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CASE REPORT

Decerebrate posturing in alcoholic coma

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SUMMARY

Two cases of alcoholic coma are presented where extensor responses to noxious stimuli are demonstrated. Decerebrate posturing normally indicates severe structural or functional depression of midbrain function but can be caused by depressant drugs. Blood alcohol measurements are a vital test in the comatose patient as the clinical picture may be caused, or temporarily significantly worsened, by severe alcohol intoxication. The preservation of pupillary light reflexes in the presence of deep coma with decerebrate posturing should alert the clinician to a possible metabolic cause for the coma, including alcohol. Nevertheless, a diagnosis of alcoholic coma should not be made unless the blood alcohol concentration is grossly elevated and other causes of coma have been excluded by careful physical examination, blood glucose and electrolyte measurement, skull radiography and, in the absence of a rapid improvement, computerized tomography.

Key words: alcohol, coma, intoxication

INTRODUCTION

The combination of coma and alcohol intoxication presents accident and emergency (A&E) department medical staff with a diagnostic challenge fraught with potential dangers. Abnormal extensor posturing in comatose patients is normally indicative of severe structural or functional midbrain depression, however, we wish to report two cases of alcoholic coma in which this abnormal response was present.

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Case one

A 20-year-old female was brought to the A&E department by ambulance with a history of having been thrown off a motorcycle. A helmet was found at her side.

On arrival she was unresponsive and tolerating an oro-pharyngeal airway. Her cervical spine was immobilized and initial observations revealed a haemodynamically stable patient. During oro-pharyngeal suction she developed an extensor response (decerebrate posturing) involving all four limbs. Her pupils were equal, dilated to 7 mm and sluggishly reactive to light. She was arreflexic with no plantar responses. Her blood glucose was 6.2 mmol L^{-1} and there was a faint smell of alcohol from her breath.

She was commenced on 100% oxygen and an intravenous crystalloid infusion. A urinary catheter was inserted draining a large volume of clear urine from a grossly distended bladder.

There were no external signs of head injury (although the patient had been wearing a helmet) and further examination did not reveal any evidence of injury elsewhere. Routine biochemistry, haematology and arterial blood gases were all normal. Blood alcohol concentration was significantly elevated at 85.7 mmol L^{-1} (395 mg dl^{-1}). Plain radiography of cervical spine, chest, pelvis and skull did not show any abnormalities.

Given the above findings, she was intubated using a rapid sequence induction technique and transferred to the regional neurosurgical centre for immediate brain computerized tomography.

During transfer, her right pupil dilated to 8 mm and became unreactive to light so she was given 100 ml of 20% mannitol intravenously. However, on arrival at the neurosurgical centre, her pupils were equal (2 mm) and reacting to light. She continued to demonstrate extensor posturing on tracheal suction but was otherwise unresponsive. Oculo-vestibular reflexes were normal. The computerized tomogram showed no abnormality.

By this stage the patient's parents had been traced and were able to confirm previous episodes of binge drinking and multiple parasuicide attempts. The police confirmed that an empty bottle of spirits had been found beside an undamaged motorcycle. A drug screen was normal. She extubated herself

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5 h later and 10 h after admission her conscious level had returned to normal. After psychiatric consultation and having made an uneventful recovery, she was discharged without follow-up 4 days later.

Case two

A 38-year-old female was brought to the A&E department by ambulance shortly after mid-day, having been found unconscious in the street. The only history available on admission was of previous alcohol abuse.

On examination she was comatose and smelling strongly of alcohol. She tolerated an oro-pharyngeal airway but was breathing spontaneously with a normal respiratory rate and pattern. She was hypotensive (90/60 mmHg) with a heart rate of 90 beats min^{-1}. There was no eye opening or verbal response but she was noted to be extending all four limbs to pain, although the right side moved more than the left. Pupils were unequal (right 6 mm, left 4 mm) both being minimally reactive to light. Eye movements were roving and dysconjugate. Her fundi were normal and there was generalized hyporeflexia with no plantar responses. There was no external evidence of head injury and no other abnormal findings were noted on physical examination. Blood glucose was 5.8 mmol L^{-1}.

Skull and chest radiography was normal and an electrocardiograph unremarkable. Biochemistry, including arterial blood gases revealed hypernatraemia (151 mmol l^{-1}), a mild metabolic acidosis, no evidence of hypoxia and a blood alcohol concentration of 108 mmol L^{-1} (498 mg dl^{-1}). Drug screen was negative.

