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Case Report

Renal Failure, Glomerulonephritis and Morbid Obesity: Improvement after Rapid Weight Loss following Laparoscopic Gastric Bypass

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There is experimental evidence but very few human studies that suggest a role for obesity in the formation and progression of some glomerular lesions. We report the case of a morbidly obese male with hematuria and proteinuria that was subsequently diagnosed with renal failure which required dialysis. Histological findings of the renal biopsy performed during a laparoscopic gastric bypass are presented. His renal failure resolved with the weight loss.

Key words: Morbid obesity, renal failure, dialysis, laparoscopic gastric bypass, IgA nephropathy

Introduction

The prevalence of obesity is increasing worldwide and the impact of obesity on metabolic and cardiovascular diseases has been well documented. However, less attention has been paid to the impact of obesity on the kidney. Obesity is a risk factor for progressive loss of renal function in patients with known kidney disease, and there is increasing evidence that obesity may also damage the kidney in otherwise healthy individuals.[1] Increased renal blood flow and glomerular filtration rate as well as microalbuminuria have been described in obesity.[2] It has previously been shown that an increased body mass index (BMI) is associated with microalbuminuria, especially in hypertensive individuals.[2] The incidence of obesity-related glomerulopathy, defined as focal segmental glomerulosclerosis (FSGS) on biopsy, has increased ten-fold over the last 15 years.[3]

In 1974, an association between massive obesity and nephrotic range proteinuria was first reported.[4] Since that time, the development of FSGS has been linked to massive obesity.[5] This obesity-related glomerulopathy (ORG) is an increasingly prevalent disease that is clinically and pathologically distinct from idiopathic FSGS (I-FSGS).[3] Previous experimental and clinical evidence suggests that excess body weight and/or obesity are associated with hyperfiltration and glomerulomegaly, with intraglomerular hypertension as the genesis of a sclerotic lesion.[2,3,6]

IgA nephropathy remains an uncommon finding in obesity-associated glomerulonephritis, and the role of excess body weight in both the clinical and pathological progression of IgA nephropathy has not been precisely investigated.[7,8] Bonnet et al[8] in their study of IgA nephropathy found a correlation between the disease and the presence of an elevated BMI.[6]

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We report the case of a super-obese patient who was diagnosed with hematuria, proteinuria and renal failure during his preoperative evaluation. A dramatic improvement in renal failure was noted after the patient underwent a laparoscopic Roux-en-Y gastric bypass (LRYGBP). We also describe the histological findings of the renal biopsy performed during the LRYGBP and a review of the relevant literature.

Case Report

A 37-year-old Caucasian morbidly obese male, with BMI 85 kg/m^2, weighing 239 kg and a height 165 cm, underwent preoperative evaluation for bariatric surgery. During that evaluation, the patient was found to be in renal failure with a creatinine of 9.9 mg/dl. He reported episodes of "cellulitis" approximately 4 times a year, almost always associated with gross hematuria for the first few days. The patient also related a cellulitis episode 2 weeks earlier, with multiple microabscesses of the skin and panniculitis involving his right thigh and abdominal surface and concomitant hematuria; the patient did not take any antibiotics for this condition. He reported that the hematuria was almost resolved when he had a relapse of the cellulitis with increased hematuria on the day of his preoperative evaluation. The past medical history was significant for hypertension and depression and was negative for diabetes.

He was admitted for intravenous (IV) antibiotics and evaluation of "acute renal failure". Laboratory analysis revealed: Hb 10 g/dl; Hct 31%; BUN 55 mg/dl; Cr 5.8 mg/dl; K^+ 4.3 mmol/L (normal 3.5-5.1); complement 3 and 4 normal and ASO 541 units (normal 0-125). Serum IgA levels were elevated to 590 units (normal 78-391). Urine collection over 24 hours revealed 3.6 g of proteinuria. The remaining serologies (antinuclear antibody, antineutrophil cytoplasmic antibodies, hepatitis panel, rapid plasma reagin, protein electrophoresis) were normal or negative. In the lipids panel, only the LDL appeared to be elevated (300 U/L).

The patient had undergone renal ultrasound; however, due to his massive body size, the left kidney was not visualized and the right side was negative for hydronephrosis. Both kidneys were well visualized on a renal nuclear medicine scan. An attempted ultrasound-guided percutaneous needle renal biopsy was unsuccessful.

