The Evaluation of Ureteral Stent Complications: Our Clinical Experience and Review of the Literature

Ureteral Stent Komplikasyonlarının Değerlendirilmesi: Klinik Deneyimimiz ve Literatürün Gözden Geçirilmesi

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Özet

Giriş: Bu çalışmanın amacı üreteral stent komplikasyonlarının değerlendirilmesidir.
Hastalar ve Yöntem: Şubat 1995 ve Aralık 2006 tarihleri arasında 146 hastada 162 üreteral stent yerleştirildi. Hastalarda 4.8F üreteral stent kullanıldı.
Bulgular: Hastaların ortalama yaşı 42.7±6.1 (21-76 yıl) yıldı. Toplam komplikasyon oranı %56.16 (n=82) idi. İlk 4 haftada görülen komplikasyonlar izole irritatif mesane semptomları (%14.38), izole hematüri (%2.05), bakteriüri ve ateş (%4.79), kombine semptomlar ve izole yan ağrısı (%7.53) iken geç semptomlar hidronefroz (%1.36), emkrustasyon (%6.16) ve fragmantasyondu (%2.05). İki olgu fragmente stent nedeniyle açık operasyon geçirdi.
Sonuç: Morbidite ve komplikasyonların erken dönemde saptanması için stentli hastalarda yakın takip çok önemlidir.
Anahtar Kelimeler: Üreter, stent, komplikasyon, tedavi.
Introduction: The aim of this study is to evaluate the complications of ureteral stents.
Patients and Methods: Between February 1995 and December 2006 a total of 162 ureteral stent were inserted in 146 patients. In patients 4.8 F pigtail ureteral stent was used.
Results: The mean age of the patients was 42.7±6.1 (range 21-76) years. Total complication rate was detected as 56.16% (n=82). Early complications during the first 4 weeks after stent insertion were isolated irritative bladder symptoms (14.38%), isolated hematuria (2.05%), bacteriuria and fever (4.79%), combined symptoms, and isolated flank pain (7.53%); late complications included hydronephrosis (1.36%), encrustation (6.16%), and fragmentation (2.05%). Two patients underwent open surgical procedure for fragmanteted stents.
Conclusions: Close follow-up of stented patients is very important in early detection of morbidity or complications.

Key Words: Ureter, stent, complication, treatment

Introduction

Ureteral stents are placed to prevent or relieve ureteral obstruction due to an intrinsic or extrinsic etiology, including ureteral calculi, ureteral stricture, congenital anomalies (Ureteropelvic junction obstruction), retroperitoneal tumor or fibrosis (1). Stents are also commonly placed before open surgical or laparoscopic procedures to help identify the ureters and prevent inadvertent ureteral injury (1,2). Due to the widely spread usage of indwelling ureteral catheters, the number of possible complications such as migration, infection, pyelonephritis, breakage, encrustation, stone formation, and fragmentation have been noted (3,4). These problems may necessitate surgical intervention and can lead to significant morbidity. The aim of this study is to evaluate the clinical complications of double-J ureteral stents with the relevant the literature.

Material and Methods

Between February 1995 and December 2006 ureteral stents were inserted in 146 renal units in 162 patients for different endications such as ureteral obstruction and after ureterorenoscopic interventions. The study group consisted of 83 male and 63 female patients. All patients were evaluated with physical examination, routine hematologic and biochemical analysis, urinary ultrasonography, and plain abdominal graphy. The left side was affected in 79 patients, the right in 67, and both ureters in 8 patients. The ureteroscopy for stone removal and extracorporeal shoch wave lithotripsy (ESWL) were the commenest indication for initial stent insertion. The other reasons for stent insertion were extrinsic malign ureteral obstruction, retroperitoneal fibrosis and ureteroureterostomy in 2 patients. The complicated stents were seen in 56.16% (n=82) cases. In all patients, a polyurethane double pigtail ureteral cathater was used. The catheters were placed with a rigid cystoscope under fluoroscopic guidance and a plain abdominal radiograph was obtained afterwards to verify proper placement of the cathater. The complicated stents were managed by a combination of endourological and some auxillary techniques. They were removed by rigid cystoscopy with viscous lidocaine. A 4.8F, 26 cm double pigtail ureteral stent was used in all patients. Prior to stent removal, a urine sample was taken from each patient for culture.

