Delayed Syndrome in Carbon Monoxide Poisoning

RS Raj*, P Abdurahiman**, J Jose***

Abstract
Acute carbon monoxide poisoning can lead to delayed manifestations. This has been called post-interval syndrome, which can present after an apparent period of normalcy following acute poisoning. We are reporting a patient who had an accidental exposure to carbon monoxide and presented later with all the manifestations of delayed poisoning. The various clinical manifestations and imaging findings are discussed. A delay in recognition in the acute stage can predispose to development of the post-interval syndrome. ©

INTRODUCTION
Carbon monoxide poisoning can have two phases of presentation – an acute phase characterized by headache, dizziness, confusion, nausea, seizures and coma, and a delayed encephalopathy clinically characterized by a recurrence of neurologic or psychiatric symptoms. This recurrence is preceded by a temporary asymptomatic period (lucid interval) of variable duration (usually 2–3 weeks) after recovery from the acute stage of CO intoxication.1 We are reporting a patient who presented late in an akinetic mute state several weeks following an acute exposure.

SUMMARY
A 55 year old man was working in the Gulf as a cleaner. One night he was exposed to smoke arising from a substance used for generating fragrance while he was sleeping in an air-conditioned room. He was found unconscious in that room the next morning. The treating physician at the local hospital made a diagnosis of acute carbon monoxide poisoning and treated accordingly. The patient became better within three days and was discharged from that hospital. Four weeks later he was found to be behaving abnormally and had developed urinary incontinence. So he was sent back home and was admitted to our hospital.

On the day of admission, the patient was conscious but not answering questions. His vitals were stable. He was in an akinetic and mute state. He had bilateral rigidity, release reflexes and brisk deep tendon jerks. He was refusing oral feeds and was on naso-gastric tube. He was on continuous bladder drainage.

His routine lab investigations were within normal limits. A head scan (CT) was taken which was also normal. MRI of brain revealed bilateral confluent white matter hyperintensities in T2 weighted images, which were hypointense on T1.

DISCUSSION
Acute CO poisoning can lead to delayed manifestations. This delayed form is also called post interval syndrome, and can present up to 1 month after initial exposure (14 – 45 days).3 Failure to suspect on the initial presentation often predisposes to late sequelae. Acute presentation is due to tissue hypoxia which predominantly affects the heart and brain.2

The chronic form (Post interval syndrome), may be due to intracellular uptake of carbon monoxide.3 Carbon monoxide binds to cytochrome oxidase and this can lead to lipid peroxidation in the brain. This injury is aggravated by the reperfusion, thereby explaining the delayed presentation. Patients present with cognitive impairment, akinetic mutism, sphincter involvement, gait ataxia and various extra-pyramidal problems in the form of chorea, dystonia and parkinsonism

MRI is the most sensitive diagnostic tool. Maximum damage is seen in the globus pallidus. Subcortical white matter changes are frequent and imaging studies roughly correlates with prognosis.1

Predictors of delayed encephalopathy at the time of acute presentation include syncope, depressed level of consciousness at the time of presentation, and abnormal mental status. Studies have shown that treatment with
Hyperbaric oxygen can offer some protection against delayed encephalopathy. Delayed encephalopathy has a very prolonged course with improvement occurring in bladder function and rigidity over months or years, but the behavioral changes can persist.

REFERENCES

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