He was placed on IV fluid and antibiotics to treat the underlying cellulites. After resolution of the cellulitis and medical clearance by the nephrologists, LRYGBP with a 200-cm alimentary limb was performed. A right renal biopsy with a Trucut needle was performed during the same procedure. The gallbladder was also removed due to the presence of cholelithiasis (serum creatinine was 6.1 mg/dl on the day of surgery).

The patient had no postoperative surgical complications. However, he remained hypertensive throughout his postoperative hospital stay and required several antihypertensive medications.

The renal biopsy report disclosed a mesangial proliferative glomerulonephritis with mild-to-moderate tubulo-interstitial injury. Electron microscopy confirmed numerous electron-dense deposits consistent with immune complexes in the mesangium and the paramesangial areas. Diagnosis was that of an immune complex mediated glomerulonephritis (Figures 1 and 2). Although no glomeruli were present in the tissue submitted for immunofluorescence, based on the light microscopic features, the studies noted above and the patient's clinical history, a diagnosis of IgA nephropathy was strongly suspected. He was discharged on the 7th postoperative day with serum creatinine 5.9 mg/dl.

Two weeks later, the patient presented to the

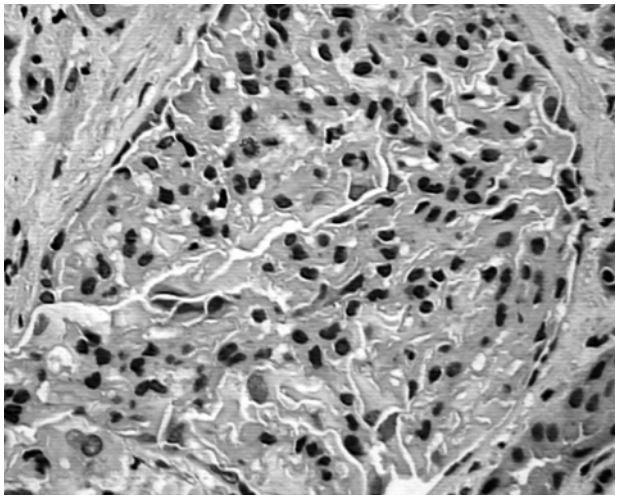

Figure 1. High power demonstrates mesangial proliferation. No necrosis, thrombi, inflammatory cells or crescents were noted (H&E x 4000).

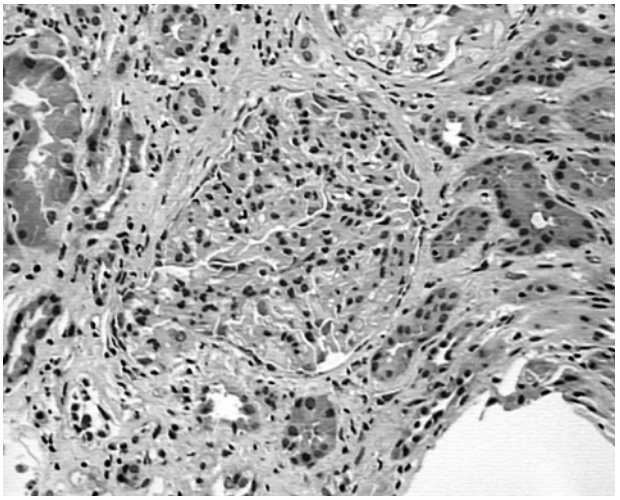

Figure 2. Scanning magnification shows an enlarged and segmentally hypercellular glomerulus (H&E x 2000).

Emergency Department with oliguria, dizziness, fatigue, and lethargy. Laboratory tests revealed creatinine 16.8 mg/dl, BUN 92 mg/dl and potassium 5.3 mmol/L. The patient reported decreased oral intake, fatigue, anuria for 2 days, and diarrhea. Intravenous fluids were started, a dialysis catheter was placed, and dialysis was initiated. His condition stabilized and renal function improved; therefore, dialysis was terminated. Creatinine at discharge was 4.6 mg/dl. Seven months after the LRYGBP, he has lost 36.4 kg, the serum creatinine is 1.6 mg/dl, BUN is 15 mg/dl, and potassium is 3.5 mmol/L.

