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Case Report

Case Report

A case of life-threatening, early postoperative refeeding syndrome in an obese young female undergoing laparoscopic sleeve gastrectomy

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Abstract

Early complications after a laparoscopic sleeve gastrectomy (LSG) include bleeding, leaks, strictures and bowel obstructions. Patients post-LSG experience rapid but intended weight loss and may be on a restricted diet before and following surgery. Thus, many of these patients are in a malnourished state, placing them at a risk of developing potentially life-threatening refeeding syndrome (RFS). We describe the case of an 18-year-old female who developed RFS 2 weeks after LSG. We examine potential causes, review literature and discuss RFS pathophysiology as well as the guidelines that could help prevent RFS in bariatric surgery. Currently, not much is known about the risk of RFS in bariatric surgery and to our knowledge, this is the first report of RFS occurring in the early postoperative phase after LSG. A globally accepted definition of RFS should be established for guidelines to encompass wider patient groups.

Keywords: colorectal surgery, upper GI surgery

Introduction

With the prevalence of obesity increasing globally, bariatric surgery (BS) has established its efficacy for sustained weight loss and reduction of obesity-related comorbidities [1]. Early postoperative morbidity can be associated with both, surgical (bleeding, infection, staple-line failure) and nutritional (micro- and macronutrient deficiencies) complications [2]. When carbohydrate rich foods are reintroduced following very low calorie diets (VLCD) as part of BS protocols, the body can emerge from this period of starvation with gross electrolyte derangements, which we now recognise as potentially life-threatening refeeding syndrome (RFS) [2, 4].

Case presentation

An 18-year-old female with a pre-operative body mass index (BMI) of 33.5 kg/m^2 (height 163 cm; weight 89 kg) underwent laparoscopic sleeve gastrectomy (LSG) due to increasing obesity secondary to longstanding polycystic ovarian syndrome and insulin resistance. Peri-operatively, the patient was placed on a VLCD consisting of 800 calories per day for a total of 4 weeks, commencing 2 weeks prior to her operation.

Two weeks post-operatively, her BMI was 29.7 kg/m^2 (weight 79 kg) and her diet was upgraded to include pureed foods. Post-prandial nausea impaired her oral intake and she presented to the local Emergency Department (ED) with dehydration. She responded well to intravenous rehydration and was discharged the following day. Within 24 hours, she re-presented to ED with a headache, fever (39.3°C) and significant electrolyte disturbances following ingestion of a carbohydrate dense meal of sushi. Her electrolytes were grossly deranged: potassium (1.8 mEq/L), magnesium (0.53 mmol/L) and phosphate (0.39 mg/dL). Full blood count revealed mildly elevated white cells. Her electrocardiograph demonstrated changes of u-waves and ST depression. An abdominal CT was performed, which excluded a post-surgical complication. In the absence of a clear surgical cause for the patient's presentation, a diagnosis of severe RFS was made and she was admitted to the ICU for 3 days of monitoring, electrolyte replacement and balanced refeeding in accordance with the National Institute for Health and Care Excellence (NICE) (2006) Guideline [5].

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Her BMI at 6-month follow-up with the dietician was 25.7 kg/m^2 (TBWL 22.4%). She was tolerating a balanced diet with no long-standing symptoms. She had adjusted to her new routine of full-time work and was consuming appropriate amounts of calories, nutrients, and fluids.

