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In-Depth Review

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Bariatric surgery and the kidney—much benefit, but also potential harm

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Abstract
Bariatric surgery is increasingly performed on overweight individuals. A significant benefit with respect to cardiovascular (CV) events and survival has been documented. After weight loss, reduction of albuminuria/proteinuria is almost consistently seen; small studies documented retardation of the glomerular filtration rate (GFR) loss after bariatric surgery; reduction of blood pressure (BP) is less consistent. It has been known for a long time that the frequency of oxalate stones is increased after bariatric surgery. The main renal threat of hyperoxaluria is renal oxalosis, often irreversible, causing persisting renal failure. The causes are reduced oxalate binding by calcium due to saponification of calcium causing fat malabsorption, increased permeability for oxalate because of increased permeability of colon mucosa triggered by increased bile salts and reduced colonization of the colon by oxalobacter formigenes. These mechanisms are susceptible to treatment.

Keywords: bariatric surgery; oxalate stones; renal oxalosis

Introduction

First cases of bariatric surgery were reported more than 50 years ago, mainly with the intention to achieve malabsorption [1]. Today, bariatric surgery is mainly performed to achieve weight loss. Most frequently the Roux-en-Y gastric bypass (RYGB) and gastric banding techniques are used, more rarely malabsorptive procedures, e.g. biliopancreatic diversion. In 2008, the cumulative frequency of bariatric surgery worldwide was 344 221 operations; laparoscopic adjustable gastric banding accounted for 42.3% and laparoscopic standard RYGB accounted for 39.7% [2]. It has become evident that part of the beneficial mechanisms accounting for weight loss are increased anorectic gut hormones, e.g. glucagon-like peptide 1 and peptide YY on the one hand, and decrease in the orexigenic hormones, e.g. ghrelin, on the other hand, [3]. Important for renal events (see below) is the fact that weight loss after bariatric surgery also causes changes in the gut microflora.

Effects of bariatric surgery on kidney function

In severe obesity, renal plasma flow (RPF), glomerular filtration rate (GFR) and filtration fraction (FF) are increased. Chagnac *et al.* [4] showed that in subjects with morbid obesity weight loss (of note, without surgical intervention) caused decreased GFR, RPF, FF and albumin excretion. Another important renal malfunction in obesity is increased proximal tubular sodium reabsorption, a mechanism contributing to hypertension in obesity [5]. New data suggest that specifically perivascular renal sinus fat is related to the intensity of microalbuminuria in the metabolic syndrome [6].

Albuminuria/proteinuria

After weight loss, specifically also after bariatric surgery, microalbuminuria and proteinuria are on average significantly decreased [7]; the reduction is independent of the decline in mean arterial pressure and was found to be $\Delta - 14$ mg/day in microalbuminuric and $\Delta - 1.7$ g/day in proteinuric obese patients; a significant decrease in creatinine clearance has been documented only in patients with bariatric surgery [8]. This finding is particularly impressive in patients with primary kidney disease, e.g. in type 2 diabetic patients treated with RYGB or other types of bariatric surgery. A small study showed that albuminuria was consistently reduced in parallel with improved insulin sensitivity and increased high-molecular-weight adiponectin [9, 10]. Such improvement in albuminuria after RYGB is also seen in non-diabetic patients as well [11]. In individual cases, the reversal of albuminuria was dramatic [12], e.g. in one 17-year-old girl with a body mass index (BMI) of 56.8 kg/m^2 with biopsy confirmed glomerulosclerosis experienced complete normalization of proteinuria after surgery and this was accompanied by complete normalization of kidney function [13].

Glomerular filtration rate

The methodological difficulties to calculate the estimated GFR (eGFR) before and after bariatric surgery with the attendant change in indices of body weight are illustrated in the recent paper of Nagelkerke (International Urology Nephrology (2012) MAR 3): using different formulae to correct for the body size resulted in substantially different GFR estimates, prompting the comment 'GFR estimation in the morbidly obese pre-and postbariatric surgery: one size does not fit all'. It is difficult to distinguish whether the reduction of GFR reflects the reversal of obesity-induced hyperfiltration or loss of renal function.

