ELSEVIER

Surgery for Obesity and Related Diseases ▮ (2017) 00–00

SURGERY FOR OBESITY AND RELATED DISEASES

Review article

Considerations regarding sarcoidosis in the bariatric surgical patient

Kimberly A. Kilby, M.P.H., M.D.[a,*], Colleen M. Kilbourne, B.S., M.S.[a], Marc A. Judson, M.D.[b], Daniel J. Bonville, D.O.[c], Scott H. Beegle, M.S., M.D.[b]

[a]*Department of Surgery, Department of Family Medicine, Albany Medical College, Albany, New York*
[b]*Department of Medicine, Division of Pulmonary & Critical Care Medicine, Albany Medical College, Albany, New York*
[c]*Division of Acute Care Surgery and Surgical Critical Care, Department of Surgery, The Houston Methodist Hospital, Houston, Texas*

Received August 7, 2017; accepted October 8, 2017

Abstract The sarcoidosis patient who seeks surgical management for obesity presents many challenges. The interaction between sarcoidosis and obesity complicates both disorders and creates special issues to consider when contemplating surgery. This manuscript will review the approach to pre- and post-operative management of the sarcoidosis patient undergoing bariatric surgery. (Surg Obes Relat Dis 2017;▮:00–00.) © 2017 American Society for Metabolic and Bariatric Surgery. All rights reserved.

Keywords: Sarcoidosis; Bariatric surgery; Obesity; Vitamin D dysregulation; Glucocorticoid use

Obesity is a national epidemic affecting over one third of U.S. adults, with a total annual cost of $147 billion. Besides these staggering costs, obesity has a major impact on morbidity and mortality [1]. This epidemic has resulted in a 24% increase in bariatric surgical procedures between 2011 and 2015 [2]. Management of the underlying medical problems in the bariatric surgery patient is paramount to minimize surgical complications and optimize outcomes.

Brief review of sarcoidosis

Sarcoidosis is defined as a noncaseating granulomatous disease that may manifest in any organ or tissue—most commonly the skin, lung, lymph nodes, liver, and eyes. The severity of the disease is variable, ranging from an asymptomatic state with no clinical consequences to a life-threatening disease. Although sarcoidosis is a ubiquitous disease that is found worldwide, it is more common in black females, but also occurs with a significant incidence in Caucasians and geographic regions far away from the equator (e.g., Northern Europeans). The disease is rare to present in persons <18 years [3].

The etiology of sarcoidosis remains elusive despite extensive investigations concerning the disease pathogenesis. Specifically, sarcoidosis is thought to result from immune mediated dysregulation of the Th1 CD4 antigen response directed by abnormal dendritic cell function, resulting in an inflammatory activation leading to granulomatous formation with epithelioid histiocytes, giant cells, and macrophages without substantial lymphocytic infiltrate [4]. Given the disease's preference for skin, lung, and eyes, it has been suspected that the causative antigens are airborne. Many infectious, occupational, and environmental exposures have been associated with sarcoidosis including mycobacteria, propionobacter acni (the common acne bacterium), firefighting, and the World Trade Center dust exposure [5,6]. There is also well-documented evidence suggesting a genetic predisposition to the disease through association studies with Human leukocyte antigen polymorphisms and sarcoidosis [6,7].

The diagnosis of sarcoidosis requires an appropriate clinical picture, histopathologic (noncaseating granulomas) investigation, and the definite exclusion of other granulomatous diseases. The lung is the most common organ

*Correspondence: Kimberly A. Kilby, M.P.H., M.D., Dept of Surgery, Dept of Family Medicine, Albany Medical College, 47 New Scotland Avenue, MC-1, Albany, NY 12208.
E-mail: kilbyk@amc.edu

http://dx.doi.org/10.1016/j.soard.2017.10.007

involved with sarcoidosis and affects up to 95% of patients. Pulmonary sarcoidosis patients will often present with cough, dyspnea, chest pain, and constitutional symptoms such as fatigue, malaise, fever, and weight loss [8]. Abnormalities on chest imaging most often include mediastinal lymphadenopathy and may reveal micronodules in a perilymphatic distribution [9]. Additionally, fibrotic change in the lung may be seen. The physiologic evaluation also includes spirometry with lung volumes and diffusing capacity. Although sarcoidosis is thought to be an interstitial lung disease, and therefore would demonstrate restriction on spirometry, it may also affect the airways and cause an obstructive or mixed pattern on spirometry [8].

