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Case Report

# A Case of Psychosis with Multiple Intra-Axial Temporal Lobe Calcifications

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## CASE REPORT

A 69 year old man married with 4 adult children 3 sons and a daughter presented to the emergency department exhibiting "bizarre behaviour" complaining that certain devils are bothering him and that he sees them, saying things to him, abusing him and have been touching his head, looking through his eyes and holding his tongue. History of being diagnosed with Schizophrenia 40 year ago on multiple antipsychotics and exhibits involuntary movements of his tongue suggestive of oro-bucco-lingual dyskinesia. Pt is a retired navy officer who served with the navy for 15 years and admitted to eating pork when in service. A physical exam was normal although patient also presented with cognitive deficits. Cognitive testing using the brief Kingston Standardized Cognitive Assessment Revised showed patient's baseline measurement score was above average and post-test scores after a two week period indicate a mild decline as scores remain above the 96 percentile. Component scores shared (Table 1). Results indicate mild degree of decline mainly in the areas for memory in immediate and delayed recall.

Table 1: pre and post-test component scores using brief kingston standardized cognitive assessment revised.

| SUB-TESTS | Pre-test August 20, 2019 | Post-test September 6, 2019 |
|---|---|---|
| 1. Orientation | 10/10 | 10/10 |
| 2. Word Recall | 7/10 | 6/10 |
| 3. Abstract Thought Score | 8/8 | 8/8 |
| 4. Spatial Reversal | 5/5 | 5/5 |
| 5. Clock Test | 6/7 | 5/7 |
| 6. Perseveration | 2/2 | 2/2 |
| 7. Delayed Recall | 7/10 | 4/10 |
| 8. Recognition | 8.5/10 | 8.5/10 |
| TOTAL SCORE /62 | 53.5 | 48.5 |
| Compared to Normals | 87.5 Percentile – Above Avg | 37.5 Percentile –Average |
| Degree of decline | | >96 percentile – mild decline |

Laboratory tests showed mild leukocytosis 13.3 (normal 3.5- 11x10 9/l) without eosinophilia which on testing and a MRI work up revealed bi-lateral intra axial calcifications. An unenhanced computed tomography scan of the head and subsequent magnetic resonance imaging scan of the brain showed no intra- or extra-axial mass hemorrhage. There were 5 or 6 scattered bilateral supratentorial and infratentorial intra-axial punctate calcifications, most being 2 mm in size. The largest could be seen

to measure 4 mm in the high posterior parietal lobe (Figure 1) on the right, 3 mm in the anterior right frontal lobe (Figure 2) and 3 mm in the posterior right temporal lobe (Figure 3). There was no associated edema or mass effect. There was no hydrocephalus. There was no midline shift or herniation. There was no evidence for acute cortical infarction. There is was no cerebral edema. There was mild diffuse atrophy. There was mild chronic microangiopathic ischemic change in the supratentorial white matter. There was no encephalomalacia. An extra-axial right frontal 7 mm calcification (Figure 4) is either a currently insignificant completely calcified meningioma or alternatively just a dystrophic dural calcification, regardless of no significance currently. There was no significant resultant mass effect or edema. The visualized portions of the paranasal sinuses and mastoid air cells were essentially clear. As the patient was from a region that is endemic for Koch's (Tuberculosis) a work up that included hematology, tuberculin test and chest X-ray was negative (Figure 5).