Of special interest is the impact of bariatric surgery on chronic kidney disease (CKD) patients; Turgeon *et al*. [14] analyzed the evolution of renal function of 27 736 patients in different stages of CKD; the complication rate rose from 4.6% (in patients with CKD stage 1) to 9.9% (CKD stage 5), but there were no fatalities. In a small study in obese type 2 diabetics undergoing RYGB, creatinine clearance was lowered by 15% in diabetic and by 21% in non-diabetic patients after 12 months of follow-up [15]. For patients with primary kidney disease, e.g. focal segmental glomerulosclerosis, membranous glomerulonephritis, diabetic nephropathy, a small study documented that in a substantial proportion of patients with kidney disease stabilization with no further progressive loss of renal function postoperatively was achieved after gastric bypass operation [16]. This finding is confirmed by a retrospective study in patients with CKD 3 undergoing bariatric surgery: after 12 months, the BMI and blood pressure (BP) had decreased and the mean GFR had even increased from 47.9 to 61.6 mL/min/1.73 m^2 [17].

Blood pressure

The impact of weight loss on BP after bariatric surgery is somewhat less consistent, but BP lowering has been documented in many studies [18]. In one prospective study (but not in all studies), the mean arterial pressure decreased 12 months after RYGP or laparoscopic sleeve gastrectomy in parallel with decrease in weight; BP decreased even more markedly in patients with impaired renal function. Lower BP was also paralleled by less systemic inflammation and improved renal function as assessed by cystatin C [19].

Diabetes mellitus type 2

Bariatric surgery has recently been used with increasing frequency in type 2 diabetes [20]. Apparently prediabetic patients progress less frequently to overt diabetes after bariatric surgery. In the USA, 22 693 persons underwent bariatric surgery in 2003–2007; after 4–5 years following bariatric surgery, the onset of type 2 diabetes was less frequent, i.e. 14.6% in individuals with bariatric surgery compared with 33% in matched controls [21]. Even in patients with overt type 2 diabetes and a BMI <35 kg/m^2, bariatric surgery reduced BMI and fasting glucose; in addition, haemoglobin A1c (HbA1c) was decreased to values <7% in 84% of patients [22]. A beneficial effect of bariatric surgery in type 2 diabetes compared with the conventional medical therapy has also recently been well documented in two large single-centre studies after both RYGP and biliopancreatic diversion [23, 24].

The spectrum of issues raised by bariatric surgery in type 2 diabetes has recently been discussed in depth by Dixon *et al*. [20]. After RYGP and gastric banding weight independent beneficial effects on diabetes have been documented. One large nationwide long-term (10 years) uncontrolled follow-up assessment of bariatric surgery in diabetes documented lower morbidity and mortality, specifically reduced Cardiovascular (CV) disease, cancer (in women) and diabetes-related mortality [25, 26], but controlled evidence is currently not available [20]. The achieved beneficial effects go beyond what is explained by weight loss per se; weight loss does play a role, but additional factors, e.g. intestinal hormones, almost certainly play a role as well.

The comparison of bariatric surgery versus non-surgical therapy in type 2 diabetic patients with BMI <35 kg/m^2 by Serrot *et al*. [27] showed even in this group a decrease of BMI (34.6 to 25.8 kg/m^2) and of HbA1c (8.2 to 6.1%) in contrast to non-surgical controls; changes in systolic BP or low density lipoprotein cholesterol were not seen in either group.

Adverse renal sequelae of bariatric surgery

Although sufficiently sized controlled studies are currently not yet available, there is no doubt that in non-diabetic individuals and type 2 diabetic patients (particularly those with morbid obesity) bariatric surgery has become an attractive therapeutic target. From a renal perspective, bariatric surgery has beneficial effects with respect to albuminuria/proteinuria as well as GFR loss [28, 29] and to a minor extent BP elevation. Major reduction of glycaemia and complications can be achieved as well [20]. A most impressive finding is the frequent observation in patients with primary kidney disease that renal function loss is progressively getting less.