Due to the systemic nature of the disease, extrapulmonary organs may be most affected by sarcoidosis and can be biopsied to secure the diagnosis. Skin manifestations are the next most common site of involvement, with 25% of sarcoidosis patients presenting with specific (granulomatous) lesions such as papular sarcoidosis, largely found within the nasolabial folds and eyelids, nodular sarcoidosis, maculopapular sarcoidosis, lupus pernio, and sarcoid granulomas in tattoos [10]. "Nonspecific skin lesions" may also occur with sarcoidosis where sarcoidosis causes a skin reaction that is not granulomatous [10]. The prototypic nonspecific sarcoidosis skin lesion is erythema nodosum—an indurated, painful, erythematous to violaceous skin lesion on extensor surfaces, such as the shins. The third most common site of sarcoid involvement is the eye, with 25% to 80% of patients developing anterior or posterior uveitis. Although a sarcoid uveitis may cause no symptoms, it is potentially vision threatening, emphasizing the importance of thorough ophthalmologic examination in all sarcoidosis patients irrespective of the presence of eye symptoms [11].

Hepatic involvement is usually asymptomatic, but granulomas can be found on biopsy in approximately 60% of patients with less than one third exhibiting abnormal liver function tests [12]. Fig. 1 shows hepatic sarcoid granulomas seen intraoperatively. Granulomatous hepatitis or cirrhosis, resulting in portal hypertension, can occur in 18% of patients. Gastrointestinal sarcoid may involve any part of the alimentary tract and as such, may present clinically with a wide and quite variable symptomatology. Esophageal involvement may result in dysmotility, ulceration, or esophagitits. While often asymptomatic, .1% to .9% of patients with gastric sarcoid develop symptoms, from upper gastrointestinal bleeding secondary to ulcerations in the antrum, pylorus, and lesser curvature, to gastric outlet obstruction secondary to infiltrative pathology. Involvement of the small bowel may result in nutritional deficiencies (B12 and folate), protein losing enteropathy, and significant unintentional weight loss. Less commonly reported, colonic involvement can result in obstruction, strictures, bleeding, and may mimic inflammatory bowel disease [13].

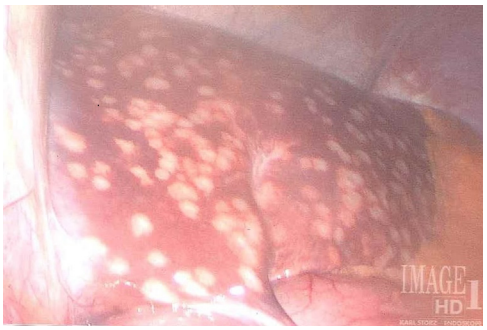

Fig. 1. Intraoperative photograph, sarcoid granulomas in liver.

Although cardiac sarcoidosis causes clinically significant disease in approximately 5% of sarcoidosis patients, it may cause life-threatening complications, such as cardiomyopathy or fatal arrhythmias. Therefore, all sarcoidosis patients should be screened for cardiac sarcoidosis, which should include at a minimum a medical history eliciting symptoms suggestive of an arrhythmia or heart failure as well as an electrocardiogram [8]. Vitamin D dysregulation due to upregulation of the 1 α-hydroxylase enzyme in activated sarcoidal macrophages and the subsequent overproduction of 1,25-dihydroxy vitamin D, the active metabolite, is fairly common in sarcoidosis and may cause hypercalciuria, hypercalcemia, nephrocalcinosis, nephrolithiasis, and renal insufficiency [11]. Sarcoidosis can also cause constitutional symptoms that are not attributable to granulomatous involvement of a specific organ but probably reflect a systemic response from release of inflammatory mediators. Such constitutional symptoms include fatigue, small fiber neuropathy, and pain syndromes [8,14].

