Syncope, the head ruling the heart

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A previously fit 53-year-old man was referred for investigation of episodes of syncope and pre-syncope. There was no history of convulsions and after one such episode his pulse rate was noted to be 44 beats/min. Examination was unremarkable. An electrocardiogram and echocardiogram were normal. Repeated 24-hour Holter monitoring did not show an arrhythmia during a collapse although his resting pulse rate fell to 39 beats/min.

Over the next few weeks he deteriorated, complaining of unsteadiness, poor short-term memory and frequent headaches. The attacks continued on a daily basis and were associated with urinary incontinence. His gait became broad-based and unsteady but otherwise neurological examination remained normal.

He was admitted after a further attack followed by confusion. On admission he had a Glasgow Coma Score of 14/15, was afebrile, had a pulse rate of 84 beats/min and a blood pressure of 120/70 mmHg. Papilloedema was noted but there were still no focal neurological signs. Over the next 24 hours he deteriorated to a Glasgow Coma Score of 8. His pulse rate decreased to 40 beats/min and his blood pressure increased to 150/100 mmHg. A computed tomography (CT) scan (figure 1) and subsequent gadolinium-enhanced magnetic resonance imaging (MRI) scan (figure 2) were performed.

Figure 1 Axial brain CT scan

Figure 2 Coronal gadolinium-enhanced MRI

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Questions

- 1 Where is the lesion and what effect is it having?
- 2 What are the ways in which lesions in this area might present?
- 3 What is the differential diagnosis?
- 4 Describe the cardiovascular response to his decreased conscious level.

Answers

QUESTION 1

The lesion is blocking the third ventricle and causing acute obstructive hydrocephalus (early periventricular lucencies can be seen on the CT scan, ie, cerebral oedema due to transudation of fluid).

QUESTION 2

Intraventricular tumours causing intermittent or permanent raised intracranial pressure have been reported to present with ataxia, memory problems, headache, vomiting, visual field defects and mental disturbance.¹ Intermittent cerebrospinal fluid (CSF) obstruction due to these tumours is frequently silent and may present with sudden death.²

QUESTION 3

The differential diagnosis of a third ventricular mass includes craniopharyngiomas,¹ colloid cysts,² choroid plexus papillomas, ependymomas, meningiomas, gliomas,³ and, very rarely, posterior communicating artery aneurysms.⁴

QUESTION 4

The decreased conscious level is due to critically raised intracranial pressure. The presentation with bradycardia and hypertension was described by Harvey Cushing in 1902 and is called the Cushing response. In his experiments Cushing infused saline into the subdural spaces of dogs via a cannula inserted through a trephine opening in the skull and dura. When the intracranial pressure exceeded the arterial blood pressure the arterial blood pressure increased in association with bradycardia and slowing of respiration. The response was abolished by cocaineinization of the medulla. The hypertensive response was prevented by section of the spinal cord and hence the descending sympathetic pathways. Vagotomy prevented bradycardia. He felt therefore that there was a regulatory mechanism in the vasomotor centre which maintained medullary perfusion.⁵ In clinical practice, the Cushing response is rarely seen except as an agonal event.

Learning points

- third ventricular tumours can cause intermittent and permanent CSF obstruction
- presentation can be with syncope, ataxia, memory disturbance, visual field defects, raised intracranial pressure and sudden death
- the triad of raised intracranial pressure is classically headache, vomiting and papilloedema
- critically raised intracranial pressure can present with the Cushing response of bradycardia, hypertension and slowing of respiration
- although third ventricular tumours are rare, complete cure is possible with surgical resection, hence rapid diagnosis is very important

Progress

After the CT scan neurosurgical referral was made. He underwent emergency external ventricular drainage which returned his Glasgow Coma Score to 15. MRI scanning to delineate the anatomy further was followed by craniotomy and macroscopic resection of the tumour with decompression of the third ventricle. Histological examination confirmed the tumour to be a craniopharyngioma.

In the immediate post-operative period the patient had a moderate memory deficit and pan-hypopituitarism necessitating hormone replacement. On follow-up 6 months later he was well, mobilising without ataxia and had improving memory. A post-operative scan confirmed that the tumour had been completely excised.

Final diagnosis

Third ventricular craniopharyngioma causing obstructive hydrocephalus.

Keywords: syncope; cerebral ventricle neoplasms; craniopharyngioma; Cushing response

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4 Smith KA, Kraus GE, Johnson BA, Spetzler RF. Giant posterior communicating artery aneurysm presenting as a third ventricular mass with obstructive hydrocephalus. *J Neurosurg* 1994;**81**:299–303.

5 Cushing H. Some experimental and clinical observations concerning states of increased intracranial pressure. *Am J Med Sci* 1902;**124**:375–400.

Tachycardia following low-tension electrocution

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A 33-year-old woman sustained an electric shock to the dorsum of her right hand from a domestic power supply. The only symptom she reported was a transient numbness in her right arm. There was no loss of consciousness. On arrival at the accident and emergency department she was well perfused and alert. Examination of her skin revealed no superficial burns. She had a regular tachycardia at a rate of 150 beats/min, and her blood pressure was 120/70 mmHg. The remainder of the examination was normal. Her admission electrocardiogram (ECG) is shown in figure 1. The patient was given 3 mg and then 6 mg of adenosine intravenously without effect: the outcome of 12 mg of adenosine is shown in figure 2.

Further investigations included serial creatinine kinase (myocardial bound fraction) and troponin I levels to exclude myocardial damage. These were within normal limits. The lung fields were clear on chest X-ray. An echocardiogram showed normal ventricular and atrial dimensions and function and no valvular abnormality.

Figure 1 Admission ECG

Figure 2 Rhythm strip during intravenous administration of 12 mg of adenosine

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Questions

- 1 Describe the features shown in the ECG (figure 1).
- 2 What does the rhythm strip in figure 2 show, and what information does this give us about the mechanism of the tachycardia?