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OVERVIEW OF TAENIA SOLIUM AND CYSTICERCOSIS CELLULOSAE: WITH REFERENCE TO EGYPT

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Abstract

Taenia solium is an intestinal cestode of worldwide distribution, especially in poorer communities where humans live in contact with pigs and eat undercooked pork. *Cysticercus cellulose*, the larval stage, primarily develops in tissues of pigs. *T. solium* clinical syndromes include neurocysticercosis (NCC) and extraneural cysticercosis. NCC, in turn, is divided into parenchymal and extraparenchymal forms. Stages of cysticercosis include an initial (viable) phase, a degenerating (enhancing) phase, and a nonviable (calcified) phase. Cysticerci may be present in more than one anatomic site and at different stages in their natural history simultaneously. Taeniasis is usually characterized by mild and non-specific symptoms. Abdominal pain, nausea, diarrhoea or constipation may arise when tapeworms become fully developed in the intestine, approximately 8 weeks after ingestion of meat containing cysticerci. Symptoms may continue until tapeworm dies by treatment, otherwise it may live for several years. It the untreated *T. solium* infections generally persist for 2-3 years. Conventional anticonvulsant therapy is a must to manage neurocysticercosis-associated seizure disorders.

Key words: Taeniasis, *Cysticercus cellulose,* Pathogenicity, Treatment, Nursing, Overview

Introduction

Cysticercosis is caused by the larval stage (metacestode) of the pork tapeworm *Taenia solium*. Clinical syndromes include neurocysticercosis (NCC) and extraneural cysticercosis (Vedantam *et al*, 2016). NCC is divided into parenchymal and extraparenchymal forms. Extraparenchymal forms include intraventricular, subarachnoid, spinal, and ocular disease. In late 20th or early 21st century showed that NC is the leading cause of epilepsy onset worldwide (Hamamoto Filho *et al,* 2022). WHO (2021) added that neurocysticercosis refers to *T. solium* cysticercosis development in human CNS causing focal epilepsy, epileptic seizures, hydrocephalus, chronic headaches, focal deficits symptoms associated with increased intracranial hypertension. Neurocysticercosis is one of the leading preventable causes of epilepsy globally, up to 30% cases in endemic areas, where symptomatic or asymptomatic neurocysticercosis people are 2.56-8.30million. But, true numbers may be underestimated as to groups' poor access at highest risk to diagnostic tests.

Principles of cysticercosis stages (White *et et al,* 2018), included initial (viable) or degenerated phase, and a nonviable phase (table 1): 1- Viable cysticerci are round, hypodense lesions (5 to 20mm in diameter) on CT that don't enhance after contrast administration, but don't cause much inflammation in surrounding tissues, 2- Cysticerci that enhance after contrast administration reflect degeneration, cyst wall increases in density forming edema, which reflect the host inflammatory response against infection and are frequently associated with seizures. In many cases, enhancing cysticerci retain viable cysticerci features, and 3-Nonviable cysticerci are solid, calcified nodular lesions (2 to 4mm in diameter; 1 to 10 mm), and are usually non-enha-

ncing but, in some cases may be associated with perilesional edema, and/or seizures.

Cysticercosis *cellulosae* may be present in more than one body site with different stages in natural history simultaneously, and patient at any time may have some viable *C. cellulosae* enhancing and/or calcified cysts (George *et al,* 2022). The main dissemination features include intractable epilepsy, dementia, enlargement of skeletal muscles, subcutaneous and lingual nodules and a relative without focal neurological signs or clinically raised intracranial pressure, at least till the disease late stage (Baily, 2003). The usual involved human organs are subcutaneous tissue, skeletal muscles, lungs, brain, eyes, liver and heart (CDC, 2024).

Clinical manifestations of NCC depend upon whether cysticerci are localized in brain parenchyma, extraparenchymal tissues, or both (White *et al,* 1997). Intraparenchymal NCC is the commonest cysticercosis form in >60% of cases (Gupta *et al,* 2012). Symptomatic parenchymal NCC usually occurs 3 to 5 years up to >30 post infection; in delayed one with nonviable or calcified lesions patients suffered from seizures and/or headache (Coyle *et al,* 2012). Seizures are the most common manifestation; less common are altered vision, focal neurologic & meningitis signs (Wilson *et al,* 2018).