Certain clinical presentations of sarcoidosis are so specific that a biopsy is not required for diagnosis [15]; these include Lofgren's syndrome and Heerfordt's syndrome. Lofgren's syndrome is characterized by acute symmetric polyarthralgia, erythema nodosum, bilateral hilar lymphadenopathy, and fever, and these patients tend to have a good prognosis [16]. Heerfordt's syndrome, also known as uveoparotid fever, is relatively uncommon and presents with fever, uveitis, and parotid swelling [17]. Unless one of these highly specific sarcoidosis presentations is present, the diagnosis of sarcoidosis requires that histopathology be obtained from an organ involved with sarcoidosis. Biopsies typically reveal compact, well-formed noncaseating granulomas containing macrophages, giant cells, and epithelioid cells encircled by lymphocytes [11]. Other causes of granulomatous inflammation, such as mycobacterial and fungal infections need to be excluded. Therefore, sarcoidosis is considered a diagnosis of exclusion, as other granulomatous disorders may also present with similar symptoms and clinical findings [4,18,19].

Once a diagnosis of sarcoidosis has been confirmed, an in-depth investigation of the extent of multisystem involvement should be performed. This should include assessment

for pulmonary, dermatologic, ophthalmologic, gastrointestinal, cardiac, and neurologic manifestations of disease, as well as vitamin D dysregulation. Given the varying clinical presentation of sarcoidosis and the varying prognosis of the disease from spontaneous remission to multiorgan dysfunction, each therapeutic plan should be personalized. Traditionally, sarcoidosis is managed in a case-by-case fashion guided by symptomatology—asymptomatic sarcoidosis is usually not treated, while the presence of active granulomatous inflammation causing significant reduction in organ function usually requires therapy [20].

Glucocorticoids (oral prednisone initially dosed at .3–0.6 mg/kg ideal weight) are considered first line therapy for sarcoidosis and have been shown to reduce symptoms by attenuating the granulomatous inflammation [21]. High-dose glucocorticoid therapy is recommended for progressive organ failure or life threatening disease. The side effects of long-term corticosteroids are well documented and have the potential to cause significant morbidity [22]. Steroid-sparing immunosuppressive agents, such as infliximab, methotrexate, or azathioprine, are used in patients with an insufficient response to glucocorticoids or with intolerable glucocorticoid side effects [20]. Antimalarial agents such as chloroquine and hydroxychloroquine are often used to treat hypercalcemia or skin involvement [11].

Considerations in the sarcoid patient undergoing bariatric surgery

Preoperative considerations

The preoperative medical evaluation of a bariatric patient with sarcoidosis should include an assessment of the extent of organ system involvement and current treatments (Table 1), and the multisystem nature of sarcoidosis should be taken into account regarding the decision to progress with surgery. This should include confirming that pulmonary and ophthalmologic evaluations are up to date. Consideration should be given to workup of vitamin D dysregulation as well as neurologic, dermatologic, and other organ involvement. A cardiac workup is warranted and includes a thorough history eliciting a past medical history of cardiac sarcoidosis and symptoms of heart failure (ankle edema, orthopnea, paroxysmal nocturnal dyspnea), cardiac rhythm disturbances, or heart block (syncope, presyncope, palpitations), and Electrocardiogram analysis. Gastrointestinal sarcoid is a rare manifestation of the disease and often asymptomatic when present, but may contribute to surgical complications. In a patient with diagnosed sarcoidosis, with clinical suspicion for concomitant gastrointestinal illness, consideration should be given to performing preoperative endoscopy.

An understanding of current and past treatments the patient has received to manage their sarcoid disease is essential, and this information should be factored in when considering whether these patients are appropriate bariatric surgical candidates and in discussion of the appropriate surgical procedure. A recent study analyzing data from the American College of Surgeons National Surgical Quality Improvement Program database indicates that chronic steroid use was an independent predictor of increased 30-day mortality risk and serious postoperative complications, regardless of type of surgery [23]. Given the well-documented increased risk of marginal ulcers with Roux-en-Y gastric bypass, it is reasonable that sleeve gastrectomy may be the most appropriate surgical procedure to offer in a patient with sarcoidosis who may require steroid treatment for sarcoidosis in the future [24,25]. An exception may be the sarcoid patient with concomitant severe gastroesophageal reflux, which can be worsened after sleeve gastrectomy [26]. As such, patients with concomitant obesity and sarcoidosis requiring chronic steroid therapy may not be appropriate candidates for bariatric surgery and other avenues to support weight loss should be pursued aggressively.

