Cerebellar Neurocysticercosis and Novel Hypotheses.

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Citation

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Abstract

Cysticercosis, a parasitic infection caused by the larval form of the pork tapeworm, Taenia solium, is increasingly recognized as a cause of epilepsy, headache, and neurological signs when it is located in the brain, optic nerve or spinal cord, known as neurocysticercosis. Anecdotal clinical presentations in patients with severe posterior fossa involvement may include signs of bilateral fourth nerve palsy, facial myokymia, upbeat nystagmus, periodic alternating nystagmus, and rhythmic oculopalatal myoclonus. In patients presenting huge cystic lesions due to posterior fossa neurocysticercosis, intermittent severe headache, epilepsy, signs of raised intracranial pressure, cerebellar signs and cranial nerves signs can be seen as well.We have developed a theoretical explanation for this mechanism based on the hemodynamic characteristic of the blood flow which to be subjected to the opinion of other authors.

INTRODUCTION

Cysticercosis, a parasitic infection caused by the larval form of the pork tapeworm, Taenia solium, is increasingly recognized as a cause of epilepsy, headache, and neurological signs when it is located in the brain, optic nerve or spinal cord, known as neurocysticercosis (NCC). A high prevalence of cysticercosis/taeniosis has been reported from the developing countries because of the coexistence of poor sanitary conditions and domestic pig rising without veterinary control or surveillance systems. It occurs mainly in Eastern and Southern Africa, China, India, Mexico, Central America, Chile, Ecuador, Colombia, Venezuela, Peru, Brazil, Papua New Guinea, and non-Islamic South East Asia where human faces reach pigs and pork is eaten raw or undercooked.

An infection which leads to extra-intestinal disease (including NCC) usually occurs as a result of eating food or drinking water contaminated by human feces containing T. solium eggs. It is a preventable and potentially eradicable neurological disease which primarily affects people living in the developing world. The disease is endemic in Central and South America, Asia and Africa.

Seizures are widely reported to be the most common symptom, occurring in 70-90% of patients. Most patients respond to praziquantel if cystic lesions are located in the parenchyma tissue and Albendazole when parasites are located in the ventricular system and subarachnoid space [1-16]. Seizure disorder is the most frequent clinical manifestation of the NCC observed in 50% to 80% of cases, in children and adults presenting intraperanchymal cystic lesions. [13-44] There is a variable time interval between point of infection and the onset of symptoms: ranging from 1-30 years. [16]

In spite of the growing number of publications related with neurocysticercosis including the non-traditional presentations, there has been no study geared to show the mechanism by which some parasites are located in these uncommon regions. Based on our observations on patients with NCC of the posterior fossa we have developed a theoretical explanation for this mechanism to be subjected to the opinion of other authors which we will try to demonstrate in an upcoming study.

NEUROCYSTICERCOSIS IN THE POSTERIOR CRANIAL FOSSA.

The posterior cranial fossa is part of the intracranial cavity, located between the foramen magnum and tentorium cerebelli. It contains the brainstem and cerebellum. This is the most inferior of the fossa. It houses the cerebellum, medulla and Pons. Anteriorly it extends to the apex of the petrous temporal. Posteriorly it is enclosed by the occipital bone. Laterally portions of the squamous temporal and mastoid part of the temporal bone from its walls.

In the posterior fossa, NCC can involve the fourth ventricle, cerebellopontine angle cistern, cisterna magna and rarely,

the cerebellum [17,18] .Cysticercosis affecting one of the above-mentioned anatomical structures are grouped under the name of NCC of the posterior fossa (PFNCC).

One of the first articles about clinical aspects of this presentation was done by Bickerstaff et al., in 1956 [19] and about clinical and surgical management by Kla in 1964. [20] Other modalities of management including the diagnostic, importance of laboratory and serological analysis of blood and cerebro-spinal fluid in PFNCC and pseudotumoral form of Cysticercus cellulose to be remembered when one attempts to characterize the type of neoplasms of the posterior fossa detected by brain scan in the infantile population were reported from 1971 and 1978. [21-24]

Anecdotal clinical presentations in patients with severe posterior fossa involvement may include signs of bilateral fourth nerve palsy, facial myokymia, upbeat nystagmus, periodic alternating nystagmus, and rhythmic oculopalatal myoclonus. [25] In patients presenting huge cystic lesions due to PFNCC, intermittent severe headache, epilepsy, signs of raised intracranial pressure, cerebellar signs and cranial nerves signs can be seen as well. [17, 26] Studies post-mortem have confirmed this correlation between signs of increased intracranial pressure, localizing cerebellar signs and even hearing loss in patients presenting PFNCC in the cerebellopontine cistern and obstructive hydrocephalus. [27, 28]