Results

The mean time of insertion was 55 days. The mean age of the patients was 42.7±6.1 (range 21-76) years. Total complication rate was detected as 56.16% (n=82). Early complications during the first 4 weeks after stent insertion were isolated irritative bladder symptoms (14.38%, n=21), isolated hematuria (2.05%, n=3), bacteriuria and fever (4.79%, n=7), and isolated flank pain (7.53%, n=11); late complications included hydronephrosis (1.36%, n=2), stent migration (6.16%, n=9), encrustation (6.16%, n=9) and fragmentation and flank pain (2.05%, n=2), combined complications (irritative bladder symptoms+hematuria+encrustation+flank pain) (12.32%, n=18). Two patients underwent open surgical procedure for fragmanteted stents.

Discussion

Ureteral stents are routinely used in urology practice as a simple, safe, and cost-effective way to re-establish or to improve drainage from kidney to bladder without external diversion (1-4). Patients with stent-related problems are common in urological practice. The ureteral stents present with a wide range of urological symptoms such as sepsis, urinary tract infection, flank pain, irrtative bladder symptoms, and loss of renal function (3,4). Early complications related to double J stents are pain, vesical irritative symptoms and fever. Irritative voiding symptoms, including frequency, urgency and dysuria, as well as flank pain, suprapubic pain and hematuria are commonly described, occurring in up to 85-90% of stented patients in contemporary studies (3,5). In a study, Ringel et al. prospectively examined 110 stented renal units in 90 patients and outlined the morbidity and complications of indwelling ureteral stents (6). In that study 103 patients (94%) had stent-related complications such as infection and flank pain on voiding. In the present study, the irritative voiding symptoms were the commonest presentation. Lower urinary tract symptoms (dysuria and frequency) are theorized to be a result of mucosal irritation of nerves located in the submucosa concentrated in the bladder trigone whereas the upper urinary tract symptoms (flank pain) are thought to be secondary to vesico-uretric reflux, especially during micturition, of urine from the bladder into the kidney generating pressure (5). Of the morbidity associated with ureteric stents, flank discomfort or pain during voiding has been reported in 35% of patients with double pigtail stents. Currently, a variety of medical treatments are available to relieve irritative bladder symptoms including a variety of analgesics and anticholinergics, which may not specifically target bladder symptoms, but rather provide global symptom relief (1,3,5).

Late complications are much more troublesome like fragmentation of the stent, calculus formation of the stent, migration, and infection. Ureteral stent fragmentation is rarely seen. El Faqih et al. reported stent fragmentation in 0.3% of their series (7). This ratio was found as 10% in study of Monga et al (8). Various different mechanisms have been proposed to explain the ureteral stent fragmentation. Breakage of a stent has been attributed to the hostility of the urine solution. Interaction with urine and extensive inflammatory reaction in situ may be important in the initiation and promotion of degradation. According to Ilker et al, it is quite common to find abundant numbers of leukocytes in urine with or without any infection, which may derive at least in part from depolymerization of biomaterials by release of lysosomal enzymes (9). Furthermore, degradation of stent polymers, and hardening of polyethylene and polyurethane can lead to fragmentation if the stents are left indwelling longer than 6 months. Mardis et al suggested that fragmentation occurs at a site previously allowed to kink during stent insertion (10). Thus, kinking during stent insertion must be avoided. In study of Zisman et al all breakage lines passed through the side holes, suggesting that this area is a weak point conducive to kinking that may predispose to fragmentation (11). For this reason, Zisman et al suggest that this location is prone to fragmentation. Another factor, which is connected with stent fragmentation, is stent composition.

