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Craniopharyngioma Presenting With Profound Hypoglycaemia and Hyponatraemia in a Child: A Case Report

Sarthak Sinha [1], Aslam Wasulasuhab [2], Isha Nijjar [2], Rajesh Sakremath [2]

1. Medicine, Shrewsbury and Telford Hospital NHS Trust, Telford, GBR 2. Paediatrics, Shrewsbury and Telford Hospital NHS Trust, Telford, GBR

Corresponding author: Aslam Wasulasuhab, aslam.wasulasuhab@nhs.net

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Abstract

Craniopharyngiomas are benign intracranial tumours arising in the sellar and suprasellar region. They may present with endocrine dysfunction, visual impairment, or neurological symptoms. A six-year-old boy was admitted with fever, vomiting, diarrhoea, and drowsiness and tested positive for influenza A. During admission, he developed profound hypoglycaemia and hyponatraemia. A hypoglycaemia screen showed a cortisol level that was inadequate for the degree of hypoglycaemia, and further endocrine evaluation confirmed central adrenal insufficiency with additional pituitary hormone deficiencies. Magnetic resonance imaging identified a 43 × 28 × 22 mm cystic sellar and suprasellar lesion causing compression of the optic chiasm, consistent with craniopharyngioma. The patient was commenced on hydrocortisone and levothyroxine replacement and had endoscopic transsphenoidal decompression. Histology confirmed the diagnosis. This case highlights the importance of performing hypoglycaemia screens and considering further endocrine investigations in children. While hypoglycaemia is often associated with infection, recurrent or severe episodes with hyponatraemia should prompt evaluation for endocrine causes. Neuroimaging should be considered in children with multiple pituitary hormone deficiencies to identify central causes such as craniopharyngioma.

Categories: Oncology, Pediatrics, Endocrinology/Diabetes/Metabolism
Keywords: central adrenal insufficiency, craniopharyngioma, hypoglycaemia, hyponatraemia, hypopituitarism

Introduction

Craniopharyngiomas are benign tumours of the central nervous system [1]. There are two main histological subtypes: adamantinomatous and papillary [2]. The most common locations are the sellar and suprasellar regions, which can result in compression of pituitary tissue, leading to pituitary deficiencies. These can present in diverse ways, including headaches, visual defects, and endocrine disturbances. In children, the commonest features are signs of raised intracranial pressure, visual impairment, and endocrine problems such as slow growth, polyuria and polydipsia [3]. They represent around 1% of all brain tumours, with an incidence of roughly 0.19 per 100,000 person-years across all ages [4]. In children and young people up to 19 years, it is similar, at about 0.21 per 100,000 [5], and in children the incidence peaks between five and 14 years of age [6]. The early symptoms are often vague, so the diagnosis is often delayed [3]. In children with brain tumours in general, endocrine and growth problems are linked with a longer time to diagnosis [7]. Cortisol and growth hormone help to keep the blood glucose up when a child is fasting or unwell, so a child without them is at risk of hypoglycaemia [8]. These problems may cause few symptoms day to day and only show up when a child becomes ill and needs more cortisol, which is why an infection can be the first sign of an underlying pituitary problem [8].

Case Presentation

History

A six-year-old boy presented to the emergency department with a brief history of fever, diarrhoea, vomiting, and drowsiness. He was found to be influenza A positive and was treated with intravenous fluids and antibiotics. He was found to be hypoglycaemic and hyponatraemic, so he was initially given oral glucose. During admission, he developed further profound hypoglycaemia with an unrecordable capillary blood glucose. In view of the hypoglycaemia, a hypoglycaemia screen was performed. His random serum cortisol performed at the time was 207 nmol/L, which was inadequate in the context of severe hypoglycaemia. On further history, his mother reported long-standing polyuria and polydipsia, with a daily fluid intake of 2.5-3 litres. To our knowledge, this had not been investigated before. He was also described as having low energy levels, lethargy, and recurrent infections. It was also reported that there was a period of slowed growth, noting that his younger sibling was of a similar height. However, no previous height measurements were available to objectively assess his growth trajectory. There were no symptoms suggestive of raised intracranial pressure.

Examination

His systemic examination, including his neurology, was within normal limits. He was age-appropriately pre-pubertal with testicular volumes of 2 mL bilaterally and normal stretched penile length.

