

## Cortical venous infarcts and acute limb ischaemia in acute carbon monoxide poisoning: A rare case report

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### Abstract

A case of carbon monoxide poisoning is presented with unusual complications; some of which have not been reported previously. A 48-years-old Asian male presented to the emergency department with dyspnoea, altered state of consciousness and pale discoloration of skin after being locked inside a factory room with burning coal. Patient was in acute respiratory distress. Arterial blood gas analysis showed respiratory acidosis with hypoxaemia. On 3rd day, patient developed dark coloured urine and right upper limb ischaemia. Acute renal failure was diagnosed. A doppler ultrasound showed stenosis of radial and ulnar arteries. On 8th day, patient regained consciousness and complained of loss of vision. An MRI of the brain revealed bilateral occipital venous infarcts. Cortical venous infarcts and arterial stenosis are rare complications of acute carbon monoxide poisoning.

**Keywords:** Carbon Monoxide, Limb Ischemia, Acute Renal Failure, Cortical Blindness.

### Introduction

Carbon monoxide is a highly toxic gas, which is colourless, odourless, and tasteless. Upon initial inhalation, it is non-irritating. Exposure typically occurs when equipment based on carbon containing fuel is used in buildings or semi-enclosed spaces malfunctions. The main manifestations of poisoning develop in the organ systems most-dependent on oxygen use, particularly the central nervous system (CNS) and the heart.<sup>1-6</sup> The initial symptoms of acute carbon monoxide poisoning may include headache, nausea, fatigue, tachycardia, hypotension, arrhythmias, delirium, hallucinations, dizziness, unsteady gait, confusion, seizures, unconsciousness, and respiratory arrest.<sup>8</sup> In this report, we present a case of carbon monoxide poisoning complicated with rare manifestations.

### Case Presentation

A 48-years-old nonsmoker man was brought to

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Figure-1: Right hand ischaemic changes.

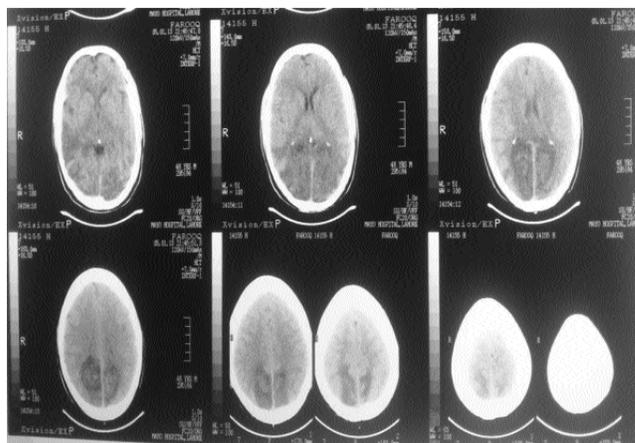


Figure-2: Showing bilateral occipital infarcts and a right posterior parietal lobe infarct.

emergency in December 2012, when found unconscious in a closed factory room for at least 12 hours with burning coal inside. Patient had ignited the coal to combat the severe winter cold. He had no past medical history and was not using any medications prior to the admission. On arrival to emergency department, he was semi-conscious, dyspnoeic and appeared pale.