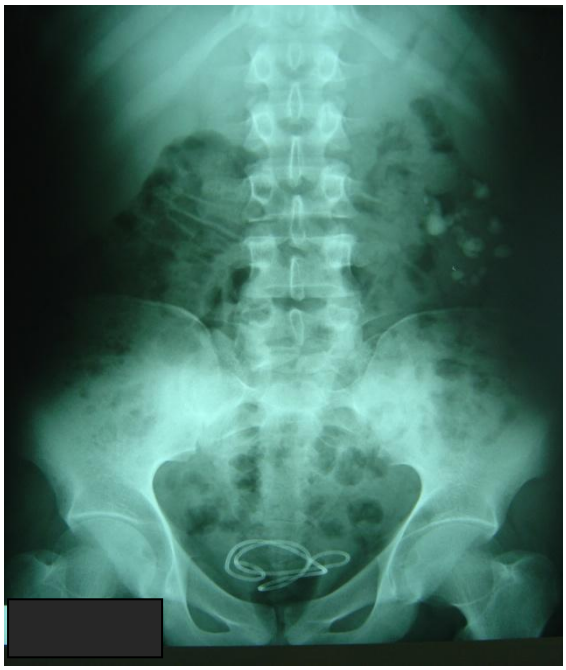
Silicone stents may be advantageous due to a lower risk of calcification and prolonged maintenance of tensile strength of up to 20 months. In our patients, we used polyurethane stent. The polyurethane stents are 4 times less likely to break than silicone stents but microscopic irregularities can predispose them to incrustation (10). There is no consensus about the ideal material for ureteral stent. Besides, some stent fragmentations occurs early period after the insertion of the stent. Some authors suggest that an accelerated aging process is an important factor leading to early mechanical failure of ureteral stents (11). In the study by Zisman et al, mechanical testing and fractography clearly showed that the stent material changed from ductile to brittle during exposure to a specific environment (11). Richter et al. (12) agree with Zisman et al (11), that accelerated aging of the stent material is an important factor leading to early mechanical failure. According to Richter et al, only this could explain why seven of their 11 fragmented stents broke only 3 months after insertion; six of these seven broken stents were made of polyurethane and one was of silicone (11). In our one patient, fragmentation was seen at third month after insertion of ureteral stent.

Stent-related infection is a significant problem in patients with indwelling stents. This can lead to an increase in bothersome symptoms. The correlation between stent colonization and the rate of a clinical urinary tract infection has not been established. Adherent bacteria in 90% of cases colonize indwelling ureteric stents. However, this translates to clinical urinary infection in 27% of patients with longer stent placement time increasing the likelihood of infection (13,14). Biofilm development can lead to urinary tract infection and subsequent sepsis (15). Newly developed biomaterials are aimed at the prevention of biofilms, thus reducing stent related morbidity. Paick et al. discovered that bacterial colonies were found on the surface of 44% of ureteral stents consisting mainly of Enterococcus species and Escherichia coli (16). Kehinde et al. followed 250 consecutive patients who received stents and determined that the risk factors for infection included female sex, diabetes, chronic renal failure, and indwelling time greater than 90 days (17). Kehinde et al. established that about 17% of patients with indwelling J ureteral stents develop significant bacteriuria and about 42% of patients have their stents colonized with time by bacteria and yeasts (17). A negative urine culture does not rule out a colonized stent, and the types of pathogens in the urine are not exactly the same as those that are adherent to the stent. Urinary tract infection is cause of considerable morbidity in patients with indwelling J ureteral stents. Antibiotic prophylaxis is ineffective in preventing stent colonization and hence is nor recommendded by some authors, while others, support its administration (18). The majority of the episodes of urinary tract infection resolve without antimicrobial treatment, just as do those associated with indwelling urethral catheters. Occassionally, however, antimicrobial treatment is required, especially when the infection is complicated by catheter-associated sepsis (16,17).

