Review article

Protein malnutrition after Roux-en-Y gastric bypass: a challenging case and scoping review of the literature

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Abstract
Although protein malnutrition (PM) is often reported after highly malabsorptive procedures, its exact incidence and mechanisms after Roux-en-Y gastric bypass (RYGB) are poorly understood. The aim of this study was to present a challenging clinical case of PM after RYGB and conduct a scoping review of the literature. Among the 18 studies with 3015 RYGB patients included in the review, the median incidence of PM was 1.7% (range, 0%–8.9%), and it was diagnosed 12 to 120 months after RYGB. The most common cause is insufficient oral intake of protein; however, in cases of persistent hypoalbuminemia, a thorough diagnostic workup needs to be performed. Risk factors for PM after RYGB include specific triggering events such as intractable vomiting and dysphagia, and a total alimentary limb length less than 250 to 300 cm. (Surg Obes Relat Dis 2023;■:1–9.) © 2022 American Society for Metabolic and Bariatric Surgery. Published by Elsevier Inc. All rights reserved.

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Protein malnutrition (PM) is a potential adverse effect of bariatric surgery, mostly encountered after severely malabsorptive procedures. Less frequently, PM can be observed after proximal Roux-en-Y gastric bypass (RYGB) and even sleeve gastrectomy (SG), pointing to other causes beyond malabsorption for this complex problem [1,2]. Mild hypoalbuminemia (serum albumin 30–35 g/L) is most often observed, whereas severe protein deficiency/PM remains an infrequent but serious metabolic disorder.

In 1998, Mason had already identified "starvation injury" as a potentially lethal consequence after gastric-reduction surgery, describing PM and other nutritional deficiencies in patients with intractable postoperative vomiting [3]. Although such severe symptoms are uncommon with modern bariatric procedures, PM is still associated with an annual hospitalization rate of 1% [4]. Most cases can be managed with dietary counseling and protein-enhanced oral supplements. If left untreated, severe PM (albumin <25 g/L) can lead to anemia, edema, Kwashiorkor starvation injury, and liver failure [5]. Such cases may be resistant to conservative treatment and require revision surgery in order to optimize absorption or even reverse to normal anatomy.

Protein and other nutritional deficiencies are more common after highly malabsorptive procedures such as biliopancreatic diversion with duodenal switch (BPD-DS), one anastomosis gastric bypass (OAGB), or distal Roux-en-Y gastric bypass (D-RYGB). However, awareness still needs to be raised for PM prevalence and management after proximal RYGB, which remains a widely performed bariatric procedure worldwide. The aim of the present study was to conduct a comprehensive literature review to summarize the existing knowledge on the incidence, risk factors, and all other clinically relevant aspects of PM after RYGB. To further illustrate this complex problem, a challenging clinical case is also reported.

Clinical case presentation

After a complete preoperative workup, a 53-year-old female with a body mass index (BMI) of 43.4kg/m^2 (111 kg) underwent laparoscopic RYGB, with a 45-cm biliopancreatic limb and a 1-m alimentary limb.

