

Findings that shed new light on the possible pathogenesis of a disease or an adverse effect

# Chronic carbon monoxide poisoning resulting in bilateral cataracts and a cystic globus pallidus lesion

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## Summary

The authors describe a case of a 43-year-old lady who developed bilateral cataracts, seizures and a unilateral cystic lesion of the basal ganglia following low-dose carbon monoxide (CO) exposure over 7 years. Cataract formation may result from sustained oxidative stress as a result of chronic environmental CO exposure.

## BACKGROUND

Carbon monoxide (CO) exposure has not directly been associated with cataract formation. This case describes a female in the fourth decade of life with bilateral lens opacities, symptoms in keeping with CO poisoning, seizures and a unilateral globus pallidus lesion. There was no relevant family or medical history. This case shows the potential effects of long term chronic exposure to CO. The temporal association between the symptoms and signs described in this case is highly suggestive of an association between CO exposure and cataractogenesis. This adds to growing evidence of environmental and metabolic influences on the development of acquired lens opacities.

## CASE PRESENTATION

We report a case of bilateral cataract formation and characteristic intracranial lesions associated with chronic CO exposure over a 7-year period. The patient was a Caucasian female with no medical or family history of ocular disease or trauma. She was not non-smoker. There were no underlying systemic co-morbidities or regular medications taken. Her initial presentation was at 37 years of age with an 11-month history of seizures occasionally resulting in loss of consciousness, persistent lethargy, headaches, personality change and depression. Neurological examination and EEG were normal. MRI of the brain 5 years after the onset of CO exposure when symptoms first began revealed a cystic lesion in the left globus pallidus and bilateral dilatation of temporal horns suggestive of hippocampal atrophy.

The patient attended ophthalmic services with gradual reduction of vision at in the right eye at 41 years of age and 2 years later with the same symptom in the left eye. Visual acuity (snellen) at the time of presentation in the right eye was 6/60 unaided; 6/36 with pin hole and 6/9 in the left eye. Clinical examination at each presentation revealed cataract; nuclear sclerosis grade 2 right eye, nuclear sclerosis grade 1 left eye. Anterior segments were normal with intraocular pressure within normal limits. The optic disc

was tilted in both eyes and a posterior vitreous detachment was evident in the right eye. The appearance of the macula, retinal vasculature and peripheral retina was within normal limits in both eyes.

## OUTCOME AND FOLLOW-UP

The patient underwent routine phacoemulsification procedures and visual acuity postoperatively improved to 6/6 in both eyes. Seven years from the onset of her symptoms her home was found to be heavily contaminated with CO due to a 'class 2 flue' in a newly built home. Engineers discovered a design fault of the system which was thought to have been present since its installation 7 years prior to its discovery. CO levels were independently measured at over 750 ppm; far exceeding than the WHO 30 ppm maximum allowable limit for domestic environments. All systemic complaints except seizures resolved once the appliance was replaced. The frequency of seizures reduced from 2 to 3 per day during the period of CO exposure to one seizure every 2–3 months. The neurologist's opinion was that her epilepsy is unlikely to be curable given the permanent intracranial lesions resulting from CO exposure.

## DISCUSSION

CO is a colourless odourless gas produced as a product of combustion. Chronic low dose environmental CO exposure has not been studied in association with acquired lens opacities. Cataracts are reported, however, in association with environmental exposure to biofuel combustion products, cigarette smoke and welding fumes, of which CO is known to comprise a significant component.<sup>1 2</sup> Data from a large case control study found cheaper cooking fuels were significantly associated with increasing rates of cataract.<sup>3</sup> Indeed animal studies also confirm that wood smoke condensates result in lens opacities forming in rats.<sup>4</sup> Data from prospective studies of smoking and cataractogenesis also adds to growing evidence that inhalation of cigarette smoke is a significant risk factor for cataract formation.<sup>3</sup>

Among specific cataract entities, nuclear cataracts are most commonly reported in association with smoking and a dose-response association demonstrated.<sup>3 5 6</sup> Furthermore, 63% greater risk of cataract was found in women who were heavy smokers compared with those who had never smoked.<sup>2</sup> These observations suggest that CO is likely to play a significant role in the pathogenesis of acquired lens opacities.

Trese reported a patient who experienced intermittent exposure to CO over 16 months.<sup>7</sup> This patient had constricted visual fields, peripheral neuropathy and tortuous retinal vessels but no lens opacity. CO and carbon disulfide have been associated with alterations in retinal and choroidal blood flow velocity and pulsation amplitude which may explain retinal vascular changes.<sup>8</sup> In the latter case, the association between optic neuropathy and CO exposure may result from mechanisms similar to that in tobacco amblyopia. Systemic symptoms such as persistent lethargy, headaches and syncope in our case are in keeping with CO poisoning. Seizures and intracranial lesions have also been reported including cortical and subcortical lesions and even confirmed by reduced metabolism of the posterior temporal and occipital lobes by <sup>18</sup>F-fluorodeoxyglucose positron emission tomography scanning.<sup>9</sup> Basal ganglia and white matter changes, and typically lesions of the globus pallidus have been reported in association with CO poisoning.<sup>9-12</sup>

This unusual case of non-fatal chronic CO poisoning demonstrates systemic manifestations of chronic CO exposure including the infrequently reported albeit recognised intracranial abnormality. The development of lens opacities however is a novel finding. This case underwent the longest period of chronic CO exposure in the literature to date. The lengthy nature of CO exposure together with multisystem involvement is highly suggestive of CO as the causative factor of cataract in this case. Possible mechanisms include increased oxidative stress leading to precipitation of lens proteins.<sup>13-15</sup> Evidence in keeping with this is that smokers are known to have low plasma antioxidant levels and the relative risk of cataract formation reduces correlates inversely with dietary antioxidant intake.<sup>1 2</sup> Future enquiry may help uncover environmental factors and metabolic processes involved in cataractogenesis.

## Learning points

- ▶ Environmental factors including cigarette smoking may be significant risk factors for acquired lens opacities.
- ▶ Early onset of cataract should prompt enquiry into patients' systemic health given the association between oxidative stress and cataractogenesis.
- ▶ CO poisoning may present with a wide spectrum of clinical signs and non-specific systemic features.

**Competing interests** None.

**Patient consent** Obtained.

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