

# Difference Between Wernicke Encephalopathy and Korsakoff Syndrome

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## Key Difference – Wernicke Encephalopathy vs Korsakoff Syndrome

Wernicke encephalopathy is caused by the [thiamine](#) deficiency and is characterized by a set of acute psychotic symptoms and ophthalmoplegia. This condition can be reversed by thiamine supplementation. But if untreated, Wernicke encephalopathy can progress into an irreversible stage called the Korsakoff syndrome. Thus, they are two extremities of a spectrum of clinical manifestations. The key difference between Wernicke Encephalopathy and Korsakoff Syndrome is that **Wernicke encephalopathy is reversible whereas Korsakoff syndrome is irreversible.**

## What is Wernicke Encephalopathy?

As previously mentioned, thiamine (vitamin B1) deficiency is the cause for the Wernicke encephalopathy. This condition is usually associated with chronic [alcoholism](#) because of the influence of alcohol on the [metabolism](#) of thiamine. It has been established that long term alcoholism can decrease the intestinal absorption of thiamine by about 70%. In addition, some non-alcoholic causes such as gastric carcinomas, persistent vomiting, and chronic gastritis can also give rise to the Wernicke encephalopathy. Some of the patients suffering from the end stage renal disease can have [hemodialysis](#) induced thiamine deficiency.

There are scepticisms over the relationship between bariatric surgery and Wernicke encephalopathy. An overwhelming majority of the clinicians believe that this particular surgical intervention carried out to counter the obesity predisposes Wernicke encephalopathy. The severe thiamine deficiency that has been observed in a staggeringly high number of individuals who have undergone the bariatric surgery is nowadays recognized as the “Bariatric beriberi”.

Starvation is another major cause of this neural disorder, especially in developing countries. [HIV/AIDS](#), [heart failure](#) and thyrotoxicosis can also give rise to this condition.

## Pathophysiology

Thiamine is an important vitamin which acts as a cofactor for several enzymes such as pyruvate dehydrogenase and transketolase, which are involved in the aerobic respiration. Our brain has a very high metabolic demand and the energy for these metabolic processes taking place in the brain comes from the [aerobic respiration](#). When the thiamine level in the body becomes inadequate, this energy producing pathway fails, resulting in the death of neural tissues and the subsequent appearance of the clinical features.

## Morphology

The unique feature observed in the Wernicke encephalopathy is the presence of foci of [hemorrhage](#) and [necrosis](#). These are seen predominantly in the mammillary bodies and in the walls of third and fourth ventricles. Initially, the capillaries are dilated and have enlarged [endothelial cells](#). Eventually, with the disease progression, these capillaries rupture forming localized micro hemorrhages.

## Symptoms

- Confusion
- Impairment of the cognitive functions
- Ataxia
- Ophthalmoplegia

## Investigations

The following set of investigations can be done to assess the nutritional status of the patient.

- Serum vitamin B1
- Serum albumin
- Transketolase activity in the red blood cells

MRI scan of the brain is done to assess the damage to the cerebral neural tissues.

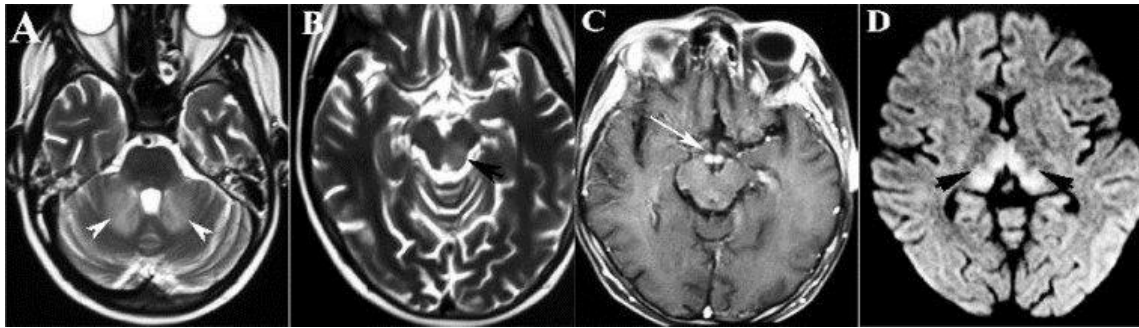


Figure 01: MRI Scans of a patient with Wernicke Encephalopathy

## Treatments

- Thiamine supplementation
- Dietary modifications
- Reducing the alcohol consumption

## What is Korsakoff Syndrome?

Korsakoff syndrome is an irreversible neural disorder characterized by the disturbances of short term memory and confabulation. Prolonged untreated thiamine deficiency is the basis for this condition; therefore any of the causes of Wernicke encephalopathy can give rise to Korsakoff syndrome also.



Figure 02: Alcoholism is a common cause for Korsakoff syndrome.

## **Morphology**

The hemorrhagic areas produced during the initial stage (Wernicke encephalopathy stage) are invaded by the macrophages. These scavenger cells destroy the damaged tissues in those regions forming cystic spaces filled with hemosiderin laden macrophages.

## **Symptoms**

- Inability to recall recent events
- Long term memory gaps
- Confabulation
- Difficulty in learning new information

## **Treatment**

There is no curative treatment for the Korsakoff syndrome. Symptomatic management is carried out in order to improve the standard of living of the patient.

- Thiamine supplementation
- Life style modifications
- Stopping the use of alcohol

## **What are the similarities between Wernicke Encephalopathy and Korsakoff Syndrome?**

- Thiamine deficiency is the basis for both conditions.
- Any factor that predisposes thiamine deficiency can give rise to Wernicke encephalopathy or Korsakoff syndrome.
- Alcohol is the commonest cause of both conditions.

# What is the difference between Wernicke Encephalopathy and Korsakoff Syndrome?

## Wernicke Encephalopathy vs Korsakoff Syndrome

Wernicke encephalopathy is a neurological disorder characterized by a set of acute psychotic symptoms and ophthalmoplegia.

Korsakoff syndrome is characterized by confabulation and short term memory disturbances.

### Reversibility

Thiamine supplementation can reverse the Wernicke encephalopathy.

Korsakoff syndrome is irreversible.

### Features

Areas of necrosis and micro hemorrhages can be observed.

In addition to areas of necrosis and hemorrhage, cystic spaces with hemosiderin laden macrophages are present.

## Summary – Wernicke Encephalopathy vs Korsakoff Syndrome

Both Wernicke encephalopathy and Korsakoff syndrome are caused by thiamine deficiency and alcoholism is the common cause for both these conditions. The main difference between Wernicke encephalopathy and Korsakoff syndrome is that Wernicke encephalopathy is reversible with thiamine supplementation whereas Korsakoff syndrome is irreversible. Both Wernicke encephalopathy and Korsakoff syndrome can be easily prevented by reducing the alcohol consumption. Alcohol has never been considered a good thing by doctors and these two disorders are horrifying examples why the excessive intake of alcohol must be discouraged.

## References:

1. Kumar, Vinay, Stanley Leonard Robbins, Ramzi S. Cotran, Abul K. Abbas, and Nelson Fausto. Robbins and Cotran pathologic basis of disease. 9th ed. Philadelphia, Pa: Elsevier Saunders, 2010. Print.
2. Kumar, Parveen J., and Michael L. Clark. Kumar & Clark clinical medicine. Edinburgh: W.B. Saunders, 2009. Print.

## Image Courtesy:

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