Against these beneficial effects, one has to weigh some relatively rare, but potentially severe, adverse renal effects [30]. Recent insights into the underlying pathomechanisms are of interest because they may suggest potential prophylactic measures to prevent the main renal threats, i.e. oxalate nephrolithiasis and renal oxalosis (which may eventually cause end-stage renal disease). In addition, there are occasional further renal side effects.

In the past, Thakar *et al*. had reported frequent occurrence of acute kidney injury (AKI) after gastric bypass [31]; cases of AKI as a result of rhabdomyolysis had also been reported [32]—but these complications had no longer been reported in more recent large series.

Following bariatric surgery, early onset of encephalopathy had been observed in cases of ornithine transcarbamylase deficiency, a rare genetic disease [33].

One initial concern had been the presumed adverse renal impact of bariatric surgery on nonalcoholic fatty liver disease; the risk of impaired renal function was thought to be particularly high in these patients, because such patients with a mild decrease in eGFR frequently have also advanced inflammation [34]—but even in this group, mortality was not increased at least during the postoperative period [35].

Another concern after bariatric surgery, particularly in patients with impaired renal function, is the potential

aggravation of disturbed calcium metabolism and development of hyperparathyroidism; the authors found that without calcium supplementation, parathyroid hormone increased and 25(OH)D decreased, so that after 1 year 15% had developed hyperparathyroidism; the authors recommended universal calcium and vitamin D supplementation [36].

Based on the follow-up of 813 patients, Schuster *et al*. [37] documented that overall bariatric surgery does not have a negative impact on renal function, at least in the short term. After bariatric surgery, the main threat to renal function is the high prevalence of hyperoxaluria which had been identified in several early studies [38–43]. It has also been known for a long time that oxalate excretion is particularly increased when dietary calcium intake is low [44]. In the 1970s, when jejunoileal bypass was still in vogue, nephrolithiasis was reported in no <39% of patients after 15 years of follow-up [45]. In 2006, Encinosa *et al*. [46] reported that 3% of patients developed renal stones in the first 180 days post-operation; 10-fold higher rates had been reported in patients with a history of nephrolithiasis [47]. Such hyperoxaluria tends to increase with time after surgery [41]. These findings had led to the concern of an incoming epidemic of oxalate stones [48]—but today it is clear that fortunately, at least to some extent, prevention is possible (see below).

Even in adult patients with no history of lithiasis, a significant increase in oxalate excretion is seen after RYGB or biliopancreatic diversion/duodenal switch when compared with controls and even when compared with idiopathic stone formers [38]. The tendency to develop high oxalate excretion rates prompted Ahmed and Byrne [28] to point to the precarious balance after bariatric surgery between benefit (mainly lower CV and diabetes risk) on the one hand and increased hazard (hyperoxaluria, oxalate nephrolithiasis, renal oxalosis) on the other hand. Fortunately, observational studies suggest that in patients after bariatric surgery, measures to prevent oxalate nephrolithiasis and end-stage kidney failure from renal oxalosis are effective.

On the one hand, nephrolithiasis is a well-known complication of obesity [49, 50]; but on the other hand, bariatric surgery nephrolithiasis is very frequent, presumably even more frequent, as well [38, 45, 47, 48, 51].

Even more serious is oxalate nephropathy and potentially its end-stage, i.e. chronic renal failure. This complication is less frequent than nephrolithiasis, but by no means absolutely rare [43, 52–57].

