International Journal of Community Medicine and Public Health
Manorenj S et al. Int J Community Med Public Health. 2016 Sep;3(9):2686-2688
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: http://dx.doi.org/10.18203/2394-6040.ijcmph20163097

Case Report

Camphor poisoning presenting as acute diffuse demyelination of brain

Sandhya Manorenj*, Snehalatha Inturi, Narayana Pancheti

Department of Neurology, Employees State Insurance Corporation, Superspeciality Hospital, Sanath Nagar, Hyderabad, India

Received: 30 June 2016
Accepted: 29 July 2016

***Correspondence:**
Dr. Sandhya Manorenj,
E-mail: drsandhyamanorenj@gmail.com

ABSTRACT

Camphor is a colorless substance available in solid and liquid form. Volatile oil form of camphor is the active ingredient of Vicks Vaporub, and Tiger Balm ointments. These ointments are commonly used as home remedies for treatment of cough, cold and for topical analgesics. They are inexpensive, over the counter remedies, hence widely used in India. Neurological complication due to camphor toxicity reported is headache, mental confusion and seizures. Here we report, a case of diffuse white matter lesions in a young female due to accidental camphor inhalation while working in factory that manufactures vicks and tiger balm. The case highlights camphor as a possible cause of toxic demyelination hence potentially exposed workers should be educated about health and safety measures to prevent such complication.

Keywords: Diffuse white matter disease, Toxic demyelination, Camphor

INTRODUCTION

Camphor is obtained by distilling the bark and wood of the camphor tree and is chemically manufactured from turpentine oil in view of its wide usage in pharmaceutics. Camphor is available as, solid crystalline colourless form with a strong aromatic odor and liquid form as volatile oils. There are wide variety of home remedies products which contain camphor. Commonly used ones are mothballs, Vicks Vaporub, and tiger balm.

Camphor poisoning is commonly reported in children due to accidental ingestion of solid form of camphor the mothball used as an insecticide. Camphor oil is the active ingredient in Vicks Vaporub (5.25gm/100gm) and Tiger balm (25% camphor). These are topical creams, used as home remedies for cough, cold, and topical rubifacient. There is few case reports of camphor poisoning following ingestion and dermal application of camphor containing products.[1-5] Neurological complication following camphor poisoning are headache, mental confusion, seizure and status epilepticus.[2,3,5] Camphor containing products affects the grey matter of brain. There are few toxins known to cause white matter disease of brain (Toxic demyelination); they are carbon monoxide, clioquinol, chemotherapeutic agents (Methotrexate, 5 Fluouracil) and amphetamine.[6] This is the first reported case of diffuse demyelination of white matter following camphor inhalation.

CASE REPORT

A 34-year-old female, presented with headache of 4 days, dysarthria, weakness of all limbs, diplopia and altered sensorium of one-day duration. Headache was, holocranial in nature associated with vomiting. There was no history of fever, diarrhoea, and recent vaccination, bleeding diathesis, joint pains and trauma. Past history was not significant.

Physical examination showed Glasgow coma scale score of 10 (eye-2, motor response -5, verbal -3) and scanning speech. Cranial nerve examination showed bilateral VIth cranial nerve palsy, bilateral facial weakness (VII) in form of drooling of saliva from both angles, but able to close the eyes. Motor system examination showed normal

tone with motor power of 4/5 Medical research council grading in all four limbs. Deep tendon reflexes were elicitable. Cerebellar system examination showed bilateral positive finger nose, heel knee test, and gait ataxia.

Planters were flexor in right and extensor on left. Clinically diagnosed as acute onset Ataxic quadriparesis with involvement of bilateral VIth and VIIth cranial nerves. Anatomical localization at pons with etiological diagnosis of acute demyelination versus stroke was considered.

