Delayed Diagnosis

Bilateral parotid enlargement as a presenting feature of bulimia nervosa in a post-adolescent male

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Summary: An unusual case of bulimia nervosa in a post-adolescent male is reported. The clinical presentation was one of painless parotid swelling of 3 years duration with marked weight loss and underlying metabolic alkalosis. The diagnostic significance of parotid salivary gland swellings is discussed.

Introduction

Bulimia nervosa is an episodic compulsive eating disorder characterized by binge eating associated with behavioural activity intended to promote weight loss. This may include self-induced vomiting, laxative or diuretic abuse, excessive exercising and periods of prolonged fasting. Bulimia is characteristically impulsive in nature, involving a lack of deliberation and a failure to consider the risks and consequences of these eating behaviours. In many respects it is similar to anorexia nervosa since in both conditions patients exhibit abnormalities of behaviour associated with eating, weight maintenance and weight regulation and also disturbances in the subjective experience of the body. The exact nature of the latter disturbances remains controversial but the DSM-III-R section on eating disorders defines the pathological body-related experiences of bulimics as involving 'persistent overconcern with body shape and weight'. There is also a high degree of associated general psychiatric disturbance with depressive symptoms and impaired social functioning being especially prominent.

The syndrome predominantly occurs in late adolescence or early adulthood mainly amongst females with only 5-10% of patients being male. Estimates of its prevalence are difficult owing to its secretive nature but about 1-3% of young women in Western society develop bulimia nervosa and the prevalence may be increasing.

Patients with unrecognized bulimia are often referred for evaluation and treatment for physical complaints of a seemingly unrelated nature, and may pose a significant diagnostic problem. Orofacial problems are commonly encountered with bulimia and as many as 50% may complain of puffy cheeks related to parotid salivary gland hypertrophy.

We present an unusual patient with parotid swelling associated with bulimia who defied diagnosis for some 3 years despite being investigated by a number of physicians and surgeons.

Case report

A 25 year old unemployed male Caucasian labourer was referred to the Department of Immunology with painless parotid swellings of 3 years duration. After initially being intermittent the swelling latterly had become constant (Figure 1). It was not associated with gustatory stimuli. He complained of nausea, vomiting associated with xerostomia and polydipsia. His weight had fallen from 112 kg to 52 kg and he had suffered two episodes of severe cramps in his arms and legs. He also described a feeling of general malaise and diffuse abdominal pain.

On questioning the patient felt his illness to have begun in early 1988 following a heavy drinking session during which he consumed some 20 pints of beer. Several episodes of vomiting with haematemesis followed. Severe abdominal pain rendered the patient unable to eat or drink for 3 weeks and he lost 9.45 kg in weight. His general practitioner referred him for endoscopy and on examination gastritis and peptic ulceration were found. Cimetidine was prescribed and he found this to be effective in relieving the pain. However, unfortunately the patient began to vomit again. He stopped drinking

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any alcohol but found the vomiting an effective means of losing weight.

The parotid glands gradually enlarged from 1988 to 1990 and the patient was seen by several physicians and surgeons with no definitive diagnosis being reached, although mumps was suggested. Throughout this period of his illness the patient's psycho-social circumstances were difficult and he had been unable to hold down a job. His father had committed suicide and had been discovered hanging by the patient when he had been a child. He was subsequently physically abused by his step father. He lived with his grandparents and uncle with whom he had a difficult relationship. He had experienced multiple heterosexual relationships but with no regular partners. He denied drug abuse.

On examination he appeared emaciated, with obvious muscle wasting on his trunk and limbs. Prominent bilateral parotid swellings were evident, including a cystic submandibular swelling. Numerous small, shotty cervical lymph nodes were palpable. Intraoral examination revealed a well-maintained dentition with erosions on the palatal aspect of the upper anterior maxillary teeth (Figure 2). General examination revealed epigastric tenderness and a 1 cm palpable liver. The spleen was not palpable. A fungal infection was present in the groin.

An open parotid gland biopsy performed in 1990 had revealed some change suggestive of chronic infection but had been otherwise unremarkable. Initial investigations included a chest radiograph and parotid sialogram, both of which were normal. The full blood picture was normal, but the ESR was raised at 31 mm/hour. Syphilis serology was negative. In view of the association between parotid hypertrophy and human immunodeficiency virus (HIV) disease and the patients gross weight loss and promiscuousness, an HIV test was performed following counselling. The result proved negative. Serum chemistry showed hypokalaemic alkalosis (Na+ 132 mmol/l, K+ 2.0 mmol/l, HCO3- 52 mmol/l), whilst the parotid saliva electrolytes showed Na+ 21 mmol/l, K+ 28 mmol/l, HCO3- 1 mmol/l, Cl- 14 mmol/l), and the urinary electrolytes, K+ 46 mmol/l, Na+ 116 mmol/l, HCO3- 48 mmol/l.

The patient was reviewed in light of these results and on further questioning admitted to self-induced vomiting. Vomiting had become habitual following the episode of recurrent vomiting with subsequent haematemesis. He admitted to periodic episodes of overeating over which he had no control, which he related to a feeling of fatness and a fear of getting back to his old weight. Oral potassium supplements were given and the patient was referred for psychiatric assessment and therapy. He subsequently began an anxiety management programme which was combined with cognitive behavioural therapy. A gradual improvement in weight gain and reduction in parotid swelling was mirrored by an increase in serum potassium levels (Figure 3).
3. Hypokalaemic alkalosis. Some $K^+$ is lost directly in the vomitus but the main effect of repeated vomiting on potassium balance is through a change in urinary potassium excretion. The metabolic alkalosis caused by the loss of gastric HCl results in reduced renal proximal tubular $\text{HCO}_3^-$ ion absorption owing to relative absence of $\text{H}^+$ ions. Proximal Na+ ($\text{HCO}_3^-$ ion's associated cation) and $\text{H}_2\text{O}$ absorption is also decreased. This results in increased NaHCO$_3$ delivery to the distal tubules, enhancing Na$^+$ secretion at this site, and increasing $\text{H}^+$ and K$^+$ secretion. A mechanism for electrolyte exchange similar to that in the kidney may well be functioning in the parotid since both the urine and parotid saliva in this patient have high K$^+$ ion concentration and a low pH.

Since 90–95% of reported cases of bulimia are in females with little change in weight it is, perhaps, not surprising that in this case a diagnosis was not arrived at earlier. The patient's sexual history, weight loss, lymphadenopathy and fungal groin infection required that the possibility of HIV-related salivary gland disease should be eliminated.

Enlargement of the parotid gland (Figure 1) and occasionally the submandibular gland is a now well recognized sequitur in bulimia. The swelling is generally asymptomatic and intermittent. The underlying pathophysiology remains uncertain, but suggested causes have included high carbohydrate intake, bulk diet, malnutrition, pancreatic damage, regurgitation of acidic gastric contents and metabolic alkalosis. In 1956, Du Plessis reported parotid swelling in severe malnutrition and suggested that an element of work hypertrophy might be involved. He proposed that prolonged stimulation of salivary glands by an exaggerated oesophago-salivary reflex originating in the oesophagitis of malnutrition was involved. Similarly in bulimic patients it seems possible that low oral pH caused by the prolonged presence of gastric acid in the mouth due to vomiting combined with episodes of binge eating stimulate repeated periods of increased salivary flow and so cause 'work hypertrophy' of salivary gland parenchyma.

References

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