In massive parenchymal cysts patients, an intense immune response with diffuse edema and clinical picture resembling encephalitis with seizures, headache, nausea and vomiting, impaired consciousness, reduced visual acuity, and occasional fever (Sinha and Sharma, 2009). Many parenchymal NCC cases are asymptomatic and identified incidentally on radiographic imaging (Lerner *et al,* 2012). Intraventricular NCC (floated-free in ventricular cavity or attached to choroid plexus) in 10 to 20% cases. Cysticerci symptoms are lodged in ventricular outflow tracks with consequent obstructive hydrocephalus and increased intracranial pressure, nausea, headache, and vomiting, altered mental status, and decreased visual acuity with papilledema and subarachnoid neurocysticercosis risky in 5% of hospitalized cases (Nash *et al,* 2018). Subarachnoid NCC may be presented with chronic arachnoiditis and/or mass effect due to cyst enlargement, and chronic arachnoiditis can develop from local inflammations; sometimes, associated with communicate hydrocephalus, vasculitis, stroke, meningitis, mass effect and focal neurologic defects causing enlarged cysticerci in the space subarachnoid >10cm (Medina *et al,* 2005).

Other NCC clinical presentations are spinal (1%) and ocular lesions (1 to 3%). Spinal one is usually located in subarachnoid space causing inflammatory and demyelinating changes in peripheral nerve roots (Bandres *et al*, 1992). Patients showed radicular pain, paresthesias and/or sphincter disturbances. Ocular one involves sub-retinal space, vitreous humor, anterior chamber, conjunctiva, or extraocular muscles (Pushker *et al,* 2001).

Cysticercosis must be suspected in patients with seizures and/or elevated intracranial pressure and relevant neuroimaging findings (cystic lesions, enhancing lesions, and/or calcifications), in areas relevant epidemiologic exposure (ingestion of *T. solium* eggs pass in patient's stool or indoors of an asymptomatic carrier). Diagnostic cysticercosis criteria are based on the clinical manifestations, neuroimaging, and epidemiologic data (Carpio *et al,* 2016). Suspected NCC patients must be evaluated with CT scan and magnetic resonance imaging (MRI) of brain. CT is useful in diagnosing calcifications and parenchymal cysticerci. MRI detects smaller lesions, evaluates degenerative changes, edema around lesions of calcified and/or scolices (Kimura-Hayama *et al,* 2010). Also, MTR (Kathuria *et al*, 1998), MRS (Pretell *et al*, 2005), and 3D constructive interference in steady state; 3DCISS can diagnose scolex in NCC certainty (Robbani *et al*, 2004).

Viable cysts are round, non-enhancing hypodense lesions usually 5 to 20mm in diameter. Cyst begins to degenerate and wall increases in density often accompanied by edema or contrast enhancement. Cyst collapse,

a residual calcified granuloma may be present; calcifications are usually solid, nodular lesions (about 2 to 4mm), and four suckers and hooks scolex in a cystic lesion. Scolices are rounded or elongated bright nodules in the cyst cavity (Guzman and Garcia, 2021).

Serologic tests must confirm cysticercosis suspected patient by Enzyme-linked immunoelectrotransferblot (EITB) with 100% sensitivity (Gekeler *et al,* 2002), or ELISA, electroimmunotransfer blot (western blot) with more 98% sensitivity than ELISA in patients more than one cyst, which dropped to 50% in one cyst (Tsang *et al,* 1989). Monoclonal antibodies-based antigen by ELISA was promising, but poor in patients with viable and/or multiple cysts in subarachnoid space or ventricular system (Garcia *et al,* 2018). The qPCR assay is useful in diagnosing subarachnoid, ventricular NCC and in assessing treatment (O'Connell *et al,* 2020).

Differential diagnosis: NCC meningitis and hydrocephalous are differentiated from chronic meningitis (Thakur and Wilson, 2018), and acquired hydrocephalus such as congenital hydrocephalus (Al-Agroudi *et al,* 2017) or posttraumatic hydrocephalus (Beyerl and Black, 1984). Also, NCC must be differentiated in patients from endemic areas, with headache and epilepsy with calcified lesions imaging and with scolex and cystic changes in subarachnoid, parenchymal and intraventricular space (Lu *et al,* 2022).