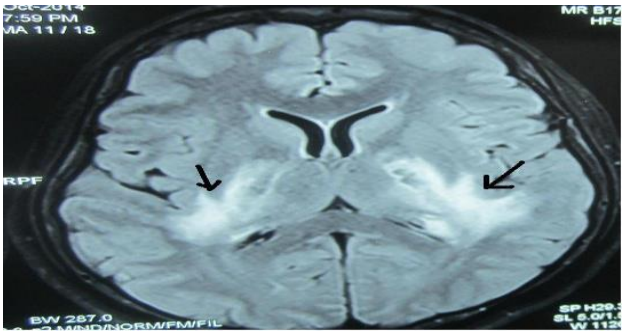

Figure 1: MRI brain T2 flair images, axial sequences showing bilateral symmetrical hyper intensities involving front parietal, periventricular, and deep white matter, posterior limb of internal capsule, lateral thalami, and posterior temporal white matter marked by black arrow.

Computed Tomography of brain was normal. Magnetic resonance imaging (MRI) brain with contrast showed bilateral symmetrical T2 flair hyper intensities involving frontoparietal, periventricular, and deep white matter, posterior limb of internal capsule, lateral thalami, and posterior temporal white matter (Figure 1).

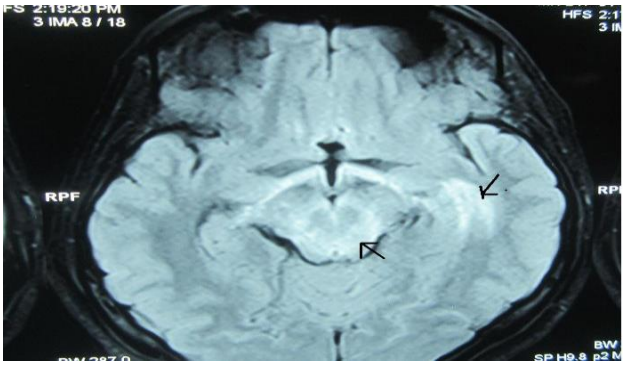

Figure 2: T2 Flair axial sequence marked by arrow showing inferior extension of the white matter signal changes into midbrain and tegmentum of the pons.

There was inferior extension of the white matter signal changes into midbrain, tegmentum of the pons, bilateral middle Cerebellar peduncles and Cerebellar deep white matter (Figure 2 and 3). There was no restricted diffusion, no haemorrhages, and no enhancement on contrast. Mild mass effect noted. MR spectroscopy was with in normal limits. Differential diagnosis considered were demyelination acute on chronic, small vessel vasculitis, haemolytic uremic syndrome, and toxic demyelination.

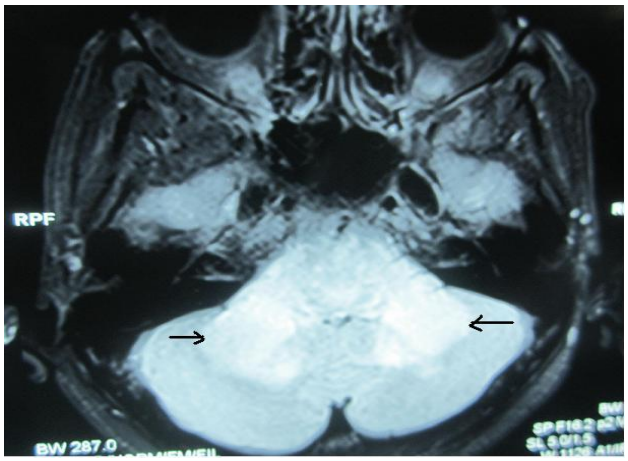

Figure 3: Flair images axial sequence marked by arrow showing involvement of bilateral middle cerebellar peduncles and cerebellar deep white matter, suggestive of cerebellar demyelinations.

Routine blood parameters showed total leucocyte count of 13100, with neutrophil of 91%, lymphocyte of 7%, and eosinophil of 1%. Erythrocyte sedimentation rate (ESR) was 55mm/hour, platelet count of 4.9 lakhs, haemoglobin of 9.6gm%, and reticulocyte count of 2.5%.

