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Delayed neuropsychiatric syndrome after carbon monoxide poisoning

SHORT CASE REPORT

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Background

Delayed neuropsychiatric syndrome (DNS) is a well-known complication following carbon monoxide (CO) poisoning and develops in up to 50 % of adult survivors. The syndrome is probably immunologically mediated. Common symptoms are slowness, Parkinsonism and cognitive impairment.

Case presentation

A woman in her forties started to show gradually increasing symptoms of DNS a few days after an episode of severe CO poisoning. She received methylprednisolone 1 g intravenously on 3 consecutive days at around 7 weeks after the poisoning, with an immediate positive response to motor deficit symptoms. Thereafter, she gradually recovered and returned to full-time employment 4.5 months after the steroid treatment.

Interpretation

The role of steroids in this patient's recovery is uncertain. However, successful high-dose steroid treatment for patients with ongoing DNS progression after CO poisoning has been reported previously in the literature. The authors recommend more attention to the risk of DNS after CO poisoning and further research on treatment options.

After acute carbon monoxide poisoning, up to half of those who survive can develop delayed neuropsychiatric syndrome. Survivors should therefore be closely monitored by healthcare professionals. The long-term prognosis for the syndrome is unknown.

A previously healthy woman in her forties suffered carbon monoxide (CO) poisoning during a camping trip. She experienced acute unconsciousness and seizures for a period of 5–10 minutes. The woman was admitted to hospital for observation and received normobaric oxygen therapy with 100 % oxygen intermittently during transportation and in the hospital setting. Around six hours after admission, her carboxyhaemoglobin (CO-Hb) level was 7.6 % (reference range < 3 %). Other blood tests showed normal findings.

At the hospital, the patient was assessed as asymptomatic, except for a headache. The relatively low CO-Hb level was considered an indication of no acute CO poisoning, and she was discharged in normal condition the next morning, with no planned follow-up.

A few days after discharge, the patient began to develop neurological symptoms: reduced cognition and psychomotor speed, concentration problems, difficulty focusing her gaze, light sensitivity, gait disturbances, fatigue and reduced fine motor skills. The symptoms led to her being medically certified as unfit for work after three weeks, and her condition gradually worsened.

Based on a published case report highlighted by the patient's family (1), the patient was admitted to the Department of Neurology seven weeks after the poisoning and was given methylprednisolone, 1 gram intravenously for three

consecutive days.

Upon readmission, reduced psychomotor speed and fine motor skills were found bilaterally, along with bilateral dysdiadochokinesia, and reflexes in the upper half of the normal range were observed in the upper and lower extremities. Clinical examination was otherwise unremarkable. Head MRI and EEG revealed no pathology. Some immediate improvement was observed in motor speed following treatment with methylprednisolone.

The first neuropsychiatric assessment was performed three weeks after the steroid treatment. Light sensitivity and problems with fine motor skills, concentration and stamina were indicated. The tests were repeated one year later, and the results were within expected performance levels, including faster psychomotor speed, improved executive functions, improved concentration and normalised visual function.

After the steroid treatment, the patient made a gradual recovery. She returned to full-time work 4.5 months after treatment, initially requiring adaptations in the form of breaks and reduced lighting.

Discussion

This patient's story illustrates a typical course of DNS after CO poisoning. This well-documented phenomenon affects up to half of all survivors (1–5). Symptoms appear days to weeks after poisoning and are caused by immunologically mediated inflammation in the central nervous system (1, 6). Adult survivors are at higher risk than younger ones (3), but otherwise there are no clear indicators of who will be affected (4, 7).

Our patient's CO-Hb level was near normal upon admission, but there may still have been an indication for hyperbaric oxygen therapy given that she had been treated with oxygen, and this shortens half-life of carboxyhaemoglobin (8). However, there is no solid evidence that acute treatment with hyperbaric oxygen reduces the risk of sequelae (4, 8).

Some studies suggest that DNS has a relatively good prognosis (2, 3). Other studies document significantly increased mortality among survivors and a high incidence of long-term work incapacity and severe psychiatric illness (9, 10).

Evaluating the impact of steroid treatment on this patient's recovery is challenging. The absence of objective neuropsychiatric test results prior to the administration of steroids complicates the interpretation. Treatment with high-dose steroids may have slowed an ongoing immunopathological process in the central nervous system. The administration of high-dose steroids to treat neuropsychiatric sequalae after CO poisoning seems to be more prevalent in Asian countries (1, 11), and this is often combined with other treatment (7, 12).

Norwegian treatment recommendations state that sequelae represent a possible complication (13), and recommend a comprehensive neurological assessment shortly after poisoning as a baseline for further follow-up. We believe that survivors of CO poisoning should be informed about the risk of

developing DNS and recommend close follow-up of patients and their families. Further research is warranted to conclude whether more patients should be given high-dose steroid treatment.

The patient has consented to publication of this article.

The article has been peer-reviewed.

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