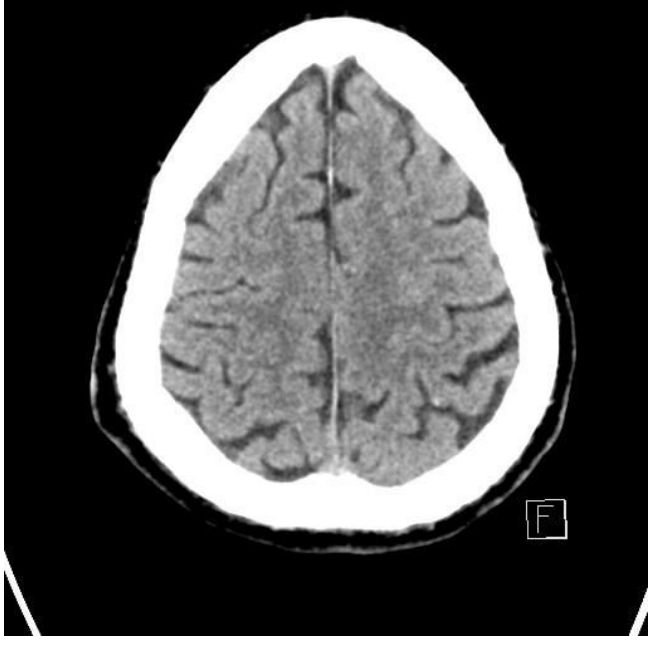

Figure 1

## KEY POINTS

- Neurocysticercosis is commonly resulted by the ingestion of Taenia solium eggs after consuming undercooked pork, or contaminated water.
- The parasite can grow in the brain and spinal cord within the nervous system.
- Patients present with severe headache and seizures along with other manifestations of the disease that include psychosis, stroke, dementia, hydrocephalus and vision loss.
- Therapeutic measures include antiparasitic drugs, surgery, and symptomatic medication.

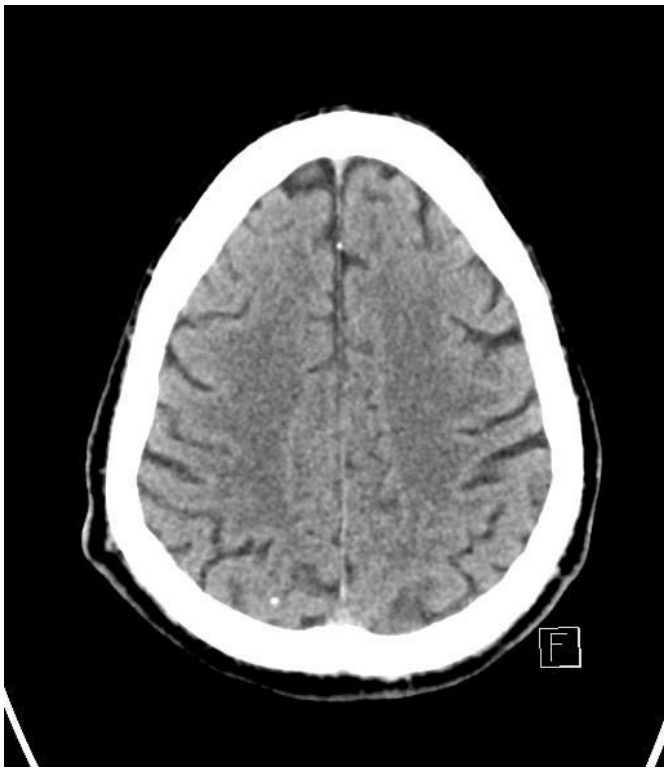

Figure 2

## DISCUSSION

All patients coming to the ED with signs and symptoms of acute psychosis must undergo a careful diagnostic workup. Apart from non-organic psychiatric syndromes themselves, the differential diagnosis must include physical trauma, drugs and toxins, organ failure (e.g., renal failure), structural lesions like intracranial hematomas or neoplasms, infections and nutritional deficiencies like vitamin B12 deficiency or pellagra [1].

Brain calcifications or granulomas which represent the most frequently observed feature in neurocysticercosis are also common in tuberculosis, sarcoidosis and toxoplasmosis [2] . Dementia syndrome observed in patients with neurocysticercosis could be a combined effect, resulting from multiple parasitic and vascular lesions, disrupting frontal-parietal-temporal networks related to intellectual functioning in patients with vulnerable brains (because of repeated epileptic seizures, low educational levels, advanced age) [3]. 80% of symptomatic



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cases present with epilepsy as the most common manifestation of neurocysticercosis. Other manifestations include focal neurological deficits (16%), increased intracranial pressure (12%), and cognitive decline (5%) [4]. Neurocysticercosis is the most common parasitic disease of the central nervous system, and also one of the most common causes of seizures in endemic areas. Globalization has caused the disease to spread around the world beyond the endemic regions. With no specific clinical symptoms of the disease, medical imaging plays an important role in the diagnosis of neurocysticercosis [5].

