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Development of Acute Gouty Attack in the Morbidly Obese Population after Bariatric Surgery

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Background: Gout is associated with increased body weight. We evaluated the prevalence of gout and acute gouty attacks in the morbidly obese population who underwent bariatric surgery.

Methods: The medical records and operative reports of 1,240 patients who underwent bariatric surgery were reviewed retrospectively for weight parameters, BMI, weight loss, medical history of gout, and onset of acute gouty attacks.

Results: Of the 1,240 patients, 5 (0.4%) had been previously diagnosed with gout. 2 of these 5 had acute attacks during the postoperative period, and responded succesfully to intravenous colchicine.

Conclusion: Although rare, gout must be considered a co-morbid illness in obese and morbidly obese patients. Surgeons should be familiar with the signs and symptoms of attacks in the postoperative period, and be knowledgeable in the management.

Key words: Gout, morbid obesity, bariatric surgery

Introduction

Gout is a common worldwide disease with an incidence of 0.2 to 0.35 per 1,000 and an overall prevalence of 1.6 to 13.6 per 1,000 in the general population.[1,2] Numerous factors may trigger acute gouty attacks. Obesity is associated with many metabolic disorders such as gout, although its true incidence is unknown. Surgery as well as surgically-induced weight loss may lead to hyperuricemia followed by acute gouty attacks.[2]

To our knowledge, there are no reports in the literature discussing the incidence of gout and gouty attacks in the obese population that have undergone bariatric surgery. The objective of this study was to determine the prevalence of gout in the morbidly obese population and to identify the incidence and cause of gouty attacks immediately following bariatric surgery and their management.

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Methods

After IRB approval, we retrospectively reviewed the medical records of 1,240 consecutive patients who had undergone laparoscopic Roux-en-Y gastric bypass (RYGBP) at the Bariatric Institute at Cleveland Clinic Florida between May 2001 and May 2004. Parameters assessed included weight, body mass index (BMI), weight loss, a medical history of gout, and any onset of acute gouty attacks.

Results

Of the 1,240 patients, five (0.4%) had been previously diagnosed with gout. These patients had a mean age of 48.5 (range 32 to 65) years and a mean BMI of 51.9 (range 35.5 to 71.6) kg/m^2. All patients were preoperatively diagnosed with gout and were taking medications such as non-steroidal anti-inflammatory drugs (NSAIDs) or allopurinol, most discontinued preoperatively (Table 1). Two patients (40%) presented with acute gouty attacks in the postoperative period manifested by monoarticular pain, predominantly in the knees and toes. Both patients were diagnosed based on the clinical manifestations and laboratory values; neither patient was febrile but one had uric acid levels >8 mg/dl. Both patients were treated with NSAIDs (Rofecoxib,

Table 1. Distribution of the patients, previously diagnosed with gout

Case	Age (yrs)	Gender	Co-morbidity	Acute Gout Attack	Treatment	Resolution
1	65	Male	Hypertension, Osteoarthritis, Diabetes, Kidney stones	No	Allopurinol	Yes
2	61	Male	Hypertension, Diabetes, Obstructive sleep apnea, Hypercholesterolemia	No	NSAIDs, Allopurinol	Yes
3	32	Male	Obstructive sleep apnea, Osteoarthritis	Yes	NSAIDs	No
4	52	Male	Hypertension, Obstructive sleep apnea, Mitral valve prolapse	No	Allopurinol	No
5	40	Male	Hypertension, Osteoarthritis, Obstructive sleep apnea, Hypercholesterolemia	Yes	Colchicine orally	No

Merck & Co., Whitehouse Station, NJ), with minimal response. A rheumatologist evaluated both patients, after which treatment with intravenous colchicine (Bedford Laboratories, Boehringer Ingelheim, Ridgefield, CT) 0.6 mg/day was started.

In our series, the prevalence of gout was of 0.4 per 1,000 and the incidence of acute attacks was 40% in the patients with a previous diagnosis of gout. There was no resolution of the disease due to weight loss in our series.

