



## Cysticercosis of the Spine: A Review

Siddharth Shah<sup>\*</sup> and Samir Dalvie

P.D. Hinduja Hospital & Medical Research Center, Mumbai, India

<sup>\*</sup>Corresponding author: Siddharth Shah, P.D. Hinduja Hospital & Medical Research Center, G-40, 3<sup>rd</sup> Floor, Sarvodaya Nagar, Mumbai-400004, India, Tel: +91 - 916 727 5533; Fax: +91 -22 -2444 0425; E-mail: siddharth88@gmail.com

Received date: June 02, 2017; Accepted date: July 20, 2017; Published date: July 25, 2017

Copyright: ©2017 Shah S, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### Abstract

Cysticercosis, caused by the helminth *Taenia solium*, is the most common parasitic infection of the central nervous system in humans, most commonly involving the brain. Spinal involvement by the cysticerci is relatively rare. However, the disease is much more common in developing nations with poor sanitation standards, and is also increasingly being reported from developed nations with a high immigrant population. Human beings, being natural definitive hosts for *T. solium*, infection occurs when humans become the accidental intermediate host, either due to consumption of poorly cooked pork or consuming food contaminated with the parasitic eggs via faeco-oral route from a carrier. Spinal cysticercosis usually presents with progressive neurological deficit, myelopathy and often bladder incontinence. Considered a focal manifestation of systemic disease, cysticercosis is an essential differential diagnosis in case of compressive spinal lesions in endemic nations. Diagnosis is accurately established based on clinical assessment, imaging studies, especially MRI being the definitive test for cysticercal lesion, and immunodiagnostic serological tests. Treatment includes medical as well as surgical line of management. Medical treatment using cysticidal anti-helminthic drugs like albendazole and praziquantel, is indicated for stable solitary cysticercal spinal lesions and as a preoperative and postoperative prophylaxis to decrease the parasitic load and prevent recurrence, respectively. Surgical management is indicated in the presence of progressively worsening neurological deficit, when the diagnosis is in doubt and in presence of severe intramedullary or extramedullary spinal cord compression. The outcome of combined modality treatment is generally favourable.

**Keywords:** Cysticercosis; Spinal cord; Spinal; *Taenia solium*; Neurocysticercosis

### Introduction

Parasitic human infections have been known since the times of Egyptian Medicine (3000 to 400 BC) [1]. Corpus hippocratorum, Hippocrates original work, makes a mention of human infestation by parasites. Aristotle (3<sup>rd</sup> century BC) in his treatise, 'The History of Animals', described the presence of cysticerci in the muscles of pigs. It was Plinio (25-79 BC) who named the adult form of the worm *Taenia* (Greek for 'lace' or 'strip') [1]. The earliest documented report of cysticercosis in humans dates back to the 16<sup>th</sup> century, when Paranoli (1550 AD), documented fluid filled vesicles at the autopsy of a man who died of stroke and Rumler (1588 AD) found similar cysts in the duramater of an epileptic patient [2]. Grisinger (1862 AD) analysed 86 patients and established a causal relationship between epilepsy and cerebral cysticercosis, outlining the first classification of the disease [1].

Since then, neurocysticercosis (NCC) has evolved to become the most common parasitic infection of the central nervous system, commonly affecting the brain [3-10]. NCC is endemic in most parts of Asia, India, Mexico, Latin America and Sub-Saharan Africa [2,11-15]. Brazil and India are the countries with the highest prevalence of reported cases [16]. In Brazil as high as 7.3% medical admissions have been attributed to NCC [17]. *Taenia solium* is a porcine parasite, where pigs are the natural intermediate hosts and humans are the natural definitive hosts. Thus, even in the endemic areas the prevalence is higher in areas where pig handling is common and where improper faecal handling and poor sanitary conditions favour humans to become the intermediate hosts [13]. With globalisation and increased

numbers of immigrants from developing nations, NCC is being increasingly reported in the developed nations, particularly where there is significant influx of immigrant population [18-20]. While 1,494 patient with NCC were reported in the United States from 1980-2004 [21], the number rapidly ascended to 18,584 hospitalizations due to NCC from 2003-2012 [22]. Travel to an endemic region also poses a risk factor for contact with the parasite, either by native pork consumption or through contaminated food or water. NCC has also been reported in individuals with no history of pork consumption or travel to any endemic area. The source of infection in such cases is usually a close household contact with a recent travel history or a domestic help from an endemic region, both acting as carriers of *T. solium* in the intestine [23].

