CLINICAL MANAGEMENT extra

Nutrient Deficiency-Related Dermatoses after Bariatric Surgery

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GENERAL PURPOSE:

To provide information on obesity, bariatric surgery, and the nutrient deficiency-related dermatoses that may result from these surgeries.

TARGET AUDIENCE:

This continuing education activity is intended for physicians, physician assistants, NPs, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES:

After participating in this educational activity, the participant should be better able to:

1. Examine issues related to obesity and bariatric surgery.
2. Identify the sources and role of specific nutrients.
3. Recognize the clinical signs and symptoms of nutrient deficiency following bariatric surgery.

ABSTRACT

Obesity is a global epidemic that increases the risk of weight-related comorbidities in modern society. It is complex, multifactorial, and largely preventable. Noninvasive treatments for obesity include diet, exercise, and medication. However, bariatric surgeries are becoming popular procedures for those who do not achieve success with noninvasive weight management treatment. Bariatric surgeries often result in dietary restriction and/or malabsorption, which lead to drastic weight loss. Individuals who had bariatric surgeries need lifelong follow-up and monitoring to ensure adequate intake of nutrients. Nutrient deficiencies can ensue when long-term vitamin and mineral supplementation is not followed. Severe nutrient deficiencies may lead to dermatoses that can be corrected by nutrient repletion and careful monitoring. A case report of nutrient deficiency-related dermatoses is followed by a review of obesity and its treatments with a focus on bariatric surgeries.

KEYWORDS: bariatric surgery, dermatitis, obesity, nutrient deficiency, nutrition, supplementation

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INTRODUCTION

Obesity is a complex, multifactorial, and largely preventable disease. In general, obesity is defined as excess body weight for height. The World Health Organization and the National Institutes of Health specifically define obesity as having a body mass index (BMI) of 30.0 kg/m^2 or higher.[1] It is further subdivided into class 1 (BMI of 30.0–34.9 kg/m^2), class 2 (BMI of 35.0–39.9 kg/m^2), and class 3 (BMI of 40.0 kg/m^2 or higher) categories.

It is estimated that more than one-third of American adults are obese and one-third of adults in the world is overweight or obese.[1,2] Obesity is a global epidemic that increases weight-related health risk and may even pose a national security threat.[2,3] Obesity is a leading cause of heart disease, stroke, type 2 diabetes, some types of cancers, and premature death.[1,4] For the first time in history, today's youth may have a shorter life expectancy than their parents because of obesity, despite advances in technology.[5] Further, Americans with obesity spent $1,429 more on medical care than people with normal weight in 2006.[6] It is estimated that annual healthcare costs attributable to obesity total $190 billion a year, about 21% of healthcare expenditures in the US.[4]

The causes of obesity are multifactorial, and risk factors are divided into those that are modifiable or nonmodifiable. Modifiable risk factors include unhealthy lifestyle habits and environments. Nonmodifiable risk factors include age, family history and genetics, race and ethnicity, and sex.[7] See Table 1 for all the risk factors of obesity.

Energy balance has always been the center stage of weight management. Unhealthy dietary choices, such as excessive consumption of added sugar, processed grains, processed meats, saturated fat, trans fat, and calories, and poor intake of fruits and vegetables can increase the risk of being overweight and obese.[7]

Sedentary lifestyle related to excessive screen time, including television, computer, video game, internet use, and social media has been associated with elevated BMI.[7] High amounts of screen time, especially television watching, is a strong obesity risk factor because it displaces time for physical activity, promotes poor diet through exposure to food and beverage marketing, and provides more opportunities for unhealthy snacking.[8]

Lack of sleep is another emerging risk factor for obesity. Studies have demonstrated a high BMI in people who are deprived of sleep.[9] It is hypothesized that sleep deprivation could increase the risk of obesity by reducing energy expenditure related to being too tired to exercise, increasing consumption of calories because those who are sleep-deprived are awake longer and therefore have more opportunities to eat, and disrupting the hormones that control appetite and hunger.[7,8]

Acute and chronic stress related to major life changes, work or school, difficult relationships, financial problems, children and family, and low self-esteem alter the brain and its production of hormones that affect appetite and eating behavior, resulting in under- or overeating.[7,10]

Built and social environmental factors can increase the risk of being overweight and obese.[7] Individuals make decisions based on their surrounding environments. Environmental factors play a significant role in people's dietary and activity preferences.[8] Food deserts are often situated in neighborhoods with low socioeconomic status, and companies aggressively market the unhealthy food and sugary drinks sold in these food deserts. A lack of access to recreational facilities for exercising; safe ways to walk around the neighborhood; and access to healthy food and snacks in school, in the workplace, or in the convenience store all affect quality of life and a healthy weight.[7,8]

Studies have suggested that individuals can be genetically predisposed to being overweight or obese. More than 50 obesity-related genes have been identified in genome-wide association studies.[11] However, most of these genes account for only small effects on obesity.[11] Although obesity can run in families, an increase in BMI was relatively small between individuals with high and low genetic risk of obesity scores. The epidemic of obesity over the past several decades appears to be beyond genetics.[4,12]

A consistent combination of healthy dietary pattern, regular physical activity, adequate sleep, appropriate stress management, and healthy environment is important for substantial health

Table 1.
RISK FACTORS OF OBESITY

Lifestyle	Environment	Genetics
• Excessive caloric intake	• Lack of access to physical activity	• Age
• Excessive empty calories	• Food deserts	• Family history
• Low physical activity	• Viruses	• Race and ethnicity
• Sedentary lifestyle	• Microbiota	• Gender
• Lack of sleep	• "Obesogens"	• Poor prenatal dietary choices and lifestyle
• Chronic stress	• Poverty	
• Increased screen time	• "Impulse marketing" by the food industry	

benefits and prevention of weight-related chronic diseases such as type 2 diabetes and heart disease.[13]

OBESITY-RELATED SKIN DISORDERS

Obesity is associated with changes in collagen structure and function, altered skin barrier function, abnormal sebaceous glands and sebum production, impaired microcirculation and microcirculation, increased skin infection, poor wound healing, and greater transepidermal water loss.[14,15] In addition, many patients with obesity experience acne, hirsutism, and androgenetic alopecia because of hyperandrogenism related to insulin resistance and hyperinsulinemia.[14,15] Moreover, it is believed that certain dermatologic disorders such as psoriasis, keratosis pilaris, seborrheic dermatitis, lichen sclerosis, acanthosis nigricans, acrochordons, striae distensae, adiposis dolorosa, scleroderma, livedo reticularis, and granular parakeratosis are more common in individuals who are obese.[14,15]

The cutaneous manifestations of obesity vary widely and include benign neoplasms and inflammatory skin disorders. Although recognizing these cutaneous findings is not required to establish a diagnosis of obesity or metabolic syndrome, these may represent downstream effects.[16] The following disorders do not constitute an exhaustive list, but include some key dermatoses associated with obesity.

