Unconsciousness and Coma
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CHAPTER 1

Case History
A 57-year-old man is found in an unconscious state at home. He was in bed when his wife left at 7.00 a.m. that morning to go to work. On her return home at 3.45 p.m., he was still in the same position in bed, unrousable, incontinent of urine, and the cup of tea she had left for him was untouched. He has been unwell recently, and prescribed a course of antibiotics and co-codamol from his General Practitioner for a discharging ear infection. He suffers from hypertension, type 2 diabetes mellitus and long-standing depression. His medication list shows that he has been prescribed gliclazide 80 mg twice daily, atenolol 25 mg once daily, ramipril 5 mg once daily and amitriptyline 25 mg once daily. He has no known allergies. His wife informs you that he has had bad headaches recently, but that no-one else at home has been unwell.

Question: What differential diagnosis would you consider from the history?

This man is in a coma, which is defined as ‘unrousable unresponsiveness’. Using the objective clinical assessment tool, the Glasgow Coma Score (see Table 1.1), coma is defined as a score of 8 or less. Those patients with a score between 14 and 9 are defined as having altered consciousness and those with a top score of 15 are normal, alert and orientated. When considering a differential diagnosis for the cause of a patient’s unresponsiveness it is important to consider those conditions that are easily reversible first.

Hypoglycaemia
The patient is a known diabetic. Hypoglycaemia or, less commonly, hyperglycaemia can result in altered consciousness and must be actively diagnosed and promptly treated. A simple bedside glucose test will identify abnormalities in blood glucose levels and will guide appropriate therapy.

It is essential that any patient with confusion, altered consciousness, coma or focal neurological signs has their blood glucose estimated as part of the initial assessment. Neurological signs resulting from hypoglycaemia usually resolve quickly with treatment, though the failure to recognise and treat hypoglycaemia promptly may lead to permanent neurological damage.

Drugs and alcohol
Excess alcohol with or without other prescription or recreational drugs is the commonest cause of altered consciousness and not quickly reversible. Of all the drugs that affect a patient’s consciousness (see Box 1.1) opiates are the only group that are readily treatable. Opiate excess leads to coma, and life-threatening respiratory depression, but thankfully can be quickly and effectively treated by the antagonist naloxone. The signs of opiate poisoning are seen in Box 1.2. Naloxone should be administered to any patient with any signs compatible with opiate poisoning.

Box 1.2  **Signs of opiate ingestion**
- Depressed conscious level
- Depressed respiratory rate
- Pin point pupils
- Needlestick trackmarks

Box 1.3  **Signs of tricyclic anti-depressant overdose**
- Dry skin and mouth
- Urinary retention
- Tachycardia
- Ataxia
- Jerky limb movements
- Divergent squint
- Altered level of consciousness

Opiate excess should be considered in this man who has had access to the simple analgesic co-codamol, which is a combination of paracetamol and the opiate codeine.

Likewise amitriptyline overdose, a common cause of coma, should be considered in the light of his depression and access to the medication. The clinical signs of tricyclic anti-depressant overdose are found in Box 1.3.

**Intracranial haemorrhage**
Vascular causes of coma are common. This man is known to have hypertension, which puts him at risk of intracranial haemorrhage. The cardinal features of an intracranial haemorrhage are sudden onset of headache, altered consciousness and focal neurological signs. Spontaneous intracranial haemorrhage usually occurs either into the subarachnoid space or into the ventricles and brain substance itself giving rise to either subarachnoid haemorrhage or intra-parenchymal haemorrhage respectively (see Figure 1.1).

Strokes due to cerebral infarction usually present differently to intracranial haemorrhages. The most important difference is that in most strokes consciousness is not impaired. There may be difficulty communicating with the patient, due to expressive or receptive dysphasia, but conscious level itself is not often altered. In brainstem infarctions, which can produce ‘locked in syndromes’ patients are aware of their surroundings, but unable to respond or communicate, so the patient can appear to be comatose.

**Infection**
Infection can lead to coma, either systemic infection as in a septicaemic illness, or intracranial infection such as meningitis or encephalitis. Patients with meningitis or encephalitis may present in coma especially if there is raised intracranial pressure.

There will often be a preceding phase characterised by symptoms suggestive of meningal irritation (stiff neck, headache, photophobia), the signs of raised intracranial pressure (irritability, altered level of consciousness, vomiting, fits) and infection (fever, lethargy). If *Neisseria meningitidis* is the causative organism, the characteristic petechial/purpural rash is seen in approximately 50% of patients (see Figure 1.2); other organisms can cause less well-defined rashes. Other causative organisms can be seen in Table 1.2.

Prompt recognition of the possibility of meningitis is vital, as if left untreated, it has a mortality rate approaching 100%.

