CASE REPORT

Stridor in an 11-year-old child

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SUMMARY

Stridor in older children can be due to diverse aetiology that includes infections, anaphylaxis and rarely systemic conditions leading to hypocalcaemia. We report the case of a previously asymptomatic 11-year-old girl who presented to the casualty with stridor due to hypocalcaemia. The aetiological investigations for hypocalcaemia uncovered previously undetected chronic renal failure, possibly due to a rare autosomal recessive condition called nephronophthisis. We report this case to highlight the importance of widening the diagnostic focus for children presenting with stridor, especially when they fall outside the usual age group for infections like croup, or when the history is atypical.

BACKGROUND

The aetiology of stridor in children can be diverse. While most of the children presenting to the emergency department with stridor do not pose diagnostic difficulties, rarely the identification of a cause can be challenging. Owing to the risk of total airway obstruction, prompt recognition and management of the underlying cause are crucial. A case of an 11-year-old child who presented with acute stridor due to hypocalcaemia secondary to previously undetected renal failure is discussed. This case is intended to highlight the importance of considering rare causes of stridor in atypical cases.

CASE PRESENTATION

An 11-year-old girl presented to the paediatric accident and emergency department with a relatively short history of cough, stridor and difficulty in breathing. On examination, she looked very pale, had stridor at rest, a soft whispering voice and mild respiratory distress. She was diagnosed as having viral laryngotracheobronchitis (croup) and was treated with oral dexamethasone and nebulised budesonide with slight improvement of symptoms. A further ear-nose-throat specialist opinion made in view of her atypical age for croup also approved the ongoing management plan. She was admitted to the general paediatric ward for observation and further investigation of anaemia.

Her medical history included dysphagia nearly a year prior to her current illness, for which she was assessed in the paediatric outpatient clinic. A full blood count at that stage had shown a mild anaemia with a haemoglobin level of 9.2 g/dL. Following this, she had a barium swallow study, which was normal. Her dysphagia improved over time and she was subsequently discharged from follow-up. Further exploration into history revealed that she had suboptimal weight gain over the preceding year associated with unexplained lethargy. She also had

generalised itching, which was attributed to eczema. There was no known history of renal disease and the family history was unremarkable.

Admission blood tests revealed a profound anaemia with a haemoglobin level of 3.3 g/dL, calcium 1.64 mmol/L (normal 2.20–2.60 mmol/L), phosphate 2.08 mmol/L, serum albumin 44 g/L and profound renal impairment with a urea level of 68 mmol/L and a creatinine of 1121 µmol/L. She was promptly referred to the paediatric renal team and an urgent renal ultrasound scan was arranged. While in the radiology department she had an episode of tetany, which responded well to an intravenous calcium bolus. Her venous blood gas during the episode showed a severe metabolic acidosis (pH 6.87, bicarbonate 5.4 and base deficit 24) presumed secondary to renal failure and had an ionised calcium level of 1.00 mmol/L (normal range 1–1.25 mmol/L).

Ultrasound scan demonstrated normal-sized kidneys with small cortical cysts, with appearances highly suggestive of nephronophthisis. After careful consideration, it was decided that a renal biopsy was unlikely to add any significant further information to assist management planning, and hence was not carried out. Additional investigations into possible alternate causes of renal failure have been negative.

She was admitted to pediatric intensive care unit and received intravenous calcium and bicarbonate infusions. She had the symptoms of intermittent respiratory obstruction due to laryngospasm, which steadily improved with calcium supplementation. She was also started on haemodialysis within 48 h of admission.

INVESTIGATIONS

Renal ultrasound scan and blood tests as outlined above.

DIFFERENTIAL DIAGNOSIS

Stridor in children can be of diverse aetiology. During infancy, stridor may be caused by narrowing of upper airway due to laryngomalacia, viral infections such as croup, subglottic stenosis or congenital anomalies like laryngeal web.¹ In older children, the causes may also include anaphylaxis, bacterial tracheitis, epiglottitis, retropharyngeal and peritonsillar abscesses and C1 esterase deficiency.² Hypocalcaemia, albeit rare outside the neonatal period, can also produce spasmodic adduction of the vocal cords resulting in upper airway obstruction and stridor.^{3–4} The causes of hypocalcaemia beyond the neonatal period can be due to parathormone (PTH) insufficiency, vitamin D deficiency, PTH-resistant states and calcium chelation.³

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Severe end organ diseases such as renal failure can cause PTH resistance leading to hypocalcaemia.⁵ Rarely, stridor due to hypocalcaemia may be the initial manifestation of an underlying systemic cause like vitamin D deficiency rickets⁶ or renal impairment.⁷

OUTCOME AND FOLLOW-UP

The patient was initially established on home peritoneal dialysis following which she had a live-related donor transplant from her father and is currently doing well.

DISCUSSION

Stridor is a common presentation in the paediatric population and the majority of cases are due to upper respiratory infections. Hypocalcaemia can present as stridor, and in the paediatric age group, is most commonly encountered in the neonatal period when it is usually secondary to transient partial hypoparathyroidism.⁸ Infants presenting with stridor due to hypocalcaemic laryngospasm has been previously described.^{6 8} Failure to recognise hypocalcaemia as a possible cause of stridor can lead to discharge of a patient at risk of serious complications such as tetany, seizures, dysrhythmias such as torsades de pointes and cardiac arrest.⁹

In our literature search we could find only one case report of a child presenting with hypocalcaemic laryngospasm secondary to renal dysplasia.⁷ Nephronophthisis is an autosomal recessive form of cystic renal disease, which can present as chronic renal failure in infancy, childhood or adolescence. The finding of normal-sized echogenic kidneys containing small cortical cysts as in this case is a common feature of nephronophthisis.¹⁰

Hypocalcaemia in renal disease is due to 1,25-dihydroxy vitamin D deficiency and hyperphosphataemia due to reduced glomerular filtration. It is also of interest that the ionised calcium level at the time of the tetanic episode was just at the lower end of the normal range, a level not normally associated with tetany, but this may have been spuriously elevated due to the concomitant acidosis.

Learning points

- ▶ Chronic renal failure can present with hypocalcaemic symptoms but very rarely with laryngospasm and stridor.
- ▶ Although rare, hypocalcaemic laryngospasm must be considered in the differential diagnosis of stridor.
- ▶ In atypical cases of stridor (atypical clinical picture, poor response to standard treatment, multiple presentations, etc) we suggest that blood calcium or ionised calcium levels should be checked to prevent missing hypocalcaemia and its potential complications.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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