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Disseminated cysticercosis incidentally diagnosed in a patient of low backache: A case report and concise review of literature

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ABSTRACT

Disseminated cysticercosis is an uncommon presentation of a common disease. Asymptomatic disseminated cysticercosis is rarely reported in literature. Here, we are reporting a case of asymptomatic disseminated cysticercosis incidentally diagnosed in a patient of low backache. Magnetic resonance imaging of lumbosacral spine and neuroimaging done subsequently during the course of evaluation revealed diffuse cysticercosis involving abdominal, paraspinal, pelvic and gluteal muscles along with neurocysticercosis. Such a disseminated cysticercosis was diagnosed incidentally in this patient of low backache with right sciatica and radiculopathy at L5-S1 prolapsed intervertebral disc and was subsequently managed by L5-S1 interlaminar fenestration and discectomy.

1. Introduction

Taenia solium infestation is of worldwide public-health importance. It is one of the 'neglected tropical diseases'[1,2] and is eradicable, as declared by 2003 World Health Assembly[3]. This infestation may present clinically as 'intestinal taeniasis' due to ingestion of undercooked pork contaminated with cysticerci, or/and as 'tissue cysticercosis' in subcutaneous tissue, muscle, or other tissues/organs due to ingestion of viable eggs released from the worm carriers (taeniasis patients). Disseminated cysticercosis is an uncommon presentation of a common disease. Widespread dissemination of cysticerci throughout the human body was reported by Krishnaswami about a century back in 1912[4]. However, report of such an entity in the literature is conspicuous by its relative rarity[5-11]. There is little information available on the natural course of human cysticercosis[12]. Disseminated cysticercosis is reported to present with a myriad of symptom-complex depending on the organ system(s) involved. However, asymptomatic disseminated cysticercosis, which sheds some light on its natural course, is rarely reported and we are presenting this case to highlight the very same phenomenon.

2. Case report

42 year old male, a security guard, presented to Orthopaedic outpatient department of Neigrihms, Shillong, India had gradual onset low backache (LBA) for 6 months with acute exaggeration and radiation to (R) lower limb for 2 weeks. There was no history of trauma, fever, bone or joint pains, abdominal pain, any swelling or weakness of limbs or loss of weight or appetite. However, he complained of numbness of (R) foot for about one week. Bowel and bladder functions were normal. His higher mental functions were normal. Clinical examination revealed features of (R) sciatica

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with (R) S1 radiculopathy. On blood examination, total white blood cells (WBC) count was 5 200 cells/cmm of blood while differential leucocyte count (DLC) showed polymorphs 66%, lymphocyte 30% and eosinophils 4%. Renal and liver function tests were normal. Radiographs of lumbosacral) (LS) spine revealed L5-S1 disc disease with no signs of mechanical insufficiency. Clinical diagnosis of discogenic LBA including (R) sciatica and (R) S1 radiculopathy was made. A trial of conservative treatment such as bed rest, analgesics and guarded physiotherapy was given, for three weeks. However, it failed to relieve the symptoms, so patient was planned for subsequent evaluation with magnetic resonance imaging (MRI) of LS spine. MRI not only corroborated our clinical diagnosis, but also revealed something completely unexpected. It reported diffuse cysticercosis involving abdominal, paraspinal, pelvic and gluteal muscles (Figure 1). In the light of this new and fortuitous information, patient was reassessed historically and clinically. He was a non-vegetarian, but non pork/beef eating and was hailing from central India but posted in north-eastern region of India, which is endemic for cysticercosis, for last 6 years. There was no history of headache or heaviness of head/vomiting/sleep disturbance/joint or muscle pain/abdomen or chest pain or any eye complaint. On repeated questioning, patient faintly recalled single episode of seizure probably generalized tonic-clonic about 9 years back for which no treatment was taken. Ophthalmologist opinion was taken to rule out any eye involvement. Fundus examination, visual acuity and perimetry were normal. X-rays of the skull and extremities were normal. There was no radiographic evidence of any calcification. 2D echocardiography did not show any cardiac cysticercosis. Electrocardiogram was normal. Microscopic examination of three stool samples over three consecutive days did not show eggs or larvae of *Taenia solium*. Enzyme-linked immunosorbent assay (ELISA) for HIV was non reactive. MRI of brain (Figure 2 & 3) revealed cysticercus cellulosae in various stages of developments (vesicular, colloidal, & granular stages). It showed multiple well-defined variable size cystic lesions, with intramural nodule (scolex) in bilateral cerebral hemispheres, right cerebellar hemisphere, left cerebral peduncle, right hemipons, bilateral lentiform nucleus, left temporalis muscle and left trapezius near the occipital insertion. Few lesions showed surrounding edema. There was no restriction of diffusion in any of the lesions. Contrast study showed nodular and ring enhancement of the lesions. Ventricles and cisternal spaces and sulcogyral pattern were normal. Serum cysticercal antibody ELISA was also positive[13-15]. Definitive diagnosis was finally revised to LBA with (R) sciatica and (R) S1 radiculopathy due to prolapsed intervertebral disc (PIVD) L5-S1 with neurocysticercosis (NCC) and disseminated cysticercosis. The treatment of anti-convulsant regimen and (R) L5-S1 interlaminar fenestration and discectomy was done. Anti-cysticercal therapy was started in the post-operative period. Patient was given prednisolone 1 mg/kg of body weight one week prior to the initiation of albendazole therapy which was given at a dose of 15 mg/kg/day and continued for one month.