On the initial examination, his pulse was 112

beats/min, blood pressure of 90/60 mmHg, respiratory rate of 38 per minute and temperature was 97°F. Patient had no visible cyanosis, oedema or jaundice. Lung examination showed vesicular pattern and decreased air entry bilaterally. CNS examination revealed Glasgow Coma Scale (GCS) score of 3/15 without a focal neurological deficit. The rest of cardiovascular and abdominal examinations were within normal limits. Complete blood count displayed leukocytosis (WBC's 17.3). Serum electrolytes and glucose were normal. Arterial blood gas analysis revealed respiratory acidosis with type II respiratory failure. On day 3 of admission, several additional abnormalities occurred; serum ALT and AST were elevated (205 U/L & 425 units/L respectively) (n= upto 400 U/L), renal functions became abnormal (BUN 110 mg/dL, creatinine 1.9 mg/dL [upto 1.0 mg/dL]), and he had hyperglycaemia, myoglobinuria, proteinuria and haematuria. CPK rose to 4956 u/l (n= 176 U/L) with CK-MB of 258 u/l (n= 10-23 U/L), and LDH was 1640 u/l (upto 248 U/L). ECG was normal. On the same day, he developed right hand ischaemia (Figure-1). A doppler ultrasound revealed stenosis of right ulnar artery, about 2 cm from its origin. There was also stenosis of right radial artery, 5 cm proximal to the wrist, with minimal post-stenotic residual flow. Carotid Doppler was normal. On day 8, patient became conscious and was weaned off the ventilator support. He complained of bilateral loss of vision. Ophthalmological examination was in favour of cortical blindness. A CT scan of the brain showed bilateral occipital infarcts and a right posterior parietal lobe infarct (Figure-2). Echocardiography showed concentric left hypertrophy and good biventricular systolic function without any intra-cardiac thrombus. An EEG was suggestive of mild cerebral dysfunction. Normal intra and extra-cranial vessels were seen on CT angiography whereas the brain MRI revealed bilateral occipital venous infarcts.

Patient had been on ventilator support for eight days for respiratory failure. Acute renal failure was treated with intravenous fluids. Regular insulin was used to control hyperglycaemia. Anti-platelet therapy was given for limb ischaemia and bilateral cortical infarcts. Patient required surgical amputation of gangrenous right hand. Subsequently, all biochemical abnormalities improved between 14th and 20th post admission day. Clinical recovery followed, and he was discharged from hospital on 35th day.

There was no visual improvement at 6-months follow up despite using anti-platelet therapy. At 1 year follow up vision had improved to light perception only.

## Discussion

The true incidence of carbon monoxide poisoning is unknown, as many non-lethal exposures go undetected. Usual manifestations of acute poisoning include dizziness, confusion, headaches and flu-like symptoms. Larger exposure can lead to significant toxicity of the central nervous system (depression, confusion, and memory loss) and cardiac arrhythmias, (hypotension) and even death. Compared to oxygen, carbon monoxide has 240 times higher affinity for haemoglobin. Once bound to haemoglobin, it shifts the oxygen-haemoglobin dissociation curve to the left, resulting in reduced delivery of oxygen to the tissues. This results in hypoxia and ischaemia. The organs most sensitive to hypoxia i.e. brain and heart are affected first leading to central nervous system depression and cardiac ischaemia.

The patient was unconscious at presentation and developed biomarkers of cardiac injury following the admission. Myocardial infarction, myocardial rupture, atrial fibrillation and supraventricular tachycardia have also been reported in acute carbon monoxide poisoning.<sup>1-4</sup>

The patient also developed rhabdomyolysis and acute renal failure, a common complication of acute carbon monoxide poisoning<sup>9,10</sup> which develops as a result of hypoxic injury to skeletal muscles.

Stenosis of right ulnar and radial arteries were other interesting features in our patient. There are no reported cases of ulnar or radial artery stenosis resulting in limb ischaemia following acute carbon monoxide poisoning. Colour Doppler of upper limb vessels and echocardiography failed to reveal any thrombus inside upper limb vessels and heart respectively. It appears that direct endothelial damage caused by carbon monoxide resulted in arterial stenosis. The mechanism is not understood fully but it probably involves lipid peroxidation by toxic oxygen species generated by xanthine oxidase. Xanthine oxidase is produced in situ from xanthine dehydrogenase via enzymes produced by white blood cells that adhere to damaged endothelial cells and causing events similar to ischaemia-reperfusion injury.<sup>7</sup>

The cause of cortical blindness in our patient was bilateral occipital venous infarcts seen on MRI. Although cortical blindness has been reported previously,<sup>5,6</sup> the cause in those cases was, leukomalacia<sup>6</sup> and diffuse hypoxic brain injury which was more marked in the occipital region.<sup>5,6</sup>

Our case is unique in a way that cortical venous infarcts and limb ischaemia after acute carbon monoxide

poisoning have never been reported before.

### Conclusion

Acute carbon monoxide poisoning can lead to acute limb ischaemia and cortical venous infarcts which are rare but possible complications.

**Conflict of Interest:** There is no conflict of interest of any author involved in case report.

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