Weight loss evolution was favorable (Fig. 1A), however hypoalbuminemia appeared on the third year post RYGB
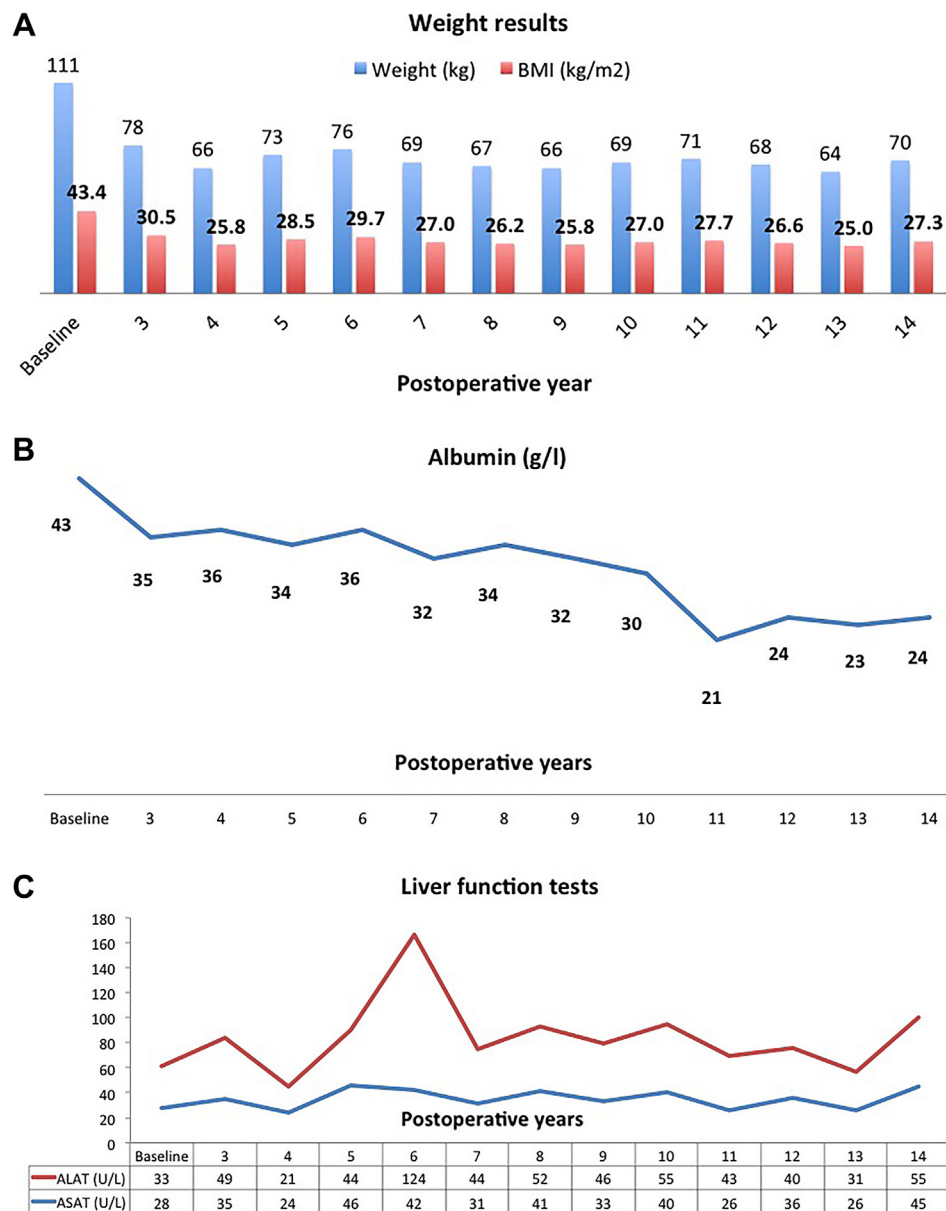

Fig. 1. Graphic representation of (A) weight loss evolution, (B) serum albumin values, and (C) liver function tests from baseline (preoperative) until the 14th postoperative year. ALAT = alanine transaminase; ASAT = aspartate aminotransferase; BMI = body mass index (kg/m^2).

and continued to worsen despite close nutritional follow-up and the introduction of oral nutritional supplements with high quality and concentrations of proteins (Fig. 1B). A moderate but persistent elevation of liver function tests was observed (Fig. 1C), while exhaustive workup repeatedly ruled out underlying infectious, metabolic, autoimmune liver diseases, or inflammatory bowel disorders. Other common post-RYGB deficiencies (calcium, vitamin D and B12, folic acid, iron, magnesium, zinc) were regularly supplemented. Extensive workup failed to identify the cause of hypoalbuminemia (Fig. 2, algorithm), while liver fibroscan did not reveal cirrhosis despite endoscopic stigmata of mild portal hypertension (stage I esophageal varices, portal colopathy). The patient presented episodic abdominal pain during the seventh postoperative year (PY). Radiologic and endoscopic workup were normal, bacterial overgrowth was diagnosed, and antibiotic treatment (metronidazole) provided transient symptomatic relief. As intermittent abdominal pain and watery diarrhea persisted, the diagnosis of protein-losing enteropathy of unknown origin was the diagnosis of exclusion. Acute abdominal pain on the 12th PY led to a new CT scan (Fig. 3), suggesting internal hernia. Laparoscopy confirmed massive chyloperitoneum and an internal hernia through the Petersen's space with clear signs of chronic venous and lymphatic stasis of the incarcerated 150-cm segment of distal small bowel. There were no signs of intestinal ischemia or necrosis, thus no resection was

necessary. The mesenteric defect was closed and postoperative course was uneventful.

