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Review article

Taenia solium Cysticercosis — The lessons of history

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ABSTRACT

Human taeniasis as well as porcine and human cysticercosis — caused by the pork tapeworm *Taenia solium* — are ancient diseases. The fact that pigs were considered impure in the ancient Greece and that the Koran prohibited the consumption of pork, were likely related to the knowledge that cysticercosis may affect swine. Evidence suggests that human cysticercosis was also present in the ancient Egypt and Rome. During the Renaissance, the causative agent was properly identified and human cases were recognized. Confirmation that both taeniasis and cysticercosis were caused by the same parasite was provided during the 19th Century by German pathologists. During the 20th Century, bouts of human cysticercosis in non-endemic regions left us valuable lessons on the mechanisms of disease acquisition and spread. These included a large series of neurocysticercosis cases in the United Kingdom that occurred after the return of troops stationed in India (which demonstrated that symptoms may occur years after infection), the epidemic of cysticercosis-related epilepsy in the Ekari people of Papua New Guinea occurring after the gift of pigs with cysticercosis received from Indonesia (demonstrating the fast establishment of endemic transmission and the impact of cysticercosis in epilepsy frequency), and the occurrence of neurocysticercosis among members of an Orthodox Jewish community of New York City, related to Latin American *Taenia* carriers working in their houses (highlighting the fact that cysticercosis transmission do not require the presence of infected pigs). These lessons of history have significantly contributed to our current knowledge on this disease.

Contents

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1. Introduction

Taenia solium belongs to the family Taeniidae, which includes three parasites affecting humans: *T. solium* (the pork tapeworm), *Taenia saginata* (the beef tapeworm) and *Taenia asiatica*, a tapeworm that is morphologically similar to *T. saginata* but biologically analogous to *T. solium*. Of these, *T. solium* is more relevant as a human pathogen since infection of the human nervous system with its larval form causes neurocysticercosis, a major cause of seizures and neurological disability worldwide [1]. As it occurs with most ancient diseases, the history of *T. solium* has been surrounded by a number of myths, fortuitous discoveries, curious experiments, and natural epidemiological scenarios [2]. We rescue here relevant episodes in the history of taeniasis and cysticercosis that left us valuable lessons on the biological cycle of *T. solium* as well as on mechanisms of disease acquisition and spread (Table 1).

2. Ancient civilizations

2.1. Pig, the impure animal

Probably the oldest source banning pork consumption was mentioned in the third book of the Hebrew Bible (Leviticus, 11:7.8, *c*600–500 BC), where it was written "And the pig, because it parts the hoof and is cloven-footed but does not chew the cud, is unclean to you. You shall not eat any of their flesh, and you shall not touch their carcasses; they are unclean to you". Ancient Greeks were familiar with the occurrence of swine cysticercosis (measly pork). Besides an anecdote mentioned in Aristophanes' (*c.*448–385 BC) comedy "The Knights", where a slave suggested that the tongue of one of the main characters should be examined in the same way it was used to do with pigs, to see if he was "measled", Aristotle (*c.* 384–322 BC) described in detail the presence of bladders or cysts in pig muscles that were compared with hailstones. He also noticed that this was a condition associated with free roaming, since nursing pigs do not suffer from the disease [2–4]. Pigs were considered impure in the ancient Greece, and it is possible that this belief inclined Muhammad (570–632 AD) to prohibit the consumption of pork at the time when the Koran was written.

2.2. Epilepsy, the sacred disease

Since there are evidences of the endemicity of porcine cysticercosis, human cysticercosis must have been present in the ancient Greece and Rome. The concept of adult-onset epilepsy being related to a structural disease of the nervous system can be traced back to the Hippocratic treatise "On the Sacred Disease" [5]. It has also been suggested that the epilepsy that distressed the Roman dictator Gaius Julius Cesar (100–44 BC) was related to cysticercosis, as it started when he was 54 years old (one year after one of his visits to Egypt) and were apparently of partial origin with secondary generalization [6,7]. While Egyptians did not eat pork with the exception of one sacred day per year, human taeniasis has been well documented in the ancient Egypt. The Ebers papyrus (1500 BC) includes descriptions of tapeworms, and both *Taenia* spp. eggs, and *T. solium* cysticerci have been found in the intestine and stomach of Egyptian mummies [8–10]. Moreover, intestinal tapeworms were a common knowledge among Arabian and Egyptian physicians, who treated them with pumpkin seeds (*Cucurbita pepo*), an herbal medicine that it is still used nowadays [11].