This man has a discharging ear infection which could potentially be the source of intracranial infection.
Unconsciousness and Coma

Post-ictal state
Following a generalised seizure, patients can be unresponsive as part of a post-ictal state. Typically, though the patient may be in coma immediately following the fit, their conscious level quickly improves within 30–60 minutes, by which time they are usually able to provide you with a history of events. Evidence of urinary incontinence and tongue biting with bleeding in or around the mouth supports the diagnosis but is not diagnostic. The duration of this man’s unconscious state would be out of keeping with a post-ictal state.

However, on examination it would be important to look for evidence of ongoing seizure activity (e.g. hypertonicity) as status epilepticus may be a possibility.

Psychogenic coma
Psychogenic coma is uncommon and accounts for less than 2% of all cases of coma and is strictly a diagnosis of exclusion. Accordingly, the patient must be assessed thoroughly to check for other causes of altered consciousness as conditions such as hydrocephalus and vertebral artery dissection have on occasions been initially labelled as psychogenic. There are a number of clinical features that may suggest that the patient is physiologically awake (see Box 1.4) but none could be said to be diagnostic.

Causes of coma not suggested by the history

Trauma
Head injury is one of the commonest causes of coma but this man’s history is not suggestive of an intracranial injury.

Structural causes
Structural causes of coma are relatively rare. Intracerebral space-occupying lesions cause coma, either as a result of their mass effect on the brain, or because of the anatomical position of the lesion.

By far the most common cause of cerebral space-occupying lesions are tumours, either primary or secondary. Other causes include cerebral abscess, cysts (e.g. cysticercosis, third ventricular colloid cysts) and granulomas (e.g. sarcoidosis, TB).

Table 1.2 Causes of meningitis/encephalitis.

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
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</thead>
<tbody>
<tr>
<td>Bacterial</td>
<td><em>Neisseria meningitidis</em>, <em>Streptococcus pneumoniae</em>, listeria (elderly), <em>Haemophilus influenzae</em>, TB</td>
</tr>
<tr>
<td>Viral</td>
<td>Herpes simplex, Coxsackie, mumps, echovirus, HIV</td>
</tr>
<tr>
<td>Fungal</td>
<td><em>Cryptococcus neoformans</em></td>
</tr>
<tr>
<td>Other</td>
<td>Drugs (trimethoprim/NSAIDs), sarcoidosis, systemic lupus erythematosus</td>
</tr>
</tbody>
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Box 1.4 Atypical clinical signs in coma

- Intact blink response
- Actively holding eyes closed
- Actively closing eyes when opened
- Presence of Bell’s phenomenon (eyes rolled up inside head when observer opens eyes)

Typically, space-occupying lesions are responsible for slowly progressive symptoms, though it is possible for acute coma to be caused by haemorrhage into a space-occupying lesion.

Carbon monoxide poisoning
Carbon monoxide poisoning is a relatively uncommon cause of coma. Smoke inhalation, fumes from poorly maintained gas appliances and car exhaust fumes are all potential causes. If the poisoning is chronic, prodromal symptoms such as fatigue and headaches may provide clues as to the cause. It is common for members of the same household to be affected, and the lack of symptoms in his wife suggests that this is not the diagnosis.

Metabolic causes
Other metabolic causes not mentioned above are listed in Box 1.5.

Case history revisited
On further questioning, the patient’s wife confirmed that he hadn’t taken any of the prescribed co-codamol or amitriptyline tablets as the bottles remained full, and that he didn’t drink alcohol.

They had a domestic carbon monoxide monitor, which had been checked recently and was in perfect working order.

Examination
Examination of the patient showed him to have a Glasgow Coma Score (GCS) of 7 (E2, M4, V1).

The patient had a clear airway and was breathing with a respiratory rate of 18 per minute.

There was no smell of alcohol or ketones on his breath. Chest auscultation revealed no abnormality. His heart rate was 94 beats/minute and regular, blood pressure was 180/105 mmHg and his temperature was 36.2°C. His bedside blood glucose level was 6.2 mmol/l.

There were no external signs of head injury, and examination of the thorax, abdomen and limbs was unremarkable. There was no visible rash.

His pupils were equally sized and reactive to light. His limbs were generally hypotonic with brisk reflexes on his right upper and lower limbs, with an upgoing right plantar reflex.
**Question: Given the history and examination findings what is your principal working diagnosis?**

**Principle working diagnosis – Intracerebral haemorrhage**

The clinical information given allows us to discount a number of the differential diagnoses.

The patient is not hypoglycaemic and there is no suggestion of opiate or amitriptyline ingestion. He is afebrile and has no evidence of meningococcal disease. The patient’s history of hypertension and the acute nature of the onset of coma strongly suggest a vascular cause such as an intracerebral haemorrhage.

**Management**

This man is in coma and requires an urgent CT scan. As his GCS is 7, his airway is vulnerable and he requires a definitive airway. Intubation and ventilation is required. No specific management is required to control his blood pressure at this time.

**Outcome**

A CT scan of the patient’s brain showed a large intracerebral haemorrhage, with intraventricular blood and hydrocephalus. Urgent neurosurgical advice was sought but unfortunately this man died during an operation to drain his hydrocephalus.

**Further reading**