The proximal and distal migration of a double pigtail ureteral stent is rare, occurring in only 0.6% to 8.2% of the cases (1,5,14) (Figure 1). El Faqih et al.

reported stent migration in 3.7% of their series (9). The incidence was 6.16% in our series. Several theories have been proposed to account for this phenomenon. It is believed that the longer the indwelling time, the more likely a stent will migrate. A double-pigtail catheter as opposed to one with a J-ending is less likely to migrate (19-21).

Figure 1. Distal migration of ureteral stent



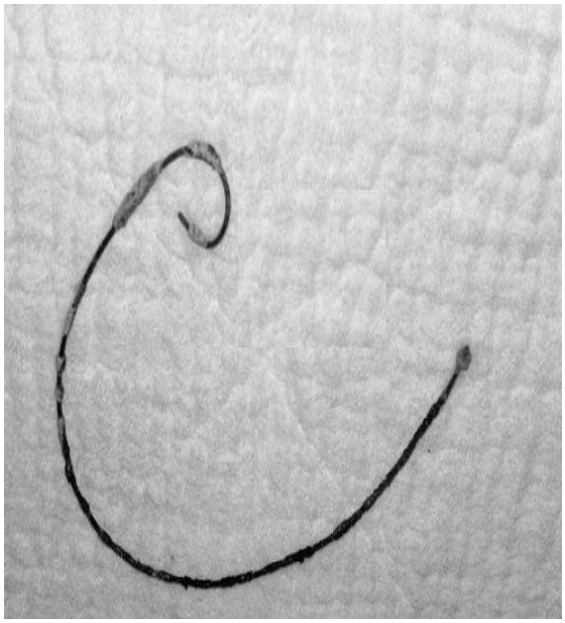
It may also be that the migration occurs when the stent length is shorter than the ureteral length. Stent movement may coincide with kidney movement during respiration. Although silicone stents have a lower risk of calcification, their smooth regular surface makes them susceptible to migration. In present study, none of patients had silicone stents. Slaton and Kropp investigated the factors that might influance stent migration (22). In their series the indwelling time of the stent was not found to be an important factor for migration. A shorter than ideal lenght, inadequate distal curl and a proximal curl placed into the upper calyx instead of renal pelvis were reported to increase the risk of upwards migration of an ureteral stent (18). Another potential factor that might influence migration is the location of the proximal curl in the renal collecting system, that is whether the curl is in the renal pelvis or an upper calix (18,23). Caudal migration can typically be treated by sinmple cystoscopic retrieval with patient under local anesthesia. Rarely, ureteric meatotomy are used to extract a stent. However, proximal migration may require one or more procedures using anesthesia. Various techniques have been described to retrieve proximally migrated stents. Bagley and Huffman removed 16 stents ureteroscopically (24). Many investigators have used cystoscopy with the patient under local anesthesia and floroscopically controlled pasage of a stone basket, Fogarthy catheter, ureteral dilating balloon or flexible forceps in a retrograde manner up the ureter to remove the migrated stent. LeRoy et al performed percutaneous nephrostomy to remove 12 migrated stents (25). In our series we used all 3 techniques to retrieve the migrated double j stents.

Many investigators have found a high occlusion rate in the subset of stented patients with obstruction secondary to extrinsic ureteral compression. It has been well documented that stent failure in extrinsic compression occurs in approximately 44% to 58% of cases (10,26,27). In a study authors report a 40.6% primary stent failure rate in 101 patients with extrinsic compression after 11 months of follow-up (27). Ureteral obstruction from malignant ureteral strictures often persists despite ureteral stent placement because of extrinsic compression from pelvic malignancy. The exact etiology of this stent failure is not

