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Neurocysticercosis Caused by Invasion of the Larvae into the Central Nervous System: A systematic Review

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Abstract

Neurocysticercosis is a disease of poverty and underdevelopment. Little is known about the natural history of the infection in humans, but some of the mechanisms whereby the parasite remains silent and evades the host immune response are understood. Symptomatic neurocysticercosis usually results from host inflammatory response after parasite death, and the clinical manifestations can be diverse. There is no evidence that cysticidal treatment does more good than harm in addition to conventional antiepileptic treatment. Population control measures involving immunisation or mass treatment have not shown long term effectiveness.

Epilepsy, similarly to neurocysticercosis, is a largely unrecognised but increasing burden on the welfare and economies of developing countries. The technology of drug treatment and psychosocial rehabilitation is well known but requires widespread and effective dissemination at low cost. There is little epidemiological data on risk factors for epilepsy in developing countries on which to base prevention strategies. The public health prioritisation of chronic disorders such as epilepsy remains a challenge for policy and practice in developing countries.

For both neurocysticercosis and epilepsy, there is a dilemma about whether limited public resources would better be spent on general economic development, which would be expected to have a broad impact on the health and welfare of communities, or on specific programmes to help individual affected people with neurocysticercosis and epilepsy. Either approach requires detailed economic evaluation.

Introduction

Taeniasis and (neuro) cysticercosis are caused by the cestode Taenia solium or pork tapeworm. T. solium is a multi-host parasite with a complex zoonotic transmission cycle, circulating between the intermediate pig host and the definitive or accidental intermediate human host. T. solium infection arises from ingestion of contaminated food or water and ingestion of raw or undercooked pork and may result in taeniasis (caused by the adult tapeworm living in the small intestine) and/or cysticercosis or neurocysticercosis [NCC; caused by invasion of the larvae into the central nervous system (CNS)] in humans[1].

When humans consume pork containing live T. solium cysts, the cysticercus develops into a mature tapeworm in the human intestine, shedding eggs that are expelled in human feces.

Cysticercosis develops when, following ingestion of T. solium eggs, T. solium larvae migrate and become encysted, typically in the muscle tissue of the host. Pigs can harbour thousands of cysts [2]. When T. solium cysticercisis develop in the human brain, the condition is defined as NCC. NCC is the most common parasitic disease of the CNS in humans affecting between 2.5 and 8.3 million people annually, accounting for a global burden of 2.8 million disability-adjusted life years (DALYs). NCC is a major clinical consequence of T. solium infection and the dominant cause of global preventable epilepsy associated with morbidity and mortality from epileptic seizures and epilepsy related death; where T. solium is endemic, 30% of epilepsy cases are estimated to be caused by NCC.

Taeniasis can cause abdominal pain, nausea, and diarrhoea, although it is often asymptomatic, at around 8 weeks post-ingestion with symptoms persisting until treatment with anthelmintic drugs or for around 2–3 years (the lifespan of the adult tapeworm) if untreated[3]. Many carriers of T. solium (taeniasis and cysticercosis) are asymptomatic and become long-term carriers of infection through self-reinfection and re-infection from others within the household. T. solium infection in pigs can be detected by meat inspection by visual inspection of cut meat and by lingual examination of the live animal; this, however, has low sensitivity as cysts can be missed[4].

Cysticerci consist of two main parts, the vesicular wall and the scolex. After entering the central nervous system, cysticerci are in a vesicular (viable) stage in which the parasites have a transparent membrane, a clear vesicular fluid, and a normal invaginated scolex. Cysticerci may remain viable for years or, as the result of the host's immunological attack, enter in a process of degeneration that ends with their transformation into calcifications. The first stage of involution of cysticerci is the colloidal stage, in which the vesicular fluid becomes turbid, and the scolex shows signs of hyaline degeneration. Thereafter, the wall of the cyst thickens and the scolex is transformed into mineralized granules; this stage, in which the cysticercus is no longer viable, is called the granular stage. Finally, the parasite remanents appear as a mineralized nodule [5].

Vesicular cysticerci elicit little inflammatory reaction in the surrounding tissue. In contrast, colloidal cysticerci are often surrounded by a collagen capsule and by a mononuclear inflammatory reaction that includes the parasite itself [6]. The surrounding brain parenchyma

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shows astrocytic gliosis, microglial proliferation, edema, neuronal degenerative changes, and perivascular cuffing of lymphocytes. When parasites enter into the granular and calcified stages, the edema subsides but the astrocytic changes in the vicinity of the lesions may become more intense, and epithelioid cells appear and coalesce to form multinucleated giant cells [7]. Meningeal cysticerci usually elicit a severe inflammatory reaction in the subarachnoid space with formation of an exudate composed of collagen fibres, lymphocytes, multinucleated giant cells, eosinophils, and hyalinized parasitic membranes leading to abnormal thickening of the leptomeninges [8]. This inflammation may be disseminated inducing damage in the optic chiasm and cranial nerves arising from the brainstem, as well as in small penetrating arteries arising from the circle of Willis [9]. The latter may cause occlusion of the lumen of the vessel with the subsequent development of a cerebral infarction. The foramina of Luschka and Magendie may also be occluded by the thickened leptomeninges and parasitic membranes with the subsequent development of obstructive hydrocephalus. Ventricular cysticerci may also elicit an inflammatory reaction if they are attached to the choroid plexus or to the ventricular wall [10]. The disrupted ependymal lining may protrude toward the ventricular cavities blocking CSF transit, particularly when the site of protrusion is at or near the foramina of Monro or the cerebral aqueduct [11].

Diagnosis

The advent of modern neuroimaging tests drastically changed our diagnostic accuracy for neurocysticercosis. CT and MRI provide objective evidence on the number and topography of lesions and their stage of involution Vesicular cysticerci appear on CT and MRI as small and rounded cysts that are well demarcated from the surrounding brain parenchyma [12]. There is no edema and no contrast enhancement. Many of these lesions have in their interior an eccentric hyperdense nodule representing the scolex, giving them a pathognomonic "hole-with-dot" appearance [13]. Colloidal and granular cysticerci appear as ill-defined lesions surrounded by edema; most of them show a ring or a nodular pattern of enhancement after contrast medium administration. This pattern correspond is commonly referred as to "cysticercus granuloma" . A particular neuroimaging pattern is that observed in patients with cysticercoid encephalitis [14]. CT and MRI show diffuse brain edema, collapse of the ventricular system without midline shift, and multiple small ring-like or nodular enhancing lesions disseminated within the brain parenchyma. Calcified cysticerci normally appear on CT as small hyperdense nodules without perilesional edema or abnormal enhancement after contrast medium administration [15].

Conclusion

Relatively little is known about the epidemiology of neurocysticercosis, and cysticidal treatments have been advocated without clear evidence for overall benefit in humans. Diagnosis may be difficult even with neuroimaging and serological facilities. Neurocysticercosis is a major cause of acquired epilepsy in Latin American countries. The economic burdens of both are not well quantified. Community prevention and eradication of neurocysticercosis depends on general infrastructural development. Most people with epilepsy in developing countries do not have access to appropriate management. The major challenge for the future will be to develop primary level services that provide appropriate interventions for these communities.

Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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