Computerized tomography of her brain was arranged but not subsequently performed. Previous hospital records revealed two recent similar presentations with alcoholic coma although without extensor posturing. In addition, within 1 h of admission the patient was demonstrating a flexor response to pain.

The patient was admitted to a general medical ward with a presumptive diagnosis of alcohol intoxication. She was closely observed until the following morning by which time she had made a complete recovery. She declined further treatment or follow-up and was therefore discharged.

DISCUSSION

The two patients by their own subsequent admission, had a diagnosis of alcoholic coma, confirmed by their grossly elevated blood alcohol concentrations and spontaneous recovery within a few hours. Both patients demonstrated abnormal extensor posturing to noxious stimulation which is highly unusual with alcohol intoxication but widely recognized with other 'metabolic' causes of coma.[1]

While features of alcohol intoxication correlate well with blood alcohol concentrations, coma should not be attributed to alcohol intoxication below a concentration of 65 mmol L^{-1}.[2]

In general depth of coma is usually measured using the Glasgow Coma Scale[3] and as coma deepens motor function in response to stimulation provides valuable localizing information and can serve as a guide to the course and severity of the illness.[4] However, as the nuclear masses and pathways that regulate motor activity are different from those mediating consciousness and arousal, caution must be exercised when gauging the depth of coma — especially with diffuse or metabolic causes. Despite this, abnormal flexor or extensor responses do appear to suggest structural or functional impairment of certain cortical and sub-cortical areas of the brain.[5]

Extensor posturing is usually seen with large or expanding lesions of the cerebral hemispheres such as intracranial haemorrhage and reflects diencephalon and mid-brain dysfunction. It can also be seen with posterior fossa or cerebellar lesions as they begin to affect parts of the mid-brain. Severe metabolic disorders including drug intoxication can depress the function of the forebrain and deeper part of the diencephalon leading to such a clinical response. In the absence of hypoxia we suggest that this was the effect of alcohol in both the cases reported here.

Neurosurgeons have long recognized the significance of abnormal motor responses in brain injury in terms of mortality and eventual outcome. In head injury, Jennett *et al.*[6] found that a withdrawal or abnormal flexion (decorticate) response was associated, in 63% of cases, with death or a persistent vegetative state. Extensor or absent responses increased this figure to 83%. It is therefore of paramount importance to determine the cause of the abnormal posturing to ensure that surgically correctable lesions are not missed. The cases reported here demonstrate that alcohol may occasionally be responsible for decerebrate posturing in cases where severe head injury is suspected.

One clue in the differentiation between structural and metabolic causes of deep coma is the state of

the pupils. Preserved pupillary light reflexes despite abnormal or absent motor responses suggest a diffuse or metabolic aetiology. The cause of the pupillary inequality in these cases is unclear but initially was a further suggestion of a structural cause.

Therefore, in comatose patients, the need for careful physical examination, blood glucose estimation, blood alcohol measurement and skull radiography is underlined.[7,8] In the absence of rapid improvement in conscious level, urgent computerized tomography is required.

REFERENCES

1. Plum F. & Posner J.B. (1980) *The Diagnosis of Stupor and Coma*, 3rd edition, pp. 184–185. F.A. Davis Company, Philadelphia.
2. Rutherford W.H. (1977) Diagnosis of alcohol ingestion in mild head injuries. *Lancet* **i**, 1021–1023.
3. Teasdale G. & Jennett W.B. (1974) Assessment of coma and impaired consciousness. *Lancet* **ii**, 81–83.12.
4. Jennett W.B. & Teasdale G. (1977) Aspects of coma after severe head injuries. *Lancet* **i**, 878–881.
5. Plum F. & Posner J.B. (1980) *The Diagnosis of Stupor and Coma*, 3rd edition, pp. 65–70. F.A. Davis Company, Philadelphia.
6. Jennett W.B. Teasdale G. & Braakman R. (1976) Predicting outcome in individual patients after severe head injury. *Lancet* **i**, 1031–1034.
7. Quaghebeur G. & Richards P. (1989) Comatose patients smelling of alcohol. *British Medical Journal* **299**, 410.
8. Tennant W.G. & Robertson C.E. (1989) Comatose patients smelling of alcohol (letter). *British Medical Journal* **299**, 790–791.