The patient's condition improved, and he was tolerating oral fluids. An urgent short Synacthen test was done as an outpatient, together with endocrinology investigations. The short Synacthen test demonstrated adrenal insufficiency (cortisol baseline 27 nmol/L, 105 nmol/L after 30 minutes, 143 nmol/L after 60 minutes) and thyroid function tests showed a low free T4 (3.4 pmol/L) with a TSH that was not raised (0.8 mU/L), in keeping with central hypothyroidism (Table *1*).

Parameters	Patient values	Reference range
Serum sodium (during acute illness)	127 mmol/L	133-146 mmol/L
Capillary blood glucose (during acute illness)	Unrecordable	Not available
Random serum cortisol (during hypoglycaemia)	207 nmol/L	Not available
Serum cortisol, short Synacthen test (0 minutes)	27 nmol/L	Not available
Serum cortisol, short Synacthen test (30 minutes)	105 nmol/L	Not available
Serum cortisol, short Synacthen test (60 minutes)	143 nmol/L	Not available
Growth hormone (initial)	0.2 µg/L	Not available
Free T4 (thyroxine) (admission)	3.4 pmol/L	11-22 pmol/L
TSH (thyroid-stimulating hormone) (admission)	0.8 mU/L	0.7-6 mU/L

TABLE 1: Laboratory investigations

Reference ranges are those of the reporting laboratory. Reference ranges were not available for the tests marked “Not available”.

Following discussion, he was admitted to the paediatric ward to commence steroid replacement prior to starting on levothyroxine. Diabetes insipidus was diagnosed before the MRI, and he was treated with desmopressin. An urgent MRI of the head was performed to identify an intracranial cause for his symptoms, as we had suspected acquired hypopituitarism.

MRI imaging of the brain revealed a large cystic sellar and suprasellar mass measuring approximately 43 × 28 × 22 mm, extending into the suprasellar region with distortion of the optic chiasm and bulging into the floor of the third ventricle (Figure *1*). This was consistent with a craniopharyngioma.

