Taeniasis and/or Cysticercosis: What Awaits Us?
Teníase e/ou Cisticercose: O que nos Espera?

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In an era of significant global changes—particularly in climate, migration, and the increasing prevalence of conflicts leading to a rise in refugees—many of whom are housed in overcrowded camps with inadequate sanitary conditions—alongside the growing phenomenon of mass tourism, which involves millions of people traveling between continents and hemispheres each year, any disease occurring anywhere in the world can potentially become relevant to us.

Diseases that were once considered remote and unlikely in our context, such as dengue, malaria, Zika, chikungunya, West Nile virus, schistosomiasis, strongyloidiasis, measles, Chagas disease, amoebic abscesses, and many others, are increasingly appearing in our clinical practice.

We must be aware that the diversity of pathologies we need to consider in our daily practice is expanding. Therefore, we must continuously improve our preparedness to be truly global physicians.

This brings us to the relevance of discussing taeniasis and cysticercosis, diseases that were once thought to be of the past or confined to poor endemic regions. Could they be re-emerging in Europe?

Taeniasis is a disease characterized by the presence of *Taenia* in the human intestine, whereas cysticercosis results from the lodging of *Taenia larvae*, viable or not, in various locations within the human body.

Cysticercosis can be symptomatic or asymptomatic. Taeniasis is usually asymptomatic. Both can coexist in the same person, but in most cases, only one or the other occurs.

Taenia is a platyhelminth (flatworm) of the *genus Taenia. T. saginata* has cattle as its intermediate host. *T. solium* and *T. asiatica* have humans as their intermediate hosts, but only *T. solium* can cause significant human disease, and thus, we will focus only on this species.

T. solium exclusively parasitizes the human small intestine. It consists of the scolex (head), which adheres to the intestinal wall using highly muscular suckers and hooks after ingestion of undercooked pork containing viable cysticerci (*larvae*). The scolex acts as an "anchor" to which the proglottids attach. As they form, the proglottids link together like train carriages, increasing the tapeworm's length, forming a ribbon-like structure that can reach up to 7 m in length, remaining in the small intestine for over 20 years and frequently causing no symptoms.

These segments, the proglottids, are hermaphroditic, containing both male and female reproductive organs, allowing them to produce up to 250 000 eggs per day. The parasite has a nervous system and an excretory canal through which eggs are expelled into the feces. Since they lack a digestive tract, they absorb nutrients through their tegument. When they reach maturity, proglottids can detach and be excreted in feces, loaded with eggs.

The proglottids and eggs excreted in feces can:

1) Be ingested by pigs, particularly when raised in open spaces contaminated with human feces in areas with poor sanitary conditions. Once in the pig's intestine, the eggs release larvae known as oncospheres, which disseminate through the bloodstream and lodge in the pig's striated muscle, where they mature into cysticerci. If humans consume undercooked pork containing cysticerci, the larvae attach to the intestinal wall via the scolex. Over several months, the tapeworm grows, potentially reaching over 1000 proglottids, which, along with eggs, are excreted in feces, repeating the cycle.

2) Be ingested by humans via fecal-oral transmission, where the eggs release small larvae (oncospheres) that cross the intestinal wall and spread through the bloodstream, lodging in various organs. The fixation of larvae (*cysticerci*) in the central nervous system can cause significant disease. Human cysticercosis is not contagious, as the cysticerci are asexual and remain in the organs where they fixate.

Neurocysticercosis can be extraparenchymal, affecting the cerebral ventricles, subarachnoid space, spinal cord, or eyes. When cysticerci lodge in the parenchyma, they can cause epilepsy. In endemic regions, neurocysticercosis accounts for 30% of epilepsy cases, reaching 70% in hyperendemic areas. Parenchymal forms can also cause intracranial hypertension, hydrocephalus, or dementia. Symptoms may appear from a few months to over 30 years after exposure.

Cysticerci can present in three forms regarding their activity: viable (typically non-contrast-enhancing and 10-20 mm

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in diameter), degenerative (with intense inflammatory activity due to the immune system's response, whether viable or not), and calcified (inactive, 4-6 mm in diameter, possibly with some perilesional edema). They can cause neurological symptoms regardless of their viability.

Taenia in humans, through egg excretion, can cause cysticercosis in many other individuals and even in the carrier, through ingestion of their own eggs via fecal-oral transmission or retrograde peristalsis. The human tapeworm is also responsible for cysticercosis in pigs (which in turn causes taeniasis in humans). Without pigs with cysticercosis, the tapeworm would eventually disappear in humans, leading to the extinction of human cysticercosis. However, as long as the tapeworm persists, the millions of eggs excreted daily and asymptomatically over many years may still result in numerous cases of human cysticercosis.

In conclusion, without human taeniasis, there is no cysticercosis in either pigs or humans and without porcine cysticercosis, human taeniasis will cease to exist.

With the arrival of travelers and migrants from endemic regions, will cysticercosis once again become an important imported and autochthonous disease in our region?

European epidemiological studies face significant challenges due to reliance on case reviews that follow different methodologies from country to country, and within each country, case definitions and diagnostic methods have varied over time.

Several high-quality studies have been conducted in Portugal and integrated into European research, which, despite their interpretative limitations, remain valuable.[1-4]

Although cysticercosis is not a mandatory reportable disease in Portugal, the number of cases appears to be declining, with 379 hospitalizations for cysticercosis reported in Portuguese hospitals over four years (1993–1996) and 293 over 19 years (2000–2019).[1,2]

However, studies unanimously confirm that 75%–90% of neurocysticercosis cases in Portugal and across European countries occurred in migrants or individuals with travel history to endemic areas. The number of cases has risen significantly in recent years.

Many undiagnosed tapeworm carriers may continue shedding millions of eggs for years, making it likely that cysticercosis could significantly increase due to fecal-oral person-to-person transmission.

Furthermore, many people with neurocysticercosis will arrive in our country. Although not contagious, the disease can be severe, requiring diagnosis and treatment.

It is also important to note that outdoor pig farming has resumed. If the number of tapeworm carriers shedding eggs increases, not only neurocysticercosis but also cases of taeniasis and consequently cysticercosis may rise.

Screening for taeniasis in high-risk individuals, reinforcing personal hygiene measures—including simple handwashing in communities, groups, or families where tapeworm carriers are identified—and maintaining pork meat inspection by veterinary authorities are among the key preventive measures.

As physicians, we must be prepared for diseases we never expected to see and should advocate for the establishment of Emerging Disease Clinics in hospitals and health centers.

These new realities make medicine even more fascinating. ■

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