Currently, at 14 years after RYGB and 2 years after internal hernia repair, the patient is closely followed by the multidisciplinary bariatric team with ongoing protein-enhanced oral supplements (Resource Ultra 200 mg, 450 Kcal/28 g whey protein, 4 g leucine, 1-2 daily), as well as intermittent enteral nutrition when needed (Fresubin 2 kcal/ml 500-750 ml/24 h through a nasojejunal feeding tube) trying to reach the recommended targets of daily protein intake 1–1.5g/kg of ideal body weight [6,7]. Even so, albumin levels remain below 24 g/L.

The combination of reduced oral protein intake, a certain metabolic resistance to protein absorption post-RYGB and low levels of physical exercise is the most plausible explanation of chronic hypoalbuminemia in this patient. The internal hernia transiently worsened PM, mostly by inducing abdominal pain and food intolerance; in addition, some reduction of the absorptive capacity of the incarcerated small bowel may have occurred, due to the venous and lymphatic stasis and resulting villous congestion. Since the impact of the internal hernia should be reversed after its correction, other underlying factors such as insufficient protein intake seem to play the predominant role of severe chronic PM in this case. Despite exhaustive workup of hypoalbuminemia in this patient and rigorous protein supplementation, severe PM persists to this day (<25 g/L)
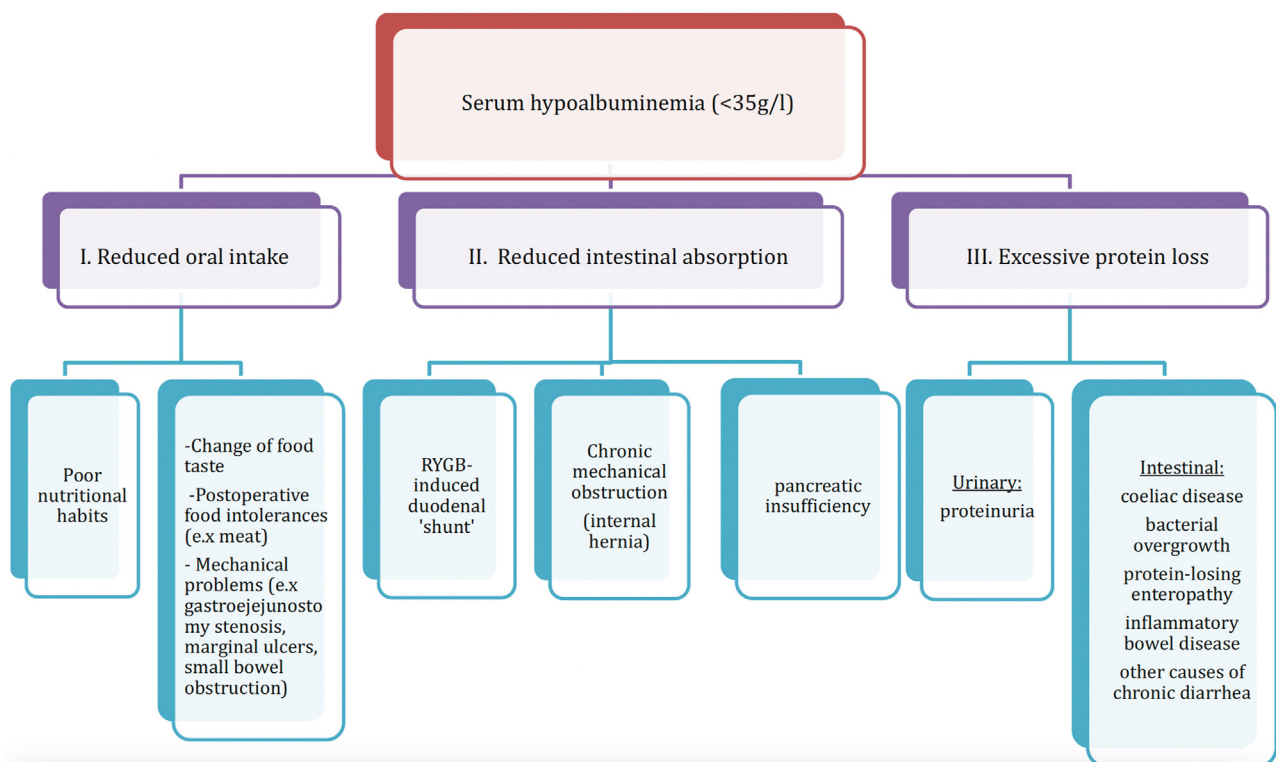