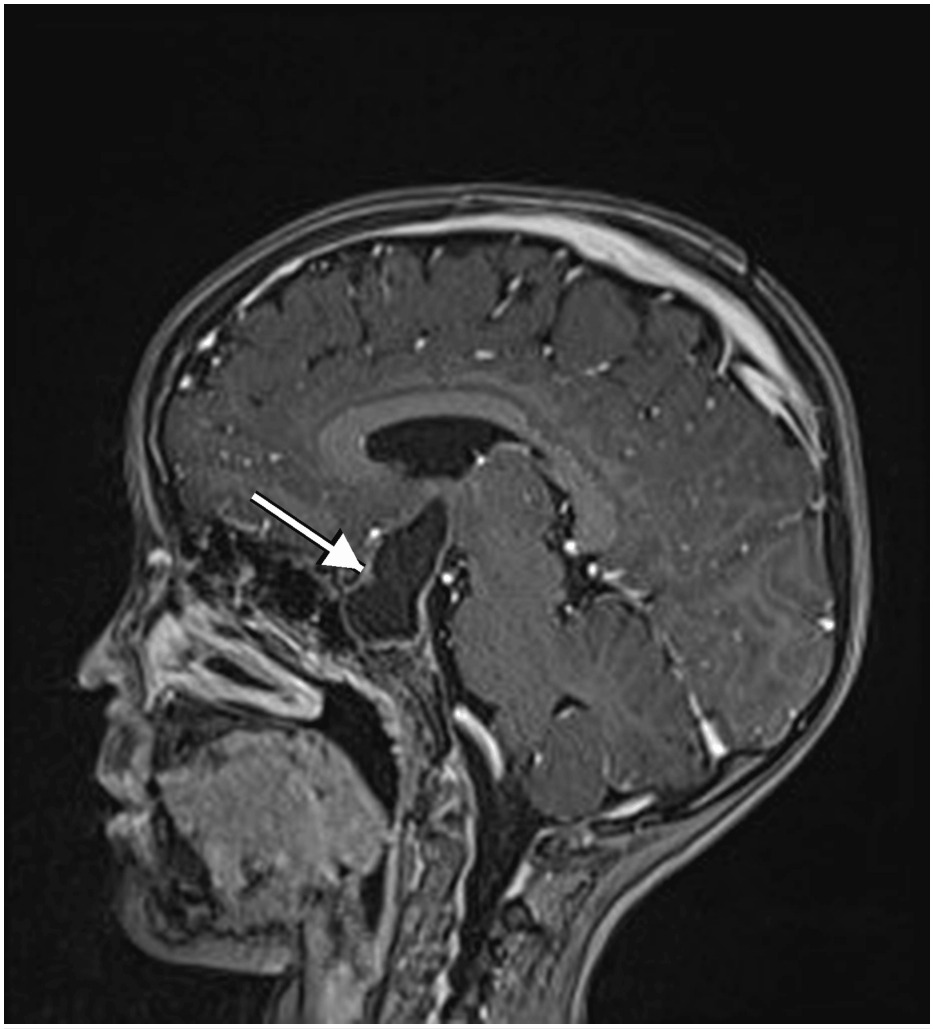


FIGURE 1: Pre-operative MRI of the brain

Sagittal T1-weighted post-contrast MRI of the brain showing a large, dumbbell-shaped cystic mass (arrow) expanding the sella turcica, with a larger suprasellar component measuring approximately 43 × 28 × 22 mm. There is thin peripheral rim enhancement, and the mass compresses the third ventricle from below.

Management

He was transferred to the nearest paediatric neurosurgical unit, where he underwent endoscopic trans-sphenoidal decompression of the lesion. Histology confirmed the diagnosis of adamantinomatous craniopharyngioma. Post-operative imaging demonstrated effective decompression of the cystic component with improvement in optic chiasm distortion (Figure *2*).

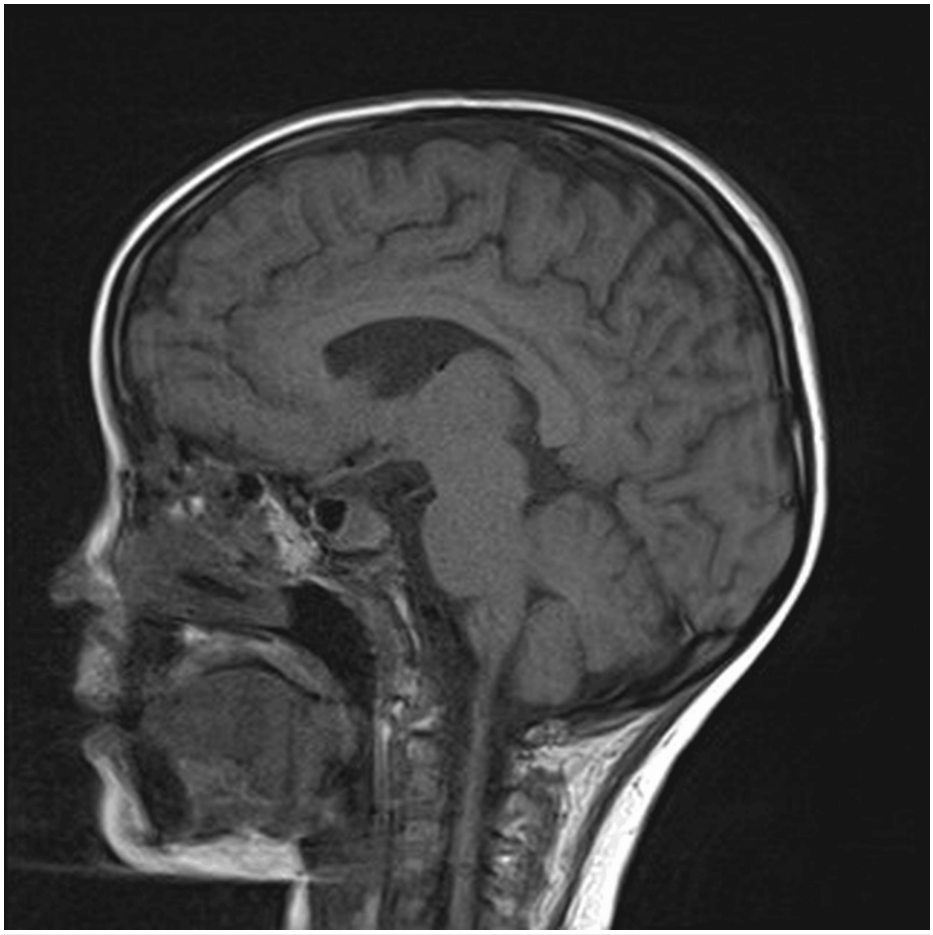


FIGURE 2: Post-operative MRI of the brain

Sagittal T1-weighted MRI of the brain five days after endoscopic trans-sphenoidal decompression. The large sellar and suprasellar cystic component has been decompressed, leaving a collapsed, fluid-filled cavity measuring approximately 26 × 20 × 36 mm. Tenting of the optic chiasm has improved significantly compared with the pre-operative scan.