In general, treatment must focus on the risky manifestations of disease and/or those with greatest likelihood response to therapy. For example, if a patient has intraventricular lesions with hydrocephalus or intraparenchymal disease, a number of interventions are needed such as shunt, endoscopic removal, anti-inflammatory, anti-parasitic, and anti-seizure therapies (Bergsneider *et al,* 2000). The initial focus must be on obstructive hydrocephalus management that was a contraindication to antiparasitic therapy (Garcia *et al,* 2014a). Albendazole treats neuro-cysticercosis in CNS, and cystic hydatidosis in the liver, lung, peritoneum and others by prevents worm from absorbing glucose, and it loses energy and dies (Mayo Clinic, 2024). Prior to initiate cysticercosis therapy, patients should have an ophthalmologic examination to exclude ocular cysticercosis (Alashry and Morsy, 2021). Inflammations around degenerated ocular cysticerci (mainly with anti-parasitic drug) can threaten vision (Dixon *et al,* 2021). Patients who required prolonged corticosteroids must also be screened for latent TB and strongyloidiasis empiric therapy (Singhi *et al,* 2004). The TB infects many organs and optic neuropathy causes vision loss from nerve infectious infiltration or basal exudates inflammation and endarteritis causes ischemia (Verma *et al,* 2019). The orbital cysticercosis must be differentiated from hydatidosis and surgery was recommended in cysts involved subconjuctiva or eyelid (Dhiman *et al,* 2017).

The initial approach to NCC patients consists of managing acute symptoms; as incresed intracranial pressure by surgical intervention and/or corticosteroids and seizures by anti-seizure drug therapy, if present. Then, a decision must be made regarding anti-parasitic and anti-inflammatory therapy; clinical decisions should be tailored to individual patient circumstances (Webb *et al,* 2018).

Managing elevated intracranial pressure:

Signs and symptoms of elevated intracranial pressure (headache, nausea/vomiting, papilledema, and somnolence) associated with parenchymal disease may occur in setting of diffuse cerebral edema on neuroimaging, where cerebral edema was treated with dexamethasone 0.2 to 0.4mg/kg/day to reduce inflammation (Duffy *et al,* 2014).

Elevated intracranial pressure associated with extraparenchymal disease is obstructive hydrocephalus with intraventricular disease and communicating hydrocephalus with subarachnoid disease (Kelesidis and Tsiodras, 2011): 1- Obstructive hydrocephalus treatment consists of a surgical approach by removal of an obstructing cysticercus or placement of an external ventricular drain or shunt. 2- Communicating hydrocephalus treatment

consists of cerebrospinal fluid diversion by ventriculoperitoneal shunt. Anti-parasitic drug is contraindicated in NCC patients with elevated intracranial pressure signs or symptoms, regardless of the cause. Such patients must be assessed for anti-parasitic drug need after the above mentioned interventions.

Anti-seizure drug therapy: Patients with seizures must be managed with anti-seizure drug, even with a single seizure; NCC lesion serves a nidus for recurrent focal epilepsy (Walton *et al,* 2021). Data on treating seizures in the NCC setting used phenytoin or carbamazepine, but levetiracetam gave same effective and better tolerated, with fewer drug-drug interactions (Kaushal *et al*, 2006).

Epileptic causes are divided into structural, genetic, infectious, metabolic, immune, and unknown: 1- Brain damage from prenatal or prenatal causes (e.g. a loss of oxygen or trauma during birth, low birth weight), 2- Congenital abnormalities or genetic conditions with brain malformations, 3- A severe head injury, 4- A stroke restricts the oxygen amount to brain, and 5- Brain infection as meningitis, encephalitis or neurocysticercosis (Stödberg *et al,* 2020).

Anti-parasitic therapy: Potential benefits of therapy include hastened resolution of active cysts, diminished seizure risk, and reduced recurrent hydrocephalus. The major potential antiparasitic therapy risk is exacerbation of neurologic symptoms caused by inflammation around the degenerating cyst, mainly in patients with so many lesions or elevated intracranial pressure (García *et al,* 2002).

Patient selection: Anti-parasitic therapy is warranted for patients with viable and/or degenerating cysts on neuroimaging (regardless of location). Anti-parasitic therapy must not be given in the following circumstances: 1- Untreated hydrocephalus, 2- High cyst burden (as cysticercal encephalitis), & 3- Presence of calcified lesion(s) only.

Regimen choice: In a randomized trial of 120 patients in Peru with viable parenchymal neurocysticercosis and seizures; patients were treated with antiepileptic drugs and with albendazole (800mg daily for 10 days) plus dexamethasone (6mg daily for 10 days) or double placebo (Garcia *et al,* 2004), followed up for 30 months or until they were six months free after tapering of anti-epileptic drug. Patients in treatment arm had fewer generalized seizures (22 vs. 68; 67% reduction), but neither significant difference in the seizures number nor patients with number seizures (Singh and Murthy, 2010). Besides, randomized trials showed that patients with a single enhancing lesion treated with antiparasitic and anti-inflammatory therapy have slightly shorter duration of seizure risk and time to radiographic resolution than patients not managed with these interventions (Otte *et al*, 2013). However, anti-parasitic therapy didn't appear to affect whether lesion would calcify (Thussu *et al*, 2008).