Table 1
Major events in the history of taeniosis and cysticercosis.

Date	Event–discovery–description
3000–1500 BC	Tapeworms recognized in mummys from the ancient Egypt.
600–500 BC	The Hebrew Bible (Leviticus) condemned pork consumption.
400–350 BC	Swine cysticercosis recognized in the ancient Greece.
600–1000	The Koran prohibited consumption of pork; Arabs and Egyptians used pumpkin seeds to treat taeniosis.
1558	First reported case of human neurocysticercosis.
1697	Recognition of cysticerci as parasites.
1792	Link between taeniasis and cysticercosis in the same patient.
1850–1900	Description of the life cycle of *Taenia solium*.
1909–1911	Introduction of the complement fixation test for diagnosis of cysticercosis.
1930–1960	Description of the natural history of human cysticercosis.
1970 to date	Introduction of neuroimaging and immune diagnostic methods; advent of specific therapy; attempts to eradicate cysticercosis.

3. Unraveling taeniasis and cysticercosis

3.1. First descriptions of human cysticercosis

After the Catholic Church stopped condemning the practice of autopsies, human cases of cerebral cysticercosis started to be recognized. Indeed, it is generally accepted that the first recorded cases of neurocysticercosis were those described by Rumler in 1558 during the autopsy of a patient with epilepsy who had liquid-filled vesicles adherent to the meninges and by Panarolus in 1652, who found similar vesicles in the corpus callosum of a priest who had suffered from seizures [2]. The parasitic nature of these vesicles was not documented until Malpighi, in 1697, described the scolex of the *T. solium* inside them. During those years, Gmelin coined the term *Taenia cellulosae* for the vesicles, and Zeder included them into a new genus, *Cysticercus* (from the Greek: *kustis*, *cystis*, bladder, and *kerkos*, *cercos*, tail). It follows that the commonly used term "cysticercus cyst" is a pleonasm, since the word "cyst" is included in the etymological definition of "cysticercus". It was initially believed that cysticerci constituted a separate parasitic species, and it was classified as *C. cellulosae* due to its tendency to develop in connective tissue; this incorrect term is still widely used nowadays.

3.2. From serendipity to rational knowledge

Simultaneous taeniasis and cysticercosis in the same person was probably first described by the Peruvian physician and journalist Hipólito Unanue in 1792, as he wrote in the journal "El Mercurio" the case of a soldier with taeniasis who died following a major seizure [12]. As noted in the original publication, Unanue was not looking to describe a particular medical condition, but the article was written as a plea for authorities to build an anatomy amphitheater in the community for a better recognition of the causes of deaths in the population.

During the 19th Century, German pathologists recognized the morphological similarities between the head of the adult *T. solium* and the scolex of cysticercus, and Küchenmeister [13] demonstrated that ingestion of cysticercus from pork resulted in human intestinal taeniasis, by feeding a convicted man, condemned to death, with sausages and a noodle soup both containing cysticerci obtained from a recently slaughtered pig. At autopsy, Küchenmeister found "a small *Taenia* that was tightly attached with its proboscis to a piece of duodenal mucosa", as well as other nine *Taenias*, one of them with the complete crown of 22 hooklets in two rows typical of the rostellum of *T. solium*. Soon thereafter, the knowledge on the life cycle of *T. solium* was completed by experiments in Belgium and Germany, demonstrating that pigs develop cysticercosis after ingesting *Taenia* eggs obtained from proglottids passed by *T. solium* human carriers. These findings were further confirmed by the seminal work of Yoshino [14–16] who infected himself with *T. solium* cysticerci to study the life cycle of the cestode.