Sang-Wook et al. [29] reported a case presenting headache, dizziness, drowsiness and ataxic gait. The magnetic resonance imaging showed hydrocephalus and an ill-defined, multicystic cerebellar mass with hyper signal on T2-weighted images, hypo signal on T1-weighted images and rim enhancement after gadolinium injection (the differential diagnoses of include abscess, tuberculosis, metastasis and other parasitic diseases). The patient underwent endoscopic third ventriculostomy and the cyst resection was done through a craniotomy. In surgical field, cysts were conglomerated in a dense collagen capsule that were severely adherent to surrounding cerebellar tissue, and transparent cysts contained white, milky fluid. Histological findings confirmed the diagnosis of cysticercosis. These authors concluded that racemose cysticercosis is rare in the cerebellar hemisphere but neurocysticercosis should be taken into consideration as a differential diagnosis of multiple cystic lesions in the cerebellum and we agree on that. As has been mentioned, T solium cysticercosis spread through the bloodstream and it may locate almost anywhere in the CNS, most frequently involves the cerebral hemispheres and sometimes ventricles, basal cisterns, subarachnoid space, and spinal cord. Cerebellar cysticercosis has been rarely reported [30, 31]. Anecdotal clinical presentations include multiple cranial nerve involvement (V, VI, VII, and VIII) and cerebellar signs resembling anterior inferior cerebellar artery (AICA) syndrome. [32]

The cerebellar lesions are an expression of this type of distribution as they generally cisticercotic lesions in the posterior fossa are only seen when there are massive infestations. (See figure 1)

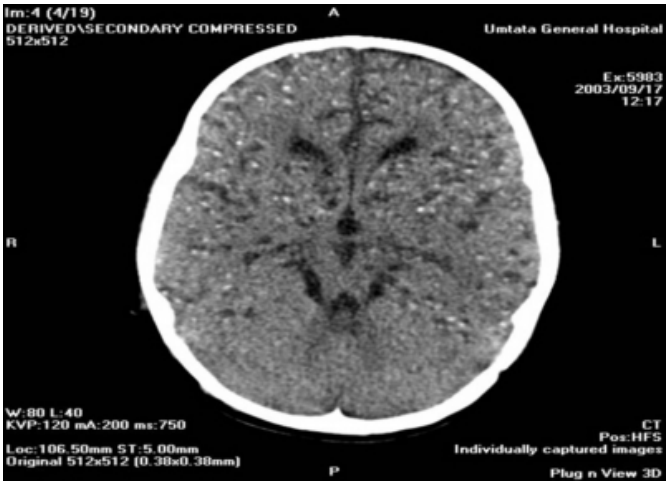
NOVEL HYPOTHESES

In the absence of a massive infestation of T solium, we believe that the presence of cysticerci in the cerebellum could be possible when the cysts travel through the bloodstream erratically and instead of move forward by the carotid territory do so by the vertebro-basilar territory infecting the cerebellum (Isolated cerebellar NCC). We think that situation happen because of the hemodynamic characteristic of the blood flow can change in different blood vessel. Let us explain what probably happen when cysticerci coincide in the same place at the same time according to our hypotheses.

We think that in some situations, between two cysticercus in movement speed gradient exists, or when one moves faster than the other for whatever reason, they develop friction forces acting tangentially to the same.

Figure 1

Figure 1: Massive neurocysticercosis showing very few active cysts on both cerebellar hemispheres.



The friction forces' trying to introduce rotation between the cysticerci in motion, but simultaneously the viscosity seeks to prevent rotation. Depending on the relative value of these

forces can be produced different flow states. When the gradient of speed is low, the inertia force is greater than the friction, the cysticerci move but do not rotate, or they make it but with very little power, the final result is a movement in which the particles follow paths defined, and all of the particles that pass by a point in the field of flow follow the same trajectory. This type of flow was identified by O. Reynolds and is called “laminar”, meaning that the cysticerci are moving in the form of layers or sheets. The increase of velocity gradient increases the friction between neighboring cysticercus to the fluid, and these become a significant energy of rotation, the viscosity loses its effect, and due to the rotation of the cysticercus change trajectory. Going from one path to another, the cysticerci collide and change course in erratic flow (“turbulent”). As is well known, turbulent flow can also be due to abnormalities in the wall of blood vessels. It can be summarized as follow:

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