### Epidemiology of Spinal Cysticercosis

Spinal Cysticercosis (SCC) is a relatively rare form of presentation of *T. solium* infection as compared to the involvement of brain. The earliest incidence of SCC was reported in 1963 by Canelas, who noted SCC in 2.7% patients with NCC [24]. The reported incidence of SCC varies from 0.7%-5.85% [1,4,5,25,26]. SCC most commonly involves the thoracic spine [27,28], and is most commonly associated with a primary NCC focus in the cranium, in approximately 75% patients (range 30%-100%) [4,5]. Isolated spinal involvement of cysticercosis is extremely rare, occurring in less than 25% patients [8,29]. Improvements in magnetic resonance imaging as well as increased awareness of SCC as a differential diagnosis for a compressive lesion in the spinal cord, especially in the endemic countries, would lead to the increase in the reported incidence of SCC than those mentioned currently [29].

SCC may be further divided, based on the anatomical location of the lesion in the spine as: extraspinal and intraspinal. While the former involves the vertebral body, the latter is further subdivided into extradural, subarachnoid (extramedullary) and intramedullary forms. Subarachnoid form is the most common location for the SCC, accounting for almost 80% of all the SCC cases [4,29]. It is also commonly associated with simultaneous cranial involvement of the NCC, which is thought to be the primary site of lodgement for the parasite, from where the larvae migrate into the spinal cord along the ventricular system and into the subarachnoid space. The intramedullary type is a rare form of SCC [7,9,30] with only 55 cases reported till 2014 [31]. Unlike the extramedullary (subarachnoid) type, it occurs from direct spread of larvae through the bloodstream, similar to intracranial parenchymal type NCC [4,29]. Extradural form of SCC is exceedingly rare, with only a few cases reported in literature [32,33]. While intracranial NCC is much more common, cerebrospinal fluid spread of the cysts can result in a high incidence of SCC; however, SCC is not reported more often in patients with NCC. The plausible hypothesis for this phenomenon include: occurrence of cerebrospinal fluid (CSF) reflux at the craniovertebral junction, which propels the cysts backwards preventing dissemination into the spinal canal [29]; "sieve effect" occurring at the transition between intracranial and intraspinal subarachnoid spaces, where the majority of cysticerci, being larger than the subarachnoid space at the cervical level, are unable to pass through [27]. However, SCC dissemination involving the whole spine cervical to lumbar levels has been reported [5].

## Etiopathogenesis

The life cycle of *T. solium* involves two hosts: pigs and humans. Pigs are the natural intermediate hosts for the larval form cysticercus, while humans are natural definitive hosts for the adult tapeworms, but accidental hosts for the larval form. The adult tapeworm is attached to the mucosa of the small intestine of humans. Gravid proglottids are released into the faeces, which liberate thousands of eggs in the environment. Pigs, coming in contact with the humans faeces, ingest the *T. solium* eggs. In the porcine intestinal wall, the eggs mature, release oncospheres, which cross the intestinal mucosa and are carried by the bloodstream to get lodged in various organs, where they evolve into cysticerci. This development of cysticerci occurs in the porcine tissues. Human consumption of poorly cooked infected porcine meat results in the release of the cysticerci in the small intestine, which attach to the intestinal mucosa and slowly evolve into mature tapeworms. Human cysticercosis occurs when humans act as the intermediate hosts, when the *T. solium* eggs are ingested directly. This occurs either due to ingestion of food contaminated with *T. solium* eggs or direct faeco-oral person-to-person transmission from individuals harbouring the adult tapeworms in their intestines. SCC results when the cysticerci migrate and get lodged in the spinal column. Several possible routes of transmission of the parasite into the spinal cord parenchyma or CSF have been postulated [27,34,35]: 1) Hematopoietic venous route: spread occurs through retrograde blood flow *via* the Bateson's vertebral venous plexus, vertebral segmental veins, intervertebral veins and intercostal veins. 2) Ventriculopendymal route: the dilated ependymal canal allows the cysticercus to migrate from the fourth cerebral ventricle into the spinal cord. 3) Subarachnoid route: although unlikely, theoretically, transpial migration of cysticerci may occur into the parenchyma from the subarachnoid space, explaining the intramedullary form of SC. 4) Contiguous spread: extremely rarely, cysticerci from the intestinal

mucosa may directly penetrate through surrounding tissue layers to reach the vertebral canal, without accessing the bloodstream.

