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CORRESPONDENCE

A novel case and review of paediatric Horner syndrome

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To the Editor:

We present a novel presentation of Horner syndrome (HS) in a child after button battery (BB) ingestion (BBI). The BB impacted in the cervical oesophagus disrupting the ipsilateral sympathetic chain. Acute presentation of HS may include ptosis, miosis and facial anhidrosis.

A 1-year-old boy, with no medical or family history, attended with a 4-week history of sore throat, pyrexia, reduced intake, vomiting and a presumed swollen left upper eyelid, despite initial treatment with co-amoxiclav for likely tonsillitis and chloramphenicol to the left eye.

Clinical assessment revealed persistent pyrexia, a left eye 3 mm ptosis with miosis but tolerating solid food. Pharmacological testing for HS with apraclonidine 1% caused a resolution of the ptosis and miosis after 20 min, confirming an HS (Fig. 1a, b). A chest X-ray revealed a 23 mm BB lodged in the oesophagus at the level of the lower cervical spine (Fig. 2a, b).

Rigid oesophagoscopy revealed the BB lodged in the oesophageal wall with circumferential white scarring noted after removal. Post-operative thoracic computer tomography (CT) scan demonstrated para-oesophageal air, consistent with a perforation (Fig. 2c). This was managed conservatively—nil by mouth, nasogastric feeding and intravenous co-amoxiclav. After 10 months, the HS has completely resolved (Fig. 1c).

Aetiologies of HS in a paediatric population are summarised in Table 1. In children with acute dysphagia, unwitnessed foreign-body ingestion must be considered.

Investigation of BBI with MRI could be catastrophic due to magnetism of BB. In five case series of 90 acquired

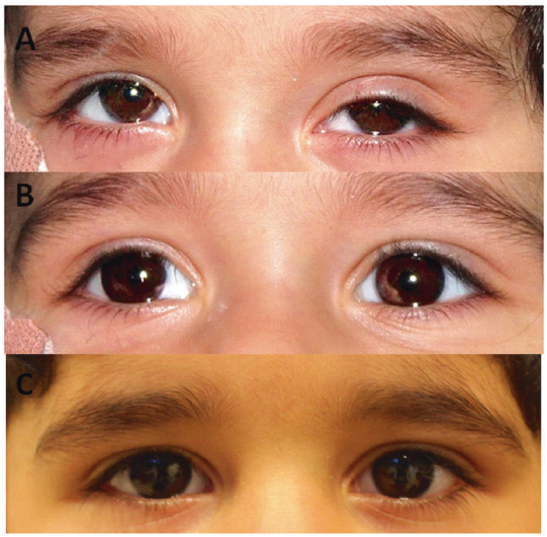

Fig. 1 **a** Before application of apraclonidine 1%. Miosis left eye and partial ptosis. **b** Twenty minute post apraclonidine 1% instillation to both eyes. Reversal of ptosis, and mydriasis of left eye. **c** 10 months after initial presentation.

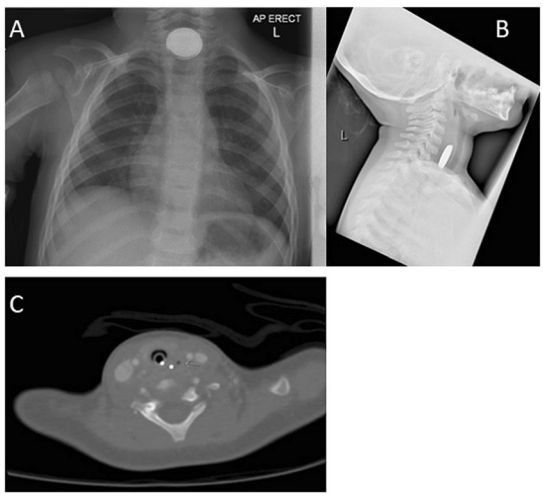

Fig. 2 **a** Antero-posterior chest X-ray revealing 23 mm button battery. Note the characteristic double rim of the battery. **b** Oesophageal impaction with button battery anterior to cervical vertebrae levels 5–7. **c** Inflammatory changes and air (arrow) within the pre-vertebral soft tissues in the mid-to-lower neck and posterior to the left lobe of the thyroid and between the left lobe of the thyroid and the left common carotid artery.

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Table 1 [a]Notable cases of paediatric case series in the literature and their recommendation.

