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Disseminated Cysticercosis with Tombstones in Portal and Splenic vein Thrombus: An Unusually Novel Location

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Keywords: *cysticerci; disseminated; portal vein; thrombus.*

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Disseminated Cysticercosis with Tombstones in Portal and Splenic vein Thrombus: An Unusually Novel Location

Shruti Thakur ^α, Vijay Thakur ^σ, Neeti Aggarwal ^ρ, Charu S Thakur ^ω & Minhaj Shaikh [¥]

Abstract- Disseminated cysticercosis is a rare complication characterized by extensive dissemination of larvae (*Cysticercus cellulosae*) of *Taenia solium* throughout the human body involving brain, subcutaneous tissue, skeletal muscles and other organs. The symptoms depend upon the parasite burden, its location, stage of cyst evolution and host immunity. We report a case of immunocompetent 47-year-old alcoholic male who presented with symptoms of chronic liver disease. Disseminated cysticercosis was an incidental finding. The emphasis is on the presence of calcified cysticerci in portal and splenic vein thrombus not yet described in literature and this makes our case worth mentioning.

Keywords: *cysticerci*; *disseminated*; *portal vein*; *thrombus*.

I. INTRODUCTION

Cysticercosis is caused by pork tapeworm; *Taenia solium* a common tropical parasite [1]. Disseminated cysticercosis is a rare manifestation that occurs due to widespread dissemination of its larvae throughout human body. We present a case of disseminated cysticercosis with unusual location of cysticerci in portal and splenic vein thrombus not described earlier.

II. CASE REPORT

A 47-year-old immunocompetent alcoholic Indian male presented with symptoms of chronic liver disease. He had a single episode of generalized tonic-clonic seizure 15 years back. His family history was insignificant. The hematologic reports revealed anemia and deranged liver functions. The patient was subjected to contrast enhanced computed tomography (CECT) abdomen. CECT was done on GE light speed Xte 64 slice machine with 100 ml of non-ionic iodinated contrast agent in portal venous phase. The imaging revealed cirrhotic liver, portal and splenic vein thrombosis, multiple collaterals, ascites and massive splenomegaly consistent with the diagnosis of cirrhosis with portal hypertension (Figure 1A, B, 2A). A noteworthy finding was presence of multiple rice grain like specks of calcification in bilateral psoas muscles, gluteal muscles, anterior abdominal muscles, paraspinal muscles and

skeletal muscles of proximal lower limbs. Multiple small calcifications were also seen in spleen. The imaging suggested the diagnosis of disseminated cysticercosis, India being endemic for this disease (Figure 1 A-D, 2 A, B). The very unusual location of these larvae in our case was in portal and splenic vein. The portal and splenic vein were distended with hypodense thrombus; a usual complication of cirrhosis, in which calcified larvae were seen (Figure 1B, 2A). Such a finding has not been described in literature yet. Although the scanned muscles were extensively infested but pseudohypertrophy of these muscles, a diagnostic hallmark of this condition was not seen because of muscular atrophy in this alcoholic male. Subcutaneous nodules another hallmark, were not palpable in our patient as interestingly the larvae were not seen in subcutaneous fat on imaging. The stool examination was done retrospectively for ova of *T. solium* which was negative. CT brain was also done which also showed calcified nodules (Figure 3). High resolution CT chest was done that showed findings of interstitial lung disease, however no nodule was seen. The muscle biopsy was not done as the diagnosis was not questionable. The patient was put on steroids for a week and albendazole was added thereafter. No antiepileptic was given as he had no seizures. Treatment for chronic liver disease and anticoagulant were started. The patient improved and was discharged and is on regular follow up on outpatient basis.

III. DISCUSSION

Human cysticercosis is a common zoonotic infection in tropical regions like South-East Asia, Africa and Latin America [2]. The causative agent is *Cysticercus cellulosae*, the larval form of *Taenia solium*; a pork tapeworm [3]. The infestation is seen in underprivileged society having poor sanitation [4]. The life cycle consists of a definite host (man; harboring adult tape worm in small intestine) and an intermediate host (pig; harboring larval form). Two distinct types of infection are seen in man; intestinal taeniasis and tissue cysticercosis. In 'intestinal taeniasis' man acts as a definite host and gets infected by eating undercooked pork infested by living cysticerci larvae. These larvae develop into adult tapeworm in human small intestine and the eggs are released in faeces. In 'tissue