completely understood. Urine is thought to flow preferentially around rather than through the stent but with extrinsic compression of the ureter urine flow through the stent lumen becomes predominant. In this scenario ureteral obstruction occurs when the stent lumen is compressed or occluded by mucus and debris (28). That the combination of extrinsic compression against the stent and aperistaltic ureteral segments may impair drainage of urine around wellplaced stents is another proposed idea. Some urologists advocate the use of larger bore, stiffer stents for ureteral obstruction from extrinsic compression. However, Hubner et al. have shown that the luminal diameter is not an important factor in physiological urine flow rates (27). We immediately used a single ureter in our patient two times. But these stents failed within 3–4 days. The placement of two ureteral stents within the same collecting system in patients in whom single ureteral stents fail has been reported with good results. Liu and Hrebinko (29) reported 4 patients with non-urinary tract malignancies for whom simple stenting for ureteral obstruction had failed. Parallel 4.8-F DJ stents were placed simultaneously and changed every 3 months, with no patient requiring percutaneous nephrostomy, to a mean follow-up of 5.8 months. The increased stiffness of 2 stents reduces kinking and luminal compression from extrinsic force, preserves some extra luminal flow between the stents and obviates percutaneous nephrostomy tubes in select patients. Placement of 2 ipsilateral parallel ureteral stents simultaneously is an easy technique. It may obviate percutaneous nephrostomy tube placement in patients in whom drainage with a single stent failed, especially in cases with extrinsic ureteral compression.

Severe encrustation (Figure 2), incrustation and stone formation in indwelling ureteral stents remains a distressing problem that can lead to severe morbidity and life-threatening urosepsis if not followed up and managed carefully (1,5). The incidence of encrustation increases with the duration that the stent remains indwelling. El-Faqih et al evaluated 299 stents in patients with calculi and noted an encrustation rate of 9.2% before 6, 47.5% from 6 to 12 and 76.3% after 12 weeks (9).

Figure 2. Stent encrustation



The etiology of encrustation is not completely clear and it may be due to multifactorial causes. The probable risk factors include poor compliance, long indwelling times, sepsis, alkaline urine, chemotheraphy, pregnancy, urinary tract infection, chronic renal failure, recurrent or residual stones, lithogenic history, metabolic abnormalities, congenital renal anomalies, and malignant ureteral

obstruction on chemotherapy with hyperuricosuria (30). Patients with malignant ureteral obstruction, as seen in one of our patients, may also be at an increased risk of stent encrustation. We believe that this may be an outcome of stasis induced by dehydration and poor intake, chemotherapy-induced hyperuricosuria associated with persistent or recurrent urinary tract infection, multiple serial stents, and poor compliance. The stent material certainly appears to have an impact on the risk of encrustation. Polyethylene is no longer used, because it is brittle and at risk of fragmentation. Silicone is inert and relatively resistant to encrustation. There are conflicting reports regarding the hydrophilic coating and the risk of encrustation. Cormio et al. reported that in a pig model, hydrophilic surfaces were less likely to encrust (31). Conversely, Desgrandchamps et al found that hydrophilic stents may carry an increased risk of encrustation (32). More recently, Choong et al validated an in vitro model using human urine and tested commonly used stents (33). Their results suggested that hydrogel coatings significantly increase the risk of encrustation compared with the same stent without the hydrogel coating or in a silicone control. It has also been well documented that stent indwelling times are strongly and directly related to the incidence of stent encrustation and obstruction. Authors suggest that stents should be changed at least within 4 months and optimally every 2 months (1,5,26). Repeated stenting should be avoided in those with significant risk factors. Encrustation deposition on synthetic materials with or without infection certainly occurs, despite the advances in stent materials, due to biofilm formation by bacteria. The presence of biofilm on a stent, particularly with urease-producing organisms, leads to hydrolysis of urea and an elevation of urinary pH in which magnesium ammonium phosphate and calcium hydroxyapatite precipitate. Scanning electromicroscopy has confirmed bacteriel presence within biofilms of encrusted catheters (30). Few reports have documented their findings of stent encrustation analysis. Biochemical and optical analyses of stent encrustations by Robert et al. revealed that these encrustations consisted mainly of calcium oxalate, calcium phosphate, and ammonium magnesium phosphate (34). They also demonstrated that calcium oxalate was the main crystalline phase, especially in the absence of ureteral stent encrustations. This difference may have been due to underlying urinary tract infection or asymptomatic bacteruria that develops more often during pregnancy. In our series, the predominant causative factor was lithogenic history. Page electrophoretic analysis of the adsorbed organic biofilms coating these stents has shown the presence of albumin, Tamm-Horsfall protein, and $alpha_1$-microglobulin. In vitro studies have confirmed that the organic biofilm layer coating the retrieved nonencrusted stents can remarkably enhance crystal precipitation and aggregation events on the surface (32). In the absence of urinary tract infection Robert et al showed that calcium oxalate is the major component of stent encrustation with calcium ammonium phosphate and calcium phosphate also present in smaller quantities (34,35). Recent studies with heparin-coated stents have shown them to reduce the incidence of encrustation and secondary calculi formation by decreasing bacterial absorption (36).