Antiparasitic drugs for patients with viable and/or degenerating cysts on neuroimaging were albendazole and combined albendazole and praziquantel (Kaur *et al,* 2009), for 10 to 14 days in most cases, and patients with subarachnoid disease warrant an extended antiparasitic drug duration (Garcia *et al,* 2011). Combined albendazole and praziquantel was associated with a radiographic higher rate of resolution than albendazole alone (Garcia *et al,* 2014b).

In a randomized trial of 124 VPN patients treated with standard albendazole (15mg/kg/day), or high-dose (22.5mg/kg/day), or combined therapy with both albendazole (15 mg/kg/day) and praziquantel (50mg/kg/day), complete radiographic resolution was done six months later in patients (68%) with more than two cystic lesions treated with combined therapy compared to 5% treated with standard albendazole and 25% treated with high-dose (Garcia *et al,* 2016). Albendazole for neurocysticercosis as 400mg was given orally twice daily for 8 to 30 day or praziquantel 100mg/kg/day for a day in 3 divided doses, then 50mg/kg/day for 2 to 4 weeks (Campbell and Soman-Faulkner, 2023)

Corticosteroids adjunctive with anti-parasitic drug were associated with fewer seizures

(deGhetaldi *et al*, 1983). The corticosteroids must be given concomitantly with antiparasitic drug (Tuckermann *et al,* 2005). Often given prior to, during, and after neurosurgical procedures to reduce inflammation and brain edema (Vedantam *et al,* 2009). There are no high-quality data on efficacy for the surgical management of IVN, but it was well accepted in other neurologic diseases (Torres-Cor zo *et al,* 2006). But, optimal adjunctive corticosteroid regimen is uncertain; commonly used ones are prednisone (1mg/kg/day) or dexamethasone (0.1mg/kg/day) started at least a day before anti-parasitic drug, continued with anti-parasitic therapy duration, and over a few days followed by a rapid taper (Romo *et al,* 2015). Garcia and Del Brutto (2017) found that cysticidal efficacy, safety, and cyst destruction impact in decreasing seizures led to anti-parasitic drug value in parenchymal brain cysticercosis cleared over risks giving substantive NCC role in causing seizures and epilepsy. Bustos *et al.* (2021) found that 38% of parenchymal cysts calcify post antiparasitic treatment, but some factors were associated with calcification modified to avoid, or to decrease seizure relapse risks. Yasir *et al.* (2024) reported that corticesteroids wrong dose and/or duration and unmindful withdrawal after prolonged treatment can have catastrophic effects and must be described by medical specialties. However, about one third of epileptic people showed drug-resistant seizures (Kwan and Brodie, 2000). Surgery was highly effective and safe for selected patients with treatment-resistant focal epilepsy, but very expensive even in high-income countries (Choi *et al,* 2008).

Specific complications as obstructive hydrocephalus due to intraventricular cyst (Matushita *et al,* 2011): Patients with intraventricular neurocysticercosis (IVN) and hydrocephalus associated with altered mental status or impending herniation should undergo emergent cerebrospinal fluid (CSF) diversion by ventriculostomy or a ventriculoperitoneal shunt placement. Patients with mild or intermittent hydrocephalus symptoms management depends on whether cysticerci were adherent (Psarros *et al,* 2003). In general, intact cysticerci without inflamed may be regarded as non-adherent: 1- For patients with non-adherent IVN in the lateral or third ventricle, cysticerci must be removed endoscopically if feasible (Suri *et al,* 2008). For patients without adherent IVN in fourth ventricle, cysticerci must be removed endoscopically or via an open approach that depends on the surgeon's experience (Bergsneider and Nieto, 2002). In most cases, fourth ventricle is more easily accessed via a suboccipital approach. Most isolated non-adherent IVN cases can be cured by removal without antiparasitic therapy if all cysticerci were removed (Husain *et al,* 2007a). There is no role for preoperative antiparasitic therapy, which can lead to an inflammatory response and reduce the likelihood of successful cyst removal. But, if not all cysticerci were removed, subsequent albendazole 15mg/kg/day in two daily doses up 1200mg/day, with food for 10 to 14 days) and anti-inflammatory therapy is warranted. 2- For adherent IVN patients, treatment consists of CSF diversion by ventriculoperitoneal shunt (rather than cyst removal with increased risk complications), followed by albendazole 15mg/kg /day in two daily doses up 1200mg/day with food for 10 to 14 days and anti-inflammatory therapy (Kelley *et al,* 2002). Shunt placement prior to antiparasitic drug is a must since hydrocephalus precipitation with anti-parasitic drug. Neurocysticercosis is the most common cause of acquired epilepsy and patients with cyst(s) causing obstruction must undergo cyst removal and anti-parasitic and anti-inflammatory drugs, and the NCC is potentially eradicable with proper sanitation, hygiene and animal husbandry are warranted (Singhi, 2011).