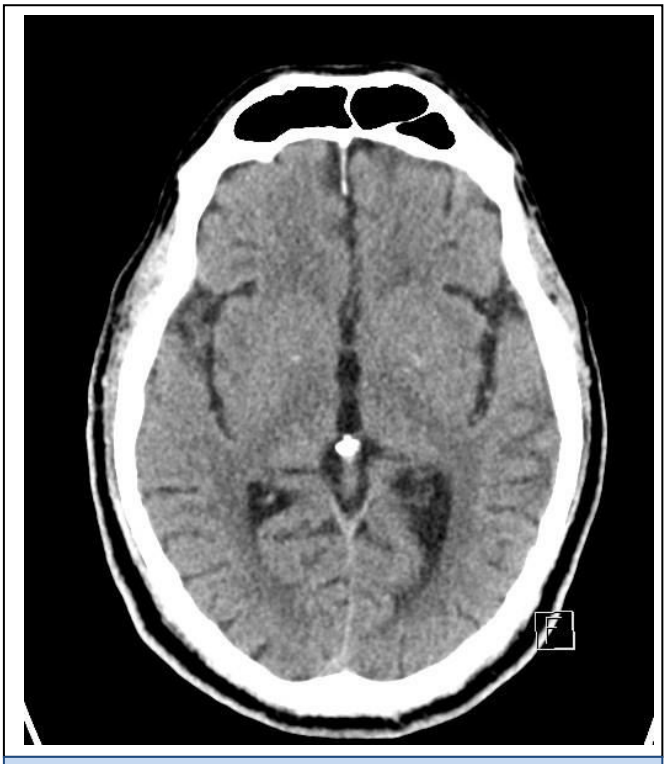

Figure 3

Adult Taenia solium tapeworms infect only human beings (the definitive host). Humans become infected with these tapeworms by eating raw or poorly cooked pork products containing viable T. solium cysts the encysted larval stage of the T. solium. Cysticercosis is a tissue infection by the encysted larval stage of the

T. solium [6]. Although pigs are the main intermediate hosts of the T. solium, humans can also be infested by the larval stage of this tapeworm through the so called fecal-oral transmission or autoinfection pathways [7]. When the Central Nervous System (CNS) of humans (the most common place) is involved by the encysted larva, it is known as neurocysticercosis. Neurocysticercosis is the most common parasitic disease of the CNS, and also one of the most common causes of seizures. Neurocysticercosis most commonly manifests in the parenchyma of the brain and typically involves the cerebral hemispheres. Basal ganglia, brainstem, and cerebellum can also be involved. The lesions are commonly found at the gray white matter junction, presumably resulting from deposition of the larvae in terminal small vessels of these regions [8]. However, some authors mentioned that the "parenchymal" location of parasites as described at cross-sectional imaging actually represented subarachnoid cysticercosis located in deep sulci or in perforating branches of perivascular spaces [9]. But, in most of the current clinical and radiologic studies, parenchymal disease is still considered to be a separate and distinct form of neurocysticercosis. Escobar's four pathological stages, depicting the natural evolution of neurocysticercosis, have been well correlated with advanced imaging findings (CT and MRI) [9,10].