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cysticercosis', man is an accidental intermediate host and procures infection by eating viable eggs from ingestion of faecally contaminated water or food or by autoinfection. These eggs develop into hexacanth embryos in small intestine. The embryos enter the systemic circulation from intestine through hepatoportal route and get disseminated in various tissues and organs where they develop into larval cysts [5]. In most instances, these larvae are destroyed by host's immunity except where body organs are devoid of immune response like nervous system [4]. The most common location is brain parenchyma where the infection is referred to as neurocysticercosis. In addition, the larval stage may infest any tissue or organ of body like eye, subcutaneous fat, skeletal muscles, diaphragm, heart, pleura, lungs, liver and peritoneum [4, 6]. The organs reported to be rarely involved are pancreas and spleen [3]. The constellation of symptoms depends upon the parasite burden, its stage of evolution, the site of lodgment and host immunity [5, 6]. The syndrome of disseminated cysticercosis is defined by pseudomuscular hypertrophy, palpable subcutaneous nodules, seizures, abnormal mentation, and relative absence of focal neurological deficit or obviously raised intracranial pressure, atleast until late in the disease [6]. The muscle involvement can be of three types- myalgic, nodular and pseudohypertrophy. The latter presentation is rarest of the three and gives a 'Herculean appearance' to the patient [3]. It signifies heavy parasite burden in muscles [6]. The exact pathogenesis of such a presentation is unclear but thought to be either an allergenic response or inflammatory response to irritant effect of dead larvae in muscles [6]. The muscle involvement is characterized by painless or painful diffuse involvement of muscles along with weakness and easy fatigability [7]. CECT is the imaging modality of choice for disseminated cysticercosis while magnetic resonance imaging (MRI) is appropriate for brain, intraocular and spinal cysticercosis that better delineates various stages of larval development. The treatment is albendazole or praziquantel both of which are cysticidal drugs. The advantage of albendazole is its better penetration into CSF for treating neurocysticercosis. Steroids are added beforehand as these cidal drugs may cite severe inflammatory response [2].

In our case, portal and splenic vein were distended and filled with thrombus; predisposing the lodgment of cysticercal larvae in this unusual location. Our case presents the extensive sites of lodgment of cysticercal larvae. The portal and splenic vein involvement is not described in literature to the best of our knowledge.

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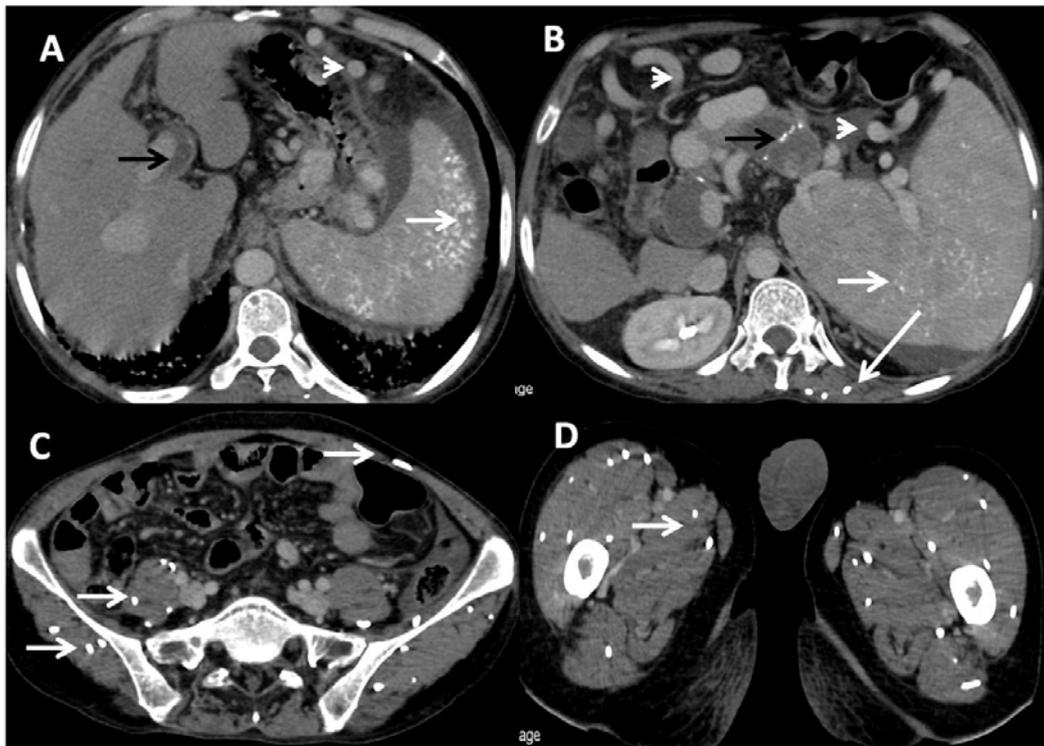


