THIAMINE REFRACTORY—WERNICKE—KORSAKOFF'S SYNDROME—A CASE REPORT

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SUMMARY

A case of Thiamine refractory Wernicke-Korsakoff syndrome, successfully treated with the combination of magnesium sulphate and thiamine is presented. The role of Magnesium Sulphate in such cases has been discussed.

Wernicke Korsakoff encephalopathy is a disorder due to thiamine deficiency usually seen in alcoholics. The cardinal features are ataxia, nystagmus, ophthalmoplegia and memory impairment (Victor et al., 1971). The syndrome responds well to treatment with thiamine. Certain patients however do not respond to thiamine alone. There is evidence in an isolated unreplicated study that administration of magnesium sulphate is useful in such Thiamine refractory cases (Traviesa, 1974).

We report a case of thiamine refractory Wernicke-Korsakoff syndrome, which responded to parenteral magnesium sulphate.

CASE REPORT

A sixty five year old male was brought with a history of altered behaviour, excessive talking, and disturbed social functioning of fifteen days duration. The patient was an alcoholic for twenty five years and was dependent on alcohol for the last five years. He had taken the last drink 15 days prior to admission.

On examination, the patient was tremulous, confused and disoriented. He had global memory impairment, confabulation, pellagrous skin changes and arthritic changes of both hip and knee joints.

Neurologically, the patient had left lateral rectus palsy, vertical and horizontal nystagmus, ataxia of limbs and trunk and peripheral neuropathy. With the diagnosis of alcohol withdrawal induced subacute delirious state and Wernicke Korsakoff syndrome, the patient was admitted in the intensive care unit and started on parenteral Thiamine 150 mgm per day in addition to symptomatic treatment. Two days later the patient showed improvement in sensorium and was found to have recent and immediate memory deficits with confabulation.

Routine laboratory investigations were normal. Pneumoencephalogram showed cortical atrophy and ventricular dilatation. The patient was given 250 mg Thiamine, intramuscularly every day for two months in addition to other vitamin supplementation. He did not show any improvement in his clinical status. The patient was reinvestigated as he was refractory to Thiamine and serum magnesium was found to be low (1.9 mEq/L.). Hence the possibility of

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hypomagnesemia as a cause of thiamine refractoriness was considered and the patient was given magnesium sulphate 6 gms intramuscularly twice in four divided doses per day, over a period of three weeks. Two days following the first administration, he started showing improvement. Three weeks later, nystagmus gradually disappeared and showed improvement in his neurological status. Memory showed improvement and confabulation disappeared. The clinical improvement in memory was in association with a rise in serum Magnesium (Figure 1). The patient continued to maintain improvement with minimal intellectual deficits.

DISCUSSION

This case was initially refractory to the conventional mode of treatment and later showed remarkable improvement after correcting the hypomagnesemia. However, the minimal intellectual deficits could be explained by the structural changes, as evidenced in the P. E. G. Hypomagnesemia has been reported in alcohol withdrawal states (Raju, 1980). The delirium like picture and refractoriness to thiamine might be due to hypomagnesia.

The mechanism of action of magnesium in thiamine refractory Wernicke syndrome remains unclear. It has been suggested that magnesium acts as a co-factor to enzymes involved in phosphate transfer such as transketolase. This enzyme is crucial to pyruvate metabolism which is disturbed in Wernicke syndrome.

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