Discussion

While RFS has no internationally standardised definition, it is widely accepted that its hallmark is hypophosphatemia following re-commencement of enteral or parenteral nutrition after a period of malnutrition (i.e. anorexia; or prolonged nil by mouth periods) [4]. With low carbohydrate intake, insulin production is decreased. The body enters catabolism, using ketosis and gluconeogenesis for energy production, consuming potassium, phosphate and magnesium. Homeostasis ensures serum electrolyte concentrations are maintained despite depleted overall body stores as intracellular concentrations plummet [6]. With the reintroduction of carbohydrates, anabolism returns, and the release of insulin from the pancreas promotes glycogenesis and inhibits lipolysis. This insulin spike drives cellular uptake of phosphate, potassium and magnesium, along a steep concentration gradient, resulting in hypokalaemia, hypophosphatemia and hypomagnesemia. To maintain osmotic neutrality, sodium and water are retained causing significant fluid shifts and resultant oedema. These severe fluid and electrolyte imbalances put patients at risk of tissue hypoxia, congestive cardiac failure, myocardial dysfunction, cardiac arrhythmias and, eventually, death [6].

A review of the literature revealed only 4 other case reports of refeeding syndrome following BS [2, 3, 7]. Two of the cases occurred after biliopancreatic diversion/duodenal switch, one after gastric banding, and the other after a Roux-en-Y bypass. No case reports have previously found RFS after an LSG.

BS currently carries an unknown risk of RFS. According to the NICE guidelines, major risk factors for RFS are having a BMI <16.5 or < 18.5, depending on other risk factors; and rapid unintended weight loss [5]. Emphasis on 'unintentional' weight loss of 10–15% over 3–6 months as a risk factor for RFS, may contribute to the lack of awareness that RFS can occur in patients with high BMIs, BS patients. While BS patients often have higher BMIs and are indeed undergoing intentional weight loss, they are at an increased risk of mineral and vitamin malnutrition [8, 9]. This is not only due to a strict diet, but also because patients following BS experience a reduced capacity, impaired tolerance, and decreased absorption of nutrients. For example, LSG treats obesity by drastically reducing stomach volume, greatly increasing gastric emptying, and small bowel transit time, limiting digestion and absorption of all dietary intake [1]. VLCD programs, which many bariatric patients are also recommended perioperatively, induce ketosis by limiting carbohydrate intake. VLCD treatment protocols vary, with programs providing 450–800 calories a day for periods ranging from 2 to 6 weeks [8]. Our patient had undergone a rapid weight loss of 11.3% in 2 weeks, and others on similar VLCD diets also presumably undergo weight loss at a rate faster than 10–15% in 3 months, putting them in a malnourished state and thus increased risk of RFS.

Current recommendations for BS patients include introducing pureed foods at two weeks post-operation, solids at five weeks, and only reintroducing a regular diet after eight to nine weeks; with ongoing vitamin and mineral supplementation [8]. It is crucial that patients are educated and remain compliant with structured nutritional plans to avoid risk of RFS. While patients undergoing BS are seemingly protected from RFS due to the anatomical restrictions of a smaller stomach compartment, carbohydrate-dense foods such as liquid carbohydrates and sugared drinks could trigger RFS. Dietitians should be closely involved in the care of pre- and post-op BS patients. While RFS and its complications are rare, this life-threatening syndrome warrants stringent monitoring and prevention.

RFS in obese individuals may also be unexpected, possibly due to their high BMI. However, many obese individuals are paradoxically malnourished. Besides a poor diet, biochemical mechanisms in obese individuals are altered, causing inadequate nutrition [9]. Some micronutrient deficiencies observed in obese individuals include vitamin D, chromium, biotin, thiamine and vitamin C, of which many are important cofactors and vitamins essential to glucose metabolism and insulin signaling [10], contributing to the development of RFS following BS in obese individuals due to already altered glucose metabolism.

Conclusion

A globally accepted definition of RFS should be established so that incidence and prevalence of the syndrome can be identified and studied in select patient populations. Guidelines should also be updated to include broader definitions of patients in at-risk groups (such as bariatric patients who have undergone prolonged periods of low carbohydrate fasting, patients with higher BMI) so that clinicians can easily identify individuals that do not fit preconceived ideas surrounding RFS.

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Conflict of interest statement

There are no conflict of interests to declare.

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Data availability

All data underlying the results are available as part of the article and no additional source data are required.

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