Ureteral obstruction following partial nephrectomy: can it be caused by fibrin glue?

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LYON TD, THEISEN KM, RYCYNA KJ, BENOIT RM. Ureteral obstruction following partial nephrectomy: can it be caused by fibrin glue? *Can J Urol* 2015;22(1):7674-7676.

Fibrin sealants are widely used during partial nephrectomy, however reports regarding the potential complications associated with their use are limited. We present the case of a 67-year-old male who developed delayed ureteral obstruction without hydronephrosis following partial nephrectomy in a solitary kidney. We hypothesize that the obstruction and absence of hydronephrosis were caused by extrinsic compression and subsequent inflammation due to the fibrin glue. Our report underscores the importance of a high index of suspicion for obstruction when acute kidney injury occurs following partial nephrectomy when fibrin glue is used, even in the absence of collecting system dilatation.

Key Words: partial nephrectomy, fibrin tissue adhesive, ureteral obstruction

Introduction

The widespread adoption of partial nephrectomy (PN) has brought new challenges in achieving intra-operative hemostasis and adequate watertight seal of the collecting system.[1] To overcome such challenges several new techniques and products have been employed to achieve hemostasis, specifically the use of various sealants and fibrin glues. Greater than 75% of urologists employ some form of hemostatic agent during partial nephrectomy.[2] To date limited data exist on the potential complications associated with these agents. We present a case of ureteral obstruction following partial nephrectomy in a solitary kidney that may have been due to the use of fibrin glue. Fibrin glue also likely prevented the collecting system from dilating which obscured the true etiology of the patient's acute kidney injury (AKI).

Accepted for publication October 2014

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Case report

A 67-year-old male with a prior history of right nephrectomy for renal cell carcinoma was referred to us after a surveillance CT scan revealed a 1.8 cm left lower pole lesion. After an extensive review of the risks and complications associated with partial nephrectomy in a solitary kidney, the patient and his family strongly desired he undergo a left partial nephrectomy.

The patient underwent an open left partial nephrectomy. The left renal artery and vein were clamped after the administration of mannitol, and the kidney was iced for 10 minutes. Retroperitoneal fat was placed in the defect and a renorrhaphy was performed using 3-0 pledgeted sutures. Prior to unclamping, Tisseel (Baxter Healthcare Corporation, Westlake Village, CA USA) fibrin glue was applied

to the defect and allowed to harden. Mannitol was infused after unclamping the renal vessels.

The kidney produced urine immediately when the artery was unclamped. Urine output remained excellent through postoperative day 3, however, the serum creatinine rose steadily and was 2.7 mg/dL by postoperative day 2. On postoperative day 3, the patient's serum creatinine increased to 6.1 mg/dL and the patient became anuric. A drain placed at the time of surgery had minimal drainage. The indwelling Foley catheter was irrigated to confirm position and patency. A CT scan and renal ultrasound did not demonstrate hydronephrosis or a perinephric collection. Renal Doppler ultrasound demonstrated preserved flow to the kidney. Given the lack of clear etiology for his renal failure and with the thought that perhaps the Tisseel had encased the renal pelvis preventing dilatation of the renal pelvis and calyces, a ureteral stent was placed. A retrograde pyelogram demonstrated no hydroureter, hydronephrosis, or filling defects. Urine output returned immediately after stent placement, and his creatinine decreased to 1.83 mg/dL 36 hours after stent placement.

Discussion

We report a case of anuria and renal failure after partial nephrectomy with use of fibrin glue. The differential diagnosis of anuria following PN in a solitary kidney includes urinary obstruction, urine leak, renovascular injury, and medical renal disease. Despite renal ultrasound having a reported 98% sensitivity for diagnosing obstruction, false negatives can occur in cases when patients are hypovolemic, are early in the course of obstruction, or have developed a process causing encasement of the ureter and/or renal pelvis.[3,4]

We believe the most likely cause of anuria in this patient is a delayed development of ureteral obstruction. Additionally, the use of fibrin glue may also have played a causative role in forming a compressive barrier around the renal pelvis and proximal ureter that prevented the development of subsequent hydronephrosis and hydroureter. While urinary retention could have caused this presentation, it does not explain our patient's presentation as he had a functioning Foley catheter throughout his hospitalization. A renal Doppler demonstrated preserved renal blood flow ruling out the possibility of renal infarction. A CT scan obtained after the onset of anuria failed to reveal a perinephric collection, and a retroperitoneal drain continued to have minimal output as his renal failure progressed, arguing against a urine leak as the cause for his anuria. Acute tubular necrosis (ATN) could also possibly explain this patient's clinical course, however brisk flow through the stent was noted intraoperatively and urine output returned immediately after stent placement which suggests obstruction as the etiology of the patient's acute kidney injury. Although theoretically possible, resumption of urine output following ATN is unlikely to have occurred at the same exact moment in time a ureteral stent was placed.

Fibrin glue has been shown to initiate a dense inflammatory reaction that can cause obstruction should it come into contact with the ureter.[5,6] The fibrin seal may also be compressive enough to prevent renal pelvic dilation or hydronephrosis, thereby eliminating radiographic evidence of obstruction. When fibrin glue is applied to a tissue, it causes local proliferation of fibroblasts and formation of granulation tissue within hours of clot polymerization.[7] Naitoh and colleagues used fibrin glue in a porcine partial nephrectomy model and found that after 1 month there were adhesions to surrounding organs and residual scar tissue when compared to a bioglue.[8] Several other animal studies of partial nephrectomy report evidence for chronic fibrous scar tissue 4-6 weeks postoperatively at sites where fibrin glue was applied.[9,10] A porcine model evaluating ureteral anastomoses using fibrin glue as an adjunct showed that at the ureteral anastomoses, the serosa had pronounced inflammation with heavy inflammatory infiltrate; there was one case of proximal ureteral obstruction as a result.[6]

To date there is only one report of a case of ureteral obstruction and urinary fistula that developed in a patient after fibrin glue was used as a sealant during partial nephrectomy.[5] Similar to our patient, their patient had a CT scan that showed no hydronephrosis. After failed retrograde stent placement, the patient underwent surgical resection of a stiff proximal ureter enfolded in fibrin glue followed by ureteroureterostomy. Histopathologic exam of the excised ureteral segment revealed fibrous scar tissue and inflammatory cell infiltration as the cause of obstruction. As a result, the authors cautioned against spillage of fibrin glue onto the ureter during partial nephrectomy. In our patient, we hypothesize fibrin glue prevented peristalsis causing obstruction.

Our experience demonstrates that when AKI occurs after partial nephrectomy when tissue sealants are utilized, obstruction should be considered even if imaging studies do not demonstrate hydronephrosis as the use of these agents may prevent dilatation of the collecting system. Clinicians should maintain a high index of suspicion in order to diagnose obstruction following PN when fibrin glue is used, especially in patients with a normal contralateral kidney who will not develop anuria. □

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