Stents that migrate, fragment, or are forgotten or encrusted may necessitate a multimodal therapeutic endourologic approach that can be performed at single or multiple sessions to render the patient stent-free. Lam and Gupta described 26 retained stents that were treated in an average of 2.7 endourologic procedures (37). There are several ways to remove a forgotten stent depending on the condition of encrustration and fragmentation. These approaches include extracorporeal shock wave lithotripsy, percutaneous nephrolithotomy and/or ureteroscopy (38). Where encrustation is minimal, correct management is by attempted removal under general anesthesia, followed by ureteroscopic intervention if the stent cannot be removed easily. Open surgery is another approach in case of severe encrustation (39). Percutaneous nephrostolithotomy and ureteroscopy are often necessary for treating a severely encrusted stent and the associated stone burden. Complications of catheter tip calcification in the renal pelvis can be treated with percutaneous management following cystolitholapaxy if the bladder tip is also involved. ESWL for treatment of stent encrustations was reported by Flan et al. in 1990 (40). They reported good results with a total of 400 shocks. They did not describe the depth of the encrustataion, however Schulze et al. described treatment of stent encrustations using Suby's solution through a nephrostomy tube (41). More recently, Singh et al. reported 15 massively encrusted stents removed successfully with a combination of treatments, including three open removals and five percutaneous procedures (19). All attempts to remove impacted stents must be under fluoroscopic control. Ureteroscopy can then be repeated as required. Using this approach to treatment, 42 (85.7%) of our 49 stents were successfully treated with an average number of procedures of 1.86 per patient. Open surgery is rarely necessary and should be reserved for resistant cases. We observed a high failure rate with in situ ESWL for severely encrusted stents; this could have been due to a large stone burden and a preponderance of radiodense hard calcium phosphate and monohydrate stones. Open surgical removal was resorted to in 3 cases. Generally, transurethrally intervention is enough for the removal of the bladder stents. Various methods such as ureterorenoscopy, percutaneous procedure have been described for the removal of the fragmented stent in renal pelvis. Nevertheless, even an open operation was reportedly required in a similar case. LeRoy et al treated 3 patients with fractured polyethylene-polyurethane stents, through a percutaneous approach (25). They recommend this approach over the retrograde approach for patients with ureteral strictures, or significant fragmented or calcified stent remnants in the renal pelvis. Rembrink et al removed a fragmented stent in pelvis renalis via open procedure (42). In our case, the catheter in the bladder was removed endoscopically under local anesthesia. We removed the stone and the broken piece of stent with an open procedure.

References

1. Lam JS, Gupta M. Update on ureteral stents. Urol. 2004;64:9-15.
2. Chew BH, Knudsen BE, Denstedt JD. The use of stents in contemporary urology. Curr Opin Urol. 2004;14:111-5.