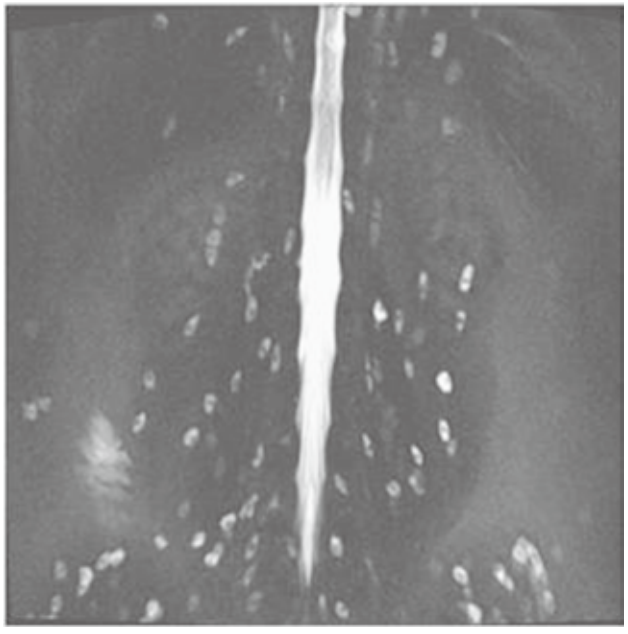

Figure 1. MR myelography with multiple cysts involving abdominal, paraspinal, pelvic and gluteal muscles.

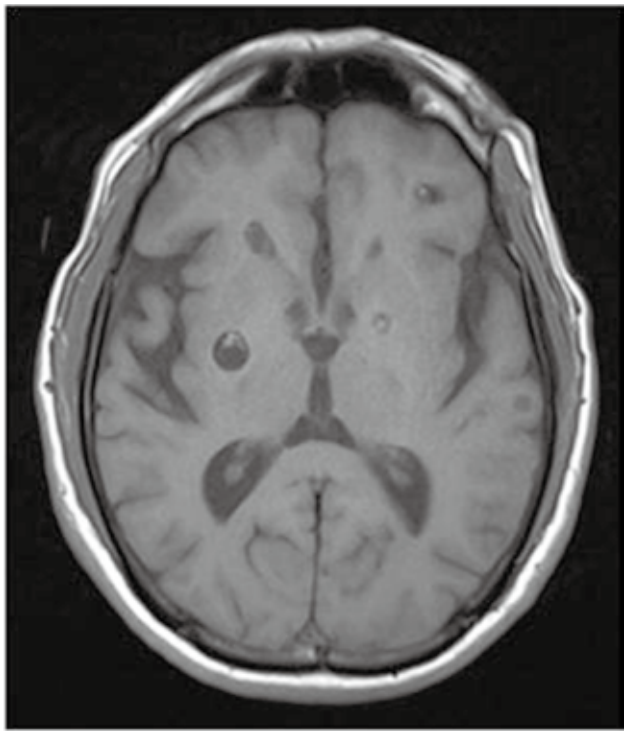

Figure 2. MRI transverse T1W1 showing multiple vesicular forms.