At follow-up, he remained on hydrocortisone, levothyroxine, and desmopressin, with ongoing endocrine surveillance.

Discussion

In children, craniopharyngioma often shows itself through endocrine problems that are already present at diagnosis. Growth hormone deficiency is the commonest (about 75%), followed by gonadotropin deficiency (about 40%) and adrenocorticotropic hormone (ACTH) and thyroid-stimulating hormone (TSH) deficiency (about 25% each) [9]. In one paediatric series, the most common complaints were headache, visual problems, and short stature [10]. In another, growth failure was seen in 11 of 18 children and started a median of about 16 months before the diagnosis [3]. Our patient was different. He came in with influenza A, and it was only the very low glucose and sodium that led to a hypoglycaemia screen and then the diagnosis. Severe hypoglycaemia has been reported as the first sign of a craniopharyngioma before [11].

The cortisol of 207 nmol/L taken during the hypoglycaemia was the first clue. Samples taken during a hypoglycaemic episode are a useful way of finding the cause [12]. On its own, the cortisol does not prove adrenal insufficiency, but a child who is unwell and hypoglycaemic should have a much higher cortisol, so it was too low for the situation. The short Synacthen test then confirmed adrenal insufficiency. In the same series, cortisol deficiency was found in about 10% of children before surgery [10], so it is not always present, and an illness can bring out a deficiency that was not causing obvious problems before [8]. Low glucose, low sodium, and an inadequate cortisol together should lead to urgent endocrine assessment and a consideration of a central cause.

Our patient also had a longstanding history of polyuria and polydipsia with a daily fluid intake of 2.5-3 litres, which had not previously been investigated. Diabetes insipidus is a recognised presentation of craniopharyngioma and has been reported in approximately 2-15% of children at diagnosis [13]. This

highlights the importance of exploring symptoms of endocrine dysfunction, especially when growth concerns or unexplained lethargy are also present.

The tumour was large, measuring 43 × 28 × 22 mm. Late diagnosis and large tumours have been linked with worse endocrine loss [10]. Looking back, he had a history of polyuria and polydipsia, low energy, repeated infections and stalled growth, so it may have been possible to pick this up earlier. Growth hormone deficiency is the commonest deficit at diagnosis [9], so poor growth together with symptoms that are not otherwise explained should make us think of a pituitary or hypothalamic cause. Earlier growth measurements and fundoscopy findings were not available to us.

Other things can cause low glucose, low sodium, and poor growth in a child. After the newborn period, the commonest causes of hypoglycaemia are diabetes and idiopathic ketotic hypoglycaemia, but endocrine disorders and inborn errors of metabolism also need to be excluded [12]. Primary adrenal insufficiency, such as congenital adrenal hyperplasia or autoimmune adrenalitis, causes low sodium, usually with a high potassium and a raised ACTH [14], and can also cause hypoglycaemia [8]. Hyperinsulinism, ketotic hypoglycaemia and inherited metabolic disorders cause low glucose [15], but would not normally explain low sodium, polyuria and poor growth as well. Other lesions in the sellar and suprasellar region can also cause hypopituitarism, which is why imaging is needed.

As in other children with craniopharyngioma, he will need lifelong endocrine follow-up with hormone replacement [3].

Conclusions

This case highlights the importance of performing hypoglycaemia screens and considering further endocrinological investigations. Hypoglycaemia in children is often associated with infection, but recurrent or profound episodes combined with hyponatraemia should prompt consideration for investigating endocrine causes. Neuroimaging should be considered in children with multiple pituitary hormone deficiencies to identify central causes such as craniopharyngioma.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Sarthak Sinha, Aslam Wasulasuhab, Isha Nijjar, Rajesh Sakremath

Drafting of the manuscript: Sarthak Sinha

Critical review of the manuscript for important intellectual content: Aslam Wasulasuhab, Isha Nijjar, Rajesh Sakremath

Supervision: Aslam Wasulasuhab, Isha Nijjar, Rajesh Sakremath

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Not Applicable issued approval Not applicable. This report describes a retrospective case report. Formal research ethics committee approval was not required. Appropriate consent for publication was obtained from the patient's parent or legal guardian. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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