Acanthosis nigricans presents with hyperpigmented, thickened plaques commonly on the neck and intertriginous areas. These areas are usually asymptomatic. It is a cutaneous manifestation of increased insulin and insulin-like growth factor 1 levels.[14] It is proposed that keratinocytes are activated, resulting in hyperplasia, in the setting of insulin-like growth factor.[17] This is the most common cutaneous finding in obesity, in up to 74% of patients.[14,18]

Acrochordons, or skin tags, are fleshy, skin-colored, pedunculated papules that commonly occur on the neck, axillae, below the breasts, and in groin folds in individuals who are obese. Skin tags can become irritated or painful if traumatized by clothing or jewelry. In one study of 156 patients, 38 had acrochordons, with an increased likelihood of acrochordons with increased BMI.[19] However, some studies suggest acrochordons may be more closely linked to insulin resistance than obesity.[20]

Hirsutism refers to terminal hair growth in areas that typically have minimal or vellus-type hairs. Terminal hair growth of the upper lip, temples, jawline, and chin is common. Hirsutism is thought to be a sign of hyperandrogenism, and facial hirsutism has been associated with obesity.[21] Additional signs of hyperandrogenism, likely from excessive androgen production from fatty tissue, include androgenic alopecia, acne, and hidradenitis suppurativa.

Psoriasis is an inflammatory skin condition that has been associated with cardiovascular disease, including dyslipidemia and diabetes mellitus.[22] Although psoriasis can occur in anyone, it is more common in individuals with obesity. Psoriasis presents with erythematous, scaly plaques and can be associated with nail changes and joint disease.

Obesity is a risk factor for hidradenitis suppurativa. Hidradenitis suppurativa is a devastating inflammatory disorder of the apocrine glands that presents with erythematous, painful papulonodules and sinus tracts. These lesions develop commonly in the axillae, below the breasts, and in the groin and buttocks.[23] Hurley staging is frequently used to characterize the clinical signs of severity.

WEIGHT MANAGEMENT AND ELIGIBILITY FOR BARIATRIC SURGERY

According to the National Health and Nutrition Examination Survey, 49% of American adults tried to lose weight over the course of 12 months from 2013 to 2016.[24] Both the World Health Organization and the National Institutes of Health recommend modest weight loss to improve general health, including improved glycemic control, BP, and cholesterol level.[25] Lifestyle modification is often the first choice for weight management because it is noninvasive. Diet, exercise, and behavior therapy reduce exposure to food, decrease cues to eat, and strengthen dietary restraint. Lifestyle modification enhances the external environment to help patients reduce their caloric intake and increase energy expenditure.[25]

Currently, a number of drugs are approved by the FDA for weight loss.[26] Pharmacotherapy is often recommended for people who

have a BMI 30 kg/m^2 or higher, or BMI 27 kg/m^2 or higher with other risk factors, or diseases who cannot lose weight with lifestyle modifications alone.[25,26] Pharmacotherapy reduces hunger, lowers food preoccupation, increases satiety, and decreases nutrient absorption.[27] Similar to other chronic diseases such as heart failure, hypertension, and diabetes, obesity is often managed with a combination of pharmacotherapy and lifestyle modification.[26]

Making lifestyle modifications is not easy and may not be enough to lose sufficient amounts of weight to achieve optimal health. Bariatric surgery is an alternative option for those who are unsuccessful with lifestyle modification and pharmacotherapy. Bariatric surgery changes the digestive system anatomically to limit the quantity of food that can be eaten and absorbed, resulting in weight loss.[28] It is increasingly accepted and accessible to the general public.[29] In fact, Medicare and many private health insurance providers now offer bariatric surgery coverage.[29]

Bariatric surgery can significantly reduce weight-related health risks via long-term weight loss.[30] In general, bariatric surgery is an option for adults with a minimum BMI of 40 kg/m^2 or a BMI of 35 kg/m^2 or greater coupled with a serious obesity-related health problem such as diabetes, high BP, severe sleep apnea, nonalcoholic fatty liver disease, osteoarthritis, lipid abnormalities, gastrointestinal disorders, or heart disease. Gastric banding is an option for adults with a BMI of 30 kg/m^2 or greater with one of the aforementioned obesity-related health problems.[30,31] Bariatric surgery is not for everyone and is not a way to avoid making lifestyle and dietary changes.[28]

COMMON TYPES OF BARIATRIC SURGERIES

The options for bariatric surgery continue to grow. It is estimated that 228,000 bariatric procedures were performed in the US in 2017, compared with 158,000 in 2011. The gastric sleeve was the most popular surgery, followed by Roux-en-Y gastric bypass.[32] Bariatric surgeries can be classified as restrictive, malabsorptive, or a combination of both.[33] These types of surgeries may also cause hormonal alterations and require dietary changes and exercise to ensure healthy, long-term, sustainable weight loss. See Figure 1 for an illustration of the four common bariatric surgeries.