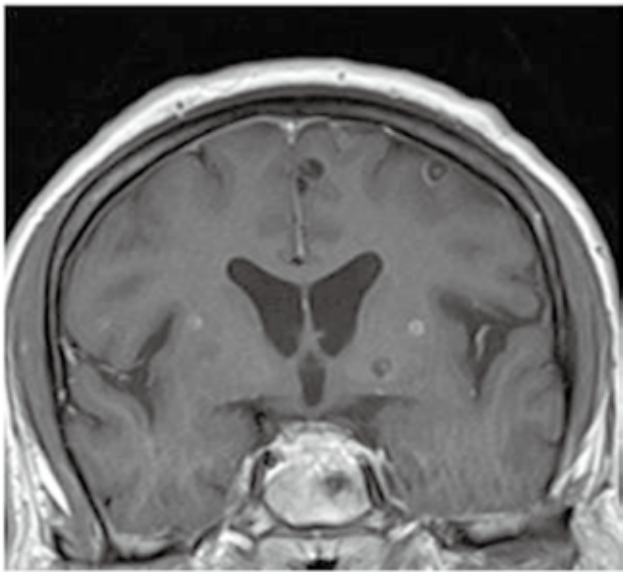

Figure 3. MRI coronal T1W1 with contrast showing colloidal cysticercosis with surrounding oedema.

3. Discussion

Life cycle of *Taenia solium* comprises two natural hosts, with generally human beings as the definite (harbors the adult tapeworm) and swine as the intermediate hosts. Intestinal taeniasis occurs when humans eat poorly cooked pork meat with living cysticerci (larvae) which develop into adult worm in the small intestine, become attached to intestinal wall and start liberating gravid proglottides in faeces. Eggs released from proglottides ingested by swine and man develops into hexacanth embryo (oncosphere) in the small intestine. Human cysticercosis is caused by the dissemination of hexacanth embryos from the intestine via the hepatoportal system to the systemic circulation and further to different organs of the host. In the tissues, oncosphere evolve into cysticerci or larval cysts. These cysts are rapidly destroyed by the host's immune system in most circumstances, except for those located in immunologically privileged sites like nervous system *etc*[16]. Cysticercosis is caused by pork-tapeworm, *Taenia solium*, but no association between pork consumption and human cysticercosis was found[17]. Sensitivity of stool examinations is poor and only about 15% of patients harbour a tapeworm at the time of diagnosis of NCC[18]. Parasite may get lodged in the central nervous system resulting in NCC[19]. The larval stage may also infest other tissues like skeletal muscle, diaphragm, heart and peritoneum, pleura and subcutaneous tissue[20]. Widespread dissemination of the cysticerci can result in the involvement of almost any organ of the body. The clinical symptoms of cysticercosis are protean, and basically reflect involvement of the affected organ(s). NCC is the most common form reported in literature with the brain parenchyma being the commonest site, followed by meninges, ventricles, eye and spinal cord. The presence of viable, living cysticerci in the central nervous system usually does not cause symptoms[21-23]. In contrast, inflammation around degenerating cysticerci may have severe consequences, including focal encephalitis, edema, and vasculitis. The manifestations of NCC are polymorphic, but most commonly presents with convulsion[14,24,25]. NCC is a leading cause of preventable epilepsy responsible for 30% of adult onset epilepsy in endemic regions[26]. Many recent review papers have put this figure around staggering 70%-90 %[27]. Ophthalmic cysticercosis may manifest with diplopia, proptosis, decreased visual acuity or loss of vision. Pulmonary and cardiac involvement by cysticercus is rare. Cardiac lesion can cause arrhythmia. Cysts in muscles may manifest as muscular pain, localized muscular swelling[28-31], weakness or pseudo-hypertrophy. Muscular pseudohypertrophy is a rare presentation due to heavy infection of the skeletal muscles, which gives the patient a "herculean appearance"[32]. Subcutaneous cysticercosis is frequently asymptomatic but may present as palpable nodules. It has been reported that *Taenia solium* cysticercosis in Asia manifests as both NCC and subcutaneous cysticercosis, but in America and Africa it manifests as NCC without subcutaneous cysticercosis[33,34]. The definitive diagnosis of NCC is made by combination of neuro-imaging procedures, immunological investigations and histological techniques because the use of any single method may give erroneous result[35]. However, invasive procedures are rarely required for establishing diagnosis[36]. A set of diagnostic criteria was proposed in 1996[14] and revised in 2001[13], based on objective clinical, imaging, immunological, and epidemiological data; these criteria consist of categories that are stratified according to their diagnostic strength. Detection of anticysticercal antibodies in serum has been reported to be a major criterion for the diagnosis of NCC[13]. More recently, Rodriguez *et al* reported that serum was better than cerebrospinal fluid for detection of specific antibody whereas, helminthic antigen was more easily detected in cerebrospinal fluid, though it was also detectable in serum. The antigen titer showed better correlation with the number of viable cysticerci[37]. Antigen detection has been reported to be an important investigation for follow-up of response to therapy[38]. Yesenia Castillo *et al* recently reported the diagnostic performance of urine antigen detection using the B158C11-B60H8 MoAbbased Ag-ELISA and ascertained 92% sensitivity of urine antigen detection for NCC with viable parasites which decreased to 62.5% in patients with a single cyst and negative assay with only calcified NCC[39]. Computed tomography (CT) and MRI have greatly augmented the accuracy in the diagnosis of NCC[40,41]. Neuroimaging studies delineate 4 stages of cyst formation. In the vesicular stage, neurocysticerci have the appearance of a clear fluid cyst with mural nodule representing the scolex, which represents one of the absolute criteria for diagnosis. Gradually continuing host's immune response may lead to disintegration of larva. Scolex starts showing early sign of degeneration with development of ring enhancing ovoid lesion and surrounding oedema, which is described as colloidal stage. The next stage is granular stage, characterized by thick capsule with some surrounding oedema. Calcified stage is the last stage. Our patient showed vesicular, colloidal and granular stages, but no calcification noted. MRI is considered the best neuroimaging tool for radiological diagnosis of NCC. The advantage of MRI is that it can differentiate the stages of the parasite, which CT fails

