Conclusion

Despite its European origin, the DEeD was found to be well accepted in this pilot study and to be a sensitive instrument to diagnose mild and moderate dementia. Further investigations among community-based populations, with minor modifications, appear justified.

REFERENCES


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Lyme disease in South Africa

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Objective. This article presents an overview of Lyme disease (LD) as it applies to neuropsychiatry and summarises research results on the epidemiology of LD in South Africa.

Method. The study is based on a review of research papers from various medical disciplines that focused on the epidemiology of LD in South Africa.

Results. Assessment of the incidence of LD in South Africa is based on a few anecdotal studies. The results of the studies are dominated by experimental weaknesses.

Conclusions. The sporadic nature of LD incidence in South Africa may either reflect a restriction of research efforts or be a true indication of the epidemiology of the disease. This review lends support to the former hypothesis. The low reported incidence of LD in South Africa is probably due to a lack of awareness and research effort.


Lyme disease (LD) is an acute and chronic inflammatory syndrome transmitted by infected female ixodes ticks. The disease has been known since the early 1900s. The aetiological agent of LD was discovered by Burgdorfer in 1981.

Burgdorfer studied ticks on Long Island, New York, and noticed spirochetes in the mid-gut smears of ixodes dammini ticks. He conducted antibody tests of the spirochetes using sera of several LD patients and found that this spirochete was the cause of LD. The spirochete was subsequently named Borrelia burgdorferi. In 1993 the first international symposium on LD was held.

Clinical manifestations of LD

LD has dermatological, arthritic, ophthalmological, cardiac, neurological and psychiatric manifestations. The disease can be divided into three clinical stages. Not all stages need to be present and some stages may overlap. Initially (stage 1), an influenza-like illness associated with an erythematous, expanding rash may appear days to weeks after the infecting bite.

Weeks to months later (stage 2), neurological or cardiac symptoms may develop. Months to years later (stage 3), arthritis frequently appears and sometimes neurological and dermatological manifestations are also found. Other accompanying symptoms of later infection include profound fatigue, chronic muscle weakness, sleep disturbance, chronic encephalitis, photophobia, auditory hyperacusis, extreme irritability or emotional lability, word-finding problems, dyslexic errors when speaking or writing and spatial disorientation.

Psychiatric manifestations

Psychiatric symptoms may be part of the clinical profile of LD. Case reports have linked LD to neuropsychiatric symptoms such as paranoia, thought disorder, delusions, auditory hallucinations, olfactory hallucinations, visual hallucinations, anorexia nervosa, obsessions or compulsions, major depression, chronic fatigue, forgetfulness, disorientation, confusion, violent outbursts, mood lability, panic attacks, mania, personality changes, catatonia and dementia. However, of all of the abovementioned symptoms or conditions, depression is the most frequently reported by LD patients.

Neuropsychological findings

Chronic neurological involvement usually occurs late in the illness. Neuropsychological testing has shown that LD...
patients often suffer from deficits of: (i) verbal fluency; (ii) executive cognitive functions; (iii) concentration; (iv) attention; (v) problem-solving and conceptual ability; (vi) immediate and delayed auditory-verbal and visual memory; (vii) the ability to learn and retrieve new information; and (viii) psychomotor and perceptual motor function. In contrast, orientation and over-learned intellectual abilities are not found to be significantly impaired. Nervous system involvement in LD is often reversible with appropriate antibiotic treatment. This underscores the necessity of recognition and treatment.

Atypically, neurological manifestations of LD include blindness, dementias, Bell's palsy, meningitis, seizure disorders, Guillain-Barre syndrome and progressive demyelinating syndromes mimicking multiple sclerosis.

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Atypically, neurological manifestations of LD include blindness, dementias, Bell's palsy, meningitis, seizure disorders, Guillain-Barre syndrome and progressive demyelinating syndromes mimicking multiple sclerosis.

Investigations which may help to identify neurological involvement in LD include serology, cranial magnetic resonance imaging, neuropsychological testing, electroencephalography, computed tomography and brain biopsy.

**Diagnosis**

The following criteria are taken into account when diagnosing LD: (i) a history of erythema migrans rash or *Ixodes* tickbite; and/or (ii) exposure to an LD-endemic area; and/or (iii) a combination of psychiatric and extraneural symptoms.

However, the diagnosis of LD is problematic as the criteria for diagnosis may pose a number of different problems for clinicians:

1. Rash/tickbite. Because the tickbite is usually painless and may go unnoticed, more than 50% of individuals with LD do not remember a bite.

2. Exposure to endemic areas. In areas where LD is endemic, the diagnosis is often made on clinical and epidemiological grounds. Cases outside the known geographical distribution may require laboratory investigations; this poses additional problems of reliability. Also, the lack of epidemiological studies in some countries restricts the use of this criterion in making a diagnosis.