Discussion

In this case, the patient's biopsy lacked glomeruli for immunofluorescence; therefore, it is impossible to know for certain that the glomerulonephritis was IgA nephropathy. Light and electron microscopy were consistent with this diagnosis, as was the patient's clinical course. The biopsy and serologies were not consistent with FSGS or post-infectious glomerulonephritis.

Bonnet et al[8] found in patients with IgA nephropathy that being overweight at the time of initial renal biopsy significantly correlated with more severe pathological lesions and increased proteinuria, which favored the subsequent development of both arterial hypertension and chronic renal failure (CRF). This was the first report of a significant influence of increased BMI on renal lesions in IgA nephropathy. There are no other published studies regarding the influence of BMI on clinical and/or pathological progression of chronic renal diseases. However, data do exist for morbid obesity and FSGS and the relation of BMI to outcome in dialysis patients with end-stage renal disease (ESRD). Although many studies describe an association between obesity and glomerular hyperfiltration[2] with subsequent FSGS,[6] the precise role of excess weight and/or obesity on renal pathological characteristics in IgA nephropathy has not been previously investigated. Additionally, the influence of excess weight on the progression of IgA nephropathy may be independent of hyperlipidemia, a frequent complication of obesity. This is highly relevant because hyperlipidemia has been shown to influence the rate of progression of various chronic renal diseases, particularly IgA nephropathy.[9] This influence of body weight on renal prognosis is consistent with our finding that being overweight is significantly associated at baseline with increased proteinuria, more severe renal lesions and a subsequent greater incidence of arterial hypertension. Proteinuria, hypertension and the degree of pathologic change in particular tubular lesions, have been shown to be important risk factors for CRF in IgA nephropathy.[9]

Decisions regarding the appropriateness of surgical procedures in renal failure patients are controversial. Although significant advances in the perioperative and intraoperative care of patients undergoing major surgery (especially vascular and cardiac) have been achieved over the past several decades, the occurrence of perioperative acute renal failure continues to plague the postoperative course of many patients. This problem is substantial when it occurs, because of its association with an increased morbidity and mortality. Anesthesiologists often use intraoperative strategies with the intention of providing renal protection; however, no substantive scientific evidence has validated any such intervention. The most commonly used techniques are intravenous hydration, mannitol, “renal dose” dopamine and loop diuretics, often in combination.[10] Maintenance of adequate intravascular volume is likely to be the most effective method for reducing the risk of perioperative deterioration in renal function.

Although the mortality rate for elective major sur-

gery is low (1-4%), in patients with dialysis-dependent chronic renal failure, the risk of perioperative complications, including postoperative hyperkalemia, pneumonia, fluid overload, and bleeding, is substantially increased. Postoperative hyperkalemia requiring emergent hemodialysis has reportedly occurred in 20-30% of such patients. Because of the increased morbidity in renal failure patients, several reports imply that major surgical procedures are contraindicated with a BUN >80 mg/dl or a creatinine >3 mg/dl.[11] In 1986, Cohen et al[12] recommended that patients with severe renal dysfunction (creatinine >4 mg/dl) who are not already on hemodialysis should be considered for preoperative dialysis in an attempt to reduce the high incidence of serious postoperative complications. Conversely, Komori et al[13] reported that none of the patients in their study, including those with severe renal failure (creatinine >4 mg/dl), required postoperative dialysis during hospitalization. Furthermore, there were no significant differences with normal renal function, chronic kidney disease and end-stage renal disease with respect to mortality rate or incidence of postoperative complications after a major operation (abdominal aortic aneurysm repair).

In our case, renal failure due to glomerulonephritis significantly improved after the dramatic weight loss following a LRYGBP. If the patient had been denied surgery due to renal failure and the initially associated high creatinine levels, it is unlikely that the renal function would have improved. Thus, renal failure should not be considered as a contraindication in the morbidly obese patient being considered for gastric bypass surgery. Therefore, although there is a risk of postoperative dehydration, gastric bypass should be considered in these patients.

Conclusion

An elevated BMI may present an independent risk factor in IgA nephropathy for progression of vascular, tubular and interstitial lesions. This effect is probably independent of associated hyperlipidemia. Recognition of obesity may alert physicians to the increased risk in these patients. Whether weight reduction may improve or delay renal disease progression remains to be prospectively evaluated. Renal failure should not be considered a contraindication to bariatric surgery.

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