Absolute contraindications to bariatric surgery should include those patients with active cardiac sarcoid with heart failure and/or arrhythmia, severe pulmonary sarcoid with limited pulmonary reserve, neurosarcoid with neurologic impairment, symptomatic gastrointestinal sarcoid, and any

Table 1
Summary of recommendations regarding bariatric surgery in patients with sarcoidosis, based on expert opinion

Preoperative	Pulmonary consultation to assess extent of sarcoid disease organ involvement and potential for current or future medical treatment, including: • Pulmonary evaluation for lung function, including spirometry and diffusing capacity of the lungs for carbon monoxide (DLCO) • Ophthalmologic exam for uveitis • Evaluation for cardiac involvement, including Electrocardiogram and symptom assessment for syncope, presyncope, or palpitation • Baseline laboratory evaluation including Complete Metabolic Profile, 25-hydroxy vitamin D, 1,25-dihydroxy vitamin D, parathyroid hormone (PTH), and 24-hour urine calcium
	Detailed preoperative counseling on the risks and benefits of each bariatric surgical procedure in light of current or future corticosteroid use titrated to lowest effective dose for disease control.
Postoperative	Encourage oral hydration in excess of 1.5 L/d. Low threshold for intravenous hydration in patients at high risk and experiencing difficulty with fluid intake.
	Dietary guidance to reduce intake of oxalate-rich and fatty foods
	No automatic recommendation for supplementation with calcium and Vitamin D. Rather, perform periodic laboratory evaluation of 25-hydroxy vitamin D, 1,25-dihydroxy vitamin D, PTH, and 24-hour urine calcium. These values should be used to guide calcium and vitamin D supplementation recommendations.
	Continued annual to biannual sarcoidosis follow-up with Pulmonologist

Table 2
Absolute contraindications for bariatric surgery in sarcoidosis

Active cardiac sarcoid, with heart failure and/or arrhythmia
Severe pulmonary sarcoid with limited pulmonary reserve
Symptomatic gastrointestinal sarcoid
Neurosarcoid with neurologic impairment
Any system involvement requiring high-dose immunosuppressive therapy, which would impair wound healing

system involvement requiring high-dose immunosuppressive therapy, which would impair wound healing (Table 2).

Vitamin D dysregulation

A standard recommendation after bariatric surgery includes supplementation with calcium citrate and vitamin D3 and routine monitoring of 25-hydroxy vitamin D levels [27]. However, this approach is inadequate in sarcoidosis patients because dysregulation of vitamin D metabolism is a relatively common problem with up to 20% experiencing hypercalcemia and nearly 50% experiencing hypercalciuria [28]. Sarcoidosis patients tend to have low 25-hydroxy vitamin D levels and normal or elevated levels of 1,25-dihydroxy vitamin D. For this reason, monitoring only 25-hydroxy vitamin D levels in sarcoidosis is inadequate, as these levels may be low when the level of 1,25-dihydroxy vitamin D, the active form of the vitamin, may actually be high. The serum parathyroid hormone is usually extremely low in this situation as it is suppressed by the elevated 1,25-dihydroxy vitamin D (see Fig. 2). Therefore, it is the opinion of these authors that routine vitamin D and calcium supplementation not be recommended postoperatively, but rather, 25-hydroxy vitamin D, 1,25-dihydroxy vitamin D, and serum parathyroid hormone levels should be monitored and used to inform supplementation recommendations.

Nephrolithiasis

Nephrolithiasis is a well-established risk after bariatric surgery; with risk of renal calculi formation increased 2- to 3-fold in the first year postprocedure secondary to hyperoxaluria as well as hypocitrituria and decreased urine volume increasing the risk of calcium oxalate stone formation [29]. Sarcoidosis similarly increases the risk of symptomatic nephrolithiasis secondary to vitamin D dysregulation-induced hypercalciuria and hypercalcemia [28,30]. The combined risk of nephrolithiasis from both bariatric surgical procedures and sarcoidosis highlights the importance of adequate hydration to increase total urine volume and decrease risk of supersaturation. In this regard, we recommend a low threshold for intravenous fluids in a patient having difficulty maintaining the recommended oral intake of >1.5 L/d who are at risk of dehydration [27]. In addition, patients can be counseled to reduce intake of oxalate-rich and fatty foods to minimize enteric absorption [29]. Although calcium supplementation with calcium citrate (rather than other forms) is recommended in those undergoing malabsorptive procedures who experience nephrolithiasis, it is generally accepted that calcium supplementation be avoided in sarcoidosis patients to avoid worsening of hypercalciuria, hypercalcemia, and calcium dysregulation [28]. We recommend obtaining a 24-hour urine relatively early in the postoperative period for determination of urinary volume, creatinine, calcium, and citrate to direct supplementation recommendations or guide