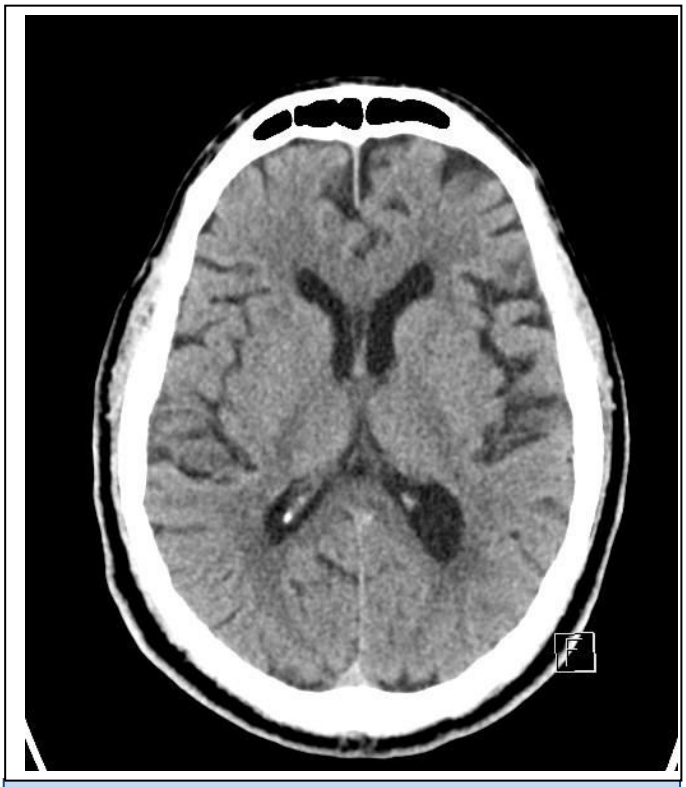

Figure 4

(i) Vesicular stage (active)

(ii) Colloidal vesicular stage (active)

(iii) Granular nodular stage (active)

(iv) Nodular calcified stage (nonactive)

Our patient's imaging findings were consistent with the nodular calcified stage. In this final stage, the lesion usually shrinks to



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one-half or one quarter of its original size, and is almost completely mineralized, with no surrounding edema. CT, better than MRI, can clearly depict the calcified nodule. As the patient only had calcified lesions, there was no role for antiparasitic agents because the cysts are already dead and underwent a routine course of antipsychotics medications.

