EMERGENCY CASEBOOKS

Glioblastoma presenting as carbon monoxide poisoning

Mark Turner, Robin J Clark

A 29 year old man who lived alone was brought to his local casualty department by his mother. He had been unwell for about three days with nausea, difficulty finding words, and headache. On the day of admission he had slept late and his symptoms had become worse. He reported to his mother by telephone that he was unwell and was not going to work. That evening she found him confused, unable to stand, and unable to answer questions. His rented flat was heated by gas appliances which had not been serviced recently.

On arrival at the accident and emergency department of his local hospital he was found to be tremulous, agitated, nauseated, and unable to answer questions. He was pale, sweaty and unable to stand, his tendon reflexes were brisk but his plantar responses were normal. Neck stiffness and fever were absent, fundoscopy was normal. Carboxyhaemoglobin (COHb) was 18%, confirming carbon monoxide poisoning. The arterial pH was 7.37.

In view of the profound neurological symptoms and signs he was transferred 50 miles by ambulance for hyperbaric oxygen therapy (HBOT). Treatment started 10 hours after presentation with 60 minutes of oxygen therapy at 18 metres depth (partial pressure of oxygen 2.8 bar) and a 30 minute decompression, during which the patient continued to breathe 100% oxygen. He remained unable to report the day, date, or year. Short term memory problems persisted and he remained drowsy and confused. Because of his incomplete recovery, further HBOT was given. After four further HBOT sessions he knew the month and year and that he was in a hospital in Gosport, but his short term memory remained poor. He correctly performed a serial 7s test from 93 to 72 but could not continue. In view of this objective improvement, further HBOT was given. After a total of 11 HBOT sessions he was fully oriented, serial 7s had improved, and short term memory was better; however, response to each treatment was decreasing.

In view of his incomplete recovery a magnetic resonance scan of the brain was performed (see fig 1). A mass was identified, which enhanced after an intravenous injection of gadolinium, in the left occipitoparietal region of the brain. The mass was surrounded by a significant amount of oedema and was causing midline shift. The

radiologist's report of glioblastoma multiforme was subsequently confirmed by brain biopsy.

This patient undoubtedly had carbon monoxide poisoning in addition to his brain tumour. The relative contribution of the carbon monoxide poisoning to his cognitive abnormalities is unclear since he had a relatively low COHb. However, the initial COHb level does not correlate well with severity of poisoning¹ and our patient may have built up substantial tissue levels of carbon monoxide during repeated and prolonged exposure. It is the tissue poisoning (for example of cytochromes) rather than blood poisoning which determines severity of carbon monoxide poisoning.² It is our practice to continue HBOT until either the patient is fully recovered or no further improvement occurs despite repeated HBOT. This patient received a total of 11 treatments, which is most unusual; the maximum number given to any patient in the period 1991–95 was three in a series of 48 cases.

We speculate that the continued improvement after multiple exposures to HBOT was not due solely to its effect in carbon monoxide poisoning and that it influenced the oedema

Figure 1 Magnetic resonance image: inversion recovery sequence showing the glioblastoma with surrounding oedema.

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around the tumour. We were unable to observe the effect of withdrawal of HBOT because corticosteroid treatment was introduced as soon as the mass lesion became apparent; however, improvement was maintained between daily treatments. Two forms of cerebral oedema are described.³ Vasogenic cerebral oedema is due to a change in capillary permeability and can occur around mass lesions. Cytotoxic cerebral oedema is secondary to cellular damage, usually hypoxia. Corticosteroids can reduce vasogenic oedema but are not thought to be of value in cytotoxic oedema. HBOT may reduce oedema by causing cerebral vasoconstriction⁴ and has been shown to reduce intracranial pressure.⁵ After carbon

monoxide poisoning any patient left with neurological sequelae should be considered for brain imaging to exclude coexistent pathology.

- 1 Norkool DM, Kirkpatrick JN. Treatment of acute carbon monoxide poisoning with hyperbaric oxygen: a review of 115 cases. *Ann Emerg Med* 1985;14:1168–71.
- 2 Goldbaum LR, Orellano T, Dergal E. Mechanism of the toxic action of carbon monoxide. *Ann Clin Lab Sci* 1976;6: 372–6.
- 3 Mendelow AD. Raised intracranial pressure, cerebral oedema, hydrocephalus and intracranial tumours. In: Walton J, ed. *Brain's diseases of the nervous system*. 10th Ed. Oxford: Oxford University Press, 1993: 146.
- 4 Hammarlund C. The physiologic effects of hyperbaric oxygen. In: Kindwall EP, ed. *Hyperbaric medicine practice*. Flagstaff, Arizona: Best Publishing Company, 1994: 20.
- 5 Lample LA, Frey G. Hyperbaric oxygen in intracranial abscess. In: Kindwall EP, ed. *Hyperbaric medicine practice*. Flagstaff, Arizona: Best Publishing Company, 1994: 664.

Covert tamponade

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Cardiac tamponade is a very uncommon complication of open cardiac surgery, whereas pericardial effusion is common.^{1,2} Studies put the incidence of effusion at 64% and tamponade at 0.4–1.9%.^{1,2}

A 58 year old man presented to the accident and emergency department with a sudden onset of fatigue and sweatiness. He had had aortic valve replacement three weeks earlier and was a known non-insulin dependent diabetic. On examination he was pale and clammy. His blood pressure was 115/70 mm Hg, pulse 100 with heart sounds I+II+ prosthetic S2 clearly audible. On detailed examination his femoral pulses were not palpable on inspiration (pulsus paradoxus).

A portable ultrasound machine in the department (Shimadzu SDL-30) was used to visualise the heart. This identified fluid within the pericardial sac. Chest radiography showed an enlarged heart (fig 1). Formal echocardiography showed 2.65 cm of fluid posterior to the heart causing tamponade (fig 2). He was taken to cardiac theatre where 50 ml of serous fluid was aspirated with dramatic clinical improvement. A further 510 ml was drained subsequently. He made an uneventful recovery.

Late tamponade presents diagnostic difficulty as clinical features are vague and atypical.³ The classical Beck's triad, distended neck veins, hypotension and muffled heart sounds, is not always present. Diagnosis is made rapidly with echocardiography.³

- 1 Pepi M, Muratori M, Barbier P, et al. Pericardial effusion after cardiac surgery: incidence, site, size and haemodynamic consequences. *Br Heart J* 1994;72:327–31.
- 2 Yilmaz AT, Arslan M, Demirklic U, et al. Late posterior cardiac tamponade after open heart surgery. *J Cardiovasc Surg* 1996;37:615–20.

Figure 1 Chest radiograph showing enlarged cardiac shadow.

Figure 2 Echocardiography showing fluid (A) in the posterior aspect of the pericardial sac.

- 3 Chuttani K, Tischler MD, Pandian NG, et al. Diagnosis of cardiac tamponade after cardiac surgery: relative value of clinical, echocardiographic and haemodynamic signs. *Am Heart J* 1994;127(4 Pt 1):913–8.

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