HIV test was negative. Renal function, liver function tests, electrolytes, blood sugar, Vitamin B12, thyroid profile, lipid profile, ECG, vasculitis work up were normal. Peripheral smear were negative for hemolysis. Visual evoked potential was normal.

Diagnosis of acute demyelination was considered, because not known, as all tests were negative. She was treated with high dose intravenous Methylprednisolone 1 gm daily for 5 days. She improved dramatically by 3rd day and was normal by a week .She was put on tapering oral steroids.

On questioning further about environmental and occupational exposure, she revealed that she stays in the locality surrounded by industries that manufactures Vicks and Tiger balm since ten years.

She has been working in one of these factories which process camphor to gaseous form which is the active ingredient for vicks and tiger balm since five years without using gloves and masks.

We suspect this as a case of diffuse toxic demyelination of brain, involving cerebrum, brainstem and cerebellar white matter fibres. At 2 months follow up, MRI brain done showed complete disappearance of lesions (Figure

3). She was followed for a period of one year and was asymptomatic.

DISCUSSION

Classically described white matter diseases of central nervous system are Multiple sclerosis, Neuromyelitis Optica and Acute disseminated encephalomyelitis. With the availability of Magnetic resonance imaging and detection of Neuromyelitis Optica IgG antibodies more cases of autoimmune inflammatory diseases are diagnosed in countries like India. Still the most common white matter diseases encountered in tropics are infectious and post infectious disorders.

Diffuse white matter involvement of brain is commonly seen with HIV encephalopathy, diffuse cytomegalovirus encephalitis, vasculitis, and Vitamin B12 deficiency and acute on chronic demyelinating diseases in tropics.[6] Present case diffuse white matter lesions of brain was due to camphor toxin, which was completely reversible after a course of steroids.

Volatile oil form of camphor is nasal irritant, damages olfactory nerves and toxin might have entered intracranially and induced demyelination. Secondly camphor when burned releases carbon monoxide that itself causes demyelination.

Recurrent exposure while working in factory and staying in locality surrounded by factories manufacturing Vicks Vaporub and Tiger balm might have increased the chance of camphor toxicity in this case.

Hence workers should be clearly educated about the safety hazards of this products Precaution should be taken for affected individual to prevent recurrence. Usage of masks, gloves and washing clothes daily are the preventive measures.

Factories should monitor air camphor level regularly to prevent health hazards. Use of inexpensive home remedies like Vicks Vaporub and Tiger balm should be cautioned in view of toxic effects of this herbicides.

CONCLUSION

Occupational and environmental exposure should be considered where aetiology is undetermined.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Uc A, Bishop WP, Sanders KD. Camphor hepatotoxicity. South Med J. 2000;93(6):596-8.
2. MacKinney TG, Soti KR, Shrestha P, Basnyat B. Camphor: an herbal medicine causing grand mal seizures. BMJ Case Rep. 2015. doi:10.1136/bcr-2014-209101.
3. Guilbert J, Flamant C, Hallalel F, Doummar D, Frata A, Renolleau S. Anti-flatulence treatment and status epilepticus: a case of camphor intoxication. Emerg Med J. 2007;24(12):859-60.
4. Manoguerra AS, Erdman AR, Wax PM, Nelson LS, Caravati EM, Cobaugh DJ et.al. Camphor Poisoning: an evidence-based practice guideline for out-of-hospital management. Clin Toxicol (Phila). 2006;44(4):357-70.
5. Ruha AM, Graeme KA, Field A. Late seizure following ingestion of Vicks Vapo Rub. Acad Emerg Med. 2003;10(6):691.
6. Lekha Pandit. Differential diagnosis of white matter diseases in the tropics: An overview. Ann Indian Acad Neurol. 2009;12(1):12-21.

Cite this article as: Manorenj S, Inturi S, Pancheti N. Camphor poisoning presenting as acute diffuse demyelination of brain. Int J Community Med Public Health 2016;3:2686-8.