Discussion

Gout is a metabolic disorder of purines, characterized by hyperuricemia and urate crystal deposition in articular and extra-articular tissues.[3] Hyperuricemia is defined as a serum urate concentration >7 mg/dl, and has been found in 2.3 to 17.6% of the population.[2] Gout is manifested by elevated serum urate concentration (hyperuricemia), recurrent attacks of acute arthritis in which monosodium urate monohydrate crystals are demonstrable in synovial fluid leukocytes, aggregates of sodium urate monohydrate crystals (tophi) deposited in and around joints (which sometimes lead to deformity and crippling), renal disease (involving glomerular, tubular and interstitial tissue and blood vessels), and uric acid urolithiasis.[2]

In our series, the incidence of gout in the morbidly obese population undergoing weight loss surgery was 0.4%. Cross-sectional studies have demonstrated an association of serum uric acid levels with obesity and overeating, alcohol consumption, serum cholesterol levels, and the use of certain drugs, particularly thiazide diuretics used in the treatment of hypertension.[4] Gout can also be precipitated by catabolic events such as major surgery. Several factors may precipitate acute gouty attacks in the perioperative period. Patients who undergo an abdominal operation may have periods of starvation, catabolism, or dehydration that can induce hyperuricemia and precipitate gout.[3,5] Multivariate analyses, including BMI, have found that the size of the visceral fat area was the strongest contributor to an elevated serum concentration of uric acid, a decrease in uric acid clearance, and an increase in the urinary uric acid to creatinine ratio.[6] Actually, as a response to major abdominal surgery and possibly to crushing of gastric tissue with release of purines, a fall in serum uric acid and an elevation in urinary uric acid occurs transiently immediately postoperatively.[7] None of the patients in our series had resolution of gout postoperatively and all continue to take their preoperative medications.

However, a study assessing the effects of weight reduction revealed that significant changes in urate metabolism occurred, with reduction in the serum urate concentration and the miscible urate pool as well as an increased renal clearance of urate. In addition, the daily production of urate had fallen, the renal excretion of labeled urate had risen, and the incorporation of glycine into urate (a reflection of *de novo* purine synthesis), had fallen to within the normal range.[8-10]

Treatment of an acute gouty attack should be initiated upon diagnosis (Figure 1). The chief objective of therapy in acute gout is rapid safe resolution of pain and functional debility.[6] This is of paramount importance in the morbidly obese population,

because lack of mobility in the postoperative period is a common predictor of respiratory complications and deep vein trombosis. Treatment options for acute gouty attacks include NSAIDs, colchicine, and corticosteroids. Uricosuric agents and xanthine oxidase inhibitors are of little value in acute attacks.

Colchicine is the treatment of choice for acute attacks; however, this drug can cause diarrhea and has undesirable effects on wound healing if administered in the postoperative period.[3] Intravenous colchicine should be used only in patients with polyarticular gout, or those unable to tolerate NSAIDs or take oral medications. An initial dose of 1-2 mg can be followed by an additional boost of a 1 mg dose 6 hours later, until a satisfactory response is achieved.[2,11] In general, the total dosage for the first 24-hour period should not exceed 4 mg.

In our study, the two patients who presented a postoperative acute gout attack were treated with I.V. colchicine. I.V. colchicine is associated with some risk if local extravasation occurs with injection, which can lead to inflammation and necrosis and may be extremely painful.[2] Although I.V. colchicine is effective, severe toxicity and death have been reported. In the 20 reported deaths attributed to the I.V. colchicine, each patient received a cumulative dose of more than the recommended maximum of 2-4 mg in 24 hrs.[1]

