Recurrent cardiac failure of environmental origin

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Generalized cellular hypoxia can precipitate cardiac failure through high demand on the heart.

CASE HISTORY

A woman of 82 with a history of hypertension and mild stable angina was admitted after 6 days of increasing shortness of breath and palpitations. There had been no chest pain and her medication had not been changed. On examination, she was dyspnoeic at rest and had an apical heart rate of 120/min, irregular. Blood pressure was 140/95 mmHg and oxygen saturation was 93% on room air. There were diffuse crepitations in both lung fields, extending to the mid-zones, but no cardiac murmurs.

Full blood count and blood chemistry was normal; cardiac enzymes did not rise. The electrocardiogram showed atrial fibrillation without ischaemic changes or voltage abnormalities. On a chest radiograph there was pulmonary oedema in the presence of mild cardiomegaly (cardiothoracic ratio 0.6). She was diagnosed as having left ventricular failure secondary to atrial fibrillation and responded well to diuretics, an angiotensin converting enzyme inhibitor and oxygen therapy. In view of the duration of symptoms we opted for rate control with digoxin, and anticoagulation, rather than chemical or electrical cardioversion. An echocardiogram showed mild left ventricular impairment with no structural abnormalities.

She was discharged after 72 hours, only to return later the same day with an identical presentation. Again she responded to standard therapy, with clinical and radiographic resolution of pulmonary oedema. The atrial fibrillation remained well controlled and anticoagulation was adequate. Again there was no rise in serial cardiac enzymes. The cause of this decompensation was unclear, but she was discharged home with a view to direct-current cardioversion after 4 weeks. However, the patient returned

that evening, for a third time, with acute cardiac decompensation. This time the paramedics who had collected the patient informed the admitting team that there was a smell of gas in the apartment.

The carboxyhaemoglobin in arterial blood was 20%. She was treated with high-flow oxygen and standard therapy for the cardiac failure. In the absence of neurological abnormalities, and in view of her previous recovery with normobaric oxygen therapy, hyperbaric oxygen was withheld (although her case was discussed with the local hyperbaric oxygen unit). She responded well, and while she was in hospital the gas company located and fixed faulty ventilation that was causing excessive ambient levels of carbon monoxide (CO) in her apartment and in nine other apartments in the same block. The patient remained well at follow-up 2 weeks and 4 months later.

COMMENT

CO is a colourless, odourless and tasteless gas that diffuses rapidly across the alveolar capillary membrane and binds tightly to iron components of haemoglobin and other haemoproteins, forming carboxyhaemoglobin (CO-Hb). The toxicity seems to result from a combination of tissue hypoxia and direct CO-mediated cellular damage. The affinity of CO for haemoglobin is 200–250 times greater than that of oxygen, causing a shift of the oxygen–haemoglobin dissociation curve to the left.¹ This results in impaired oxygen release and cellular hypoxia. Other effects are oxidative stress on cells with the production of oxygen radicals, and lipid peroxidation with reversible demyelination in the central nervous system.²

Most cases of CO poisoning present with flu-like symptoms. Other common features are tachycardia and tachypnoea, which are compensatory mechanisms for cellular hypoxia. Nausea and vomiting, presyncope, syncope and seizures are less frequent manifestations. An increased cardiac output due to the cellular hypoxia can result in or exacerbate acute coronary syndromes, pulmonary oedema and arrhythmias. Despite having no acute signs of cerebral impairment, about 15% of patients develop the 'delayed neuropsychiatric syndrome', which typically occurs between 2 and 28 days after exposure and can include parkinsonism, dementia, incontinence, psychosis and personality changes.³ The severity of any of the above symptoms does not correlate well with CO-Hb concentrations; duration of exposure seems more important.⁴ What is clear is that one rarely observes the classic findings of cherry-red lips, cyanosis and retinal haemorrhages.²

Most modern blood gas machines can measure CO-Hb. However, if there is a delay between CO exposure and

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measurement of CO-Hb, levels may have fallen substantially. Measurement of CO either in the exhaled breath or in the ambient air at the scene can be helpful. The cornerstone of treatment is high-flow oxygen. This reduces the half-life of CO-Hb to 4–6 hours. With hyperbaric oxygen this comes down to 15–30 minutes.³ Whilst hyperbaric oxygen confers theoretical advantage, the absolute indications for its use remain in question. In England, Northern Ireland and Wales, the British Hyperbaric Association runs a 24-hour telephone helpline (01752 209 999) to discuss cases and advise on referral. CO poisoning is avoidable, and household detectors are widely available at modest cost.

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A not-so-simple collapse

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In unexplained collapse, the possibility of carbon monoxide poisoning must not be neglected.

CASE HISTORY

A man of 40 was admitted having collapsed. His only medical history was treatment for depression. Examination was unremarkable except for complete short-term memory loss to such an extent that he was unable to recall his address. Blood tests and electrocardiography were normal. CT and MRI (Figures 1 and 2) revealed symmetrical lesions in the globus pallidus and posterior putamen. There were

also ill-defined lesions in the posterior periventricular white matter. The following day the patient's neighbour reported finding a hosepipe attached to his car's exhaust pipe.

The radiological findings are characteristic of carbon monoxide poisoning. The patient was referred for hyperbaric oxygen therapy. The results of psychometric testing improved but he was left with defects that limited his everyday function.

Figure 1 CT of brain

Figure 2 MRI of brain

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