

# Tuberculous Encephalitis Associated With Basal Ganglia Lesions And Movement Disorders

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## Resumen

Un número creciente de desórdenes autoinmunes que afectan predominantemente los ganglios basales, provocando trastornos del movimiento y síntomas psiquiátricos, han sido descritos. Reportamos el caso de un paciente de 26 años de edad, quien tres semanas después de iniciado el tratamiento para meningitis tuberculosa, presentó de manera aguda, hemichorea-balismo derecho y confusión. Una semana más tarde el paciente presentó hemiparkinsonismo izquierdo agudo. El líquido cefalorraquídeo mostró bandas oligoclonales y la IRM lesiones bilaterales en los ganglios basales en secuencias T1W y FLAIR. Conjuntamente con la terapia antituberculosa el paciente recibió levodopa y esteroides mostrando mejoría clínica. A nuestro conocimiento este es el primer reporte de encefalopatía tuberculosa asociada con compromiso de ganglios basales y trastornos del movimiento. Este caso sugiere la implicación de los ganglios basales a través de una patología inmunológicamente mediada.

**Palabras clave:** encefalitis tuberculosa, parkinsonismo, corea, lesión de ganglios basales, patogénesis

## Abstract

An increasing number of autoimmune disorders with predominant involvement of the basal ganglia which result in movement disorders and psychiatric symptoms have been described. We report a 26 year old patient who, three weeks after initiation of treatment for tuberculous meningitis, presented with acute right hemichorea-ballism and confusion. One week later the patient presented acute left hemiparkinsonism. The CSF showed oligoclonal bands. The MRI showed bilateral lesions in the basal ganglia in the T1W and FLAIR sequences. Antituberculous therapy with concomitant steroids and L dopa treatment resulted in clinical improvement. To our knowledge this is the first report of tuberculous encephalopathy associated with involvement of the basal ganglia and movement disorders. This case suggests involvement of the basal ganglia through an immune mediated pathogenesis.

**Keywords:** tuberculous encephalitis, parkinsonism, corea, basal ganglia lesion, pathogenesis

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## Background

There is circumstantial evidence that immune/hypersensitivity reaction mechanisms are involved in tuberculosis.<sup>1-3</sup> An immune-mediated causal association between pulmonary tuberculosis and demyelinating syndromes has been suggested.<sup>1</sup> The paradoxical response to antituberculosis treatment may be the result of an immune reaction or hypersensitivity caused by lysing of mycobacterium or related to treatment.<sup>4,5</sup> Over the past few years, it has been recognized that there are autoimmune or immune-mediated post-infectious disorders that predominantly or in isolation affect the basal ganglia and typically appear with neuropsychiatric and movement disorders.<sup>6,7</sup> We describe a 26-year-old male who, after receiving a three-week treatment for tuberculous meningitis, presents neuropsychiatric and movement disorders, with lesions in the basal ganglia.

## Case Report

A 26-year-old male patient, with no prior medical history, was admitted to hospital two weeks after presenting an intermittent night-time fever, asthenia, headache, general discomfort and vomiting. On admission he has fever of 38.5 C, episodes of agitation, intense diaphoresis and left hemiparesis. The CSF exam showed a WBC count of 222, with 98% predominance of lymphocytes, 20 mg/dl of glucose and 98.5 mg/dl of proteins. Brain MRI with and without gadolinium was within normal limits. Chest CT and X-ray were normal. The Mantoux test and acid fast bacilli testing in CSF were positive. The patient was diagnosed tuberculous meningitis and treatment began with rifampicin, isoniazid, and pyrazinamide. Two weeks later the patient was without fever and ambulatory with 4/5 left hemiparesis. Serological testing for human immunodeficiency virus (HIV) and dengue infection

were negative. CSF culture was negative. Three weeks after antituberculosis treatment started, the patient presented acute right hemichorea-hemiballismus. On neurological examination, the patient was confused and, the following days, he had a disorder in his sleep-wake cycle, with cardiac arrhythmia and unstable blood pressure, with episodes of aggressiveness, psychosis, obsessive-compulsive disorder and emotional lability. CSF testing showed a WBC count of 130, 70% lymphocytes, 30 mg/dl glucose and 72 mg/dl proteins. Blood and urine tests were within normal limits. One week later, the patient presented with left hemiparkinsonism with rigidity, bradykinesia and intermittent resting tremor. T1W and FLAIR MRI images of the brain showed bilateral basal ganglia lesions (Figure 1A). In the CSF testing positive oligoclonal bands were present. The EEG showed diffuse slow theta and delta activity. Treatment began with 1 gram/day of methylprednisolone for five days and afterwards 80 mg/day of prednisone, which was gradually suspended over four weeks. He had a score of 36 in the UPDRS motor assessment. We started treatment with levodopa-carbidopa until reaching 750 mg/day, leading to progressive improvement of his hemiparkinsonism. The patient can walk with assistance, although he remains inattentive and apathetic, with difficulties in terms of memory skills, calculation, reading and writing and with a limited understanding of simple verbal commands. The hemichorea improved significantly without any pharmacological treatment. Two months after being admitted to hospital, when the patient was stable, he obtained a score of 15 in the mini-mental state examination (MMSE). He was discharged was still taking L-dopa and anti-tuberculosis medication

