

## Mini Review

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# Good Response to Thiamine in Bilateral Thalamic Infarction Simulating a Wernicke Syndrome: Does it Has a Role in Acute Stroke?

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## Abstract

The classic clinical triad of Bilateral Thalamic Infarction is consciousness compromise, ocular motility disturbances, and cognitive deterioration; and would be an obligatory differential diagnosis of Wernicke's Syndrome, which usually has, as clinical findings, altered mental status, ataxic gait and ophthalmoplegia. While Wernicke's Syndrome is frequently associated with alcohol intake, it is known that there are some cases not related to alcohol consumption, these subtypes are called atypical non-alcoholic Wernicke's Syndrome and are provoked by malnutrition as their most important etiology. Clinical case: female patient, admitted to our hospital with sudden installation of postural instability, tendency to drowsiness, nausea, dysarthria and provoked-type confabulation, initially diagnosed as an atypical non-alcoholic Wernicke's Syndrome, with a first cerebral tomography without alterations and good response to thiamine infusion. In a second image study, a Bilateral Thalamic Infarction was evidenced. According to many reviews, the use of thiamine in acute stroke is not useful; and would be recommended in the neuro-rehabilitation phase. The clinical regression of symptoms and signs, in our patient, would be the natural history of some series of patients with Bilateral Thalamic Infarction described in the literature; but, we propose that it could also be explained in a bilateral thalamic dysfunction previously described in Wernicke's Syndrome, and its fast response to thiamine use; so it would be interesting publishing more clinical reports or trials using thiamine in this specific type of stroke.

**Keywords:** Bilateral Thalamic Infarction; Wernicke Syndrome; Thiamine; Acute Stroke

## Introduction

The Wernicke Syndrome (WS) is a serious and potentially lethal neuropsychiatric disease [1], of acute onset, caused by a deficiency of vitamin B1(thiamine); with an estimated prevalence between 0.4% and 2.8% [1,2,4]. It is characterized by ophthalmoplegia, ataxia and compromised consciousness [1-3]; this classic triad is observed only in 16%-33% of patients; and is more frequently observed in alcoholics patients [2]. About 19% of patients do not

have any of these symptoms initially. The most frequent symptom is the alteration of mental state, that is present in 34%-82% of all cases, and is explained by the compromise of the reticular system at thalamic nuclei or mammillary bodies level; and is followed by the alteration of the ocular motility that is due to a lesion of the pontine tegmentum [2]. On the other hand, the ataxia can be explained by the compromise of the cerebellar vermis and vestibular alteration

[2]. The diagnosis of WE is clinical, so it requires a high degree of suspicion [1].

Alcoholism is described as the main cause, but only accounts for 50% of cases. Non-alcoholic WS cases have atypical symptoms, leading to late or under-diagnosis [1]; and is mainly related to malnutrition, chemotherapy, hyperemesis gravidarum, gastrointestinal surgeries, dialysis, parenteral nutrition, thyroid pathology, and psychiatric disorders such as anorexia. The mainstay of treatment is the soon administration of thiamine [1]; and, since diagnostic confirmation of WS can often be difficult and ever late, when WS is suspected, treatment should be started early [2]. This consideration is due to the fact that the administration of thiamine is safe, inexpensive, and the prompt supplementation prevents the progression of WS to irreversible deficits, leading a rapid improvement of symptoms, especially alterations in mental status, and ocular motility [1,2]. On WS, the cerebral tomography (CT) can show hypodense areas in the periaqueductal gray matter, and the medial region of the thalamus; but in most cases, there are no alterations in the acute phase of WS. On the other hand, magnetic resonance imaging (MRI) is highly specific and has great value in the diagnostic confirmation of WS, showing a symmetric and bilateral hyperintensity at the paraventricular level of the thalamus, hypothalamus, mammillary bodies, periaqueductal region and floor of the fourth ventricle [1,2].

One of the rarest types of stroke is bilateral thalamic infarction (BTI); with an estimated incidence of 0.6% of all strokes, and between 22%-35% of all thalamic strokes [5]; and is associated with an anatomical variant in the cerebral posterior irrigation, named Percheron's artery [5,6]; and has some usual symptoms like depression of wakefulness, amnesia, confabulation, aphasia (if

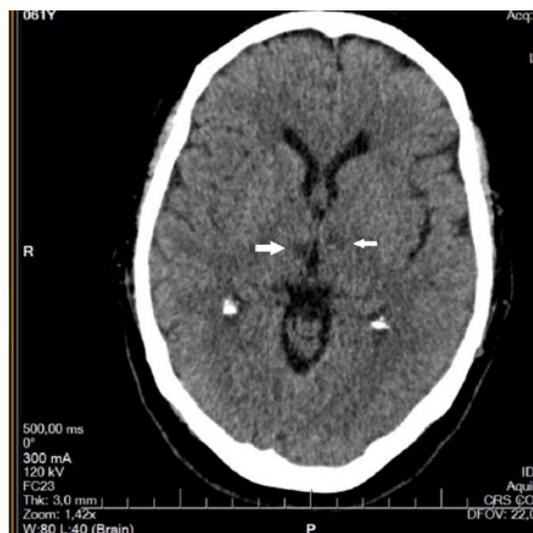
lesion is in the dominant hemisphere), apathy and agitation, among others [6]; and also paralysis of ocular motility [5]. The natural evolution of BTI is very diverse; showing cases of spontaneous resolution of symptoms in hours to days [5], secondary dementia [7,8] and death [5].