After lodging into the neural tissue, the cysticerci are in the vesicular stage of development. They then pass through other successive stages: colloidal stage, granular stage and the final calcified stage. In the colloidal stage, the cysticerci elicit an intense inflammatory response from the host, with mononuclear infiltration, surrounding astroglial proliferation, edema, perivascular lymphocytic cuffing and neuronal degeneration. In the calcified stage, the edema subsides but the astrocytic gliosis becomes intense and multinucleated giant cells appear surrounding the lesion [36]. Inflammatory reaction ensues against the dead parasite causing perilesional edema, damaging the parenchyma and causing worsening of symptoms [7]. Inflammatory reaction in the subarachnoid space also leads to leptomeningeal thickening, which coupled with exudates of inflammatory cells and parasitic membranes, occludes the CSF flow, leading to obstructive hydrocephalus.

SCC lesions can cause symptoms by one or more pathological mechanisms: firstly, the intense host inflammatory reaction occurring surrounding the lesion leads to acute neuronal dysfunction, presenting with a radiculopathy-like clinical presentation. Secondly, the calcified lesions produce a mass effect onto the spinal cord, producing compressive spinal cord dysfunction. Thirdly, chronic neuronal degeneration occurring along with astroglial and microglial proliferation, produce irreversible cord changes leading. The pathological process producing symptoms would also determine response to treatment. Inflammatory pathology would have a good response to medical management with antihelminthic drugs, along with corticosteroids given to suppress the release of inflammatory mediators. Surgical decompression would benefit the patients whose symptoms arise predominantly from a mass effect due to the cysticercal lesion. The outcome in the case of chronic neuronal degeneration is usually unpredictable and often partial or none, as there is rapid neuronal loss and the spinal cord parenchymal changes are often irreversible.

## Clinical Presentation

The mode of presentation of SCC depends on a number of factors [29,37]: 1) Spinal level of the lesion; 2) Anatomical location (intramedullary/extramedullary/extradural); 3) Size of the lesion; 4) Stage of development of cysticerci; 5) Host immune reaction with inflammation or scarring around the neural structures. The common clinical presentations include pain, progressive weakness, spasticity, bowel and bladder dysfunction and myelopathy [7,16,28,38,39]. Thoracic canal being narrower, the spinal cord affection in this region occurs in the earlier stages of the cysticercal development, with relatively smaller size of the calcified cysticerci causing greater degrees of symptoms. Thoracic level involvement is most commonly presents as progressive myelopathy with patients presenting with lower limb neurological deficit, often with bowel and bladder involvement. In contrast lumbar vertebral canal is greater in diameter and the contents include the conus and cauda equina, thus lesions in this region become symptomatic relatively later, once the lesion grows significantly to compress the cord. Lumbar SCC may present with backpain, radicular symptoms with varying grades of lower limb weakness. Besides the spinal location, the anatomical location of the lesion also influences the duration and presentation of SCC. Smaller intramedullary lesions become symptomatic early, whereas extramedullary lesions may become very large and exhibit relatively late and insidious onset of

symptoms [8,29]. In cases with intramedullary lesions, the most common clinical signs are myelopathy and progressive weakness, induced by spinal cord compression; whereas, back pain and radicular pain are the most common symptoms associated with extramedullary SCC [8]. Inflammation occurring as a consequence of reactive host immune response may produce symptoms of spinal cord and nerve root irritation, and progressive accumulation inflammatory mediators may produce spinal compression. In certain patients of SCC, the neurological symptoms may follow a temporal pattern. Initially they may present with a clinical picture of recurrent meningitis with episodes of intracranial hypertension, associated with hydrocephalus [27]. The spinal symptoms may develop months or years after the cranial symptoms, as a consequence of secondary spread of the cysts.