3. Auge BK, Preminger GM. Ureteral stents and their use in endourology. Curr Opin Urol. 2002;12:217-22.
4. R. Damiano, A. Oliva, C. Esposito, M. De Sio, R. Autorino and M. D'Armiento, Early and late complications of double pigtail ureteral stent, Urol Int. 2002;69:136–40.
5. Dyer RB, Chen MY, Zagoria RJ, Regan JD, Hood CG, Kavanagh PV. Complications of ureteral stent placement. Radiographics. 2002;22:1005-22.
6. Ringel A, Richter S, Shalev M, Nissenkorn I. Late complications of ureteral stents. Eur Urol. 2000;38:41-4.
7. el-Faqih SR, Shamsuddin AB, Chakrabarti A, Atassi R, Kardar AH, Osman MK, Husain I. Polyurethane internal ureteral stents in treatment of stone patients: morbidity related to indwelling times. J Urol. 1991;146:1487-91.
8. Monga M, Klein E, Castaneda-Zuniga WR, Thomas R. The forgotten indwelling ureteral stent: a urological dilemma. J Urol.1995;153:1817–9.
9. Ilker Y, Türkeri L, Dillioğlugil O, Akdaş A. Spontaneous fracture of indwelling ureteral stents in patients treated with extracorporeal shock wave lithotripsy: two case reports. Int Urol Nephrol. 1996;28:15-9.
10. Mardis HK, Kroeger RM, Morton JJ, Donovan JM. Comparative evaluation of materials used for internal ureteral stents. J Endourol. 1993 ;7:105-15.
11. Zisman A, Siegel YI, Siegmann A, Lindner A. Spontaneous ureteral stent fragmentation. J Urol. 1995;153:718-21
12. Richter F, Irwin RJ, Watson RA, Lang EK. Endourologic management of malignant ureteral strictures. J Endourol. 2000;14:583-7.
13. Franco G, De Dominicis C, Dal Forno S, Iori F, Laurenti C. The incidence of post-operative urinary tract infection in patients with ureteric stents. Br J Urol. 1990;65:10.
14. Farsi HMA, Mosli HA, Al-Zemaity MF, Bahnassy AA, Alvarez M. Bacteriuria and colonization of double-pigtail ureteral stents: longtermexperience with 237 patients. J Endourol. 1995;9:469–74.
15. Reid G, Denstedt JD, Kang YS, Lam D, Nause C. Bacterial adhesion and biofilm formation on ureteral stents in vitro and in vivo. J Urol. 1992;148:1592–4.
16. Paick SH, Park HK, Oh SJ, Kim HH. Characteristics of bacterial colonization and urinary tract infection after indwelling of double-J ureteral stent. Urology. 2003;62(2):214-7.
17. Kehinde EO, Rotimi VO, Al-Hunayan A, Abdul-Halim H, Boland F, Al-Awadi KA. Bacteriology of urinary tract infection associated with indwelling J ureteral stents. J Endourol. 2004 ;18:891-6.
18. Kawahara T, Ito H, Terao H, Yoshida M, Ogawa T, Uemura H, Kubota Y, Matsuzaki J. Choosing an appropriate length of loop type ureteral stent using direct ureteral length measurement. Urol Int. 2012;88:48-53.
19. Singh I, Gupta NP, Hemal AK, Aron M, Seth A, Dogra PN. Severely encrusted polyurethane ureteral stents: management and analysis of potential risk factors. Urology. 2001;58:526.
20. K. Mohan-Pilllai, F.X. Keeley, Jr, S.A. Moosa. Endourological management of