The serological tests most often used to diagnose *B. burgdorferi* infection are enzyme-linked immunosorbent assay (ELISA), indirect immunofluorescence assay (IFA) and Western blot. The reliability of the serological findings is often dependent on disease duration and clinical manifestation.

The complexity introduced by diagnostic tests is exacerbated by the diverse manifestations of the disease. The diversity of clinical manifestations of LD may be mimicked by many other illnesses. Early LD may suggest other multisystem diseases such as serum sickness, rheumatic fever or hepatitis B infection. Neurological manifestations of LD may resemble meningitis, Bell's palsy and multiple sclerosis.
Geographical spread of LD

LD is increasing in incidence and geographical spread. Current serological and clinical evidence indicates that LD has a worldwide distribution in humans, domestic and wild mammals and birds. The disease is endemic in northern Europe and the USA. Worldwide reporting also includes cases from the Soviet Union, Australia, eastern Europe, China, Japan and other Scandinavian nations.

LD in South Africa

The incidence of LD in South Africa is unknown. The country has a wide variety of tick species. These include five tick genera in which spirochaetes have been found elsewhere in the world. Tick-borne diseases affecting humans and animals are common in South Africa. Although ticks of the Ixodes family are uncommon in southern Africa, B. burgdorferi is reported also to have been isolated from mosquitoes (which the country has in abundance).

The South African literature on LD to date consists of a few anecdotal cases and two postgraduate studies.

1982/3. In 1982 an outbreak of arthritis in horses at a riding school in KwaZulu-Natal was reported. Clinical and serological data provide speculative evidence that this disease might have been LD.

1984. A 25-year-old woman tourist camped on the KwaZulu-Natal south coast between June and August 1984. She sustained numerous tick bites on her trunk and forearm. When she returned to Germany she was investigated for fatigue and malaise. High titres of antibodies against B. burgdorferi were found, but the possibility that the infection had been acquired outside South Africa could not definitely be excluded.

1986. All domestic animals at the riding school in KwaZulu-Natal (mentioned above) were tested for antibodies against B. burgdorferi in 1988. Seventy-one of 117 horses (61%) and 6 of 11 dogs (55%) tested positive. These data provide suggestive evidence that LD may exist in South Africa.

October 1988. The owner of the riding school in KwaZulu-Natal developed severe headache following a tickbite. She was diagnosed with tickbite fever, but failed to respond to treatment with tetracycline. Her condition deteriorated over the following few weeks. She developed severe arthralgia, depression and slurred speech. All serological tests conducted by the Department of Medical Microbiology, University of the Orange Free State, were negative except for a positive IFA test for B. burgdorferi. The result was later confirmed by an ELISA at the Hygiene-Institute der Universität (Vienna).

1989. Evidence of infection in several groups of South Africans was investigated. Patients who were identified on clinical grounds as possible LD cases were included in the study. Four hundred and twenty-
seven individuals in the Gauteng area were tested for antibodies. IFA was the serological technique used. Only 1 patient met the criteria for the diagnosis of LD. However, it is clear from the literature study that negative serological tests may not exclude the presence of the condition.

1992. Norton1 did a study to investigate the capacity of some of our local tick genera to transmit B. burgdorferi, and to find serological evidence implicating local vertebrates as possible hosts. Four genera of tick that commonly occur in South Africa were tested for their capacity to acquire and transmit B. burgdorferi. Tick transmission experiments were carried out using rabbits as the host of infection. Only one rabbit was successfully infected. All other animals failed to become infected with B. burgdorferi.

To investigate local vertebrates as possible hosts, murine and equine studies were done. The data showed tentatively that B. burgdorferi occurs in South Africa. Norton concluded that further investigations are required to confirm the findings of the study.

Conclusion

Because of the increase of LD in incidence and geographical spread, it seems logical that the risk of contracting and hosting the spirochaetes in South Africa will increase. However, the literature survey indicates that only a few cases have been identified to date. The sporadic nature of LD cases may reflect restriction of research efforts or may be a true indication of the epidemiology of the disease in South Africa.

There are a number of significant limitations in the studies discussed in this article. A major difficulty in diagnosing LD is that the serological tests available for diagnosis have, to date, been deficient. Serodiagnosis in early LD is reported to have low sensitivity and often produces false-positive results.11 For this reason, the research data may not be an accurate measure of LD in South Africa. A further problem is that the studies focused on restricted areas. Therefore, for much of the area outside the Witwatersrand and Natal there is no data available. Also, in those areas where LD is likely to be present there has been no monitoring or long-term investigation. Finally, clinicians in South Africa need to know more about LD and to consider it more frequently in differential diagnosis.

This review lends support to the hypothesis that the low incidence of LD in South Africa is probably due to a lack of awareness and research effort. Large community studies should be undertaken before any firm conclusions may be drawn about the prevalence of LD.

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