## Diagnostic Evaluation

Magnetic resonance (MR) imaging is the diagnostic modality of choice for diagnosing cysticercal lesions in the spine. MR imaging provides a highly effective non-invasive modality of imaging since it allows detailed assessment of spinal cord and any pathologic entity, while simultaneously allowing screening of the whole spinal axis to quantify the extent of disease. The lesion of SCC appears as a cystic lesion which is hypointense on T1 weighted images, while on T2 weighted images, it shows high signal intensity due to high protein content within the cyst [7,8]. Surrounding edema and peripheral rim enhancement can be seen on post contrast images [40,41]. The diagnostic sign of cysticercal lesion, although seen infrequently, is the presence of scolex within the cyst cavity, seen on MR imaging as a 'mural nodule', which is hyperintense on T1 image and hypointense on T2 image [40,42-44]. In the presence of calcification in the lesion, computed tomography provides better outline and details of the calcified cyst. The differential diagnosis of an intramedullary cystic lesion includes some other cysts such as arachnoid cyst, ependymal cyst, neurenteric cyst, sarcoidosis, neoplasms such as ependymoma, and infections such as abscess [7].

Immunodiagnostic tests of serum samples have been widely used to confirm the diagnosis of cysticercosis. Some cysticercal surface antigens stimulate formation of antibodies as a part of the host immune response [36]. These antibodies can be assayed, and this forms the basis for immunological testing for diagnosis of cysticercosis. Enzyme linked immunosorbent assay (ELISA) of both, the serum and CSF, is reported to be highly sensitive and specific for diagnosis of SCC [45-48]; however, certain reports claim CSF ELISA to be more accurate than serum; with its sensitivity and specificity for SCC being 87% and 95% respectively [29]. The drawback of immunotesting is the high (50%) false-positive rate seen when SCC occurs purely intramedullary or parenchymal, in solitary cysts or in cysts with calcification alone [8, 36]. Another drawback is the false-positive response in patients who are exposed to adult parasite without developing cysticercosis [49]. Monoclonal antibody based parasitic antigen assays of CSF samples, reflect the presence of live parasites, establish presence of ongoing viable infection and can be used to quantitatively verify successful treatment [49].

## Treatment of Spinal Cysticercosis

### Medical management

Albendazole and Praziquantel are anticysticidal drugs of choice which have been effectively used to treat spinal cysticercosis

[6,17,46,50,51]. Medical therapy is considered imperative as a postoperative prophylaxis to prevent recurrence after surgical excision of the lesion since cysticercosis is considered to be a generalised disease with a focal manifestation. The usual dose of albendazole and praziquantel are 15 mg/kg/day and 50 mg/kg/day respectively, with the usual duration of prophylaxis being 4-6 weeks [7,31]. Albendazole can also be used preoperatively to consolidate the lesion, and thus provide a clear delineation of the cyst during surgery [7]. Albendazole has been found to be superior to praziquantel in trials comparing both the drugs [17,52]. However, Albendazole given in large doses in the presence of viable vesicular cysts, may cause aggravate the inflammatory reaction around the cysts as they degenerate, which could lead to neurological worsening [3]. Thus, corticosteroids are used in the management of cysticercal arachnoiditis, encephalitis and angitis [8]. Steroids control the inflammatory reaction around the cystic lesion as well as prevent deterioration in spinal cord function after treatment. Besides preoperative and postoperative use, medical therapy is indicated independently for the treatment of highly suspected intramedullary SCC, for SCC with stable neurology and for multifocal, unresectable lesions [7,9,30,46,51]. However, the effectiveness of medical cysticidal therapy does not obviate the need for surgical management in symptomatic SCC patients, especially when the symptoms are progressive.

