Key points

- Right heart free floating thrombi are associated with increased mortality if left untreated.
- Thrombolysis or surgical embolectomy should be employed.
- Bedside echocardiography can diagnose high risk pulmonary embolism and guide appropriate treatment.
- Prolonged bed rest, tachycardia and syncope are important associations of pulmonary embolism in the elderly.
- Elderly patients are less likely to receive thrombolysis compared with younger individuals.

Conflicts of interest

None declared.

Supplementary data

Supplementary data mentioned in the text are available to subscribers in Age and Ageing online.

References


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Urinary incontinence a first presentation of central pontine myelinolysis: a case report

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Abstract

An 84-year-old lady was treated for hyperosmolar hyperglycaemia with IV insulin, fluids and catheterisation for fluid balance monitoring. Trial without catheter failed as the patient complained of new-onset urinary incontinence and lack of awareness of bladder filling. In light of her breast cancer history, we excluded cauda equina. Ultrasound KUB showed an enlarged bladder. Whole-body MRI revealed a lesion in the pons which was highly suggestive of central pontine myelinolysis (CPM). Her electrolytes were normal throughout her admission; thus, the rapid fluctuation in osmolality, secondary to her hyperglycaemic state, was the likely cause of CPM. CPM has been reported secondary to hyperglycaemia; however, this is the first reported case of CPM presenting as urinary incontinence and loss of bladder sensation.

Keywords: central pontine myelinolysis, urinary incontinence, hyperosmolar hyperglycaemic state, older people
Case report

An independent 84-year-old lady living in a residential home was admitted with decreased appetite for 3 days and delirium. The patient did not volunteer any respiratory, bowel or urinary symptoms. Her past medical history included hypertension, colostomy (perforated diverticular disease), breast cancer (1991) and T2DM treated with gliclazide.

Her examination was unremarkable aside from her BP (90/65), suprapubic tenderness, CAM test positive for delirium and GCS 15/15 with no focal neurology.

Bedside tests revealed hyperglycaemia (BM 27) and a positive urine dipstick for urinary tract infection (UTI).

Our working diagnosis was UTI and hyperosmolar hyperglycaemic state. Treatment included antibiotics, insulin sliding scale, IV fluids and a catheter for fluid balance.

Lab findings showed increased WCC (23.0) and CRP (208). Her electrolytes showed raised urea (16.1), creatinine 180 and a reduced eGFR (23). However her sodium (133) and potassium (3.9) remained normal throughout her admission; [Na+] ranged 133–140, serum osmolarity on admission 316.9 mmol/l (280–295), Day 5 304 mmol/l and Day 13 300 mmol/l.

On Day 11, the catheter was removed and the patient complained of new unawareness of bladder filling resulting in overflow urinary incontinence. Her neurological exam demonstrated upper and lower limb power 5/5, reflexes were difficult to elicit, down going planters, anal tone and saddle sensation were intact. Patient’s cranial nerve exam was normal. A PV exam revealed a small vesicocele.

In light of the patients past medical history of breast cancer, we urgently wanted to rule out spinal cord compression secondary to bone metastases. US KUB showed an enlarged bladder (capacity 1,200 ml) and no hydronephrosis.

An urgent diffusion-weighted brain and total body MRI showed degenerative changes, lumbar spinal stenosis, a degree of right nerve root compression at L3/L4 and no signs of cauda equina. The unexpected finding was a high attenuation signal seen in the pons, consistent with central pontine myelinolysis (CPM); comparing this to a previous scan 3 years ago, this was a new finding (Figure 1).

Discussion

CPM is a non-inflammatory demyelination within the central basis pontis, which presents in a variety of ways, most commonly as pseudobulbar palsy (dysarthria, dysphagia), quadriplegia and reduced consciousness. The absence of classic features of CPM cannot be explained anatomically, especially with a bilateral lesion like in this case, making this a unique presentation of CPM.

CPM is classically associated with rapid correction of chronic hyponatraemia, which is defined as hyponatraemia known to be present for >48 h or developed at a rate of <0.5 mmol/h [1]. CPM develops when there is rapid correction with hypertonic solution that exceeds rate of synthesis and transport of osmoles back into oligodendrocytes resulting in cell death. Most recent recommendation is to increase the sodium by a maximum of 8 mmol/l/day. Other causes of CPM include hyperglycaemia, uraemia, starvation and burns [2, 3].

Diabetic hyperglycaemia has been reported previously to cause CPM, but this is uncommon. It is thought that the hyperglycaemia disrupts the blood–brain barrier and the endothelial lining of the cells leading to demyelination due to vasogenic oedema. After the initial metabolic insult the radiological signs can take up to a week to evolve; however, diffusion-weighted MRI may identify acute CPM [4].

Key points

- CPM is a non-inflammatory demyelination within the central basis pontis.
- This is the first case of CPM presenting as urinary incontinence and loss of bladder sensation.
- Diffusion-weighted MRI may identify acute CPM.
- Chronic hyponatraemia should not be corrected too quickly.
- CPM can resolve spontaneously in a few cases.
Conflicts of interest

None declared.

References


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