Laparoscopic Adjustable Gastric Banding

Adjustable gastric band (AGB) is a laparoscopic procedure that places an inflatable band around the upper portion of the stomach.[34] This creates a very small reservoir above the band for holding food, while the rest of the stomach remains intact below the band. The AGB is thought to aid in weight loss because smaller portions are needed to satisfy hunger and cause fullness. The band can be adjusted to create different-sized openings between the pouch and the rest of the stomach. This procedure is solely restrictive; there is no malabsorption of nutrients, and digestion and absorption continue as normal. Ultimately, AGB procedures seem to cause decreased feelings of hunger, early satiety, and fewer calories consumed, but they result in slower weight loss than other surgeries.

Sleeve Gastrectomy

Laparoscopic sleeve gastrectomy (SG), or simply "the sleeve," is a surgery that removes about 80% of the stomach.[34] The remaining portion is a narrow tube that looks similar to a banana. This surgery is meant to aid in weight loss by simply reducing the amount of food that can be consumed. However, studies have shown that the large impact of the sleeve on weight loss is attributable to the changes in intestinal hormones that take place after the surgery.[35] Both of these factors can contribute to decreased hunger, sensations of fullness, and better blood sugar control. Patients who undergo SG require lifelong vitamins and mineral supplementation to ensure optimal nutrition. Over time, many patients experience significant weight regain because of pouch expansion, further alterations in gut hormone levels, lack of nutrition follow-up, and poor dietary choices.

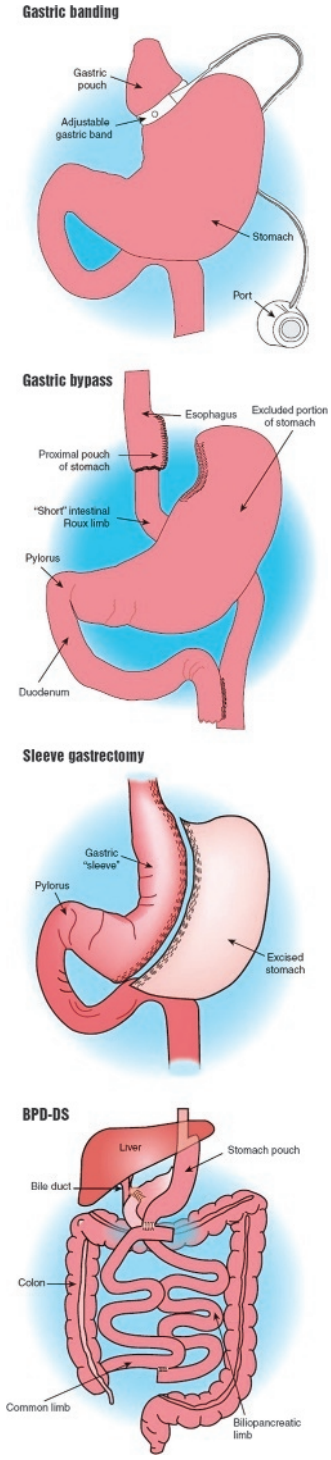
Roux-en-Y Gastric Bypass

In the world of weight loss surgery, Roux-en-Y gastric bypass is often considered the criterion standard and has been the most commonly performed operation for weight loss in the US. It is a more complex procedure than AGB or SG and consists of a surgical anastomosis between the stomach and small intestine. First, the surgeon staples off the upper section of the stomach, forming a pouch usually about 30 mL in volume. Next, the first portion of the small intestine is divided, and the distal portion, called the Roux limb, is connected to the small stomach pouch. Finally, the top portion of the small intestine is connected to a portion further down so that stomach acid and other digestive enzymes can act on the food. This procedure is both restrictive and malabsorptive and results in significant long-term weight loss. The pouch restricts the amount of food consumed, and the rerouting of food past a portion of the small intestine limits the absorption of calories as well as some vitamins and minerals. In addition, this anatomical change causes hormonal adjustments, which lead to alterations in feelings of fullness and hunger.[35] Complications such as gastric distress and discomfort (dumping syndrome), vitamin and mineral deficiencies, and food intolerances can occur after this surgery.[36] These can be managed with dietary compliance and lifelong supplementation. Although Roux-en-Y gastric bypass is a more complicated surgery, some studies show that amount and rate of weight loss are similar to those achieved with SG.[37,38]

Duodenal Switch with Biliopancreatic Diversion

Similar to Roux-en-Y gastric bypass, the biliopancreatic diversion with duodenal switch (BPD/DS) is a two-step surgery that involves a reduction of the stomach and rerouting of the small

Figure 1.
COMMON TYPES OF BARIATRIC SURGERY



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intestine.[34] However, in the BPD/DS, food ultimately bypasses a much larger portion of the small intestine. First, a narrow, banana-shaped pouch is created by removing a large portion of the stomach. Second, the duodenum is cut very close to the gastric outlet. Next, a piece of the last part of the small intestine is connected to the stomach. Finally, the bypassed portion that holds the digestive juices is sewn to the most distal portion, so that the bile and pancreatic enzymes such as lipase and protease can mix with the food. This surgery is considered restrictive and malabsorptive, but over time, the stomach can stretch so that the patient can eat normal-sized meals. Therefore, the long-term effect of this surgery is primarily malabsorptive because of the significantly diminished capacity to absorb protein and fat. Similar complications can result from the BPD/DS as in the Roux-en-Y. This surgery also results in significant weight loss requiring dietary modifications and supplementation.