Fig. 2. Diagnostic algorithm in patients with protein malnutrition (PM) after Roux-en-Y gastric bypass.
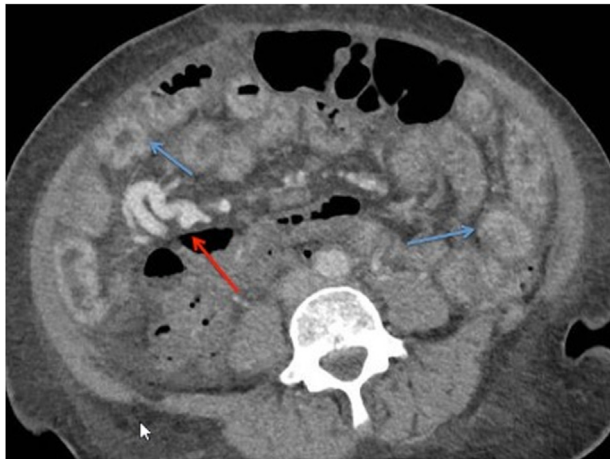
Fig. 3. Radiologic image of chronic venous and lymphatic occlusion of the small bowel after Roux-en-Y gastric bypass. Abdominal computed tomography showing dilated venous collateral circulation in the superior mesenteric vein territory (red arrow) and diffuse thickening of the small bowel (blue arrows) with free intra-abdominal fluid. Laparoscopy confirmed a Petersen space internal hernia, with a twist of the mesenteric root responsible for the chronic venous and lymphatic obstruction.

and the exact cause remains unclear. In such refractory cases, reversal of the RYGB might be an option but was not considered in this patient, who is compliant to follow-up and supplementation and prefers to continue close surveillance rather than taking the risk of weight regain following reversal.

Scoping methodology

A comprehensive literature review was conducted to answer the question: "What is known on PM as a metabolic complication of proximal Roux-en-Y gastric bypass?." As PM after RYGB is a rare complication with no clear consensus on a clinically relevant cutoff value, and because variable follow-up across published studies may have a significant impact on reported PM rates, a meta-analysis would be plagued by a considerable amount of bias and would be of little clinical value. Instead, a scoping review was preferred with the aim to map the existing literature on the subject and summarize information on the following aspects: incidence, potential risk factors, and clinical consequences of PM after RYGB [8]. Of note, proximal RYGB will be referred to as RYGB to avoid confusion with distal (D-RYGB) and very distal Roux-en-Y gastric bypass (VD-RYGB).

A database search was performed in MEDLINE via PubMed and Embase with the terms "protein malnutrition" OR "hypoalbuminemia" AND "gastric bypass" OR "RYGB." Study selection was performed according to pre-specified criteria, as stated by the SPIDER framework [9] (Online Appendix 1). The study is reported according to the Preferred Reporting Items for Systematic reviews and Meta-Analyses for Protocols extension for Scoping Reviews (PRISMA-ScR) guidelines [10]. The patient of the clinical case provided written informed consent for research purposes (Protocol No 12_001 BGC).

For quantitative analysis, the pooled median of the primary endpoint was reported (incidence of PM after RYGB) considering the absolute number of RYGB patients in all included studies.

Review results

From the 396 search results initially retrieved, 18 studies were included in the review (Online Appendix 2, PRISMA flowchart). Overall, 3015 RYGB patients were analyzed in 14 comparative [1,11–23] and 4 noncomparative [24–27] studies. Postoperative follow-up varied between 12 and 120 months, with PM rates reported at the latest timepoint in all studies. PM presented a wide range of definitions (serum albumin 24-40 g/L), with no clear consensus of which cutoff should be considered most clinically relevant.