In the ensuing months, he improved gradually and progressively, although he continued to be apathetic. The CSF checkup six months after starting treatment showed WBC count of 30, 49 mg/dl glucose, and 62.2 mg/dl proteins. After completing nine months of treatment, the CSF showed a WBC count of 20, 42 mg/dl glucose, and 45 mg/dl proteins. Twelve months after antituberculo-

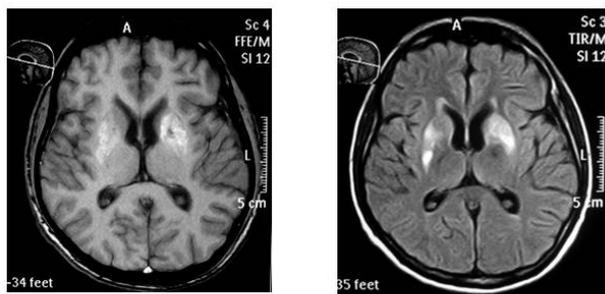
sis treatment, the patient presented mild right hemichorea, without any left motor impairment with a motor score of 18 on the UPDRS scale and CSF within normal limits. An MRI taken 18 months after starting antituberculosis treatment yielded T1 and T2W images showing central frontal atrophy and basal ganglia encephalomalacia (Figure 1B). An assessment with neuropsychologist tests showed scores of 22/30 in the MMSE, 15/32 in the MMP, 13 for depression and 9 for anxiety. The EEG carried out at 9 and 12 months was within normal limits. The patient completed 12 months of antituberculosis treatment and 18 months of L-dopa. Six months after levodopa treatment, the patient was asymptomatic.

### Discussion

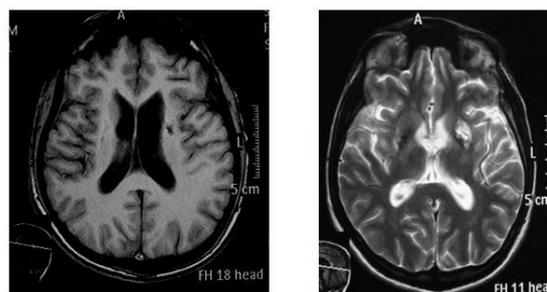
Encephalitis associated with movement disorders due to basal ganglia involvement can be due to infection.<sup>7</sup> Encephalitis involving the basal ganglia includes autoimmune or immune-mediated clinical syndromes that predominantly or solely affect the basal ganglia and typically present with movement disorders and neuropsychiatric disease.<sup>6</sup> It has been suggested that there is an immune-mediated pathogenic association between pulmonary tuberculosis and white brain matter diseases.<sup>1</sup>

Autoimmune basal ganglia encephalitis is associated with movement disorders, sleep disturbance, dysautonomia and neuropsychiatric sequelae following an infectious prodrome.<sup>7</sup> It has been suggested that basal ganglia encephalitis may be autoimmune, because of its postinfectious onset, response to steroids, presence of oligoclonal bands in CSF and negative CSF testing for infectious agents.<sup>7</sup>

Our patient had severe hypersomnolence with disorders of the sleep-wake cycle and dysautonomia with labile average blood pressure and diaphoresis. In addition, he presented psychosis and obsessive-compulsive symptoms with emotional lability, hemichorea-ballismus and contralateral hemiparkinsonism. The MRI yielded symmetric basal ganglia hyperintensity on T2 and fluid attenuated inversion recovery sequences, which are the characteristic



**Figure 1A.** T1W and FLAIR MR images in a 20-year-old patient with acute encephalopathy, right hemichorea and left hemiparkinsonism, show bilateral basal ganglia lesions, secondary to Tuberculous Encephalitis.



**Figure 1B.** T1 and T2W MR images 18 months after antituberculous treatment, show central frontal atrophy and basal ganglia encephalomalacia.

features of autoimmune basal ganglia encephalitis. The good response to therapy with methylprednisolone, postinfectious onset, selective damage to the basal ganglia, presence of neuropsychiatric and movement disorders, and spontaneous remission with partial recovery of cognitive functions, along with the MRI taken 18 months after anti tuberculosis treatment started showing bifrontal atrophy and atrophy of the basal ganglia, suggest that the present case could be similar to those reported as autoimmune basal ganglia.<sup>7</sup> However the presence of inflammatory CSF with positive mycobacterium tuberculosis, acute onset delayed after initial clinical improvement as a result of anti tuberculosis treatment, may suggest that the pathogenesis of this case could be immune-mediated. In the present case, allergic or delayed hypersensitivity owing to mediated cellular immunity induced by the tuberculoprotein or lysis of the mycobacterium<sup>1</sup> could be the pathogenic mechanism that triggered the cerebral lesion.<sup>1</sup> A wide range of hypoxia/ischemia, infection toxin and antituberculosis drug-related factors of the infection may contribute to this pathological reaction in the brain.<sup>2,4,5</sup>

### Conclusion

Our case suggests that there is a heterogeneous group of clinical pathological entities with multifactor pathogenic mechanisms that may manifest themselves after systemic tuberculosis or CNS infection.

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