BARIATRIC SUGERY-RELATED DERMATOLOGIC DISORDERS

An individual with obesity is not necessarily well nourished. Many are already suffering from nutrient deficiencies that can be exacerbated by bariatric surgery given its nature of food restriction and/or nutrient malabsorption.[15] For example, glossitis, angular cheilitis, erythematous desquamative dermatitis, and reduced niacin (vitamin B_3) levels were reported in a young woman 3 months post Roux-en-Y gastric bypass procedure.[15]

Further, alopecia has been observed following Roux-en-Y, AGB, and SG.[15] Hair loss associated with bariatric surgery could be related to telogen effluvium, in which anagen follicles prematurely enter the telogen phase, causing excessive hair loss.[15] Hair and nutrition are interrelated. Protein malnutrition may cause dry and brittle hair. Zinc deficiency and essential fatty acid (EFA) deficiency may cause progressive hair loss.[15] Selenium and biotin deficiencies may cause pseudoalbinism and dermatitis in addition to alopecia. Low iron level may reduce division of hair follicle matrix cells.[15]

Excess skin is a problem for individuals who lose extensive amount of weight following bariatric surgery.[15] Research has shown that fungal infection, eczema, pruritus, excessive perspiration, and hygiene issues are common after Roux-en-Y gastric bypass because of excess skin related to extensive weight loss.[15]

POSTBARIATRIC SURGERY CARE

After surgery, patients are required to follow a specific diet progression to allow for healing, promote weight loss, and help mediate any adverse symptoms and nutrition-related complications.[39] The first phase of the diet consists of thin liquids such as water, broth, skim milk, low-sugar protein shakes, and unsweetened tea. It is important to avoid carbonation and concentrated sweets, because these may cause dumping syndrome. Patients are advanced to pureed foods and eventually to soft foods as tolerated.

Adequate protein is encouraged in all phases of the diet progression to promote healing and the maintenance of lean body mass.

Supplementation

Nutrient deficiencies after bariatric surgery are common.[40] Approximately 50% of patients who have undergone bariatric surgery are nutrient deficient.[41] The level of nutrient deficiency is affected by the complexity of the surgery. Deficiencies are more common after malabsorptive surgery, such as Roux-en-Y gastric bypass and BPD/DS, than restrictive procedures, such as AGB and SG.[40,42] See Table 2 for the properties of vitamins and minerals in the body.

Lifelong supplementation should begin at the time of discharge from the hospital.[40,42] Chewable or liquid vitamin and mineral supplementation is preferred in the first 3 to 6 months postoperation because of ease of absorption.[38,40] Afterward, tablets can be consumed.[40,42] Postoperative vitamin supplementation should include one to two adult multivitamins with minerals, 1,200 to 2,400 mg elemental calcium, 3,000 IU vitamin D or more, and 250 to 350 μg vitamin B_{12} daily.[40] Annual blood work should be conducted to assess nutrition status and prevent deficiency.[40]

CASE REPORT

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

A.B. was a chronically ill-appearing 42-year-old woman with a history of obesity, pancreatitis, and iron deficiency anemia requiring multiple transfusions who underwent a Roux-en-Y gastric bypass 14 years prior to presentation. She was in her usual state of health until 2 months prior to this admission when she developed bilateral lower extremity edema, pain, and blistering. She presented to various other hospitals and was thought to have a diuretic-induced blistering disorder. Following her discharge with topical glucocorticoids, she then presented to the authors' hospital for further evaluation.

Based on her skin condition and history of gastric bypass, dermatology and bariatric surgery consultations were requested. Dermatology performed a total body skin examination that revealed pale conjunctiva with diffusely thin hair (Figure 2). Perioral skin demonstrated hypopigmented patches with scaling. Bilateral upper extremities had faintly erythematous macules and patches (Figure 3). Bilateral hands appeared to be re-epithelializing following desquamation. Bilateral legs had 3+ pitting edema to the midthigh, and lower legs had scattered erosions and intact bullae. A 4-mm punch biopsy of a bulla was performed for hematoxylin-eosin and direct immunofluorescence analysis. Given A.B.'s medical and surgical history, dermatology was concerned about nutrition deficiency. Bariatric surgery and dermatology recommended a retinol (vitamin A), thiamine, vitamin B_3, vitamin B_6 (pyridoxine), vitamin B_{12}, biotin (vitamin B_7), 25-hydroxyvitamin D, α-tocopherol (vitamin E), vitamin K, and iron panel.

Dermatology service followed up with A.B. the next day. Her rash appeared stable over the day following admission. The preliminary results of her biopsy were suggestive of nutrition deficiency with superficial keratinocytic pallor and subcorneal separation. Both necrolytic migratory erythema associated with glucagonoma and acrodermatitis enteropathica related to severe zinc deficiency were considered. A.B. was started on zinc supplementation, vitamin C, and multivitamins, whereas laboratory tests were pending.

Clinical nutrition services provided consultation based on dermatology findings during A.B.'s stay in the CCU. A.B. reported that she did not follow up with her bariatric surgeon and did not take the recommended vitamin and mineral supplements for multiple years. In addition, A.B. had inadequate protein consumption. Prior to admission, she had a poor appetite and minimal dietary intake for several months. Her appetite did not markedly improve during this admission. Preferred foods and high-protein, high-calorie oral nutrition supplements (ONSs) were provided but were often seen untouched during mealtime rounding.

On physical examination, A.B. was noted to have generalized edema, but overall appeared to be thin, with temporal wasting. The registered dietitian nutritionist (RDN) diagnosed A.B. with severe protein-calorie malnutrition in the context of chronic illness related to bariatric surgery, as evidenced by 75% or less of estimated energy requirement for 1 or more months and moderate to severe fluid accumulation. Nutrition recommendations included continuation of a regular diet to accommodate patient's food preferences, encouragement of high-protein, high-calorie ONS; vitamin and mineral supplementation (folic acid, vitamin C, zinc, and multivitamins) to correct deficiencies; additional copper supplementation given aggressive zinc repletion; an appetite stimulant; and regular checks for levels of retinol, 25-hydroxyvitamin D, α-tocopherol, vitamin K, biotin, and EFAs to assess for any deficiency.

A.B. had progressively worsening mental status over the next day with no clear etiology and was unresponsive to pain during dressing changes. Head computed tomography scan was performed, which showed no acute intracranial abnormality, so the critical care team consulted neurology and neurocritical care for further assessment. At this time, A.B. was not following any commands and only turned her head slightly to noxious stimulus during neurologic examination. These findings were most consistent with diffuse encephalopathy with a Glasgow Coma Scale score of 8 without clear focality. Despite A.B.'s serum thiamine level within normal limits (173 nmol/L on admission), high-dose IV thiamine was administered for the acute unexplained encephalopathy. At the same time, A.B.'s skin condition was improving, and she showed improvements in pitting edema as well as re-epithelialization of her hands and feet.