Among the 18 aforementioned studies, the pooled median incidence of PM after RYGB was 1.7% (range, 0%–8.9%); 6 of those studies report a 0% rate (Table 1). In the 3 studies displaying results after D-RYGB, PM rates were .4% [14] , 5% [18], and 21.1% [21], but after VD-RYGB they escalated to 63.6% 3 years postoperatively [21]. Definition of D-RYGB and VD-RYGB was self-reported by the authors of included studies, with considerable variation of limb lengths as illustrated in Table 1. The incidence of PM after OAGB is 2.8% to 14% [11,13,19,20,22], whereas BPD has similar rates of 3.6% to 14.9% [15–17,23]. Finally, in the 5 studies including patients with SG, PM rates were between 0% and 1.2% [1,12,15,20,22]. Among RYGB patients, 0% of revisional surgery for intractable PM was reported in 7 studies [11,14,16,23,24,26,27], whereas it was 1.7% [25] and 1.9% [20] in 2 other series. RYGB limb lengths are detailed in Table 1, illustrating heterogeneity in surgical technique among included studies.

Some studies assessed specific risk factors for PM after RYGB; Chen et al. [20] identified male sex, higher baseline BMI, short total bowel length, short Roux limb, and total alimentary limb length (TALL), whereas Jammu et al. [22] showed a vegetarian diet, pre-existing diabetic nephropathy, and alcoholic and nonalcoholic fatty liver disease as risk factors. Older age (with no precise cutoff) demonstrated a higher risk for PM in the study by Suàrez-Llanos [17].

Faintuch et al., in a series of banded RYGB, identified specific triggering events in 63.6% of all who presented PM, most of whom (70%) had developed intractable vomiting or severe dysphagia due to local complications of the band around the pouch [25]. Ghiassi et al. analyzed a series of patients undergoing revisional RYGB for weight loss failure, and reported significantly higher rates of PM when TALL measured less than 250 to 300 cm. Similarly, Chen

Table 1
Overview of included studies

First author and year	Number of RYGB patients	Procedure(s) studied	Definition of limb lengths			PM rate (%)	Revisional surgery for PM (%)	Albumin cutoff	Follow-up time after surgery
			BP limb (cm)	RL length (cm)	TALL (cm)				
Bhandari 2019 [11]	122	RYGB versus OAGB	80 250	120 –	NA NA	7.3 10.5	0 1.1	<30 g/L	60 mo
Billeter 2015 [24]	20	RYGB	75	150	NA	0	0	<35 g/L	24 mo
Chen 2019 [20]	377	RYGB versus OAGB versus SG	100 200	≈250 –	NA NA	1.8 2.8 1.2	1.9 1.2 0	<35 g/L	12 mo
Faintuch 2004 [25]	236	RYGB	70	100	NA	4.7	1.7	<25 g/L	68 mo
Ghiassi 2018 [21]	96	RYGB versus D-RYGB	25 NA	100 NA	NA 400–450	2.1 21.1	NA	<32 g/L	36 mo
Gracia 2009 [23]	115	RYGB versus BPD/DS	NA NA	150 NA	450 250	0 11	0 3.2	NA	120 mo
Jammu 2015 [22]	295	RYGB versus OAGB versus SG	50 200	75 –	NA NA	2 13.1 0	NA	<35 g/L	NA
Lin 2019 [1]	79	RYGB versus SG	100	100	NA	8.9 1.2	NA	<40 g/L	12 mo
Khalaj 2020 [19]	145	RYGB versus OAGB versus OAGB	50 200 160	150	NA	0 4.7 0.3	0 2.6 0	NA	12 mo
Murad 2018 [26]	102	RYGB	200	50	NA	0	0	<35 g/L	65 mo
Peterli 2013 [12]	110	RYGB versus SG	50	150	NA	0.9 0	0 0	NA	36 mo
Ritz 2009 [27]	110	RYGB	100	120	NA	0.9	0	<30 g/L	24 mo s
Risstad 2016 [18]	61	RYGB versus D-RYGB	50 50	150 NA	NA 150	2 5	0 3.3	NA	24 mo
Robert 2019 [13]	63	RYGB versus OAGB	50 200	150 NA	NA	0 14	0 3.4	NA	24 mo
Shah 2019 [14]	671	RYGB versus D-RYGB	60 200	150 60	560 420	0 0.4	0 0	NA	96 mo
Skroubis 2011 [15]	227	RYGB versus BPD/DS versus SG	50 NA	150 NA	NA 500	2.2 3.6 0	NA	NA	36 mo
Skroubis 2014 [16]	65	RYGB versus BPD/DS	60 175	100 NA	NA 100	1.5 4.6	0 1.5	<25 g/L	96 mo
Suàrez-Llanos 2015 [17]	121	RYGB versus BPD/DS	50–150 50	50–250 NA	NA 350	8.7 14.9	NA	<35 g/L	24 mo