4. Natural epidemiological scenarios

4.1. The legacy of British military doctors

By the end of the 19th Century and the first two decades of the 20th Century, isolated case reports mentioned the occurrence of cysticercosis

in persons with dementia or epilepsy living in or returning from different outposts of the British Empire [17–20]. At that time, the worldwide prevalence of cysticercosis was largely unknown, but it was suspected to be present in some of the regions where those British colonies were settled, particularly the sub-Saharan Africa [21] and the Indian subcontinent, where the disease was first recognized in 1888 during the autopsy of an inmate patient from the lunatic asylum in Madras [22].

In the 1930s — working at Queen Alexandra's Military Hospital (Millbank) in London — Colonel (later Lieutenant General) Sir William Porter MacArthur (1884–1964) and Colonel (later Brigadier) Henry Brian Frost Dixon (1891–1962) with some of their co-workers, brought to the attention of the medical community that a high number of British soldiers had been discharged during the previous decades due to epilepsy [23–28]. Further study of these soldiers showed that a sizable proportion of them had cysticercosis, a disease that was considered to be almost inexistent in England by that time. Interestingly, most of these soldiers started having seizures during or after serving in India, one of the countries where this parasitic disease was suspected to be endemic. While a total of 450 cases were reported [28], these numbers were clearly an underestimate, and it was estimated that the incidence of symptomatic cysticercosis ranged from 1.2 to 2 per 1000 men among troops stationed in India from 1921 to 1937.

This unique epidemiological scenario, in which the circumstances of the infection could be estimated, allowed — for the very first time — a description of the natural history of human cysticercosis as well as the recognition that a sizable number of infected persons may be asymptomatic for several years or may never develop clinical manifestations at all. More than 50% of patients initiated with seizures between three and five years after returning from India. Indeed, MacArthur stated in one of his papers "For every case of cysticercosis immediately diagnosticable there are a large number which will defy diagnosis for years" [26].

MacArthur [24] as well as Dixon and Smithers [25] suggested that cysticerci might live longer in the brain than in muscles and subcutaneous tissues. This was based on three main facts: 1) intracranial calcifications were detected by X-ray films at a later stage than elsewhere in the body, 2) a sizable proportion of patients develop neurological symptoms (seizures) years after muscle cysts have been calcified, and 3) in some necropsies or patients experiencing surgery, cysts in the brain showed no evidence of calcification while muscle cysts had been calcified for up to five years. The latter had already been described several years before, in the seminal work of Henneberg [29].

The British military doctors also noticed that treating the adult tapeworm might exacerbate the symptoms of cysticercosis, giving the first note of caution for the potential risks associated with mass treatment of human taeniasis at the population level [23]. It could also be recorded that some of the patients with cysticercosis were family members of the soldiers but had never been abroad, suggesting locally transmitted disease in a household contact of a soldier who was also a *T. solium* carrier [28].

4.2. The epidemic of burns in West New Guinea

Rural inhabitants of West New Guinea have always had a swine-breading culture. Nevertheless, the country was free of porcine cysticercosis until 1972, when people living in the Enarotali region (near the Wissel Lakes area) received a gift of infected pigs from the Indonesian government in Java. Soon thereafter, cysticerci were noticed in the flesh of local pigs slaughtered in this region, and intestinal taeniasis was diagnosed in humans. By 1974, an epidemic of burns was observed among Ekari natives of the region [30]. This epidemic of burns resulted from seizures occurring when persons were sleeping, causing them to fall into bonfires used to warm-up their huts during cool nights. A sizable proportion of these patients also had subcutaneous nodules that were confirmed as cysticercus by biopsy and one of them (a girl dying after a major seizure episode) had thousands of parasites in the brain parenchyma [31]. Nine years later, a new survey showed that cysticercosis still remained endemic in the Ekari people, and started to spread to neighboring villages [32], and even nowadays, the disease is still prevalent in the region [33].