Subarachnoid NCC can present with communicating hydrocephalus, meningitis, stroke, or focal neurologic deficits and NCC cases (91%) presented with parenchymal infection, subarachnoid cysts (2%), ventricular cysts (6%), and hydrocephalus (16%) of patients (Shereef *et al,* 2021). NCC cases bet-

ween (1997-2005) from Texas, USA, included 111 patients of whom 60 (54%) had parenchymal disease 22 (20%) with intraventricular involvement, 13(12%) had subarachnoid disease, and 13 (12%) had calcifications only, two patients had hydrocephalus, and one patient developed ocular cysticercosis (Serpa *et al,* 2011).

Subarachnoid neurocysticercosis and communicating hydrocephalus or CSF diversion treatment was ventriculoperitoneal shunt, followed by anti-parasitic & anti-inflammatory corticosteroid drugs (Proaño *et al,* 2001). However, endoscopic removal of subarachnoid cysts was by third ventriculostomy but, the intervention role was controversial causing bleeding (Proaño *et al,* 2009). Hydrocephalus is a serious and common risk in clinical subarachnoid hemorrhage course and continued to vague, various background and circumstances ranged from 6 to 67% (Garton *et al,* 2016).

Subarachnoid cysts didn't respond to typical durations of antiparasitic therapy. Therapeutic options include prolonged administration of albendazole (15mg/kg/day) or combined albendazole; 15mg/kg/day and praziquantel; 50mg/kg/day (Göngora-Rivera *et al,* 2006). Antiparasitic therapy must be continued until radiographic cysticerci resolution, a year or more of treatment may be required. Albendazole treated patients for >14 days warrant monitoring for hepatotoxicity and leukopenia (Husain *et al,* 2007b). CBCs and liver enzymes must be checked weekly during initial therapy month and monthly after. Follow-up neuroimaging is at least every six months to monitor response to therapy.

Concomitant corticosteroids administration is essential, as antiparasitic therapy exacerbates inflammation. A reasonable approach was prednisone (up to 60mg/day), or dexamethasone (up to 12 to 24mg/day), few days before anti-parasitic therapy initiation. Patients required a therapy course more than two weeks; methotrexate was used as a steroid-sparing agent (Mitre *et al,* 2007). Methotrexate doses usually start as 7.5mg weekly and can be increased, using rheumatoid arthritis protocols (Rubio-Romero *et al,* 2024). This antiparasitic drug was supported by a total of 33 patients with subarachnoid cysts at least 5cm in diameter and intracranial hypertension (Torres-Corzo *et al,* 2006). All received albendazole for four weeks; ten patients subsequently received praziquantel for 4 weeks, 17 received a 2nd albendazole course, 3 received a 3rd course, and 1 received a 4th course. After 59 months follow-up, all patients were improved and cysts disappeared or calcified. Of 22 patients with seizures, 11 continued to receive antiepileptic drugs.

Giant subarachnoid cyst: In giant subarachnoid cysticerci (>5 cm in diameter, usually accompanied by cerebral edema and mass effect), management consists of corticosteroids; rarely, surgical decompression was required (Zymberg *et al,* 2003).

Cysticercal encephalitis: Cysticercal encephalitis (diffuse cerebral edema with multiple inflamed cysticerci) is a contraindication for antiparasitic therapy, since enhanced parasite killing can exacerbate patient inflammatory response, led to diffuse cerebral edema and potential transtentorial herniation (Rangel *et al,* 1987). Most cysticercal encephalitis cases cured with high dexamethasone dose of 0.2 to 0.4mg/kg/day. Therapy duration must be individualized based on clinical and radiographic resolution of edema. Most cysticerci cases resolved without antiparasitic drug.

