Kiambu	28 (21)	F	Kikuyu	H/wife	x	7 yrs	x	3.5	10
2. FM	Meru	27 (22)	F	Meru	Accountant	√	5yrs	√	1.0	0.0
3. JM	Nairobi	28 (26)	F	Kikuyu	Nurse	√	2 yrs	√	1.0	6.0
4. LN	Machakos	29 (24)	F	Kamba	Agr-officer	√	Syrs	x	3.0	3.0
5. RM	Mombasa	39 (35)	F	Asian	Teacher	√	4yrs	√	3.0	4.0
6. SM	Molo	18 (17)	M	Kikuyu	Student	x	1 yrs	x	1.0	0.0
7. AK	Nairobi	24 (20)	M	Asian	Student	√	4yrs	√	5.5	?
8. MM	Nyahururu	35 (31)	F	Kikuyu	Teacher	x	4yrs	x	4.5	3.0
9. AK	Murang'a	22 (17)	F	Kikuyu	Student	x	5yrs	x	4.5	10

Key: DS = EDS rating; CS = Current EDS Status (1999); Travel = travel outside the country; Agr. = Agriculture

Table 3

Pattern of clinical presentation of Kenyan MS

Symptoms	Patients
Visual disturbances	9
Blurring of vision	9
Diplopia	9
Optic neuritis	9
Paraesthesia of limbs	7
Sensory loss (tactile, vibration)	9
Dysaethesia (painful)	2
Posterior column disturbances	9
Weakness (upper motor neuron type)	9
Deafferentation	3
Cerebellar dysfunction (ataxia, tremors, dysarthria)	4
Sphincter disturbances	1
Higher cerebral dysfunction (emotional disturbances, fatigue)	2
Cranial nerve dysfunction (hearing loss, facial weakness)	2

Table 4

Clinical types of MS

Category	Clinical type of MS	Expected age %	No. of patients	Age %
I	Benign MS	10	(3)	33
II	Relapsing-remitting MS	40	(3)	33
III	Secondary chronic progressive (initially category II)	40	(3)	33
IV	Primary progressive MS	10	(0)	0

The degree of disability when the patients first presented is shown in Table 2. All of them except patient number 7 were initially independent and the majority of them have remained fully functional in their individual pursuits. One patient however has had her responsibilities scaled down and she currently requires support to walk. One of them has since died and another lost to follow up. The clinical MS types of the nine patients were almost equally distributed amongst the benign, relapsing and secondary progressive varieties Table 4. There were no patients with primary progressive type.

DISCUSSION

Although multiple sclerosis has always been considered a disease of white races, experiences in different non-white communities have shown a steady increase in its prevalence. This appears to be the case in Kenya although there are no proper epidemiological studies. This increase in apparent prevalence may well reflect the improvement in health services, which in turn increases the probability of diagnosis of MS. It may also represent an actual increase in the number of cases. Environmental alterations in Kenya that have followed the general tendency towards a more western European way of life may also contribute to this change in prevalence if confirmed to be real. The majority of our MS patients were Kikuyus. This may simply be a reflection of the distribution of clientele patronising the clinic together with the close geographical proximity of the clinic to Kikuyu land but it could also easily be related to the degree of adoption of the western way of life. Similarly, the bigger representation of school based occupations may have a similar explanation. This can only be tested by well-designed epidemiological surveys or prospective studies since retrospective studies will be grossly erroneous. That notwithstanding however, MS can no longer be said not to occur amongst the black Bantu people as it has been reported in many such communities(4,11). The observed tendency of MS to occur more frequently in females seen among the Kenyan patients is similar to the patterns seen in other countries. Where epidemiological studies have been done, the mean age of onset of MS is early thirties and the sexes are represented at a ratio of 2:1 in favour of females in all forms of MS. So far, there is nothing to suggest that the clinical presentation of MS in Kenya is any different. Indeed, it has earlier been observed that the clinical presentation is similar to that observed in western countries(4). However, the small numbers so far seen suggest that the condition may be occurring earlier in Kenya.

In Kenya proper epidemiological studies are currently unavailable. Undoubtedly, many cases must go unnoticed, probably carrying wrong diagnostic labels and therefore the picture seen in retrospective studies cannot be accurate.

Thus the emerging picture of prevalence of MS in Kenya arising from the publications must be a gross underestimate.

None of the patients had a family history of a similar illness among either the immediate family members or first and second-degree relatives. Although this last aspect of family history was not exhaustively evaluated to include earlier generations, the cases presented would appear to have been sporadic. The finding that the prevalence rate of MS among Africans and Asians increases when they emigrate to high risk areas(13), tends to support the existence of a causal environmental factor, which presumably the high risk people take with them whenever they migrate to low risk areas(14).

The numbers among Africans are too small to allow an adequate estimation of the genetic risk amongst relatives of MS patients, but where such studies have been done amongst the white races, the risk is put between two per cent and five per cent for first degree relatives. This is 10 to 25 times higher than in the general population(15). It would be of interest to see whether a genetic risk exists in the indigenous non-white African population. It has been noted amongst Indian MS patients that the commonest initial clinical presentation was optic neuritis(16). This is observed less often amongst European and Arab patients, whose commonest symptoms at initial presentation, are attributable to disorders of the pyramidal tracts(17). These two apparently different modes of presentation have been dubbed the Asian and European types of MS respectively(18). As is to be expected, as the number of systems involved in MS increases the longer the duration of the illness. Our patients, the majority of whom had illnesses for longer than four years, presented with widespread system involvement. The commonest symptoms however were still visual, brainstem and sensory which occurred in all of them. Sphincter disturbances and disturbances of higher cortical functions were distinctly uncommon.

The majority of our patients were all independent when initially seen and some (two) have entered a prolonged remission with no noticeable disability but the majority have either remained the same or have moderately deteriorated while two have died. The two deaths have both occurred in patients in their twenties which would suggest a more aggressive course in these two if their deaths were shown to be MS related. In western countries, the majority of MS patients belong to the relapsing and secondary progressive types. However, our patients were evenly distributed among the benign, relapsing and secondary progressive types. There was no patient with the primary progressive type of MS. This observation would most likely change as larger numbers of MS are observed in Kenya. However, it has been observed that the Asian type of MS runs a milder course blending into the monophasic de Vic's disease or neuromyelitis optica and our observations may suggest this inclination.

There are numerous unanswered questions concerning MS that findings from the emerging disease in Africans may help evaluate. This would include the role of genetic versus the environmental factors, the role played by specific factors in diet, childhood illnesses, vaccination, under-nutrition, over-nutrition, avitaminosis and many others. The identity of the environmental factor that the white people carry with them when they migrate would probably be found amongst those communities with more exposure to the western culture but not amongst those who have retained their traditional cultures. Both types of communities are to be found living side by side in Kenya. The possibility of MS being a heterogeneous disease or even several disease types with a common clinical presentation, will also need to be examined as the disease evolves in the black Africans.

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