4.3. Disappearing CT enhancing lesions in Indian patients: an evolving concept

Soon after the introduction of CT in India, an increasing number of young adults with recent onset epilepsy presenting with a single, small, enhancing parenchymal brain lesion was noticed. While these lesions were initially suspected to be tuberculomas [34], their spontaneous resolution on serial imaging studies created confusion to the point that it was considered that they just represented rupture of the blood–brain barrier and thus, were seen as the consequence and not the cause of the seizure disorder [35]. By that time, reports from Mexico also brought to the attention of the medical community the same disturbing images, commenting on their possible cysticercotic nature and suggesting that they represented acute phases of infestation of the nervous system by these parasites [36,37]. Controversies on etiological considerations were solved after pathological studies of biopsy specimens revealed that cysticercosis was the cause of this peculiar CT finding [38,39]. Subsequent reports from different cysticercosis endemic areas confirmed that this form of neurocysticercosis is not confined to the Indian subcontinent [40,41]. Nevertheless, it remained to be understood why many of these parasites spontaneously disappear on neuroimaging studies. After years of debate, recent position papers presented arguments favoring the fact that these lesions most likely represent young or immature forms of cysticerci that are destroyed by the host immune system soon after entering the nervous system [42–44].

4.4. Cysticercosis in an Orthodox Jewish Community in New York City

Improved neuroimaging led to a sudden increase in the numbers of neurocysticercosis cases observed in the US after 1970, mainly occurring in Mexican immigrants to the Southwestern States of Texas and California [45]. A decade after, the occurrence of neurocysticercosis among members of an Orthodox Jewish community disturbed the medical community of New York City. As expected, these people did not eat pork for religious reasons, and most of them had never been in cysticercosis-endemic countries. When experts from the Center for Disease Control and Prevention (Atlanta, GA) conducted an investigation to determine the source of infection, the most probable source of infection were *T. solium* carriers from Latin America. They had most likely been infecting people for whom they worked — as housekeepers — via fecal–oral contamination [46]. Further studies showed an increased prevalence of anti-cysticercus antibodies in members of this community which were related to widespread employment of domestic workers from cysticercosis-endemic regions [47]. The same scenario has more recently been noticed in some countries of the Arab World, where autochthonous cases of neurocysticercosis have been increasingly recognized in the context of wealthy Muslim families employing babysitters and housekeepers from disease-endemic areas [48]. Both, in the US and in the Arab World (and probably in other non-endemic regions as well), *T. solium* carriers entering those countries every year may infect local people and may be increasing the prevalence of neurocysticercosis without the need of infected pigs [49].

5. Comment

These curious tales on the history of taeniasis and cysticercosis are much valued as they significantly contributed to our current knowledge on the life cycle of *T. solium* and on the mechanisms of disease acquisition by humans. In particular, the information revealed along the three natural epidemiological scenarios recounted in this paper greatly

added to our understanding on the clinical expression of the disease in migrant populations, on the dynamics of transmission in newly endemic regions, and on the possibility of local transmission in non-endemic regions.

Many important lessons were derived from the bout of human cysticercosis in British soldiers, including the assessment of the contribution of neurocysticercosis to the burden of seizures and epilepsy, the understanding that brain infection survives muscle and subcutaneous infection for years, and the knowledge that in many cases seizures occur years after infection, likely reflecting late degeneration of one or several brain parasites [23–28]. From the West New Guinea epidemic, we learned the fast, widespread, and clinically expressive introduction of infection and clinically symptomatic disease in a previous non-endemic region [30–33]. Finally, the bout of cysticercosis among Orthodox Jews in New York City is a clear reminder that infected pork is not required for cysticercosis transmission, and migrating tapeworm carriers may be responsible for the development of foci of infection even under good sanitary conditions in a developed country [46,47]. This also explains the occurrence of locally acquired cysticercosis in areas where pig-husbandry is adequate or where it is prohibited by religious laws [48,49]. These lessons have provided basic information to better understand the mechanisms of disease transmission and should eventually contribute to the reduction of the burden of human cysticercosis in this global world.

Conflicts of interest

Nothing to disclose.

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