Clearly, the lithogenicity of bariatric surgery is multifactorial [30], but this diversity also provides a spectrum of preventive approaches. (i) Saponification of calcium as a result of fat malabsorption reduces binding of oxalate by calcium. (ii) In addition, increased bile salts in the colon (as a result their decreased upstream absorption of bile salts) increase the permeability of colon mucosa, thus permitting oxalate to escape into the blood stream. (iii) Finally, a finding in these patients is a potential target for prevention: presumably as a result of frequent antibiotic therapy [58–61], the colonization of the colon by oxalobacter formigenes is often reduced. Consequently, it is sensible in these patients to withhold unnecessary antibiotic treatment. Dog experiments clearly showed that decreased enteric colonization with oxalobacter formigenes is a risk factor for urolithiasis in dogs [62]. Administration of oxalobacter reduced plasma oxalate concentrations in children [63] and had no side effects when administered to patients with primary hyperoxaluria [64]; the same was found in healthy subjects as well [65]. (iv) Finally, vitamin C administration is another potential cause of oxalate nephropathy [66] and this should be avoided.

The injurious impact of oxalate on the kidney is further illustrated by cases of hyperoxaluria, recurrent urolithiasis and even systemic oxalosis in the absence of bariatric surgery, i.e. in patients with no primary renal disease such as liver transplantation [67], lung transplantation [68], chronic pancreatitis [69], Crohn's disease [70] or similar.

Conclusion

Bariatric surgery usually has a beneficial impact on renal malfunction (proteinuria, glomerular filtration) in obese patients and even in obese patients with primary kidney disease. A major long-term risk, however, is increased urinary oxalate excretion which may cause oxalate lithiasis or even renal oxalosis. Preventive measures are advisable.

Conflict of interest statement. None declared.

References

1. Baker MT. The history and evolution of bariatric surgical procedures. *Surg Clin North Am* 2011; 91: 1181–1201, viii
2. Dixon JB, Straznicky NE, Lambert EA *et al*. Surgical approaches to the treatment of obesity. *Nat Rev Gastroenterol Hepatol* 2011; 8: 429–437
3. Sandoval D. Bariatric surgeries: beyond restriction and malabsorption. *Int J Obes (Lond)* 2011; 35(Suppl 3): S45–S49
4. Chagnac A, Weinstein T, Herman M *et al*. The effects of weight loss on renal function in patients with severe obesity. *J Am Soc Nephrol* 2003; 14: 1480–1486
5. Chagnac A, Herman M, Zingerman B *et al*. Obesity-induced glomerular hyperfiltration: its involvement in the pathogenesis of tubular sodium reabsorption. *Nephrol Dial Transplant* 2008; 23: 3946–3952
6. Wagner R, Machann J, Lehmann R *et al*. Exercise-induced albuminuria is associated with perivascular renal sinus fat in individuals at increased risk of type 2 diabetes. *Diabetologia* 2012; 55: 2054–2058
7. Navarro-Díaz M, Serra A, Romero R *et al*. Effect of drastic weight loss after bariatric surgery on renal parameters in extremely obese patients: long-term follow-up. *J Am Soc Nephrol* 2006; 17: S213–S217
8. Afshinnia F, Wilt TJ, Duval S *et al*. Weight loss and proteinuria: systematic review of clinical trials and comparative cohorts. *Nephrol Dial Transplant* 2010; 25: 1173–1183
9. Navaneethan SD, Kelly KR, Sabbagh F *et al*. Urinary albumin excretion, HMW adiponectin, and insulin sensitivity in type 2 diabetic patients undergoing bariatric surgery. *Obes Surg* 2010; 20: 308–315
10. Izzedine H, Coupaye M, Reach I *et al*. Gastric bypass and resolution of proteinuria in an obese diabetic patient. *Diabet Med* 2005; 22: 1761–1762
11. Mohan S, Tan J, Gorantla S *et al*. Early improvement in albuminuria in non-diabetic patients after Roux-en-Y bariatric surgery. *Obes Surg* 2012; 22: 375–380
12. Cuda SP, Chung MH, Denunzio TM *et al*. Reduction of proteinuria after gastric bypass surgery: Case presentation and management. *Surg Obes Relat Dis* 2005; 1: 64–66
13. Fowler SM, Kon V, Ma L *et al*. Obesity-related focal and segmental glomerulosclerosis: normalization of proteinuria in