A.B. continued to be unresponsive and was found to have an elevated serum ammonia level of 129 umol/L without any clear evidence of hepatic dysfunction. At this time, she was

Table 2.
PROPERTIES OF VITAMINS AND MINERALS

Vitamin/Mineral	Role	Primary Absorption Site	Signs and Symptoms of Deficiency	Dosage for Deficiency	Food Source
Vitamin A	• Formation and maintenance of healthy teeth, bones, muscles, skin and vision[43] • Promotes a healthy immune system[43]	• Duodenum[44] • Jejunum[44]	• Night blindness[45] • Xeropthalmia[45] • Skin drying/scaling • Growth retardation[45] • Infections[45]	• Without corneal changes: 10,000–25,000 IU/d orally[40,42] • With corneal changes: 50,000–100,000 IU IM for 3 d followed by 50,000 IU/d for 2 wk IM[40,42]	• Sweet potato,[46] carrot, spinach, broccoli, cantaloupe, peppers, tomato, beef
Vitamin B_1 (thiamine)	• Enzymatic cofactor[47] • Nerve structure and function[47] • Brain metabolism[47]	• Upper jejunum[48]	• Lactic acidosis[42] • Delayed gastric emptying[42] • Peripheral neuropathy[42] • Ataxia[42] • Ocular changes[42] • Beriberi[42] • Wernicke encephalopathy[42]	• IV 500 mg/d for 3–5 d, then 250 mg/d for 3–;5 d or until symptoms resolved. Then further treatment by oral administration of 100 mg/d as needed[40,42]	• Whole grains[49] • Meat • Fish
Vitamin B_6 (pyridoxine)	• General cellular metabolism[50] • Cofactor[50]	• Proximal jejunum[51]	• Muscle weakness[51] • Paresthesia[51] • Abnormal gait[51]	• 50–100 mg daily[52]	• Fish,[53] beef, potato
Vitamin B_9 (folate)	• Biosynthesis of DNA/RNA[54] • Cell division and repair[54] • Red blood cell formation[54]	• Jejunum[55]	• Neutral tube defects[56] • Macrocytic anemia[54] • Weakness[54] • Fatigue[54] • Shortness of breath[54] • Heart palpitations[54] • Red, swollen tongue[54]	• 1,000 µg daily[40,42]	• Spinach[57] • Liver • Asparagus • Brussels sprouts
Vitamin B_{12}	• Maintenance of normal neurological function[54] • Formation of red blood cells[54] • Metabolism of fatty and amino acids[54]	• Terminal ileum[54]	• Peripheral neuropathy[58] • Megaloblastic anemia[58]	• Sublingual: 1,000–2,000 µg/d[40,42] • IM: 1,000 µg[40,42]	• Meat[59] • Milk • Eggs • Fish
Vitamin C (ascorbic acid)	• Cell growth[60] • Tissue repair[60] • Collagen synthesis[60] • Absorption of nonheme iron[60] • Immune function[60]	• Intestine[60]	• Scurvy[60] • Bleeding gum[60] • Fatigue[60] • Impaired wound healing[60]	• 100 mg three times a day or 500 mg/d for 1 mo[40]	• Bell pepper,[60] broccoli, sweet potato, tomato, strawberry, orange

(continues)

Table 2.

PROPERTIES OF VITAMINS AND MINERALS, CONTINUED

Vitamin/Mineral	Role	Primary Absorption Site	Signs and Symptoms of Deficiency	Dosage for Deficiency	Food Source
Vitamin D	• Bone health and calcium-phosphate metabolism[61]	• Duodenum[62] • Jejunum[62]	• Thin/brittle bones[42] • Osteoporosis[42] • Frequent fractures[42] • Muscle weakness[42] • Fatigue[42] • Mood changes[42]	• 50,000 IU weekly for 8 wk, followed by maintenance therapy of 1,500–2,000 IU/d to achieve normal concentrations[40]	• Egg yolks[61] • Oily fish • Fortified orange juice • Fortified dairy
Vitamin E	• Protects the cells of the body from oxidative damage from free radicals[63] • Healthy immune system[63] • Production of red blood cells[63] • Prevents platelet aggregation[63]	• Duodenum[63] • Jejunum[63]	• Spinocerebellar ataxia[63] • Muscle weakness[63] • Hemolytic anemia[63] • Retinopathy[63] • Impaired immune response[63]	• 800–1,200 IU/d[40]	• Wheat germ oil[64] • Sunflower seeds • Hazelnuts • Peanut butter • Almonds
Iron	• Hemoglobin and myoglobin synthesis[65] • Formation of enzymes involved in electron transfer and oxidation-reductions[65]	• Duodenum[66] • Proximal jejunum[66]	• Anemia[67] • Fatigue[67] • Pale skin[67]	• 150–200 mg elemental iron/d[40,42]	• Red meat[67] • Beans • Legumes
Selenium	• Metabolism in liver, kidney, heart, skeletal muscle, lens of eyes, and in erythrocytes[68] • Free radicals damage protection[68]	• Duodenum[68]	• Cardiomyopathy[69] • Myopathy[69] • Osteoarthropathy[69] • Keshan disease[69]	• 100 µg/d[70]	• Seafoods[71] • Organ Meats
Copper	• Neurotransmitter synthesis[47,72] • Superoxide synthesis[47,72] • Respiratory oxidation[47,72] • Iron absorption[47,72]	• Duodenum[47,62] • Proximal jejunum[47,72]	• Neuropathy[47] • Anemia[47] • Neutropenia[47] • Optic neuropathy[47] • Fatigue[47] • Myelopathy[47]	• Oral: 3–8 mg daily[42] • Parenteral: 2–4 mg daily for 6 d[40,42]	• Whole grains[73] • Beans • Nuts • Potato • Shellfish
Zinc	• Cellular metabolism[47] • Catalyst[47] • DNA synthesis[47] • Protein synthesis[47] • Immune function[47] • Wound healing[47]	• Duodenum[47] • Proximal jejunum[47]	• Hair loss[47] • Diarrhea[47] • Glossitis[47] • Hypogonadism[74] • Taste alteration[74] • Delayed wound healing[47]	• 60 mg elemental zinc twice daily[40,42]	• Red meat[74] • Oysters • Poultry • Baked beans • Chickpeas • Nuts

thought to have a previously undiagnosed urea cycle disorder and hyperammonemia-induced encephalopathy related to gastric bypass. Rifaximin and lactulose were started to remove ammonia-producing bacteria from her gastrointestinal tract to lower her serum ammonia and to optimize her urea cycle, but with poor response. Her serum ammonia was lowered to 95 μmol/L, but plateaued despite continued treatment. Therefore, renal replacement therapy was initiated to remove ammonia from her blood.