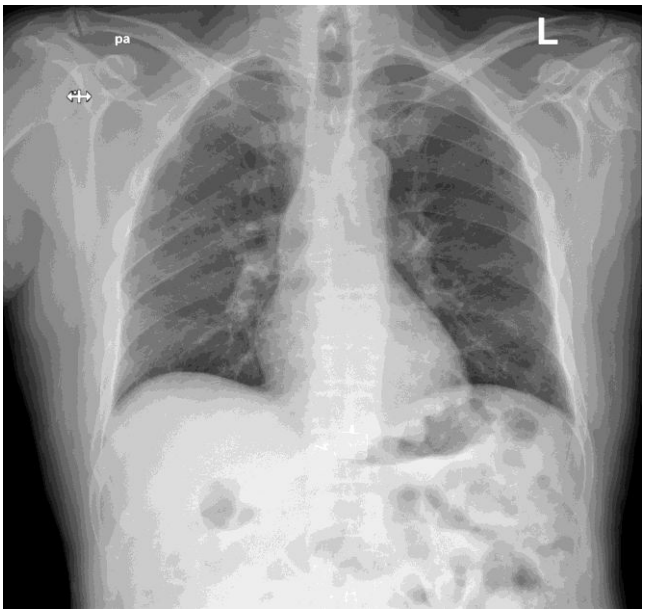


Figure 5

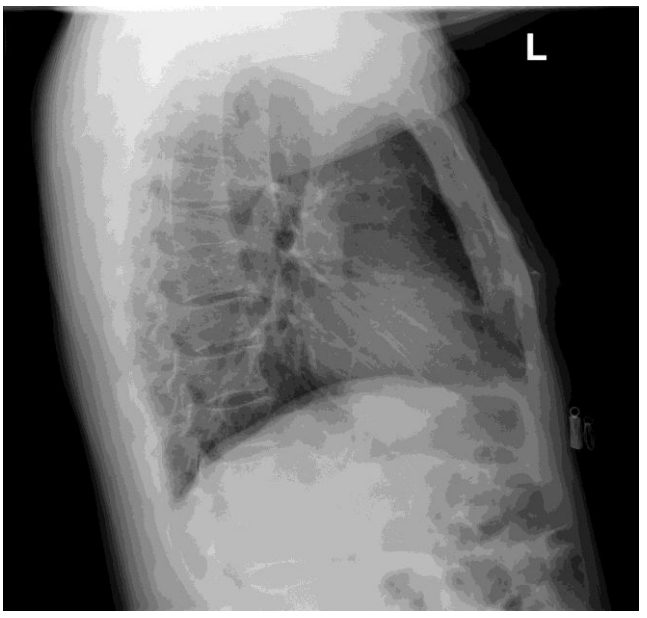


Figure 6

## REFERENCES

1. Xavier M, Correa B, Coromina M, Canas N, Guimarães J. (2005). Sudden psychotic episode probably due to meningo encephalitis and Chlamydia pneumoniae acute infection. Clin Pract Epidemiol Ment Health. 1: 15.
2. Carabin H, Ndimubanzi PC, Budke CM, Nguyen H, Qian Y, et al. (2011). Clinical Manifestations Associated with Neurocysticercosis: A Systematic Review. PLoS Negl Trop Dis. 5: e1152.
3. Ramirez-Bermudez J, Higuera J, Sosa AL, Lopez-Meza E, Lopez-Gomez M, et al. (2005). Is dementia reversible in patients with neurocysticercosis? J Neurol Neurosurg Psychiatry. 76: 1164-1166.
4. Garcia HH, Nash TE, Del Brutto OH. (2014). Clinical symptoms, diagnosis, and treatment of neurocysticercosis. Lancet Neurol. 13: 1202-1215.
5. Zhao JL, Lerner A, Shu Z, Gao XJ, Zee CS. (2015). Imaging spectrum of neurocysticercosis. Radiology of Infectious Diseases. 1: 94-102.
6. Nash TE, Garcia HH. (2011). Diagnosis and treatment of neurocysticercosis. Nat Rev Neurol. 7: 584-594.
7. Garcia HH, Del Brutto OH. (2000). Taenia solium cysticercosis. Infect Dis Clin N. 14: 97-119.
8. Lerner A, Shiroishi MS, Zee CS, Law M, Go JL. (2012). Imaging of neurocysticercosis. Neuroimaging Clin N Am. 22: 659-676.
9. Kimura-Hayama ET, Higuera JA, Corona-Cedillo R, Chavez-Macias L, Perochena A, et al. (2010). Neurocysticercosis: radiologicpathologic correlation. Radiographics. 30: 1705-1719.
10. Zee CS, Go JL, Kim PE, DiGiorgio CM. (2000). Imaging of neurocysticercosis. Neuroimaging Clin N Am. 10: 391-407.

## Appendix

Collected : 30/09/19 1215 Doctor :

Received : 30/09/19 1320 Spec # : 3009:H00044R

Comments : Campus: BCH

**Department of Hematology**

| CBC WITH DIFF | | | | | |
|---|---|---|---|---|---|
| > LKC | | 13.3 | H | 3.5-11.0 X10 9/L | BC |
| > HGB | | 100 | L | 120-175 g/L | BC |
| > HCT | | 0.28 | L | 0.35-0.52 L/L | BC |
| > PLTS | | 332 | | 140-400 X10 9/L | BC |
| > ERC | | 3.4 | L | 3.8-5.8 X10 12/L | BC |
| > MCV | | 83 | | 78-98 fl | BC |
| > MCH | | 29 | | 27-34 pg | BC |
| > MCHC | | 355 | | 320-360 g/L | BC |
| > RDW CV | | 12.7 | | 11.8-15.3 % | BC |
| > ABS NEUT | | 11.2 | H | 1.5-7.5 X10 9/L | BC |
| > ABS IMM GRAN | | 0.06 | | < 0.10 X10 9/L | BC |
| > ABS LYMPH | | 1.0 | L | 1.1-3.9 X10 9/L | BC |
| > ABS MONO | | 1.1 | H | 0.1-0.8 X10 9/L | BC |
| > ABS EOS | | 0.0 | | 0.0-0.6 X10 9/L | BC |
| > ABS BASO | | 0.0 | | 0.0-0.2 X10 9/L | BC |
| > ABS NRBC | | 0.0 | | < 0.1 X10 9/L | BC |