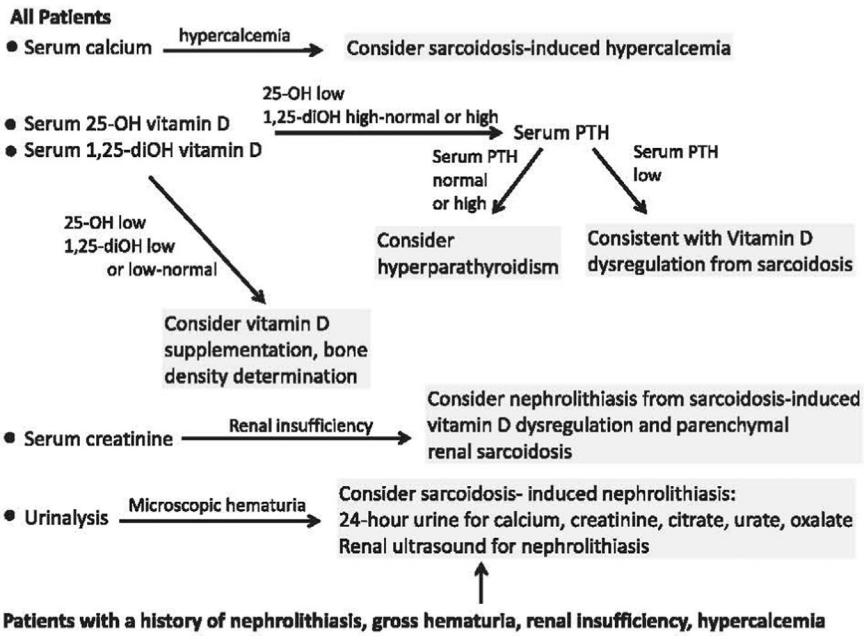

Fig. 2. Screening for sarcoidosis-induced vitamin D dysregulation and its consequences. OH = hydroxyl; PTH = parathyroid hormone (Reproduced with permission from Judson, MA. Judeson MA. The 3 tiers of screening for sarcoidosis organ involvement. Respir Med 2016;113:42–9).

prophylactic therapies to minimize nephrocalcinosis and nephrolithiasis.

Medical management of sarcoid disease

In the years after bariatric surgery, it is possible that a patient previously asymptomatic with sarcoid disease could experience a natural progression of their disease resulting in the need for treatment. As aforementioned, the mainstay treatment of sarcoidosis is corticosteroid therapy, but this recommendation conflicts with the general postbariatric surgical recommendations to avoid gastric irritants, such as oral steroids and nonsteroidal anti-inflammatory drugs [11,23,27]. Immediately postbariatric surgery, patients on chronic steroids have an increased mortality and an increased risk of postoperative complications, owing to the increased risk of infection from immunosuppression [23]. From a long-term perspective, chronic steroid use may increase risk of gastric ulcer formation and diminish the effectiveness of bariatric surgery in morbidly obese patients due to a risk of Cushingoid habitus and weight gain, compounded by increased appetite, insulin resistance, avascular necrosis, and osteopenia/osteoporosis leading to vertebral fractures and inactivity [31,32]. Second line agents can also present with similar side effects that negatively affect the postbariatric surgical patient [20]. Specifically, gastrointestinal toxicity is a common complication of immunomodulators and as such, may contribute to the malabsorptive disorders, which may present in most postbariatric surgery patients.