Five days following admission, A.B. was found to have normal levels of thiamine, vitamin B_{12}, vitamin C, and iron. A.B. had low levels of total 25-hyroxyvitamin D and serum vitamin B_6, α-tocopherol, and copper. She was started on IV biotin, niacin, and vitamin B_6. Given the possibility of a urea cycle disorder, the RDN also recommended a very low-protein, nonessential amino acid-free, metabolic disease enteral nutrition formula via a nasogastric feeding tube. Despite IV vitamin repletion, nutrition provision, and decreasing ammonia levels, A.B. showed no improvement in mental status.

Computed tomography and MRI scans of A.B.'s chest, abdomen, and pelvis were ordered to identify any possible occult malignancies that may be causing the acute changes. She was found to have an ill-defined hypodense lesion in the head of her pancreas (likely adenocarcinoma or glucagonoma) despite a normal plasma glucagon level (141 pg/mL). Gastroenterology and endocrinology were consulted for further evaluation of her pancreatic mass.

A.B.'s mental status eventually started to improve, but she remained lethargic at times. She was stable enough to be transferred out of the CCU. The medical geneticist stated that A.B.'s hyperammonemia and urine amino acid studies were more consistent with a severe catabolic state. Her whole blood ornithine transcarbamylase sequencing was underway but eventually returned a negative result. She was resumed on a regular diet with high-protein, high-calorie ONS but continued to have poor appetite and dietary intake, often only drinking some juice as a meal. Given the unknown absorptive capacity of A.B., the RDN also recommended IV multivitamins, in addition to her dietary supplementation.

Her skin continued to make progress; the ulcerations of her right posterior thigh were healing and appeared healthy, the web space erosions of her bilateral hands were healing, and her bilateral arms re-epithelialized.

A.B.'s mental status, appetite, and dietary intake continued to wax and wane. She participated in a goals-of-care discussion and decided to continue treatment for curable conditions such as nutrient supplementation and antimicrobial therapy for infection, but not incurable conditions, such as a biopsy of her pancreatic mass. At the time, her retinol (0.14 mg/L), vitamin B_6 (12.9 nmol/L), 25-hydroxyvitamin D (8 ng/mL), and α-tocopherol (4.2 mg/L) levels were still low, although the zinc level (78 μg/dL) was corrected. Her goals were to go home and spend as much time with her children as possible.

A.B.'s mental status continued to improve, and she could participate in physical and occupational therapy sessions. The medical team planned to discharge her to an acute inpatient rehabilitation facility when medically optimized. However, A.B. then reported to have multiple loose, foul-smelling stools. A fecal fat test was ordered and showed steatorrhea. Clinical nutrition services recommended substituting conventional multivitamins with water-miscible fat-soluble vitamins to enhance absorption, pancreatic enzyme supplementation, and checking A.B.'s fatty acids profile to assess EFA deficiency.

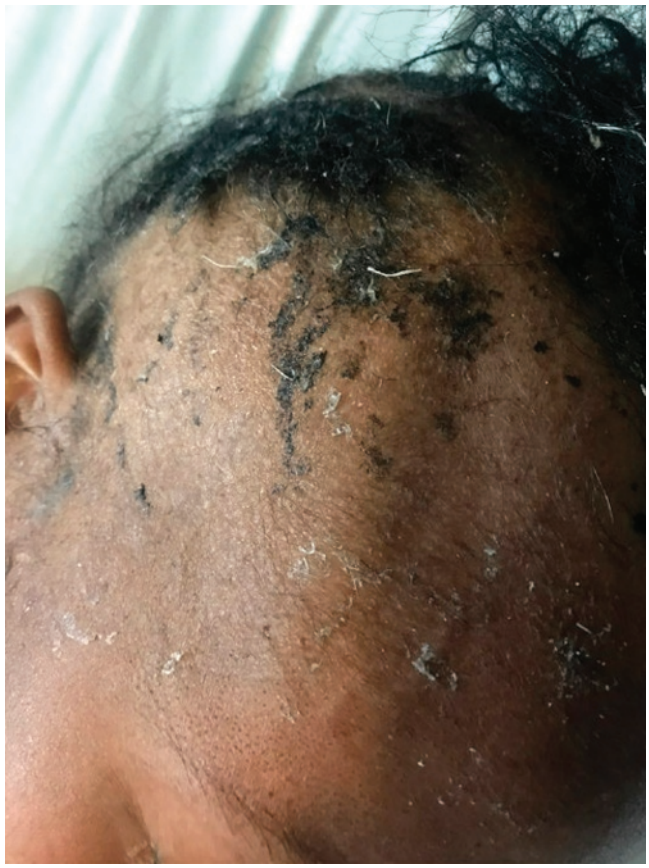
Despite previous improvement, A.B.'s skin rash appeared to worsen even with continued nutrition supplementation. Her left axilla macerated and eroded with foul odor and yellow discharge, but her hands were completely re-epithelialized. The discovery of her pancreatic mass, gastrointestinal bleed, weight loss, and encephalopathy raised concern for glucagonoma. Although A.B.'s serum glucagon level was normal, and her pancreatic mass was at the head of the pancreas, it did not rule out glucagonoma. A trial of low-dose octreotide therapy was started and titrating up as tolerated per endocrinology. Alternative diagnoses for her worsening rash included overdiuresis with zinc wasting and newly acquired malabsorption worsening her chronic malabsorption from the Roux-en-Y gastric bypass.