severely encrusted ureteral stents. J Endourol. 1999;13:377–9.
21. M. Kumar, M. Aron, A.K. Aggarwal. Stenturia: an unusual manifestation of spontaneous ureteral stent fragmentation. Urol Int. 1999;62:114–6.
22. Slaton JW, Kropp KA. Proximal ureteral stent migration: an avoidable complication? J Urol. 1996;155:58-61.
23. Kilciler M, Erdemir F, Bedir S, Coban H, Erten K, Guven O, Topac H. Spontaneous ureteral stent fragmentation: a case report and review of the literature. Kaohsiung J Med Sci. 2006;22:363-6.
24. Bagley DH, Huffman JL. Ureteroscopic retrieval of proximally located ureteral stents. Urology. 1991;37:446-8.
25. LeRoy AJ, Williams HJ Jr, Segura JW, Patterson DE, Benson RC Jr. Indwelling ureteral stents: percutaneous management of complications. Radiology. 1986;158:219-22.
26. Chung SY, Stein RJ, Landsittel D, Davies BJ, Cuellar DC, Hrebinko RL, Tarin T, Averch TD. 15-year experience with the management of extrinsic ureteral obstruction with indwelling ureteral stents. J Urol. 2004;172:592-5.
27. Hübner WA, Plas EG, Stoller ML. The double-J ureteral stent: in vivo and in vitro flow studies. J Urol. 1992;148:278-80.
28. Rotariu P, Yohannes P, Alexianu M, Rosner D, Lee BR, Lucan M, Smith AD. Management of malignant extrinsic compression of the ureter by simultaneous placement of two ipsilateral ureteral stents. J Endourol. 2001;15:979-83.
29. Liu JS, Hrebinko RL. The use of 2 ipsilateral ureteral stents for relief of ureteral obstruction from extrinsic compression. J Urol. 1998;159:179-81.
30. Sıngh I, Gupta NP, Hemal AK, Aron M, Seth A, Dogra PN. Severely encrusted polyurethane ureteral stents: management and analysis of potential risk factors. Urol. 2001;58:526-31.
31. Cormio L, Talja M, Koivusalo A, Mäkisalo H, Wolff H, Ruutu M. Biocompatibility of various indwelling double-J stents. J Urol. 1995;153:494-6.
32. Desgrandchamps F, Moulinier F, Daudon M, Teillac P, Le Duc A. An in vitro comparison of urease-induced encrustation of JJ stents in human urine. Br J Urol. 1997;79:24-7.
33. Choong S, Whitfield H. Biofilms and their role in infections in urology. BJU Int. 2000 ;86:935-41.
34. Robert M, Boularan AM, El Sandid M, Grasset D. Double-J ureteric stent encrustations: clinical study on crystal formation on polyurethane stents. Urol Int. 1997;58:100-4.
35. H. Hedelin, C.G. Bratt, G. Eckerdal and K. Lincoln, Relationship between urease producing bacteria, urinary pH and encrustation on indwelling urinary catheters. British J Urol. 1991;67:527–31.
36. Cauda F, Cauda V, Fiori C, Onida B, Garrone E. Heparin coating on ureteral Double J stents prevents encrustations: an in vivo case study. J Endourol. 2008;22:465-72.
37. Lam JS, Gupta M. Tips and tricks for the management of retained ureteral stents. J Endourol. 2002;16:733-41
38. Mohan-Pillai K, Keeley FXJr, Moussa SA, Smith G, Tolley DA. Endourological management of

severely encrustedureteral stents. J Endourol. 1999;13:377.
39. Murthy KV, Reddy SJ, Prasad DV. Endourological management of forgotten encrusted ureteral stents. Int Braz J Urol. 2010;36:420-9.
40. Flam TA, Brochad M and M. Zerbib, ESWL to remove calcified ureteral stents. Urology. 1990;36:164–6.
41. Schulze KA, Wettlaufer JN, Oldani G. Encrustation and stone formation: complication of indwelling ureteral stents. Urology. 1985;25:616-9.
42. Singh I, Gupta NP, Hemal AK, Aron M, Seth A, Dogra PN. Severely encrusted polyurethane ureteral stents: management and analysis of potential risk factors. Urology. 2001;58:526-31.
43. Rembrink K, Goepel M, Meyer-Schwickerath M. The forgotten double J stent. Case report of a multifractured ureter stent. Urol Int. 1992;49:119-20.