### Surgical therapy

According to the American Society for Microbiology Current Consensus Guidelines for Treatment of Neurocysticercosis, the treatment of the spinal cysticercosis, intra- or extramedullary, is primarily surgical [3]. Surgical treatment is indicated as the definitive mode of treatment of SCC with severe progressive neurological deficit, irrespective of prior medical therapy. Extramedullary lesions warrant surgical treatment with laminectomy and excision of the compressing cystic lesions. In most of the cases the cyst excision is technically simple since the lesions adhere to the parenchyma even in the degenerative stage [27]. However, in certain cases, intense inflammations and arachnoidal scarring may cause adhesion to the neuronal structures, making the dissection and excision difficult; which would then be possible only by meticulous dissection, gentle irrigation and valsalva manoeuvres which help in extirpating adherent cysts [53]. Some cysts may not be completely excised because of the parenchymal adhesions and extensive scarring. The excision of intramedullary lesion can be done after a myelotomy and microsurgical dissection from surrounding parenchyma, although the approach and surgical procedure adds to the morbidity of the patient. The outcome of intramedullary SCC excision is not poor and neurological improvement in upto 85.7% patients has been reported after surgical treatment [26]. Surgery does not provide the expected outcome in all patients since multiple pathogenic mechanisms are responsible for the neurological symptoms in SCC [6]. Surgical treatment is the procedure of choice when the diagnosis is in doubt [7], and should be performed at the earliest, to prevent permanent neurological sequelae. Leptomeningeal inflammation and arachnoiditis may cause obliteration of the subarachnoid space, causing obstruction in CSF flow; it requires treatment with duraplasty to re-establish CSF flow [29]. Upto 50% patients undergoing spinal surgery for SCC experience persistence or recurrence of symptoms due to arachnoid inflammation [3]. Besides the conventional surgical complications, one peculiar complication of surgical cyst excision is cyst rupture during surgical manipulation. Cyst rupture would lead to postoperative inflammation and arachnoiditis, which would lead to postoperative

neurological worsening. This can be effectively minimized by hypertonic saline irrigation during cyst removal, use of black gauze pieces surrounding the cyst wall and perioperative use of steroids [27].

## Conclusion

Spinal cysticercosis is rare form of human infection by the helminth *T. solium*. SCC is endemic in the developing nations with high-risk populations, commonly presenting with radiculopathy, myelopathy or cauda equina syndrome. MR imaging is the investigative modality of choice for diagnosis, with immunodiagnostic tests providing ancillary evidence of infection. Medical management is indicated in preoperative and postoperative prophylaxis and as definite treatment in stable solitary cysts or as salvage therapy in multiple inoperable cysts. Steroids play an important role in controlling inflammatory reaction around the lesion. Surgical resection is indication in presence of progressive neurological deficit, extramedullary compressible cysts and selected intramedullary cysts amenable to resection.

## Conflict of Interest

None.