RYGB = Roux-en-Y gastric bypass; PM = protein malnutrition; BP limb = biliopancreatic limb; RL = Roux limb; TALL = total alimentary limb length; OAGB = one-anastomosis gastric bypass; NA = not available; SG = sleeve gastrectomy; D-RYGB = distal Roux-en-Y gastric bypass; BPD/DS = biliopancreatic diversion/duodenal switch.

et al. identified a TALL less than 400 cm in 100% of RYGB patients needing revision for PM [20].

Discussion

Protein malnutrition is a rare but potentially serious metabolic complication of proximal RYGB, reported in a pooled median of 1.7% (range, 0%–8.9%) of patients in the present review. Although PM is much less common after RYGB than malabsorptive procedures such as OAGB, BPD/DS, and D-RYGB, it still concerns a substantial number of patients given the total number of RYGB performed worldwide. Several factors have been suggested to predispose to PM after RYGB. Some of them are nonmodifiable, such as age, male sex, and higher BMI at baseline, or pre-existing liver or kidney disease [17,20–22,25]. Specific attention is needed to detect changes in food preferences and intolerances that may develop after bariatric surgery, avoidance of solid and dense food such as meat, and shifting to softer foods containing little or no proteins. The diagnostic, and hence therapeutic, approach to a patient with PM after RYGB is rather complex and mandates

multidisciplinary assessment, as illustrated in the algorithm (Fig. 2). Some of the most frequent causes of PM are further detailed in the following three sections.

Insufficient protein intake

Even in modern bariatric surgery programs pre-existing hypoalbuminemia has been reported in up to 6.2% [27,28] of patients, and up to 9.2% in low-income countries [29]. Patients with obesity can be malnourished, and PM may stem from long-standing poor alimentary habits. Thus, in case of hypoalbuminemia after bariatric surgery it is of prime importance to monitor nutritional and dietary protein intake, especially in vegetarian/vegan patients where potential sources of protein may be reduced.

The latest American Society for Metabolic and Bariatric Surgery (ASMBS) guidelines suggest that at least 10% to 35% of the daily postoperative caloric intake should consist of proteins, with a minimal intake of 60 g/d [6]. This should be individualized, assessed, and guided by a specialized dietician with regard to sex, age, and weight [6]. In case of massive weight loss or after a malabsorptive procedure such as BPD/DS, up to 1.5 to 2 g protein/kg/d may be needed to prevent lean mass reduction and PM [6,30]. Giusti et al. demonstrated a drastic reduction of protein intake after RYGB, from 87 ± 4 g/d preoperatively to 29 ± 2 g/d 1 month postoperatively [31]. This tendency improved up to 5 years later, with a median daily intake of 57 ± 3 g, without reaching preoperative levels in most patients. A robust explanation for this phenomenon is difficult to provide, however several factors may be held responsible. In addition to the expected restrictive effect of surgery, a change of taste and food preferences may be observed in some patients after RYGB further modifying food ingestion [32]. However, inadequate compliance to follow-up or poorly structured nutritional follow-up are often the leading causes of low protein ingestion in bariatric surgery patients [25].