Spinal lesions: Management of spinal neurocysticercosis must be individualized based on symptoms, cysticerci anatomic site, arachnoiditis degree, and surgical expertise with good responses (Alsina *et al,* 2002). Spinal neurocysticercosis with compressed spinal cord and myelopathy (paraparesis or incontinence), high corticosteroids treated dose is warranted, either alone or combined with antiparasitic drug. Spinal NC micro-surgical removal plays an important role if the spinal compression signs led to neurological deficits (Barrie *et al,* 2020).

Ocular lesions: Treatment of intraocular

cysticercosis surgical removal of 45 patients' retinal reattachment was eyes accomplished 22(87%) and 67% recovered ambulatory vision (Sharma *et al,* 2003). Cysticercal extraocular muscles involvement was treated by albendazole15mg/kg/day for 2days up 1200 mg/day, with food for 10 to 14 days & corticosteroids; prednisone 1mg/kg/day or dexamethasone 0.1mg/kg/day (Murthy and Samant, 2008). In a retrospective study of 32 cases with viable extraocular muscle cysticercosis treated with albendazole and steroids, cure rate was in 87% and 13% had residual motility limitation (Sundaram *et al,* 2004).

Extra-central nervous system disease: Generally, management of patients with symptomatic subcutaneous or intramuscular cysticerci consists of nonsteroidal anti-inflammatory medication. Excision is good for symptomatic solitary lesion (Zhao *et al,* 2016), but asymptomatic patients don't require specific therapy. Patients with extraneural cysticercosis must undergo radiographic brain imaging to evaluate for neurocysticercosis and followed up imaging must be every six months followed completion of initial anti-parasitic drug, until no cystic lesion resolution (Gonzalez *et al,* 2021). Patients with persistent viable or enhancing lesions on follow-up imaging warrant a repeat antiparasitic drug course, and optimal antiseizure drug duration is uncertain; many patients continue to have seizures following antiparasitic treatment (Stelzle *et al,* 2022). Recurrent seizures risk factors were residual cystic lesion or calcification on radiographic imaging, breakthrough seizures, and >2 seizures during disease course (Rajsshekhar and Jeyaseelan, 2004). Multiple lesions' patients, anti-seizure therapy must continue for at least 24 months. Tapering and discontinuation of anti-seizure therapy is reasonable for patients with few seizures prior to antiparasitic drug, resolution of cystic lesion(s) on radiographic imaging, and no seizures for 24 consecutive months (Del Brutto, 1994).

Calcified nonviable lesion patients: Calcified parenchymal neurocysticercosis patients, management with neither viable nor enhancing lesions are symptomatic therapy. Calcified lesions without parasites neither antiparasitic nor anti-inflammatory therapy were given, and corticosteroid was not warranted for perilesional edema, but if corticosteroid was tapered or stopped, rebound edema can occur (Garg *et al,* 2018). Patients with seizures warrant antiseizure therapy; in such patients, calcified lesions increase risk of recurrent seizures and chronic epilepsy (Shorvon *et al,* 2011). No anti-seizure drug is warranted for asymptomatic patients. Patients with a single seizure and not recurence within six months, tapering and stopping antiseizure drug was reasonable (Nash *et al,* 2008). Patients with CPN and refractory epilepsy warrant epilepsy subspecialty were evaluated as candidacy for surgical seizure resection.

Neurocysticercosis children may be treated as previously given and dosing must be weight based. Pregnant women with elevated intracranial pressure must be treated and corticesteroids may be used if necessary. Choice of antiseizure therapy should take into account potential teratogenicity and pharmacokinetics in pregnancy, and antiparasitic drug was rarely required urgently and deferred to delivery (Gedzelman and Meador, 2012).

Historically: Adult-onset epilepsy related to a structural CNS disease was traced back to the Hippocratic treatise “on Sacred Disease” (Engel and Pedley, 2008). It was suggested that epilepsy distressed the Roman dictator Gaius Julius Cesar (100-44BC) was due to cysticercosis, as it started when he was 54 years old a year after one of his visits to Egypt and was of partial origin with secondary generalization (McLachlan, 2010). Though, Egyptians didn't eat pork with the exception of one sacred day per year; human taeniasis was well known in the ancient times (Grove, 1990). Li *et al.* (2012) in China added that tapeworms were known by Egyptian and Arabian physicians and treated infection with pumpkin seeds (*Cucurbita pepo*), an herbal medicine used nowadays. Cox (2004) reported that Egyptian Ebers papyrus (1500BC)