Although A.B.'s triene-to-tetraene ratio was within normal limits, her serum linoleic acid was low, and her α-linoleic acid was borderline low. Although A.B. took pancreatic enzymes, she was encouraged by the RDN to include foods that were rich in EFAs and α-linoleic acid to help correct any deficiencies. She was instructed to consume each meal with extra margarine and mayonnaise, which are made with soybean oil naturally rich in linoleic acid. A.B. continued on the trial of octreotide therapy, and her skin appeared to improve again.

Because of this chronic fat malabsorption and vitamin and mineral deficiencies, the RDN suggested total parenteral nutrition (TPN) or peripheral parenteral nutrition for aggressive nutrition repletion. A.B. was provided TPN that contained dextrose, amino acids, EFAs, vitamins, and minerals. However, A.B. reported an unexplained burning sensation of her vagina when TPN was infused, but had no soybean allergy or other known contraindication to the formulation. Parenteral nutrition support was eventually discontinued. Based on her improvement with octreotide, it was suspected that her skin condition was mainly necrolytic migratory erythema in the setting of pseudoglucagonoma. However, there was likely some component of nutrient deficiency, and A.B. remained on zinc and other nutrient supplementation.

Before A.B. was discharged, her retinol (0.15 mg/L) and 25-hydroxyvitamin D (11 ng/mL) levels remained low, but her retinol level significantly improved from baseline, and her other previously deficient vitamins including vitamin B_6 (55.7 nmol/L), α-tocopherol (8.9 mg/L), copper (98 μg/dL), and zinc (86 μg/dL)

Figure 2.
THINNING HAIR RELATED TO ZINC DEFICIENCY



were corrected to within normal limits. Her skin experienced significant improvement with the start of octreotide such that her bilateral thighs with indurated healing plaques and her plantar were healed. A.B. eventually went back home to spend time with her family. She was referred to the weight management center to follow up with her vitamin and mineral deficiency. A.B. continued to decline biopsy of her pancreatic head mass, which continued to be an obstacle to discovering the exact cause of her unexplained skin condition (whether glucagonoma or severe zinc deficiency).

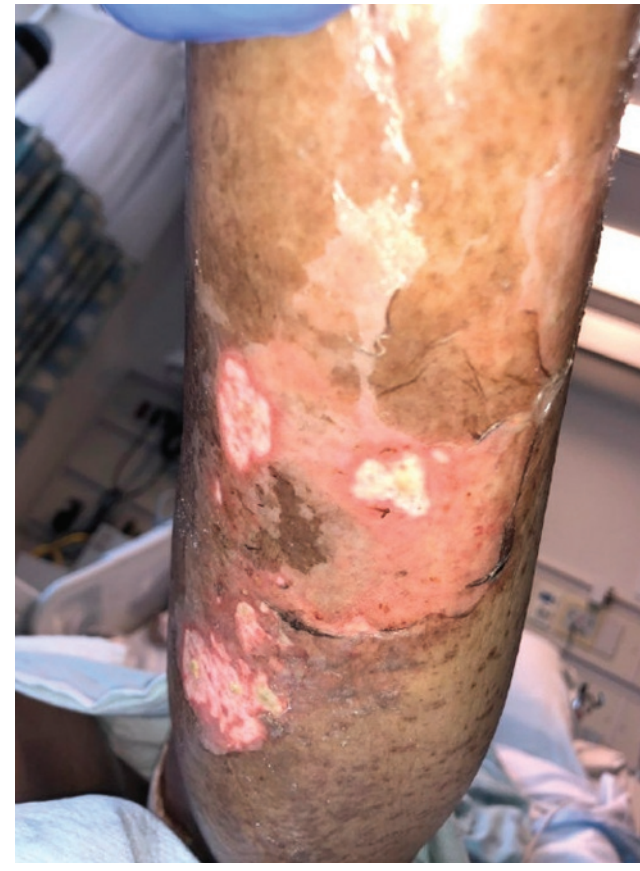
DISCUSSION

There were a couple of differential diagnoses in this case report, primarily zinc deficiency and glucagonoma. Zinc deficiency, or acrodermatitis enteropathica, results from either acquired or genetic causes. It is clinically characterized by cutaneous erythema, erosions, and blisters as well as alopecia and diarrhea. Zinc deficiency is thought to be less common in developed countries (just 1%–3% of the population in the US), although current epidemiologic data are lacking.[75] In some countries, however, manifestations of zinc deficiency are seen in up to 40% of preschool children.[76] There does not appear to be a gender or racial predilection.[77]

The genetic form of acrodermatitis enteropathica is an autosomal recessive disorder of gene *SLC39A4* on chromosome 8q24 that leads to a deficiency of zinc absorption via Zip4 transporter protein in the duodenum and jejunum.[77] Manifestations can present within 1 to 2 weeks after cessation of breastfeeding, or by age 4 to 10 weeks in bottle-fed infants.

Acquired forms of zinc deficiency are more common, however, and result from lack of intake.[78] Because meats and poultry are the main sources of zinc in the diet, vegetarian/vegan diets can provide insufficient daily zinc, as can poor nutrition seen in alcoholism or anorexia nervosa.[78] Diets high in mineral-binding phytates, commonly seen in cereals, can cause binding and a decrease in the uptake of zinc.[79] Conditions such as celiac disease,

Figure 3.
HYPOPIGMENTED PATCHES OF SKIN WITH SCALING RELATED TO ZINC DEFICIENCY



inflammatory bowel disease, cystic fibrosis, HIV, and renal disease can all cause decreased zinc levels.[78,79] In addition, insufficient zinc supplementation with TPN has been reported.[80]

Some studies suggest that morbid obesity may predispose patients to zinc deficiency, although this has been called into question.[81] Bariatric surgery, however, is known to increase the risk of acquired zinc deficiency, likely because of alterations in anatomy of the proximal intestine.[82] One study suggests that it can be seen in more than 35% of patients postoperation. A DPS/DS has been found to cause more dramatic decreases in zinc levels than Roux-en-Y gastric bypass or SG.[81]