Limb length matters

Standard RYGB induces caloric restriction through gastric volume reduction with only a moderate degree of malabsorption, as a long common channel is kept functional. However, the present review clearly demonstrates that not all "proximal" RYGB procedures are alike, and substantial variations may be observed in the different limb lengths (Table 1). Of note, no cases of PM were observed in the 2 RYGB studies where TALL was measured (450 and 560 cm, respectively) [14,23]. In contrast, PM was reported in more than 5% D-RYGB patients [18], reaching 29% to 63% in very distal procedures with a TALL of 250 to 350 cm [21,33]. A TALL greater than 300 cm has been suggested as the minimum threshold of safety to avoid severe PM in RYGB patients [20,33] while Ghiassi et al. suggested elongation of TALL to 400 to 450 cm to combine satisfactory weight loss with low rates of hypoalbuminemia

[21]. A recent Dutch series assessing the benefit of RYGB distalization in cases of insufficient weight loss reported that 43% of patients developed PM and 89% suffered from refractory vitamin deficiencies despite regular multivitamin supplementation, with a TALL of 300 cm. The rate of debilitating diarrhea that adversely affected overall quality of life was also high [34]. The authors suggest a TALL of more than 450 cm (250 cm AL and 200 cm common channel) as a safer option if a distalization process is considered [34]. As the present literature review suggests, the TALL is rarely measured and reported in RYGB series, mostly to avoid the time-consuming intraoperative bowel measurement and the related risk of inducing iatrogenic small bowel injuries. Although a standard proximal RYGB may not necessarily mandate such a measurement, precise measurement of all limb lengths and mostly the TALL should be performed in all cases of revisional surgery, especially if PM is present or bypass distalization is planned.

In general, the most effective bariatric procedures for weight loss and co-morbidity control also carry the highest risk of nutritional deficiencies. To this day, BPD/DS offers the best long-term results for weight loss and improvement of obesity-related morbidities, but it also results in more severe nutritional and metabolic complications than other procedures [35,36]. In the 4 comparative studies including RYGB and BPD/DS patients [15–17,23], BPD/DS induced significantly higher rates of PM than RYGB (3.6%–14.9% versus 0%–8.9%, respectively). OAGB represents an alternative to RYGB to improve weight loss and metabolic benefit [13]. Jammu et al. [22] reported PM in 13.1% (3.8% severe cases), while Bhandari et al. reported PM rate of 10.5% after OAGB [11]. Notably, the YOMEGA trial found a 14% PM rate after OAGB versus 0% after RYGB [13]. Chen et al. reported low rates of PM after 1 year in a series of 2048 patients who underwent OAGB (2.8%) versus RYGB (1.8%) and SG (1.2%) [20]. However, a recent meta-analysis identifies severe PM as the leading cause of reversal after OAGB, with a high rate of postoperative complications including liver failure in these patients [37]. A recent consensus of OAGB experts reveals large variations in terms of surgical technique (BP limb length, the need for total bowel measurement), as well as routine nutritional recommendations that could largely influence PM [38]. The malabsorptive nature of OAGB warrants close monitoring of patients for nutritional deficiencies, especially those with BP limb greater than 200 cm, as was chosen by the majority of surgeons, in the consensus survey. In the current review, OAGB patients were found to have higher rates of PM than RYGB.

The belief that restrictive procedures do not result in protein deficiency is common. Although PM after SG is infrequent (0%–4.2% 5 yr after surgery) it can still be observed due to restricted dietary protein intake, as well as underlying poor alimentary habits [1,12,15,20,22]. Thus, long-term surveillance for nutritional deficiencies after SG is as

important as for any other bariatric procedure, as deficiencies may develop much later after surgery.

Specific triggering factors

Over the years, functional results and quality of life after bariatric surgery have shown some significant improvements. However, food intolerance, functional abdominal pain, and mechanic obstruction (anastomotic stricture, intestinal obstruction) still occur. Severe hypoalbuminemia after bariatric surgery in often precipitated by food intolerance, anastomotic ulcers, chronic diarrhea, intestinal or gastric outlet obstruction. Indeed, disturbance in normal alimentary habits may not only reduce food intake altogether, but also favor consumption of liquid or semi-liquid foods, leading even to a paradoxical weight regain in a patient with PM [39]. This seemed to play a role, though transient, in the case of our patient, who experienced worsening of PM while food tolerance was impaired due to the internal hernia.