Figure 1 : Contrast enhanced computed tomography (CECT) axial images show (**Figure 1 A, B**) shrunken liver with widened ligament falciparum fissure. The spleen is massively enlarged and shows multiple small specks of calcification (white arrows). The portal vein and splenic vein are distended with thrombus in which foci of calcification are evident (black arrows). Multiple collateral vessels are also seen (white arrow head). (**Figure 1 C, D**) - Rice grain like calcifications are seen in bilateral psoas, gluteal muscles, paraspinal muscles and thigh muscles



Figure 2 : Contrast enhanced computed tomography (CECT) coronal images show (**Figure 2A**) cirrhotic liver, thrombosed portal and splenic vein with calcified cysticercal larvae within it (black arrows), splenic enlargement with multiple calcifications (white arrows). **Figure 2B** shows calcified larvae in paraspinal muscles and gluteal muscles

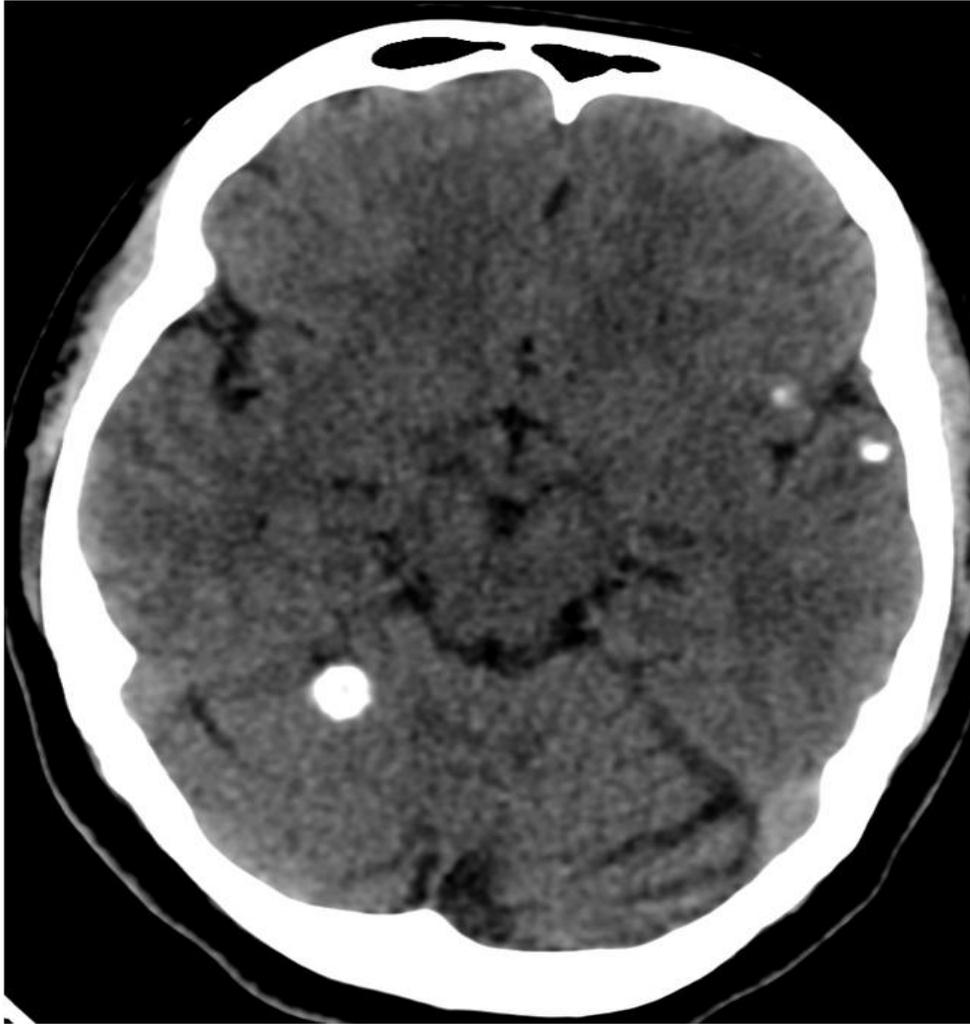


Figure 3 : Plain CT brain axial image show calcified nodules in right cerebellar hemisphere and left frontal and temporal lobes