gave descriptions of tapeworms, both *Taenia* spp. eggs, and *T. solium* cysticerci were in intestine & stomach of Egyptian mummies. Bruschi *et al.* (2006) described an ancient cysticercosis case in an Egyptian mummy of about 20 years aged woman who lived in the Ptolemaic period (2nd to 1st Centuries B.C.), and that in Hellenistic Egypt, swine farms were present. Tiwari *et al.* (2013) reported that the Indian Nursing History dated back to about 1500 B.C.E., in scriptures of Hindu teaching of the Samhite period (2000e 1100 B.C.E.), Atharva Veda, Sushruta (500 B.C. E.) and Charaka (300 B.C.E.), that led to ayurveda authorities (science of life), its eight parts covered the entire medical field of science, including nursing treatments. More details and descriptions about nursing appear in the old Indian records than in any other country worldwide. Sushruta defined the ideal relations of doctor, nurse, patient, and medicine as four feet upon a cure must rest. Shulman *et al.* (2002) in USA reported that nurses played a significant role in primary NCC prevention and care during treatment and follow-up. Ma *et al*. (2011) in China reported that education was more heavily influenced by models from U.S.A., but Egypt looked to those from Britain and France. Most striking, however, was what they now share. Both countries' systems of nursing education are now clearly located in an increasingly global world of health, and health care recognized that more educated nursing workforce remains the critical component of any initiative to better health care needs.

Most Egyptian people don't consume pig's pork for religious reasons, *T. solium* & *C. cellulosae* were not common (Youssef and Uga, 2014). In Cairo Governmental Abattoir of 6,434,039 animals (1994-1997), individual infection rate was 0.2% in local cattle 7.3% and imported 7.3%, in buffaloes 0.1% and 0.1% in pigs, *C. bovis* & *C. cellulosae* were 0.7% (Haridy *et al,* 1999). In Mansoura City human *T. saginata* was 1.1% (El Shazly *et al,* 2006). Basem *et al.* (2009) in Assiut found that cysticercosis *bovis* was endemic among cattle and buffaloes. Abdel-Hafeez *et al.* (2015) in Minia City among 100 of each cattle, goats, and pigs in governmental abattoir found *C. bovis* (20%) in cattle and *C. cellulosae* (12%) in pigs. ELISA human positive *T. solium*/*C.cellulosae* in Assiut and Sohag Governorates was 8.1% & 3.33% respectively. Fahmy *et al.* (2015) in Qalyoubia G. reported high prevalence rate of bovine cysticercosis and *T. saginata* by the bioinformatic tools. Elkhtam *et al.* (2016) in Menofia reported that the annual cysticercosis losses were 87032 Egyptian Pound among cattle. El-Sayad *et al.* (2021) in North Egypt, found *C. bovis* detected by the meat inspection was overestimated compared with PCR, and that a more specific molecular test could give an accurate diagnosis. El-Dakhly *et al*. (2023) in a retrospective study (2018-2020) showed cycticercosis *bovis* allover Egypt except Luxor and North Sinai. Phylogenetic analysis indicated that they originated from the same global clade of *Taenia saginata* GB isolates. Mansour *et al.* (2023) in Cairo reported an uncommon case of trapped (locked-in) lateral ventricle caused by an isolated cysticercus trapped at ipsilateral foramen of Monro, an atypical location for neurocysticercosis, adding more challenges to diagnosis and during the process of surgical extraction.

Conclusion

Clinical syndromes related to this parasite include neurocysticercosis (NCC) and extraneural cysticercosis. NCC is divided into parenchymal and extraparenchymal forms. Cysticercosis stages include an initial (viable) phase, a degenerating (enhancing) phase, and a nonviable (calcified) phase. Cysticerci may be found in more than one anatomic site with at different stages in their natural history may be present simultaneously.

Before treating cysticercosis, all patients must have an ophthalmologic examination to exclude ocular cysticercosis. Patients who are likely to require prolonged corticesteroids must also undergo screening for latent TB and screening or empiric therapy for strongyloidiasis.

The initial NCC patients approach consists of treating acute symptoms, if present, such as increased intracranial pressure and seizures. Antiparasitic and anti-inflammatory therapies determination must be done as to management and clinical decisions must be tailored to each patient circumstances.

Antiparasitic therapy is warranted for patients with viable and/or degenerated cysts on neuroimaging regardless site. Parasitic drug is not allowed to a patient with hydrocephalus, cysticercal encephalitis, or calcified lesions only. Antiparasitic drug depends on disease burden. Patients with one to two cysts are albendazole treated, but with more than two cysts are albendazole and praziquantel treated for 10 to 14 days; patients with subarachnoid disease warrant an extended antiparasitic therapy duration.

