CASE REPORTS

Life threatening airway obstruction: a hazard of concealed eating disorders

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Abstract

The ingestion of unusual objects is not uncommon in florid mental illness. Less common is the accidental ingestion of a foreign body which has been used to induce vomiting. A case is reported of complete dysphagia that resulted from impaction of a plastic fork in the hypopharynx. The patient had been attempting to induce vomiting and, as a result of the presentation, was found to be suffering from a previously concealed eating disorder (bulimia). Self induced vomiting is one criterion for the diagnosis of bulimia and a review of the literature indicates that accidental ingestion of large foreign bodies is an increasingly familiar hazard of occult bulimia.

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Keywords: bulimia; foreign body; dysphagia; ingestion

Case report

A 22 year old woman attended the accident and emergency department. Conscious but distressed, she was suffering from complete dysphagia, having partially swallowed a plastic eating fork. Sedation with intravenous midazolam allowed indirect laryngoscopy which revealed a white plastic eating fork impacted in the hypopharynx (see fig1). Shortly after admission, the patient underwent general anaesthesia during which the intact 15 cm long fork was removed under direct vision. The head and prongs were lying anteroposteriorly across the laryngeal inlet with the tips of the prong impinging on the laryngeal surface of the epiglottis. The handle was lying within the upper oesophagus but the shoulders of the fork were caught by the cricopharyngeus so the

head and prongs remained within the hypopharynx. Both ingestion and removal of the fork resulted in negligible damage to the pharyngeal or laryngeal tissues.

On waking, the patient claimed that she had been using the fork to remove another (unidentified) foreign body which had allegedly lodged in her throat after a previous meal. None the less, a collateral history from her parents revealed a year long history of eating disorder involving self imposed starvation alternating with food binges. Purging would be induced soon after completion of a binge and it transpired that this particular incident resulted from an overzealous attempt to induce emesis. Postoperative physical recovery was unremarkable and follow up was arranged with the local psychiatric services.

Discussion

The ingestion of foreign bodies by mentally ill patients and children is commonplace. The majority pass harmlessly through the gastrointestinal tract but a few become impacted at various levels and require active intervention to remove them.^{1,2} Rarely, ingestion of foreign bodies results in perforation of the oesophagus or more distal alimentary canal with severe complications including death.^{3,4} Bulimia is a serious eating disorder that predominantly affects young and middle aged women. Originally classified with anorexia nervosa, it is now regarded as a distinct entity in which patients suffer from an urge to consume food, often in inappropriate amounts, and then attempt to regain weight control by self induced vomiting or laxative abuse. Patients' fingers are probably the most common instruments used to induce vomiting but a review of the literature reveals several cases where toothbrushes have been used with subsequent accidental ingestion.^{5,6} Riddlesberger *et al* have suggested that the toothbrush is popular due to its usual presence in the bathroom where self induced vomiting is most likely to occur.⁶

This case appears to be the first reported where such potentially serious consequences have resulted from accidental swallowing of a fork. It serves to reinforce the growing awareness of the medical hazards of bulimia; formerly believed to be an innocuous disorder, it is now recognised that as many as 40% of bulimics developed significant medical problems ranging from electrolyte imbalance to

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Figure 1 Plastic fork removed from patient.

sudden death.⁷ Accidental ingestion of foreign bodies, while rare, may now be added to the list of serious complications.

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Serotonin syndrome due to venlafaxine overdose

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Abstract

A case is presented of serotonin syndrome after deliberate overdose of the antidepressant venlafaxine. The mechanism, diagnosis, and management of this disorder is discussed.

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Keywords: serotonin syndrome; overdose; venlafaxine

Case report

A 28 year old male with a previous history of chronic fatigue syndrome and depression presented one hour after taking an overdose of 40, 75 mg venlafaxine tablets. On arrival he was conscious, but unable to speak, with a coarse tremor of his upper limbs and rigid lower limbs, with marked clonus present in his ankles. He was not taking any other prescribed or illicit drugs, did not smoke, and seldom used alcohol. He was tachycardic with a rate of 160 beats/min, hypertensive with blood pressure 174/83 mm Hg, flushed, and diaphoretic. His temperature was 36.6°C. An electrocardiogram showed sinus tachycardia with normal morphology. Arterial blood gases were normal.

On the basis of the history and clinical findings a diagnosis of serotonin syndrome was made. He was resuscitated with 2 litres of 0.9% saline over two hours and charcoal given via a nasogastric tube. Chlorpromazine 12.5 mg was given intravenously to control the tremor. Fifteen minutes later his condition had improved such that he could answer questions by nodding or shaking his head. At this stage he was pyrexial with an axillary temperature of 38.5°C.

Thirty minutes after administration of the chlorpromazine the patient suffered a seizure, treated with an intravenous bolus of 2 mg midazolam. A second dose of chlorpromazine was given three hours after the overdose when rigidity returned. His pyrexia responded to cooling and the chlorpromazine. Creatine kinase peaked at 1307 U/l (normal range 20-260) at 16 hours after the overdose. Renal function was normal throughout and urinalysis unremarkable. He recovered completely and was discharged after 48 hours.

Discussion

This is the second reported case of serotonin syndrome after venlafaxine overdose,¹ although in the previous case the patient had taken paroxetine two weeks previously.

Venlafaxine (Efexor, Wyeth Pharmaceuticals) is a chemically distinct antidepressant with unique action and efficacy. It has a faster onset of action than both selective serotonin reuptake inhibitors and tricyclic antidepressants and appears to inhibit reuptake of serotonin, noradrenaline, and dopamine. The main indication for venlafaxine is major depressive disorder, particularly seriously depressed melancholic patients. Overdosage with venlafaxine has been reported to cause profound central nervous system depression requiring intubation,² generalised seizure,³ and serotonin syndrome as mentioned above, although most patients are asymptomatic. Of patients who report symptoms, somnolence is the commonest.

Serotonin syndrome is a symptom complex resulting from increased biological activity of serotonin. Symptoms include altered mental status, neuromuscular irritability, and autonomic instability. Previously it has been seen almost exclusively in the context of polypharmacy in patients on serotonergic medication, most frequently monoamine oxidase inhibitors, tricyclic antidepressants, or selective serotonin reuptake inhibitors, all of which increase the half life of serotonin in the synaptic cleft. Most reported cases have occurred shortly after an increase in the dose of a serotonergic drug or the addition of another. Diagnosis is clinical, since laboratory abnormalities are non-specific. Symptoms are due to synaptic serotonin concentration alone, hence blood serotonin values are unhelpful.

Diagnostic criteria⁴ are:

(A) The recent addition of increase in the dosage of a drug which enhances serotonin activity or availability;

(B) The absence of abused substances as well as metabolic or infectious causes;

(C) No recent addition or increased dose of neuroleptic;

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