Epilepsy caused by Parasitic Parasites in an Onchocerciasis Endemic Area

Henriette Kirstine Christensen*

Editorial Office, Neurology and Neurorehabilitation, Germany

Corresponding Author*

Henriette Kirstine Christensen
Editorial office
Neurology and Neurorehabilitation
Germany
E-mail: nneurorehabilitation@gmail.com

Received date: 12 January 2022; Manuscript No. NNR-22-52031; **Editor assigned:** 17-January-2022; Pre QC No. NNR-22-52031(PQ); **Reviewed:** 21-January-2022; QC No. NNR-22-52031(Q); **Revised Date:** 27-January-2022; Manuscript No. NNR-22-52031(R); Accepted: 28-January-2022; Manuscript No. NNR-22-52031; **Published:** 29-January-2022; DOI: 10.37532/nnr.22.4.1.9-10

Abstract

In Africa, where parasitological infections are common, epilepsy is prevalent. A door-to-door study in 2016 found an epilepsy prevalence of 4.6 percent in an Onchocerciasis endemic area in the Logo health zone, Ituri province, the Democratic Republic of the Congo, and 50.6 percent of epileptics were infected with Onchocerca volvulus. In the current study, the serum of 195 people infected with O. volvulus and suffering from epilepsy was tested to determine the proportion of co-infections with Taenia solium, Toxocara Canis and Strongyloides. Neuro-inflammation may be linked to Onchocerciasis-Associated Epilepsy (OAE), although little immunological research in children with this type of epilepsy has been conducted to date. In a preliminary investigation, we examined cytokine levels in the Cerebrospinal Fluid (CSF) of people with OAE from Maridi, South Sudan, and Mosango, DRC, and compared them to cytokine levels in the CSF of Africans with non-OAE neurological illnesses and Europeans with epilepsy or other neurological conditions. In an ideal scenario, cytokine levels in serum and CSF have taken at the time of the first seizure should be measured in prospective research.

Keywords: Neuro-inflammation • Epilepsy • African trypanosomiasis • Neuro-cysticercosis

Introduction

Epilepsy is more common in low- and middle-income nations where parasitological diseases are common. Plasmodium falciparum, which causes cerebral malaria, and Taenia solium, which causes neurocysticercosis, are the two parasites that are well-known causes of epilepsy in Sub-Saharan Africa (NCC). Other parasites, such as schistosomiasis, human African trypanosomiasis, and toxocariasis, are linked to several neurological disorders, including seizures. Epidemiological studies have linked the Onchocerca volvulus parasite to epilepsy, although the etiology has yet to be determined. In children, the skin snip microfilaria (mf) load appears to be the most important driver of the chance of developing epilepsy. Mf, on the other hand, are rarely found in the Cerebrospinal Fluid (CSF) and have never been confirmed to cross the Blood Brain Barrier (BBB). mf were found in the CSF of some people before widespread ivermectin administration programs were implemented. Certain epidemiological and clinical criteria have been proposed to identify Otoacoustic Emissions (OAE). The person must have lived in an onchocerciasis-endemic region for at least 3 years, and the onset of seizures must have occurred between the ages of 3 and 18 [1]. There is a high prevalence of epilepsy in the village, with several families having multiple children with epilepsy. There is no obvious cause of epilepsy, such as perinatal trauma, recent head trauma, cerebral malaria, or encephalitis [2]. However, tau deposits were not discovered in the brains of two people who died with OAE, including five people who died with nodding syndrome [2] in a more recent post-mortem investigation on nine people who died with OAE, including five people who died with nodding syndrome. Tau deposits have also been seen in people with refractory epilepsy, hence tau deposits in people with OAE are almost certainly the result of repeated seizures. In all but one person, the second post-mortem analysis revealed localized evidence of neuro-inflammation defined by gliosis and characteristics of the previous ventriculitis and/or meningitis [3]. We tested CSF samples of ivermectin-naive persons with OAE from South Sudan using a select panel of 9 inflammatory cytokines and chemokines, based on pro-inflammatory, anti-inflammatory and vascularisation response, to identify a cytokine profile that could provide additional information for pathology studies in people with OAE [4]. We retested all samples in Europe for the presence of T. solium, *Toxocara Canis* antibodies to explore the validity of the OAE definition and to estimate the proportion of people infected with *O. volvulus* who is also infected with another parasite [5].

Only a lower level of IL-13 expression in the CSF of people with OAE was seen when compared to African controls. IL-6, IL-4, and TNF- were not detectable in either the patients or the controls. When compared to Europeans with epilepsy, Africans with non-OAE neurological illnesses had a higher level of IL1- For IL-5 and CCL3 (Mip1-), there was no significant difference between the groups. The expression profile of VVCAM differed significantly between African controls and Europeans with neurological disorders but no epilepsy. However, there was no discernible difference between the cases and the controls. Furthermore, there were no differences in expression between any of the groups.

Discussion

When compared to Europeans with epilepsy, African controls with non-OAE neurological illnesses had a greater level of IL1-. In comparison to Europeans with neurological disorders, African controls with non-OAE had a high amount of VCAM. However, given the study's small sample size and the lack of cytokine serum level measurements, it's challenging to interpret these findings. It's crucial to point up the study's shortcomings. The biggest drawback is that we did not include healthy controls or people with epilepsy who did not have O. volvulus infection. Furthermore, the results could have been impacted by the cross-reactivity of the Onchocerca, Strongyloides, and Toxocara serological tests. However, correlation analysis revealed no substantial positive link between the tests.

Conclusion

In conclusion, based on the findings of our pilot investigation and a review of the literature, the causative involvement of neuro-inflammation in OAE, including nodding syndrome, cannot be determined. Because the local population raises pigs, a more thorough examination of the neurocysticercosis prevalence in the area should be investigated. Ideally, cytokine levels should be measured in prospective research, with serum and CSF samples taken at the time of the first seizure.

References

1. Colebunders, R., et al. "Onchocerca volvulus and epilepsy: A comprehensive review using the Bradford Hill criteria for causation." *PLoS Negl Trop Dis* 15.1 (2021).
2. Gumisiriza, et al. "Onchocerciasis elimination drastically reduces epilepsy prevalence and incidence in Kabarole, Western Uganda". In Proceedings of the 4th African Epilepsy Conference, Entebbe, Uganda, (2019) 22-24.
3. Chesnais, C. B., et al. "A second population-based cohort study in cameroon confirms the temporal relationship between onchocerciasis and epilepsy." *Open Forum Infect Dis* 7.6 (2020).
4. Hotterbeekx, A., et al. "Neuroinflammation and not tauopathy is a predominant pathological signature of nodding syndrome." *J Neuropathol Exp Neurol* 78.11 (2019): 1049-1058.
5. Duke, B. O., et al. "Microfilariae in the cerebrospinal fluid, and

neurological complications, during treatment of onchocerciasis with diethylcarbamazine." *Tropenmed Parasitol* 27.2 (1976): 123-132.

Cite this article: Christensen HK. Epilepsy caused by Parasitic Parasites in an Onchocerciasis Endemic Area. Neurol Neurorehabilit. 2022, 4 (1), 007-008.