## References

- Sibat HF, Valdés LFI (2013) Introduction to Cysticercosis and Its Historical Background, Novel Aspects on Cysticercosis and Neurocysticercosis, Prof. Humberto Foyaca Sibat (Ed.), InTech.
- Olivé JI, Angulo-Rivero P (1962) Panel discussion. Cysticercosis of the nervous system. I. Introduction and general aspects. *J Neurosurg* 19: 632-634.
- Bansal S, Suri A, Sharma MC, Kakkar A (2017) Isolated lumbar intradural extra medullary spinal cysticercosis simulating tarlov cyst. *Asian J Neurosurg* 12: 279-282.
- Izci Y, Moftakhar R, Salamat MS, Baskaya MK (2008) Spinal intramedullary cysticercosis of the conus medullaris. *WMJ* 107: 37-39.
- Lim BC, Lee RS, Lim JS, Cho KY (2010) A case of neurocysticercosis in entire spinal level. *J Korean Neurosurg Soc* 48: 371-374.
- Kasliwal MK, Gupta DK, Suri V, Sharma BS, Garg A, et al. (2008) Isolated spinal neurocysticercosis with clinical pleomorphism. *Turk Neurosurg* 18: 294-297.
- Qi B, Ge P, Yang H, Bi C, Li Y, et al. (2011) Spinal intramedullary cysticercosis: a case report and literature review. *Int J Med Sci* 8: 420-423.
- Jang JW, Lee JK, Lee JH, Seo BR, Kim SH, et al. (2010) Recurrent primary spinal subarachnoid neurocysticercosis. *Spine* 35: 172-175.
- Ganesan S, Acharya S, Kalra KL, Chahal R (2015) Intradural Neurocysticercosis of Lumbar Spine: A Case Report. *Global Spine J* 5: 1-4.
- Kim KS, Weinberg PE (1985) Spinal cysticercosis. *Surg Neurol* 24: 80-82.
- Fleury A, Garcá JM, Aguerrebere PV, De Sayve DM, Rodriguez PB, et al. (2010) Neurocysticercosis, a persisting health problem in Mexico. *PLoS Negl Trop Dis* 4: e805.
- Prasad KN, Prasad A, Verma A, Sing AK (2008) Human neurocysticercosis and India Scenario: a review. *J Biosci* 33: 571-582.
- White AC. Epidemiology, transmission, and prevention of cysticercosis.
- Nash TE, Neva FA (1984) Recent advances in the diagnosis and treatment of cerebral cysticercosis. *N Engl J Med* 311: 1492-1496.
- Weatherly NF (1988) Medical helminthology. Joklik WK, Willett HP, Amos DB, et al. *Zinsser Microbiology*, Edn 19, Norwalk, Appleton & Lange 970-996.
- Cárdenas G, Guevara-Silva E, Romero F, Ugalde Y, Bonnet C, et al. (2016) Spinal *Taenia solium* cysticercosis in Mexican and Indian patients: a comparison of 30-year experience in two neurological referral centers and review of literature. *Eur Spine J* 25: 1073-1081.
- Takayanagui OM, Jardim E (1992) Therapy for neurocysticercosis. Comparison between albendazole and praziquantel. *Arch Neurol* 49: 290-294.
- Del la Garza Y, Graviss EA, Daver NG, Gambarin KJ, Shandera WX, et al. (2005) Epidemiology of neurocysticercosis in Houston, Texas. *Am J Trop Med Hyg* 73: 766-770.
- Ong S, Talan DA, Moran GJ, Mower W, Newdow M, et al. (2002) Neurocysticercosis in radiographically imaged seizure patients in U.S. emergency departments. *Emerg Infect Dis* 8: 608-613.
- Sorvillo FJ, DeGiorgio C, Waterman SH (2007) Deaths from cysticercosis, United States. *Emerg Infect Dis* 13: 230-235.
- Wallin MT, Kurtzke JF (2004) Neurocysticercosis in the United States: review of an important emerging infection. *Neurology* 63: 1559-1564.
- O'Neal SE, Flecker RH (2015) Hospitalization frequency and charges for neurocysticercosis, United States, 2003-2012. *Emerg Infect Dis* 21: 969-976.
- Sorvillo F, Wilkins P, Shafr S, Eberhard M (2011) Public health implications of cysticercosis acquired in the United States. *Emerg Infect Dis* 17: 1-6.
- Montano SM, Villaran MV, Ylquimiche L, Figueroa JJ, Rodriguez S, et al. (2005) Neurocysticercosis: association between seizures, serology, and brain CT in rural Peru. *Neurology* 65: 229-234.
- Sheehan JP, Sheehan J, Lopes MB, Jane JA Sr (2002) Intramedullary spinal cysticercosis: case report and review of the literature. *Neurosurg Focus* 12: e10.
- Mohanty A, Venkatrama SK, Das S, Bhabani S, Rao BR, et al. (1997) Spinal intramedullary cysticercosis. *Neurosurg* 40: 82-87.
- Colli BO, Valença MM, Carlotti CG Jr, Machado HR, Assirati JA Jr, et al. (2002) Spinal cord cysticercosis: neurosurgical aspects. *Neurosurg Focus* 12: e9.
- Colli BO, Assirati Júnior JA, Machado HR, Dos Santos F, Takayanagui O, et al. (1994) Cysticercosis of the central nervous system. II. Spinal cysticercosis. *Arq Neuropsiquiatr* 52: 187-199.
- Alsina GA, Johnson JP, McBride DQ, Rhoten PR, Mehringer CM, et al. (2002) Spinal neurocysticercosis. *Neurosurg Focus* 12: e8.
- Ahmad FU, Sharma BS (2007) Treatment of intramedullary spinal cysticercosis: report of 2 cases and review of literature. *Surg Neurol* 67: 74-77.
- Chaurasia RN, Mishra VN, Jaiswal S (2015) Spinal cysticercosis: an unusual presentation. *BMJ Case Rep*.
- Mohanty A, Das S, Kolluri VR, Das BS (1998) Spinal extradural cysticercosis: a case report. *Spinal Cord* 36: 285-287.
- Quiroz LDS, Filho AP, Callegaro D, De Faria LL (1975) Intramedullary cysticercosis: case report, literature review and comments on pathogenesis. *J Neurol Sci* 26: 61-70.
- Rossi LA, Sestari A, Cerioni Jr. M (2006) Intradural-extradural spinal cysticercosis with brain involvement: a case report and literature review. *Radiologia Brasileira* 39: 379-382.
- Rossitti SL, Roth-Vargas AA, Moreira ARS, Sperlescu A, Araujo JFM, et al. (1990) Cisticercose espinhal leptomenígea pura. *Arq Neuropsiquiatr* 48: 366-370.
- Del Brutto OH (2012) Neurocysticercosis: a review. *ScientificWorldJournal* 2012: 159821.
- Sotelo J, Marin C (1987) Hydrocephalus secondary to cysticercotic arachnoiditis A long-term follow-up review of 92 cases. *J Neurosurg* 66: 686-689.
- Cabieses F, Vallenat M, Landa R (1959) Cysticercosis of the spinal cord. *J Neurosurg* 16: 337-341.
- Canelas HM, Ricciardi-Cruz O, Escalante OAD (1963) Cysticercosis of the nervous system: less frequent clinical forms. *Arq Neuropsiquiatr* 21: 77-86.
- Gaur V, Gupta RK, Dev R, Kathuria MK, Husain M, et al. (2000) MR imaging of intramedullary spinal cysticercosis: a report of two cases. *Clin Radiol* 55: 311-314.

