Case report

An undiagnosed stupor in the acute medical unit: a case of malignant catatonia


From the Hammersmith Hospital, Imperial College Healthcare NHS Trust, London, UK

Address for correspondence to Dr G. Adams, Hammersmith Hospital, Du Cane Road, London W12 0HS, UK. email: g.adams@doctors.org.uk

Learning Point for Clinicians

Malignant catatonia is a rare cause of a reduced Glasgow Coma Score. It is difficult to diagnose as it has no specific investigation findings, and it may mimic a number of other conditions. A dramatic response to benzodiazepines however is characteristic and may assist in the diagnosis.

Case

A 45-year-old man was brought to our hospital by Ambulance having been found lying unconscious on the pavement. On admission he was obtunded with a Glasgow Coma Score (GCS) of 6 (E1, V1, M4), but maintaining his airway. He was pyrexial (temperature 38.5°C) and tachycardic with a pulse rate of 106 beats per minute. His blood pressure was 142/96 mmHg and capillary blood glucose 6.2 mmol/l. There was no evidence of trauma or needle marks. Neurological examination revealed mildly increased tone and brisk reflexes. His pupils were 4 mm, equal and reactive to light. Fundoscopy showed normal optic discs. Cardiovascular, respiratory and abdominal examinations were normal.

Blood tests for full blood count, urea and electrolytes, liver function, bone profile, vitamin B12, folate, thyroid function and creatine kinase (CK) were normal. HIV, serum cryptococcal antigen, hepatitis, syphilis and mumps serology tests were negative. His c-reactive protein was 30 mg/l. Toxicology screens for drugs (paracetamol, salicylates, alcohol, cocaine, amphetamines, opiates and cannabis) and heavy metals (lead, mercury and cadmium) were negative. Contrast CT and MRI brain imaging found no abnormality. He was treated empirically for meningitis and encephalitis with intravenous ceftriaxone, acyclovir and ampicillin. However, lumbar puncture opening pressure was 21 cmH2O (normal range 8–21 cmH2O) and cerebrospinal fluid (CSF) white cell count was <1, protein 0.42 g/l and glucose 3.8 mmol/l (serum 5.2 mmol/l). CSF PCR found no evidence of HSV, VZV or enteroviruses. His autoimmune serology, including anti-VGKC, anti-NMDA receptor, anti-Hu, anti-Yo and anti-Ri antibodies, ANA and ANCA, was also negative. Multiple blood cultures grew no organisms.

The patient remained unresponsive and pyrexial, requiring intensive nursing and nasogastric (NG) feeding. On Day 5, as part of our workup for non-convulsive epilepsy, a trial of intravenous diazepam (5 mg) was given. Remarkably the patient became completely alert (GCS 15/15), albeit briefly (<5 min) after which he reverted back to stupor. Further small diazepam boluses elicited similar responses but intravenous phenytoin and levetiracetam had no effect. His EEG, while unresponsive, was normal. Whilst awake he gave a history of depression. He took no medications. The clinical picture, fever, hypertension, tachycardia and the
response to benzodiazepines with otherwise normal investigation findings supported a diagnosis of malignant catatonia. He was started on regular lorazepam that was titrated up over 3 days to 2 mg QDS. Within 4 days of this treatment the patient was fully awake. When stable, the psychiatry team took over his ongoing care, ultimately diagnosing him with an underlying affective disorder.

Catatonia is a neuropsychiatric syndrome characterized by motor disturbance. Common features include rigidity, posturing and mutism but excessive motor activity and automatism may also be present. The diagnosis of malignant catatonia requires the additional features of fever, delirium and autonomic instability.\textsuperscript{1–3} The pathophysiology of catatonia is unclear, however it is often associated with psychiatric conditions, notably mood disorders.\textsuperscript{2} Epilepsy, infection and drug overdose are important differentials. A typical feature of catatonia is a rapid, temporary relief of signs after a benzodiazepine challenge.\textsuperscript{4} For ongoing treatment, lorazepam 1–4 mg/day is recommended initially. Higher doses (8–24 mg/24h) can be used if symptoms do not fully respond after 48 h.\textsuperscript{5} Approximately 80\% of cases respond to this treatment. If no resolution after 5–7 days, electroconvulsive therapy should be considered. This has proved efficacious in even those cases resistant to benzodiazepines.\textsuperscript{4}

Antipsychotics and other dopamine antagonists (e.g. antiemetics) should be avoided due to the risk of the neuroleptic malignant syndrome. Without prompt treatment malignant catatonia has a significant morbidity and mortality, mainly from sequelae of the prolonged stupor and immobility.\textsuperscript{2} In conclusion, malignant catatonia is an unusual diagnosis but should be considered in all patients with an unexplained reduced GCS.

Conflict of interest: None declared.

References

3. Fink M, Taylor MA. The catatonia syndrome: forgotten but not gone. \textit{Arch Gen Psychiatry} 2009; \textbf{66}:1173.