an adolescent after bariatric surgery. *Pediatr Nephrol* 2009; 24: 851–855
14. Turgeon NA, Perez S, Mondestin M *et al*. The impact of renal function on outcomes of bariatric surgery. *J Am Soc Nephrol* 2012; 23: 885–894
15. Saliba J, Kasim NR, Tamboli RA *et al*. Roux-en-Y gastric bypass reverses renal glomerular but not tubular abnormalities in excessively obese diabetics. *Surgery* 2010; 147: 282–287
16. Alexander JW, Goodman HR, Hawver LR *et al*. Improvement and stabilization of chronic kidney disease after gastric bypass. *Surg Obes Relat Dis* 2009; 5: 237–241
17. Navaneethan SD, Yehnert H. Bariatric surgery and progression of chronic kidney disease. *Surg Obes Relat Dis* 2009; 5: 662–665
18. Montereggi A, Leone A, Castelli G *et al*. Prevalenza e tipologie di ipertensione arteriosa in una popolazione di pazienti non selezionati affetti da obesità morbigena in previsione di intervento di chirurgia bariatrica [Prevalence and subtypes of systemic hypertension in an unselected patient population with severe obesity undergoing bariatric surgery]. *G Ital Cardiol (Rome)* 2012; 13: 291–296
19. Fenske WK, Dubb S, Bueter M *et al*. Effect of bariatric surgery-induced weight loss on renal and systemic inflammation and blood pressure: a 12-month prospective study. *Surg Obes Relat Dis* 2012; doi: 10.1016/j.soard.2012.03.009
20. Dixon JB, le Roux CW, Rubino F *et al*. Bariatric surgery for type 2 diabetes. *Lancet* 2012; 379: 2300–2311
21. Bolen SD, Chang HY, Weiner JP *et al*. Clinical outcomes after bariatric surgery: a five-year matched cohort analysis in seven US states. *Obes Surg* 2012; 22: 749–763
22. Reis CE, Alvarez-Leite JI, Bressan J *et al*. Role of bariatric-metabolic surgery in the treatment of obese type 2 diabetes with body mass index <35kg/m2: a literature review. *Diabetes Technol Ther* 2012; 14: 365–372
23. Mingrone G, Panunzi S, De Gaetano A *et al*. Bariatric surgery versus conventional medical therapy for type 2 diabetes. *N Engl J Med* 2012; 366: 1577–1585
24. Schauer PR, Kashyap SR, Wolski K *et al*. Bariatric surgery versus intensive medical therapy in obese patients with diabetes. *N Engl J Med* 2012; 366: 1567–1576
25. Sjöström L, Lindroos AK, Peltonen M *et al*. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med* 2004; 351: 2683–2693
26. Sjöström L, Peltonen M, Jacobson P *et al*. Bariatric surgery and long-term cardiovascular events. *JAMA* 2012; 307: 56–65
27. Serrot FJ, Dorman RB, Miller CJ *et al*. Comparative effectiveness of bariatric surgery and nonsurgical therapy in adults with type 2 diabetes mellitus and body mass index <35kg/m2. *Surgery* 2011; 150: 684–691
28. Ahmed MH, Byrne CD. Bariatric surgery and renal function: a precarious balance between benefit and harm. *Nephrol Dial Transplant* 2010; 25: 3142–3147
29. Zalesin KC, McCullough PA. Bariatric surgery for morbid obesity: risks and benefits in chronic kidney disease patients. *Adv Chronic Kidney Dis* 2006; 13: 403–417
30. Currie A, Chetwood A, Ahmed AR. Bariatric surgery and renal function. *Obes Surg* 2011; 21: 528–539
31. Thakar CV, Kharat V, Blanck S *et al*. Acute kidney injury after gastric bypass surgery. *Clin J Am Soc Nephrol* 2007; 2: 426–430
32. Bostanjian D, Anthone GJ, Hamoui N *et al*. Rhabdomyolysis of gluteal muscles leading to renal failure: a potentially fatal complication of surgery in the morbidly obese. *Obes Surg* 2003; 13: 302–305
33. Hu WT, Kantarci OH, Merritt JL II *et al*. Ornithine transcarbamylase deficiency presenting as encephalopathy during adulthood following bariatric surgery. *Arch Neurol* 2007; 64: 126–128