Author (year)	Patients (*n*)	Aetiology *N* (%)					Recommendation
		Idiopathic	Birth trauma	Surgery/ acquired trauma	Neuroblastoma/ mass lesion	Other	
Giles and Henderson 1958 [1]	32	0 (0)	12 (37)	15 (47)	4 (13)	1 (3)	Emphasise on thorough history.
Saur and Levingohn 1975 [1]	7	0 (0)	1 (14)	0 (0)	5 (72)	1 (14)	Complete neurologic exam, full examination of head and neck. Routine radiography of chest, cervical spine and skull, 24-h urinary assays, particularly in cases of a known cervical/pulmonary mass.
Weinstein et al. 1980 [1]	11	3 (27)	5 (46)	2 (18)	0 (0)	1 (9)	Nil.
Woodruff et al. 1988 [1]	10	4 (40)	0 (0)	2 (20)	2 (20)	2 (20)	Unless strongly associated with surgical manipulation, congenital and acquired onset warrants CXR, CT head and neck, 24-h urinary assays.
Jeffery et al. 1998 [1]	73	15 (21)	16 (22)	32 (44)	6 (8)	4 (5.5)	Acquired Horner syndrome without diagnosed mass lesion or surgical trauma should undergo neuroimaging of abdomen, chest, neck and head and physical examination of the neck and abdome + 24-h urine assays.
George et al. 1998 [1]	23	16 (70)	4 (17)	0 (0)	3 (13)	0 (0)	Thorough examination for cervical or abdominal masses and involvement of other cranial nerves. In truly isolated manifestations, 24-h urine assays. Further investigation is warranted if the Horner syndrome is acquired or associated with other signs such as progressive heterochromia.
Mahoney et al. 2006 [1]	56	21 (75)	1 (2)	10 (18)	13 (23)	11 (57)	Brain, neck and chest MRI contrast and 24-h urine analysis are recommended in all cases of paediatric Horner syndrome without a surgical history.
Smith et al. 2010 [1]	20	7 (35)	7 (35)	6 (30)	0 (0)	0 (0)	Obtaining imaging studies should not be considered the standard of care for paediatric patients with Horner syndrome after surgical trauma. As idiopathic Horner syndrome found to eventually have a background of malignancy is small and neuroimaging should be proceeded with caution due to risk of general anaesthetic and radiation exposure.
Miquel et al. 2017 [2]	3	3 (100)	0 (0)	0 (0)	0 (0)	0 (0)	Neurological examination and imaging should be performed to rule out structural lesions.
Kadom et al. 2015 [3]	38	32 (84)	0 (0)	0 (0)	2 (5)	4 (11)	All patients with paediatric Horner syndrome should be investigated with neuro-imaging of the oculosympathetic pathway. Cross sectional angiographic studies were helpful identifying vascular dissections.
Martin et al. 2017 [3]	26	24 (8)	0 (0)	0 (0)	2 (8)	0 (0)	Perform neuro-imaging for all cases of miosis with associated signs of Horner Syndrome; and in isolated miosis and a positive cocaine test.
Total	299	125 (42)	46 (15)	67 (22)	37 (12)	24 (8)	

Summary of major case series of paediatric HS reported in the literature and their recommendations

CXR chest X-ray, *CT* computed tomography, *MRI* magnetic resonance imaging

[a]Table adapted and expanded on from Smith et al. [1–3]

paediatric HS where MRI was clinically available, clinicians chose to exclusively investigate with MRI 63 (70.0%), CT 8 (8.9%), plain-XR 3 (3.3%) or no imaging 8 (8.9%) [1–3]. With a 6.7-fold increase of BBI in 25 years [4], this unique presentation demonstrates the importance of plain-XR prior to MRI imaging, particularly in cases with a history of upper gastrointestinal symptoms.

BBI can result in tissue damage from several mechanisms: electrical discharge, pressure necrosis, leakage of alkaline content and metal toxicity. The caustic injury is generated from hydroxide radicals in the mucosa resulting from high pH. Animal models have shown a rise in pH from 7 to 13 at the BB's negative terminal and necrosis of oesophageal lamina propria within 15 min of ingestion, with extension to the outer muscular layer within 30 min [5].

The National Capital Poisons Center cohort of BBI (8648 cases) reported death in 13/8648 (0.15%) and a major morbidity in 73 (0.8%) patients [6]. The major risks for these are BB diameter >20 mm, lithium cells (longer shelf life, increased voltage capacity and stability at cool temperatures) and oesophageal impaction [1].

In this case, the exact mechanism of injury to the sympathetic chain is unknown, however we presume that changes to the electrical environment, or associated inflammation disrupted conduction, which at 10 month follow-up, made complete recovery (Fig. 1c).

The administration of topical apraclonidine, in a paediatric setting, can cause central nervous system depression. National recommendations from the Royal College of Ophthalmologists have been published after this case, that advise against, for paediatric HS diagnosis: apraclonidine 1%, apraclonidine 0.5% below 6 months of age and extra caution below 2 years of age. Monitoring should be for 2 h following administration. We report no drowsiness following close observation after administration.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Publication consent statement The authors confirm that they have obtained informed consent for publishing this case including all photographs.

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