### Case Report

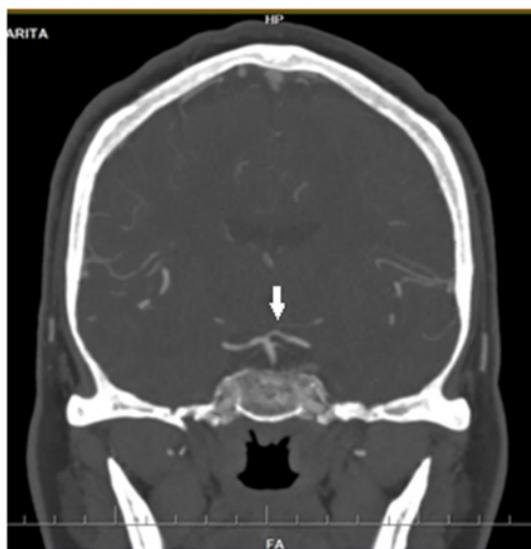
Female patient, 61 years old, active smoker, with diagnosis of chronic depression, and Rankin Scale at 0. The patient consults to our hospital due to 24 hours of sudden onset of postural instability, tendency to drowsiness, nausea and dysarthria; without history of headache or fever. At emergency room (ER), the blood pressure, glycemic value, and other biochemical parameters were normal. An electrocardiogram was performed that did not show alterations. The neurological examination is described like alertness with tendency to drowsiness, unable to invert automatic series; dysarthria, provoked type confabulation, pupillary anisocoria reactive to light, limitation of vertical gaze without nystagmus; and bilateral dysmetria of great intensity to right side. At ER, a therapeutic test with flumazenil was made, because a benzodiazepine poisoning was suspected; but without response. A CT, shown in Figure 1, and a lumbar puncture were made, all without pathological findings. Due to suspicion of probable WE or non-alcoholic cause, thiamine is started at dose of 1500mg / day with good response at 24 hours; and was maintained for 5 days. Because the unusual and rapid onset of clinical symptoms, a second CT was made showing a subacute bilateral thalamic ischemic lesion, as seen in Figure 2; associated to a proximal left cerebral posterior artery stenosis as is evidenced in Figure 3. The patient's mental status, speech, dysmetria and ocular motility evolves to total regression. There were no complications during hospitalization.



**Figure 1:** Initial CT without pathological findings.  
CT: Cerebral tomography.



**Figure 2:** Arrows showing a Bilateral Thalamic Infarction in a second CT.  
CT: Cerebral tomography



**Figure 3:** Proximal cerebral posterior artery, as the arrow points.

## Discussion

As previously stated, WS may or may not be associated with alcohol (OH) intake [1] and should be suspected in patients with rapid and progressive onset of altered mental status, ocular motility palsy and ataxia [2]; and usually in that clinical sequence. This triad of symptoms was present in our patient, and although there was no history of OH ingestion, a WS was suspected, especially when an easy provoked-type confabulation was evidenced in the patient. This last finding, the confabulation, has been associated with WS since it was initially described by Korsakoff in 1889, in amnesic alcoholic patients; however, it has been described also in other conditions such as anterior communicating artery rupture, traumatic brain injury, Alzheimer's disease and brain tumor [9].

It is well recognized that early onset of thiamine administration in WS is recommended because it has low cost, general safety, rapid improvement in symptoms, and prevention of progression to irreversible brain damage [1]. Actually there is no consensus on the therapeutic dose, but the recommendation of the European Federation of Neurological Societies (EFNS) is for intravenous infusion of thiamine, 200mg diluted in 100cc of 5% dextrose or normal saline; in approximately 30 minutes [1]; however, according to our experience, it has to be supplemented for a minimum of 5 days. This treatment was done in this clinical case, showing improvement in 24 hours. The good evolution of our patient in response to thiamine use, the absence of alcohol intake, and the early pupillary compromise in this clinical case did caught our

attention, and it made us suspect another condition like BTI in this case. Although thiamine is not recommended in acute stroke, there is some evidence of usefulness in post-cardiorespiratory arrest encephalopathy [10], and there are also reports of benefits in the neuro-rehabilitation stages of stroke, by improving post-stroke fatigue of this type of conditions [11].

On the other hand, although hypodense areas in the periaqueductal gray matter, and the medial region of the thalamus has been described in CT of patients with WS [14], how was found in our patient, segmental stenosis of the posterior cerebral artery is not a finding as it was evidenced in this clinical case. It is possible that the improvement of our patient may be due the natural evolution of some patients with BTI, described as 3 to 24 hours [5]; but we theorize that it could be related to the action mechanism of thiamine in some WS cases reports, in which dysfunction in bilateral thalamic perfusion had been found [12-15], just like patients with BTI.

## Conclusion

The differential diagnosis of WS, especially in cases not associated to OH intake, must be broad and rapid for an early onset of thiamine; and among these diagnoses, BTI should be included due to the similarity in the clinical characteristics. A possible way to make the differential diagnosis is the more acute onset and the pupillary alterations in BTI. Finally, although the thiamine is not useful in the acute management of stroke –in BTI– for having some similarity with WS in the pathophysiology, it could be possible that it has a role in this subtype of stroke. It would be recommended to increase the report of this type of case with thiamine supplementation in acute BTI.

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## Conflict of Interest

No conflict of interest.

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