to do. Moreover, MRI with gradient echo sequence phase imaging has been reported as good as CT for the detection of the scolex in cystic lesions and also the calcified stage of the parasite[42]. A failure to see the scolex in neuroimaging studies should prompt a search for alternative diagnoses. Central nervous system tuberculosis remains the single most important differential diagnosis in developing countries. One may be confronted with enigmatic diagnostic workup in countries where both are endemic[43]. Other granulomatous central nervous system diseases such as histoplasmosis, sarcoidosis, fungal infections, toxoplasmosis and primary central nervous system neoplasias or metastatic tumors should be considered as well[44]. Asymptomatic subcutaneous or intramuscular cysticerci do not require treatment[45]. Management of NCC includes antiparasitic drugs, surgery, anti-seizure prophylaxis and symptomatic medication. The potential efficacy of antihelminthic therapy in patients with neurocysticercosis is controversial but more recently treatment with anticysticercal drugs is favored, which results in better resolution of single enhancing lesions, but antihelminthic therapy and steroids do not affect the development of calcification and risk of chronic epilepsy[46-48]. Recently two articles reported rapid resolution of multiple viable cysticerci with antihelminthic therapy but only 30%-50% of lesions showed resolution[47,49]. Praziquantel and albendazole are effective antiparasitic drugs against *Taenia solium* cysticerci. Recently, a critical review concluded that albendazole is more effective than praziquantel regarding clinically important outcomes in patients with neurocysticercosis[50]. Albendazole has been used in a 15-30 mg/kg doses for 15, 28-30 and even 60 days[51-54]. Involution of cysticercus has been found to be more significant in the groups treated with higher doses of albendazole[55]. More recently, Singhi *et al* in a prospective, randomized, double blind study with 122 children with neurocysticercosis, concluded that 1 week treatment was as effective as the treatment for 28 days[56]. In our patient, we used 15 mg/kg dose for 30 days. It has recently been proposed that intraparenchymal calcifications, which are ominous and epileptogenic, can be reversed by calcium chelation with bisphosphonates[57]. Two to five days after antiparasitic therapy, there is usually an exacerbation of neurological symptoms, attributed to local inflammation due to the death of the larvae. For this reason, both albendazole and praziquantel are generally given simultaneously with steroids in order to control the edema and intracranial hypertension that may occur as a result of therapy. The role of surgical therapy in the management of neurocysticercosis has significantly decreased over time and is now mainly restricted to placement of ventricular shunts for hydrocephalus secondary to neurocysticercosis. Seizures secondary to neurocysticercosis usually respond well to first-line antiepileptic[34].

Conflict of interest statement

We declare that we have no conflict of interest.

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