34. Machado MV, Gonçalves S, Carepa F *et al*. Impaired renal function in morbid obese patients with nonalcoholic fatty liver disease. *Liver Int* 2012; 32: 241–248
35. Smith MD, Patterson E, Wahed AS *et al*. Thirty-day mortality after bariatric surgery: independently adjudicated causes of death in the longitudinal assessment of bariatric surgery. *Obes Surg* 2011; 21: 1687–1692
36. Flores L, Osaba MJ, Andreu A *et al*. Calcium and vitamin D supplementation after gastric bypass should be individualized to improve or avoid hyperparathyroidism. *Obes Surg* 2010; 20: 738–743
37. Schuster DP, Teodorescu M, Mikami D *et al*. Effect of bariatric surgery on normal and abnormal renal function. *Surg Obes Relat Dis* 2011; 7: 459–464
38. Patel BN, Passman CM, Fernandez A *et al*. Prevalence of hyperoxaluria after bariatric surgery. *J Urol* 2009; 181: 161–166
39. Patel SR, Penniston KL, Iwicki L *et al*. Dietary induction of long-term hyperoxaluria in the porcine model. *J Endourol* 2012; 26: 433–438
40. Whitson JM, Stackhouse GB, Stoller ML. Hyperoxaluria after modern bariatric surgery: case series and literature review. *Int Urol Nephrol* 2010; 42: 369–374
41. Sinha MK, Collazo-Clavell ML, Rule A *et al*. Hyperoxaluric nephrolithiasis is a complication of Roux-en-Y gastric bypass surgery. *Kidney Int* 2007; 72: 100–107
42. Nordenvall B, Backman L, Larsson L. Oxalate metabolism after intestinal bypass operations. *Scand J Gastroenterol* 1981; 16: 395–399
43. Nelson WK, Houghton SG, Milliner DS *et al*. Enteric hyperoxaluria, nephrolithiasis, and oxalate nephropathy: potentially serious and unappreciated complications of Roux-en-Y gastric bypass. *Surg Obes Relat Dis* 2005; 1: 481–485
44. Stauffer JQ. Hyperoxaluria and calcium oxalate nephrolithiasis after jejunoileal bypass. *Am J Clin Nutr* 1977; 30: 64–71
45. Clayman RV, Williams RD. Oxalate urolithiasis following jejunoileal bypass. *Surg Clin North Am* 1979; 59: 1071–1077
46. Encinosa WE, Bernard DM, Chen CC *et al*. Healthcare utilization and outcomes after bariatric surgery. *Med Care* 2006; 44: 706–712
47. Durrani O, Morrisroe S, Jackman S *et al*. Analysis of stone disease in morbidly obese patients undergoing gastric bypass surgery. *J Endourol* 2006; 20: 749–752
48. Miller NL. Modern bariatric surgery and nephrolithiasis–are we on the verge of a new epidemic? *J Urol* 2008; 179: 403–404
49. Semins MJ, Shore AD, Makary MA *et al*. The association of increasing body mass index and kidney stone disease. *J Urol* 2010; 183: 571–575
50. Taylor EN, Stampfer MJ, Curhan GC. Obesity, weight gain, and the risk of kidney stones. *JAMA* 2005; 293: 455–462
51. Annuk M, Backman U, Holmgren K *et al*. Urinary calculi and jejunoileal bypass operation. A long-term follow-up. *Scand J Urol Nephrol* 1998; 32: 177–180
52. Kleinman JG. Bariatric surgery, hyperoxaluria, and nephrolithiasis: a plea for close postoperative management of risk factors. *Kidney Int* 2007; 72: 8–10
53. Montagnac R, Schendel A, Vuiblet V *et al*. Chirurgie bariatrique, lithiase oxalo-calcique et insuffisance rénale par néphropathie oxalique [Bariatric surgery, calcium oxalate urinary stones and oxalate nephropathy]. *Nephrol Ther* 2011; 7: 38–45
54. Hassan I, Juncos LA, Milliner DS *et al*. Chronic renal failure secondary to oxalate nephropathy: a preventable complication after jejunoileal bypass. *Mayo Clin Proc* 2001; 76: 758–760
55. Moutzouris DA, Skaneli G, Margellos V *et al*. Oxalate nephropathy in a diabetic patient after gastric by-pass. *Clin Nephrol* 2011; 75: 16–19
56. Nasr SH, D'Agati VD, Said SM *et al*. Oxalate nephropathy complicating Roux-en-Y Gastric Bypass: an underrecognized