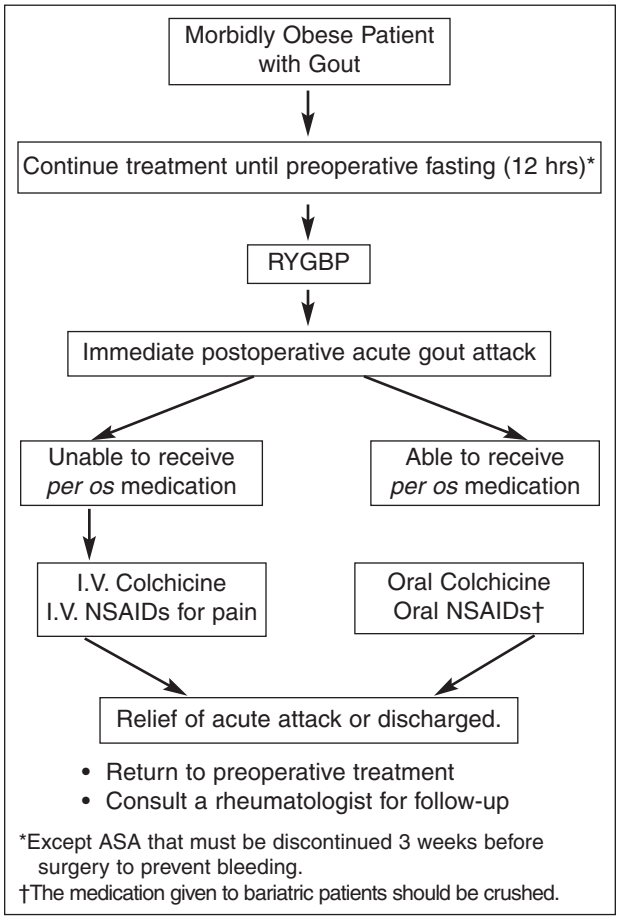

Figure 1. Algorithm for treatment of morbidly obese patients with gout undergoing bariatric surgery.

NSAIDs can be highly effective in the treatment of acute gout. However, the effects of NSAIDs in the production of gastric ulcers are well recognized. In these cases, we recommend concomitant proton pump inhibitors to prevent gastric ulcer formation.

Our study suggested that bariatric surgery is a factor in the development of acute gouty attacks. Resolution of the disease may not be related to weight loss, as seen in our series. Therefore, obese or morbidly obese patients with gout may not respond positively to medical or surgically-induced weight reduction, specifically in patients with high uric acid levels.

References

1. Becker M. Treatment of gout. UpToDate® version 12.2 May 4, 2004. http://www.utdol.com/application/topic.asp?file=rheumati/32001&type=A&selectedTitle=2~35
2. Wortmann RL, Kelley WN. Gout and hyperuricemia. In: Ruddy S, Harris ED, Sledge CB, eds. Kelley's Textbook of Rheumatology, 6th Edn. St. Louis: WB Saunders 2001: 339-1371.
3. Craig MH, Poole GV, Hauser CJ. Postsurgical gout. Am Surg 1995; 61: 56-9.
4. Sillman AJ, Hochberg MC. Epidemiology of the Rheumatic Diseases. Oxford: Oxford University Press 1993.
5. Becker M. Clinical manifestations and diagnosis of gout. UpToDate® version 12.2. May 4, 2004. http://www.utdol.com/application/topic.asp?file=rheumati/26808&type=A&selectedTitle=1~35
6. Terkeltaub RA. Gout. N Engl J Med 2003; 349: 1647-55.
7. Deitel M, Thompson DA, Saldanha CF et al. "Pink urine" in morbidly obese patients following gastric partitioning. Can Med Assoc J 1984; 130: 1007-11.
8. Emmerson BT. Alteration of urate metabolism by weight reduction. Aust NZ J Med 1973; 3: 410-2.
9. Takahashi S, Yamamoto T, Tsutsumi Z et al. Close correlation between visceral fat accumulation and uric acid metabolism in healthy men. Am Fam Physician 1999; 59: 1799-806, 1810.
10. Wise CM, Agudelo CA. Gouty arthritis and uric acid metabolism. Curr Opin Rheumatol 1996; 8: 248-54.
11. Star V, Hochberg MC. Prevention and management of gout. Drugs 1993; 45: 212-22.

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