Due to the varying range of severity present in sarcoidosis patients and the unique surgical and postsurgical risks in bariatric surgery, a case-by-case assessment of the risks and benefits of various treatments is best for each postbariatric surgery patient with sarcoidosis. If steroids are deemed absolutely necessary, the lowest effective dose and shortest length of treatment should be used to minimize side effects and achieve optimal management of organ dysfunction. A 3-month course of prednisone dosed at 20 to 40 mg per day followed by a brisk taper to 5 to 15 mg per day, combined with a proton-pump inhibitor, is a reasonable therapeutic approach, with second line immunosuppressant therapy on hand either for insufficient disease control or excessive side effects.

Conclusions

The incidence of patient populations with co-morbidities that require conflicting treatments is not uncommon in today's medical realm. The obese patient with sarcoidosis poses such a challenge. Surgical treatment of obesity in this population should be considered individually and heavily depends on the sarcoid disease severity and active treatment. It is important to note that if the decision is made to pursue surgical intervention for weight loss in a patient with sarcoidosis, there is no evidence base on how to manage both of these conditions simultaneously and efficaciously. This highlights the importance of the multidisciplinary team approach involving pulmonology, nutrition, and surgery to weigh the risks and benefits of surgical intervention for weight loss and reduction of obesity-related co-morbidities.

Diagnosis of sarcoidosis can be difficult given the varying clinical presentations and the spectrum of severity from asymptomatic subclinical disease to overt disease impacting multiple organ systems. The prevalence of obesity and associated bariatric surgery treatments is undoubtedly rising in epidemic proportions, with more than one third of U.S. adults currently classified as obese, leading to a 6-fold increase in bariatric surgery over the last decade [29]. Bariatric surgery patients with concomitant sarcoidosis need to be uniquely and more carefully managed to ensure positive postoperative results both in the short and long term. As well, in the setting of a multisystem disease such as sarcoidosis, addressing and optimizing the patient's chronic disease should remain paramount to any elective procedure. We are hopeful that this review may contribute to optimal management based on the best available expert opinions regarding both conditions.

Disclosures

Dr. Marc A. Judson reports potential conflict of interest as a consultant for Biogen and has received grant funding for the institution (not personally) from Novartis and Mallinckrodt. The remaining authors have no conflicts of interest to report.

References

[1] Finkelstein EA, Trogdon JG, Cohen JW, Dietz W. Annual medical spending attributable to obesity: payer-and service-specific estimates. Health Aff (Millwood) 2009;28(5):w822–31.

[2] Estimate of Bariatric Surgery Numbers, 2011-2015 [homepage on the Internet]. Gainsville: American Society for Metabolic and Bariatric Surgery; c2017 [updated 2016 Jul; cited year mo day]. Available from https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers. Accessed November 8, 2017.

[3] Baughman RP, Field S, Costabel U, et al. Sarcoidosis in America. Analysis based on health care use. Ann Am Thorac Soc 2016;13(8):1244–52.

[4] Zaba LC, Smith GP, Sanchez M, Prystowsky SD. Dendritic cells in the pathogenesis of sarcoidosis. Am J Respir Cell Mol Biol 2010;42(1):32–9.

[5] Jordan HT, Stellman SD, Prezant D, Teirstein A, Osahan SS, Cone JE. Sarcoidosis diagnosed after September 11, 2001, among adults exposed to the World Trade Center disaster. J Occup Environ Med 2011;53(9):966–74.

[6] Muller-Quernheim J, Prasse A, Zissel G. Pathogenesis of sarcoidosis. Presse Med 2012;41(6 Pt 2):e275–87.

[7] Newman LS, Rose CS, Bresnitz EA, et al. A case control etiologic study of sarcoidosis: environmental and occupational risk factors. Am J Respir Crit Care Med 2004;170(12):1324–30.

[8] Judson MA. The clinical features of sarcoidosis: a comprehensive review. Clin Rev Allergy Immunol 2015;49(1):63–78.

[9] Spagnolo P CP, duBois RM. *Sarcoidosis: interstitial lung disease*. 5th edition. Shelton: People's Medical Publishing House; 2011.