41. Parmar H, Shah J, Patwardhan V, Patankar T, Patkar D, et al. (2001) MR imaging in intramedullary cysticercosis. *Neuroradiology* 43: 961-967.
42. Martinez HR, Rangel-Guerra R, Arredondo-Estrada JH, Marfil A, Onofre J, et al. (1995) Medical and surgical treatment in neurocysticercosis a magnetic resonance study of 161 cases. *J Neurol Sci* 130: 25-34.
43. Zee CS, Segall HD, Boswell W, Ahmadi J, Nelson M, et al. (1988) MR imaging of neurocysticercosis. *J Comput Assist Tomogr* 12: 927-934.
44. Palasis S, Drevelengas A (1991) Extramedullary spinal cysticercosis. *Eur J Radiol* 12: 216-218.
45. Rosas N, Sotelo J, Nieto D (1986) ELISA in the diagnosis of neurocysticercosis. *Arch Neurol* 43: 353-356.
46. Corral I, Quereda C, Moreno A, Lopez-Velez R, Martinez-San-Millan J, et al. (1996) Intramedullary cysticercosis cured with drug treatment A case report. *Spine* 21: 2284-2287.
47. Garg RK, Nag D (1998) Intramedullary spinal cysticercosis: response to albendazole: case reports and review of literature. *Spinal Cord* 36: 67-70.
48. Tsang VC, Brand JA, Boyer AE (1989) An enzyme-linked immunoelectrotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis. *J Infect Dis* 159: 50-59.
49. Garcia HH, Del Brutto OH, Nash TE, White AC Jr, Tsang VC, et al. (2005) New concepts in the diagnosis and management of neurocysticercosis (*Taenia solium*). *Am J Trop Med Hyg* 72: 3-9.
50. Hawk MW, Shahlaie K, Kim KD, Theis JS (2005) Neurocysticercosis: a review *Surg Neurol* 63: 123-132.
51. Chhiber SS, Singh B, Bansal P, Pandita KK, Razdan S, et al. (2009) Intramedullary spinal cysticercosis cured with medical therapy: case report and review of literature. *Surg Neurol* 72: 765-768.
52. Sotelo J, Del Brutto OH, Penagos P, Escobedo F, Torres B, et al. (1990) Comparison of therapeutic regimen of anticysticercal drugs for parenchymal brain cysticercosis. *J Neurol* 237: 69-72.
53. Kim SK, Wang KC, Paek SH, Hong KS, Cho BK, et al. (1999) Outcomes of medical treatment of neurocysticercosis: a study of 65 cases in Cheju Island, Korea. *Surg Neurol* 52: 563-569.