Corticosteroids must be given side by side with antiparasitic therapy, optimal regimen is uncertain; commonly used are prednisone (1mg/kg/day) or dexamethasone (0.1mg/kg/day) begun at least one day prior to antiparasitic therapy, continued for antiparasitic drug duration, and followed by a rapid taper.

Follow-up neuroimaging must be done every six months following completion of antiparasitic therapy, until radiographic resolution. Patients with persistent lesions warrant a repeat course of antiparasitic therapy.

Optimal duration of antiseizure therapy is uncertain; many patients continue having seizures after antiparasitic treatment. For patients with viable and/or multiple lesions, antiseizure drug therapy should continue for at least 24 months. For patients with a single enhancing lesion, antiseizure drug must continue for six months after radiographic resolution of active infection.

Spinal neurocysticercosis treatment must be individually based on symptoms, cysticerci site, arachnoiditis degree, and surgical expertise. Intraocular cysticercosis is surgical removal. Symptomatic subcutaneous or intramuscular cysticerci patient is neither steroidal nor anti-inflammatory treated.

Excision is reasonable for symptomatic sigle lesion. Emphasizing medical compliance is vital and need long-term treatment.

Critical seizure education, such as driving or activity is restricted and follow-up after discharge as well as proper hand washing, sanitary practices, and food processing.

Recommendations

Careful meat inspection, strict hygienic control and treatment of patient to stop infective eggs passage is a must for welfare.

Spine and other organs must be examined to rule out cysticercosis disseminated when multiple infections detected in the brain.

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Table 1: Classification of neurocysticercosis as to location and parasite appearance surrounding host tissue on neuroimaging

Form*	Characteristic on neuroimaging	Histopathology
Parenchymal in brain parenchyma ¶		
Nonviable calcified	Nodular calcifications <20 mm in diameter (1 to 5 mm) with or without edema and/or contrast enhanc-ement.	Calcified granuloma with or without surrounding inflammation and/or gliosis.
Single, small enhancing	Cystic or nodular with lesion <2 cm in size	Single parenchymal parasites in degeneration pro-cess surrounding by inflammation and variable opacification or without cyst fluid.
Viable parenchymal	Vesicular lesions often associated with contrast enha-ncement and/or surrounding edema. Scolex visible on high-definition imaging.	Parasites with intact cyst wall, vesicular fluid, & scolex, with variable inflammation amounts sur-rounding parasite sometimes invading cyst wall.
Extraparenchymal in CNS		
Intraventricul-ar	Cysticerci within ventricles, obstructive hydrocephal-us or loculated hydrocephalus with disproportionate, CT/MRI ventricles dilatation suggestive cysticercus.	Viable cysticercus cyst within ventricle and/or obstructive hydrocephalus.
Subarachnoid	Cysticerci in sylvian fissure, in basilar cisterns, or interhemispheric spaces. Strokes or meningitis with-out discrete cysts.	Cysticerci in subarachnoid space with arachn-oiditis, vasculitis. Often in clusters, with prolifr-ating membranes (racemose), no scolex.
Spinal	Cysticerci within spinal subarachnoid space with or without inflammation/ diffuse spinal arachnoiditis. Intramedullary cysticerci within spinal cord.	Subarachnoid cysticerci often with associated ara-chnoiditis. Intramedullary cysticerci similar patho-logically to parenchymal cysticerci.

CT: computed tomography; MRI: magnetic resonance imaging.

* Patients with more than one form classification found lower on chart, with exception of single enhancing lesions and viable grouped with single enhancing lesions. Ocular cysticercosis classified separately.

¶ Small cysticerci in gyri over cerebral convexity behave clinically like parenchymal cysticerci, grouped with parenchymal cysticerci. Rarely neurocysticercosis with multiple inflamed parenchymal cysticerci with diffuse cerebral edema or cysticercal encephalitis, large parenchymal cysticerci (>20 mm)

Explanation of figures

Fig. 1: Neurocysticercosis an important cause of epilepsy in children.
Fig. 2: Cystiocercosis cellulose in an eyelid.
Fig. 3: Cysticercosis cellulosae multiple subcutaneous nodules in breast and abdomen.
Fig. 4: Cysticercosis cellulosae disseminated to child tongue.
Fig. 5: *Taenia solium* gravid segment produces up to 50,000/segment.
Fig. 6: *Taenia saginata* gravid segment produces up to 100,000/segment.
Fig. 7: Eggs morphologically more or less similar, of *T. saginata* slightly larger and always ovoid, *T. solium* eggs smaller and mostly round, a- Iodine mount, b- MZN stained.