

Case Report

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Neuronal Migration Disorder Mimicking Tuberculosis

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Abstract

A previously healthy 20-year-old patient presents to the ER with a tonic-clonic seizure. His siblings had been diagnosed with pulmonary tuberculosis, one of them within the previous six months. His chest x-ray shows an image suggestive of tuberculosis but CT scan and lumbar puncture did not supported CNS infection. A contrast-enhanced MRI was then performed, showing pachygyria and absence of corpus callosum, establishing the diagnosis as a neuronal migration disorder.

Case Report

A previously healthy 20-year-old man presents to the ER with a tonic-clonic seizure. His past medical history is not contributory except by the fact he has lived in the highlands of Peru during his childhood. His family history reveals that he has 3 brothers with pulmonary tuberculosis; being one of them recently diagnosed 6 months ago. No abnormalities were found in his physical exam. A chest X-ray and a non-contrast cerebral CT scan were performed (Figures 1 and 2). A suggestive image of pulmonary tuberculosis was obtained in the chest X-ray but acid-fast bacilli sputum smears were negative repeatedly. Cerebral CT scan showed asymmetry of the posterior horns of lateral ventricles, and non-accurate delimitation of the white matter along the frontal and parietal right hemispheres. Since these exams were not conclusive, a lumbar puncture and a contrast-enhanced cerebral MRI (Figures 3 and 4) were performed. Cerebrospinal fluid was free of acid-fast bacilli, had normal adenosine deaminase levels and a negative Neurocysticercosis western blot assay. Contrast-enhanced cerebral MRI showed thickening in the cortex at the right inter-hemispheric

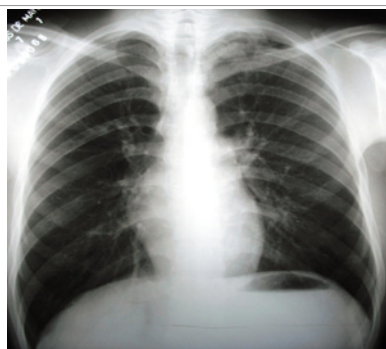


Figure 1: Chest X-ray shows an increased opacity at the left apical lung zone.

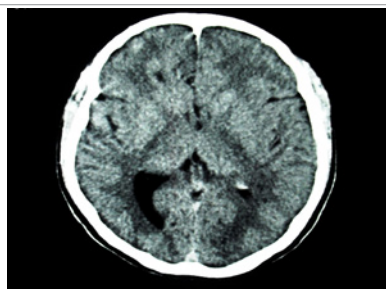


Figure 2: Non-contrast cerebral CT scan shows asymmetry of the posterior horns of lateral ventricles, and non-accurate delimitation of the white matter along the frontal and parietal right hemispheres..

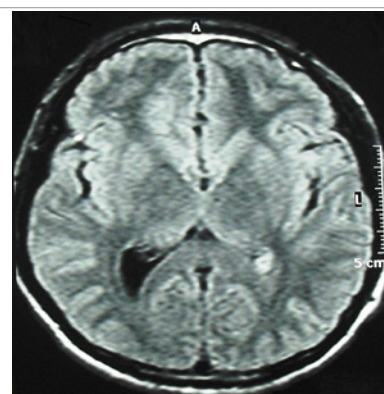


Figure 3: Contrast-enhanced cerebral MRI shows thickening in the cortex at the right inter-hemispheric sulcus and lack of the corpus callosum.

sulcus, a thick circumvolution at the left hemisphere, compatible with pachygyria and absence of the corpus callosum, finally establishing the diagnosis as a neuronal migration disorder. The patient was discharged with phenytoin prophylaxis. The chest image was cataloged as a tuberculosis scar, probably from a previous infection.

Discussion

This patient presented to the ER with a single seizure and no previous history of neurological disease. A vascular disorder was considered primarily in the ER due to the sudden presentation, so a non-contrast cerebral CT scan was executed. Since there were no signs of warning, he was admitted to the internal medicine ward. His past medical history was analyzed and directed our possibilities towards a convulsive disorder secondary to an infectious etiology, such as CNS tuberculosis or neurocysticercosis. As the first entity has different manifestations, including *Tuberculous meningoencephalitis* (the most frequent), and masses like tuberculomas (most frequent parenchymatous manifestation, usually presenting with seizures) and abscesses [1,2], we decided to perform a lumbar puncture. Moreover,

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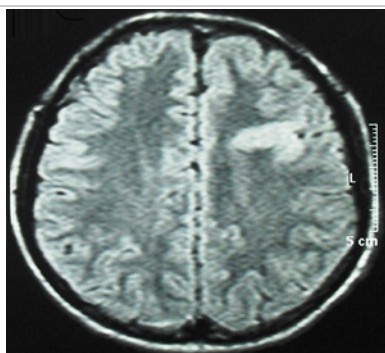


Figure 4: Contrast-enhanced cerebral MRI shows a thick circumvolution at the left hemisphere, compatible with pachygyria.

neurocysticercosis in Peru is highly prevalent; serological and imaging studies in people with seizures living in endemic zones of this country have found that 30 to 50% of these cases are associated with neurocysticercosis [3,4], so a Western blot assay for neurocysticercosis was performed in cerebrospinal fluid.

No meningeal compromise or acid-fast bacilli were found at the cerebrospinal fluid. Western-blot assay for neurocysticercosis was also negative. The chest X-ray showed a possible infiltration compatible with tuberculosis and that was the main reason for which further imaging was needed in order to definitively rule out a tuberculoma as the main cause of the convulsive disorder.

A cerebral contrast-enhanced MRI demonstrated pachygyria and absence of the corpus callosum, a neurological entity considered into neuronal migration disorders. These disorders are not common, and its origin is embryological (12th to 24th weeks of gestation), after dorsal and ventral induction and neuronal proliferation. It is possible that genetic and environmental factors play a role in the development of these disorders: some mutations have been identified and it was reported that viral infections, maternal diabetes, ethanol and drugs use can influence this process [5].

Classification of these disorders is based on morphological criteria

and includes schizencephaly, porencephaly, lissencephaly, argyria, macrogyria, pachygyria, microgyria, and polymicrogyria [5]. The classic manifestation of these disorders is epilepsy, which presents in childhood and is usually intractable [6].

The absence or agenesis of corpus callosum is among the most frequent human brain malformations, reaching an incidence of 0.5 to 70 in 10000. Its origin is heterogeneous, but several genetic causes have been identified, as monogenic syndromes or complex chromosomal disorders [7]. Manifestations involve visual impairments, delay in motor milestones, and alterations in inter-hemispheric transfer of tactual-motor learning when a spatial component is involved [8].

This case report presents an uncommon manifestation of these disorders, especially taking into account the patient's family history and risk to develop tuberculosis.

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