Clinics in Surgery

Case Report
Published: 16 Jan, 2020

Acute Reversible Blindness in a Patient with Wernicke's Encephalopathy

Vasant Raman[1] and Roland Ling[2]*

[1]Royal Eye Infirmary (Level 3), University Hospitals of Plymouth, UK

[2]West of England Eye Unit, Royal Devon & Exeter Hospital Foundation Trust, UK

Abstract

The typical triad of Wernicke's disease includes ataxia, ocular signs and a confusional state. We present a case of Wernicke's encephalopathy, presenting with acute loss of vision. There are many causes of Wernicke's disease. Nevertheless, alcohol toxicity continues to be the main etiological agent. Commonly consumed alcohol (ethanol) is not known to cause acute blindness. Whereas methylated spirit (Methanol) can cause acute loss of vision. This can be consumed accidentally or intentionally with disastrous consequences. Patients with alcohol dependence, presenting with Wernicke's disease, the possibility of methanol poisoning needs to be considered and excluded, among the important differential diagnosis. Prompt treatment in our patient completely reversed the blindness.

Keywords: Wernicke's encephalopathy; Blindness; Thiamine deficiency

Introduction

Chronic alcohol abuse is the most common cause of Wernicke's encephalopathy in the developed world. It can also occur during pregnancy, starvation states, gastric plication surgery, and cancer and with the use of certain drugs [1-3]. Acute visual loss is not a well-recognized feature of Wernicke's encephalopathy, but has been reported in the English literature [3,4]. In the context of alcohol abuse, acute visual loss can occur in methanol poisoning, and alcoholic ketoacidosis [5,6]. The visual loss in the above two conditions is due to severe acidosis. Although, blood levels of methanol will establish the diagnosis in methanol poisoning, arterial blood ph, and serum bicarbonate levels can be used as a surrogate marker to establish the acidosis, which is an important feature of methanol poisoning and alcoholic ketoacidosis. Excellent visual prognosis has been reported following correction of metabolic acidosis in patients with alcoholic ketoacidosis. Whereas methanol poisoning can potentially lead to blindness and death [6-8].

We present a case of a patient presenting with acute visual loss is in Wernicke's disease. Since blindness is uncommon in this condition, consumption of products contaminated due to methanol, a common ingredient in household products, or the possibility of purchase of spurious methanol was entertained as in our patient.

Case Presentation

A 63-year-old white man with a history of chronic alcohol abuse, accompanied by a friend, presented to the eye emergency department with a two-day history of sudden loss of vision from both eyes. He had been drinking alcohol continuously, during the previous week, with a poor dietary intake. He smoked one packet of cigarette a day for nearly 40 years.

Medical history included previous admissions on three occasions for alcohol-related seizures, unsuccessful attempts at alcohol rehabilitation, and treatment for pulmonary tuberculosis. Past ophthalmic history was unremarkable.

On ocular examination, his visual acuity was reduced to hand movements in both eyes. Ocular motility revealed restricted elevation of both eyes. Abduction of eyes beyond midline was not possible, and he manifested a horizontal jerky nystagmus on attempted abduction. Pupils were sluggish to direct and consensual reaction. Intraocular pressure measured 6 and 8 mmHg respectively by applanation tonometry. Rest of the eye examination was unremarkable. General examination revealed poor nutritional status and personal hygiene. Recent memory was impaired, but the patient denied any hallucinations. He had ataxia with positive Romberg's sign. Deep tendon

OPEN ACCESS

Correspondence:

Vasant Raman, Royal Eye Infirmary (Level 3), University Hospitals of Plymouth, Plymouth, UK, Tel: 00-44-1752-439359/0044 7720718667; E-mail: vasant.raman@nhs.net

Received Date: *09 Dec 2019*

Accepted Date: *06 Jan 2020*

Published Date: *16 Jan 2020*

Citation:

Raman V, Ling R. Acute Reversible Blindness in a Patient with Wernicke's Encephalopathy. Clin Surg. 2020; 5: 2709.

reflexes were normal.

Urgent arterial blood gas analysis was done to rule out acidosis, secondary to methanol intoxication in view of the acute visual loss.

pH, P02, pC02 and bicarbonates levels were within normal limits. Having ruled out methanol poisoning, the patient was admitted and treated as Wernicke's encephalopathy with I/V pabrinex (ascorbic acid 500 mg, nicotinamide 60 mg, Pyridoxine 50 mg, and Thiamine hydrochloride 250 mg) and chlordiazepoxide 10 mg for three days. This was followed by administration of oral thiamine 300 mg for four days. He was then maintained on multivitamin tablets. Blood results showed a macrocytic type of anemia. His liver functions were abnormal due to alcohol-related liver damage. Serum Vit B12 level was normal, but his serum folic acid level was very low- 1.2 ng/100 ml. Hence 5 mg oral folic acid supplementation was started. MRI scan of the brain showed widening of sulci with large ventricles, cerebellar atrophy and a small pons. Following administration of I/V pabrinex, the patient reported a subjective improvement in his vision the following day and Snellen acuity was recorded as 6/24 in both eyes. Two weeks following his admission, vision had improved to 6/9 both eyes unaided. Extraocular movements had improved, but gaze-evoked nystagmus persisted in the horizontal gaze.