Consequences of PM after bariatric surgery: from hair loss to liver failure

Initial clinical manifestations of PM include altered hair quality and hair loss, and in more advanced stages exercise intolerance, generalized weakness, edema, osteoporosis, and muscle wasting. In addition, patients with PM are very likely to present other micro-and macro-nutrient deficiencies [40]. Another subtle but potentially disastrous metabolic complication of PM is liver toxicity. Although bariatric surgery is known to improve pre-existing steatosis and steatohepatitis [41], new-onset liver injury can also appear postoperatively [42]. The jejuno-ileal bypass, once widely performed as a weight loss procedure, has nowadays been abandoned due to its severe metabolic consequences including liver failure and long-term mortality reaching 10% [43]. Biliopancreatic diversion with duodenal switch (BPD/DS) has also been related to transient or permanent liver damage, leading even to cirrhosis and transplantation after 5 postoperative years [44]. Similarly, liver toxicity after OAGB has been reported in the literature [45].

Although liver failure is observed after highly malabsorptive procedures, its exact mechanism remains complex and poorly understood. In the immediate postoperative period, rapid weight loss and peripheral fat mobilization produce an excessive free fatty acid load in the bloodstream and portal circulation, resulting in hepatotoxicity [46]. In the long-term, chronic protein deficiency may induce autophagy/autolysis of liver cells, in order to retrieve amino-acids for albumin synthesis [4,42]. In addition, small-intestinal bacterial overgrowth induces bacterial translocation into the portal venous system, resulting in hepatocellular damage [47–49]. When PM is observed after bariatric surgery, extensive workup is needed, especially in presence of other concomitant liver aggressors (viral/autoimmune hepatitis, alcohol intake, intestinal bacterial overgrowth),

as liver fibrosis and failure may appear with few or no clinical manifestations until late in the course of the disease. As bariatric surgery and RYGB in particular has a protective role against obesity-related liver toxicity, PM should be proactively researched and corrected to prevent its deleterious effect on long-term liver function [46].

The management of an RYGB patient presenting PM is complex, and mandates prompt multidisciplinary assessment (Fig. 2). In addition to understanding the causal factors, adequate protein intake is needed, either by oral protein supplements or even through more invasive means, such as a nasojejunal feeding tube. In severe, refractory cases or when patient compliance is limited or uncertain, parenteral nutrition may be considered. Surgical revision may also be an option to verify the absence of mechanical issues affecting the small bowel, to measure the entire bowel length if not clarified during primary surgery, and eventually to elongate the TALL to at least 400 to 450 cm, with a minimal common channel length of 200 to 250 cm. In patients where the aforementioned steps fail to improve severe protein/nutritional deficiencies, proximalization of D-RYGB or reversal of RYGB to normal anatomy need to be discussed. Adequate information and preoperative dietary counseling are key to minimize the risk of weight regain in this case.

Limitations of the study

The present study has some limitations that need to be discussed. A statistical synthesis of results (meta-analysis) was precluded by the limited number of comparative studies between RYGB and other techniques in terms of PM, as well as the heterogeneity of reported thresholds for hypoalbuminemia. A pooled analysis was deemed suitable, reporting the arithmetic median of PM after RYGB. Notably, there is some significant variation in reported PM rates (0.8%–9%). This may be partly explained by the difference in thresholds used to define PM (serum albumin <25–35 mg/dL). In addition, the substantial variability in rates of long-term follow-up across included studies may influence PM detection. Series with a short (<12 mo) follow-up were excluded from the review to limit this source of bias, however as both the duration and completeness of follow-up is variable among bariatric surgery centers, some heterogeneity is to be expected. A scoping review was deemed the most appropriate design to summarize published literature on PM after RYGB, aiming to raise awareness and provide an overview of the incidence, potential causes, and consequences of this complex problem. The resulting diagnostic algorithm proposed in our study could serve as a valuable tool to optimize diagnosis and management of PM in patients after RYGB.

Conclusion

Protein malnutrition is a rare complication after RYGB, reported in 1.7% of patients in the present pooled analysis.

Rigorous and long-term postoperative follow-up is mandatory in order to detect this deficiency, as it may appear several years after the operation. Although it is most often related to insufficient dietary intake and/or other acute triggering factors, a thorough workup is needed to identify potential causes of PM and guide adequate management. Along with correction of all potential causative factors, aggressive protein supplementation up to 1.5 to 2 g protein/kg/d with high-quality anabolic amino acids is recommended to compensate for the deficiency and prevent its complications, such as edema, muscle wasting, and liver damage.

Disclosures

The authors have no commercial associations that might be a conflict of interest in relation to this article.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.1016/j.soard.2022.12.032.

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