[10] Haimovic A, Sanchez M, Judson MA, Prystowsky S. Sarcoidosis: a comprehensive review and update for the dermatologist: part I. Cutaneous disease. J Am Acad Dermatol 2012;66(5):699. e1–18; quiz 717–8.
[11] Iannuzzi MC, Rybicki BA, Teirstein AS. Sarcoidosis. N Engl J Med 2007;357(21):2153–65.
[12] Vatti R, Sharma OP. Course of asymptomatic liver involvement in sarcoidosis: role of therapy in selected cases. SarcoidosisVasc Diffuse Lung Dis 1997;14(1):73–6.
[13] Dulai PS, Rothstein RI. Disseminated sarcoidosis presenting as granulomatous gastritis: a clinical review of the gastrointestinal and hepatic manifestations of sarcoidosis. J Clin Gastroenterol 2012;46 (5):367–74.
[14] de Kleijn WP, De Vries J, Lower EE, Elfferich MD, Baughman RP, Drent M. Fatigue in sarcoidosis: a systematic review. Curr Opin Pulm Med 2009;15(5):499–506.
[15] Judson MA. Advances in the diagnosis and treatment of sarcoidosis. F1000 Prime Rep 2014;6:89.
[16] Newman LS, Rose CS, Maier LA. Sarcoidosis. N Engl J Med 1997;336(17):1224–34.
[17] Petropoulos IK, Zuber JP, Guex-Crosier Y. Heerfordt syndrome with unilateral facial nerve palsy: a rare presentation of sarcoidosis. Klin Monatsbl Augenheilkd 2008;225(5):453–6.
[18] Haramati LB, Lee G, Singh A, Molina PL, White CS. Newly diagnosed pulmonary sarcoidosis in HIV-infected patients. Radiology 2001;218(1):242–6.
[19] Chalayer E, Bachy E, Occelli P, et al. Sarcoidosis and lymphoma: a comparative study. QJM 2015;108(11):871–8.
[20] Beegle SH, Barba K, Gobunsuy R, Judson MA. Current and emerging pharmacological treatments for sarcoidosis: a review. Drug Des Devel Ther 2013;7:325–38.
[21] Schutt AC, Bullington WM, Judson MA. Pharmacotherapy for pulmonary sarcoidosis: a Delphi consensus study. Respir Med 2010;104(5):717–23.
[22] Paramothayan S, Jones PW. Corticosteroid therapy in pulmonary sarcoidosis: a systematic review. JAMA 2002;287(10):1301–7.
[23] Kaplan JA, Schecter SC, Rogers SJ, Lin MYC, Posselt AM, Carter JT. Expanded indications for bariatric surgery: should patients on chronic steroids be offered bariatric procedures? Surg Obes Relat Dis 2017; 13(1):35–40.
[24] Azagury DE, Abu Dayyeh BK, Greenwalt IT, Thompson CC. Marginal ulceration after Roux-en-Y gastric bypass surgery: characteristics, risk factors, treatment, and outcomes. Endoscopy 2011;43 (11):950–4.
[25] Iannelli A, Martini F, Gugenheim J. Laparoscopic conversion of Roux-en-Y gastric bypass to sleeve gastrectomy for intractable marginal ulcer. Surg Obes Relat Dis 2015;11(1):262–4.
[26] Iannelli A, Sans A, Martini F, Santonicola A, Iovino P, Angrisani L. Hiatal hernia, GERD, and sleeve gastrectomy: a complex interplay. Obes Surg 2016;26(10):2485–7.
[27] Mechanick JI, Youdim A, Jones DB, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient–2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. Obesity (Silver Spring) 2013;21(Suppl 1):S1–27.
[28] Berliner AR, Haas M, Choi MJ. Sarcoidosis: the nephrologist's perspective. Am J Kidney Dis 2006;48(5):856–70.
[29] Canales BK, Hatch M. Kidney stone incidence and metabolic urinary changes after modern bariatric surgery: review of clinical studies, experimental models, and prevention strategies. Surg Obes Relat Dis 2014;10(4):734–42.
[30] La Rochelle JC, Coogan CL. Urological manifestations of sarcoidosis. J Urol 2012;187(1):18–24.
[31] Curtis JR, Westfall AO, Allison J, et al. Population-based assessment of adverse events associated with long-term glucocorticoid use. Arthritis Rheum 2006;55(3):420–6.
[32] Kumagai S, Kawano S, Atsumi T, et al. Vertebral fracture and bone mineral density in women receiving high dose glucocorticoids for treatment of autoimmune diseases. J Rheumatol 2005;32(5):863–9.