cause of irreversible renal failure. *Clin J Am Soc Nephrol* 2008; 3: 1676–1683
57. Duffey BG, Pedro RN, Makhlouf A *et al*. Roux-en-Y gastric bypass is associated with early increased risk factors for development of calcium oxalate nephrolithiasis. *J Am Coll Surg* 2008; 206: 1145–1153
58. Mittal RD, Kumar R. Gut-inhabiting bacterium Oxalobacter formigenes: role in calcium oxalate urolithiasis. *J Endourol* 2004; 18: 418–424
59. Mittal RD, Kumar R, Bid HK *et al*. Effect of antibiotics on Oxalobacter formigenes colonization of human gastrointestinal tract. *J Endourol* 2005; 19: 102–106
60. Lange JN, Wood KD, Wong H *et al*. Sensitivity of Human Strains of Oxalobacter formigenes to Commonly Prescribed Antibiotics. *Urology* 2012; 79: 1286–1289
61. Kharlamb V, Schelker J, Francois F *et al*. Oral antibiotic treatment of Helicobacter pylori leads to persistently reduced intestinal colonization rates with Oxalobacter formigenes. *J Endourol* 2011; 25: 1781–1785
62. Gnanandarajah JS, Abrahante JE, Lulich JP *et al*. Presence of Oxalobacter formigenes in the intestinal tract is associated with the absence of calcium oxalate urolith formation in dogs. *Urol Res* 2012; 40: 467–473
63. Hoppe B, Dittlich K, Fehrenbach H *et al*. Reduction of plasma oxalate levels by oral application of Oxalobacter formigenes in 2 patients with infantile oxalosis. *Am J Kidney Dis* 2011; 58: 453–455
64. Hoppe B, Groothoff JW, Hulton SA *et al*. Efficacy and safety of Oxalobacter formigenes to reduce urinary oxalate in primary hyperoxaluria. *Nephrol Dial Transplant* 2011; 26: 3609–3615
65. Jiang J, Knight J, Easter LH *et al*. Impact of dietary calcium and oxalate, and Oxalobacter formigenes colonization on urinary oxalate excretion. *J Urol* 2011; 186: 135–139
66. Lamarche J, Nair R, Peguero A *et al*. Vitamin C-induced oxalate nephropathy. *Int J Nephrol* 2011; 146927
67. Beloncle F, Sayegh J, Duveau A *et al*. An unexpected cause of progressive renal failure in a 66-year-old male after liver transplantation: secondary hyperoxaluria. *Int Urol Nephrol* 2012; doi:10.1007/s11255-012-0140-1
68. Dheda S, Swaminathan R, Musk M *et al*. Acute irreversible oxalate nephropathy in a lung transplant recipient treated successfully with a renal transplant. *Nephrology (Carlton)* 2012; 17: 12–15
69. Cartery C, Faguer S, Karras A *et al*. Oxalate nephropathy associated with chronic pancreatitis. *Clin J Am Soc Nephrol* 2011; 6: 1895–1902
70. Hueppelshaeuser R, von Unruh GE, Habbig S *et al*. Enteric hyperoxaluria, recurrent urolithiasis, and systemic oxalosis in patients with Crohn's disease. *Pediatr Nephrol* 2012; 27: 1103–1109

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