Zinc deficiency is clinically characterized by a cutaneous eruption, alopecia, and diarrhea. The cutaneous eruption presents with symmetric erythema, erosions, pustules, and/or bullae in a periorificial and acral distribution. The periorificial eruption classically spares the upper lip.[79] Other cutaneous features include hypopigmentation, paronychia (infection at nail cuticle), brittle hair, and poor wound healing. Stomatitis, conjunctivitis, blepharitis, dysgeusia, hypogonadism, irritability, and growth impairment can also be seen.[79,83] The diagnosis of zinc deficiency can be established with laboratory evaluation, preferably with blood collected in metal-free tubes.[84] Plasma zinc levels should reveal deficiency, but these levels can vary secondary to inflammation and diurnal variation.[79] Low levels of zinc-dependent alkaline phosphatase can also aid in establishing the diagnosis.[85] Histopathology of cutaneous lesions can suggest a nutrition deficiency with varying degrees of spongiosis, epidermal hyperplasia, epidermal pallor, and dyskeratosis.[79]

Acrodermatitis enteropathica characteristically responds well to zinc repletion. The genetic variation requires lifelong supplementation.[79] Both parenteral and enteral supplementations are available, and in adults, 60 mg/d of elemental zinc twice daily has been recommended.[40,42] Zinc is better absorbed while fasting rather than with a meal.[86] There are no clear recommendations for he dosing of supplementation following bariatric surgery, although consensus suggests it is necessary.[78]

A.B.'s other differential diagnosis, glucagonoma, is a slow-growing, rare tumor of the islet cells of the distal pancreas that secrete glucagon.[87–89] Glucagonoma is usually malignant, and about 50% to 80% are metastatic at the time of diagnosis.[88] The cause of glucagonoma is unknown, but genetic factors may play a role in some cases. Up to 10% of glucagonoma develops in individuals with the multiple endocrine neoplasia type 1 syndrome.[88]

Glucagonoma syndrome is thought to be related to an excess level of glucagon in the blood. Glucagon elevates blood glucose, promoting glycogenolysis by the breakdown of glycogen from hepatic storage and gluconeogenesis by producing glucose from amino acid substrates.[87,88] Signs and symptoms of glucagonoma syndrome include hyperglycemia, anemia, involuntary weight loss, chronic diarrhea, venous thrombosis, neuropsychiatric disorders, cheilitis, and necrolytic migratory erythema; of these, involuntary weight loss and necrolytic migratory erythema are the most common at the time of diagnosis. However, these signs and symptoms are not specific and have been associated with other disorders, including zinc deficiency, pellagra, and kwashiorkor.

Glucagonoma can be diagnosed based on an increased fasting plasma glucagon level (>500 pg/mL).[88] Diagnostic imaging, such as multiphasic helical contrast-enhanced computed tomography or MRI, is required to locate the tumor and examine its extent. Endoscopic ultrasound or functional imaging with somatostatin-receptor scintigraphy should be considered if cross-sectional imaging is inconclusive.[88]

Management of glucagonoma syndrome includes glycemic management, supportive care, and octreotide to decrease hormone secretion and manage signs and symptoms of hyperglucagonemia.[88,89] Nutrition support may be warranted in malnourished patients to treat catabolism related to hyperglucagonemia. Resection of the primary pancreatic tumor is indicated if the tumor is not metastasized at the time of diagnosis.[88]

In patients with a metastasized tumor, liver resection can be an option if hepatic function is normal, there is no presence of diffused metastatic bilobar hepatic involvement, and the tumor is localized.[88] For patients with unresectable liver metastases, hepatic artery embolization may be performed as a palliative measure to relieve symptoms. Efficacy of radiofrequency ablation and cryotherapy is uncertain in the long term, and liver transplantation is only investigational for metastatic pancreatic neuroendocrine tumors.[88] Octreotide may have cytostatic activity in well-differentiated neuroendocrine tumors. Further, cytotoxic chemotherapy, molecularly targeted agents, and peptide receptor radioligand therapy are under evaluation as options for patients with unresectable disease.[88]

CONCLUSIONS

Obesity is an increasingly common epidemic around the globe. It is a serious medical condition with serious complications. Obesity is associated with changes in skin physiology and aggravation of dermatologic disorders. For people who do not achieve success with noninvasive treatment of obesity, bariatric surgery may be an option. Similar to other major procedures, bariatric surgery carries significant risk. It requires lifelong commitment to dietary and lifestyle changes and medical care and is not for everyone.

Additional skin disorders may present after bariatric surgery. Nutrient deficiencies may develop if long-term postoperative monitoring is not followed. Vitamin and mineral deficiencies play an important role in dermatologic complications after bariatric surgery. Nutrient deficiency-related dermatoses after bariatric surgery can generally be corrected with nutrient repletion.

PRACTICE PEARLS

- Obesity is a global epidemic and is a leading cause of heart disease, stroke, type 2 diabetes, some types of cancers, and premature death.
- Obesity and bariatric surgeries are both associated with dermatologic disorders.
- Nutrient deficiencies after bariatric surgery are common because of the nature of food restriction and/or malabsorption.
- It is important to begin lifelong vitamin and mineral supplementation after bariatric surgery to prevent deficiency.
- Severe nutrient deficiencies may lead to dermatoses and other skin disorders.

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- Read the article beginning on page 443. For nurses who wish to take the test for CNE contact hours, visit http://nursing.ceconnection.com. For physicians who wish to take the test for CME credit, visit http://cme.lww.com. Under the Journal option, select *Advances in Skin and Wound Care* and click on the title of the CE activity.
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- There is only one correct answer for each question. A passing score for this test is 13 correct answers. If you pass, you can print your certificate of earned contact hours or credit and access the answer key. Nurses who fail have the option of taking the test again at no additional cost. Only the first entry sent by physicians will be accepted for credit.

Registration Deadline: September 30, 2021 (physicians); September 3, 2021 (nurses).

PAYMENT

- The registration fee for this CE activity is $17.95 for nurses; $22.00 for physicians.