Serial visual field analysis showed gradual improvement, over the first two weeks and was normal at the end of the third week. The patient developed pyrexia during his stay in the hospital. No definite cause could be found on investigation, but nonetheless responded well to empirical oral antibiotic treatment. The patient was awaiting rehabilitation with the social support system, before being discharged for living independently on his own.

Discussion

The characteristic brain lesions of Wernicke's encephalopathy include symmetrical discoloration of structures surrounding the third ventricle, cerebral aqueduct and fourth ventricle, with petechial hemorrhages in some acute cases and atrophy of the mammillary bodies in chronic cases. Microscopic examination reveals endothelial proliferation, demyelination but with relative preservation of neurons.

How thiamine deficiency causes the CNS manifestations in Wernicke's disease is not clear. Nevertheless, the central nervous system manifestations of Wernicke's disease are reversible with the administration of thiamine. Deficiency of thiamine in alcoholics is due to poor dietary intake, GI malabsorption and inadequate hepatic storage. Thiamine is an essential cofactor for transketolase, ά-ketoglutarate dehydrogenase, and dehydrogenase, enzymatic activity and in addition thiamine is also necessary for oxidation of glucose in the brain. It is involved in axonal conduction and synaptic transmission. The dramatic visual recovery following administration of thiamine suggests, thiamine deficiency related optic neuropathy [9,10]. The visual recovery in this patient paralleled the improvement in neurological signs and other ocular features. Pierre Dreyfus report two patients with a subacute visual loss from tobacco-alcohol amblyopia, which recovered following parenteral administration of thiamine [11]. Although described as tobacco-alcohol amblyopia, the clinical features described were consistent with Wernicke's encephalopathy. They also found low levels of blood transketolase, a thiamine-dependent transferase enzyme. Rodger showed experimentally that thiamine deficiency could result in degenerative changes in the optic nerve of rats [12].

The possibility of classical tobacco-alcohol amblyopia in this patient is unlikely in view of the acute onset of symptoms, absence of typical field changes and the dramatic response to thiamine therapy. The other biochemical abnormality noted in the investigation was a deficiency of serum folic acid. Golnik reported visual improvement in six of their patients with tobacco-alcohol amblyopia, following administration of folic acid [13]. The precise mechanism of folic acid deficiency and the visual improvement following administration is not clear but it seems to be involved in repair of the adult central nervous system [14].

In conclusion, we would like to submit that, acute visual loss is an uncommon presentation of Wernicke's encephalopathy; hence the possibility of methyl alcohol poisoning should be considered in the differential diagnosis and ruled out by appropriate investigation. The acute visual loss in Wernicke's disease seems to respond well to thiamine administration. Concomitant folic acid deficiency should be investigated and treated if necessary.

References

1. Watson AJ, Walker JF, Tomkin GH, Finn MM, Keogh JA. Acute Wernicke's Encephalopathy Precipitated by Glucose Loading. Ir J Med Sci. 1981;150(10):301-3.
2. Kwee IL, Nakada T. Wernicke's Encephalopathy Induced by Tolazamide. N Engl J Med.1983;309(10):599-600.
3. Tesfaye S, Achari V, Yang YC, Harding S, Bowden A, Vora JP. Pregnant, vomiting, and going blind. Lancet. 1998;352(9140):1594.
4. Timmings PL, Carroll GJ, Donaldson IM. Wernicke's encephalopathy presenting with Blindness. N Z Med J. 1993;106(954):159-60.
5. Yanagawa Y, Kiyozumi T, Hatanaka K, Itoh T, Sakamoto T, Okada Y. Reversible Blindness Associated with Alcoholic ketoacidosis. Am J Ophthalmol. 2004;137(4):775-7.
6. Naeser P. Optic Nerve Involvement in a Case of Methanol Poisoning. British J Ophth. 1988;72(10)778-81.
7. Lushine KA, Harris CR, Holger JS. Methanol Ingestion: Prevention of Toxic Sequelae after Massive Ingestion. J Emerg Med. 2003;24(4):433-6.
8. Teo SK, Lo KL, Tey BH. Mass Methanol Poisoning: A Clinico-Biochemical Analysis of 10 Cases. Singapore Med J. 1996;37(5):485-7.
9. Charness ME, Simon RP, Greenberg DA. Ethanol and the Nervous System. N Engl J Med. 1989;321(7):442-54.
10. Bronte-Stewart J, Pettigrew AR, Foulds WS. Toxic Optic Neuropathy and its Experimental Production. Trans Ophthalmol Soc UK. 1976;96(3):355-8.
11. Dreyfus PM. Blood Transketolase Levels in Tobacco-Alcohol Amblyopia. Arch Ophthalmol. 1965;74(5):617-20.
12. Rodger FC. Experimental Thiamin Deficiency as a Cause of Degeneration in the Visual Pathway of the Rat.Br J Ophthalmol. 1953;37(1):11-29.
13. Golnik KC, Schaible ERJ. Folate-Responsive Optic Neuropathy. Neuroophthalmol. 1994;14(3):163-9.
14. Iskandar BJ, Nelson A, Resnick D, Pate Skene JH, Gao P, Johnson C, et al. Folic Acid Supplementation Enhances Repair of the Adult Central Nervous System. Ann Neurol. 2004;56(2):221-7.