



Polypoid Cystitis Unrelated To Indwelling Catheters: A Report Of Eight Patients

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Purpose: To evaluate the patients with polypoid cystitis, who did not have a catheterisation history and to review the related literature.

Materials and Methods: A retrospective analysis of the records of Turgut Özal Medical Center revealed 8 patients with aforementioned qualification.

Results: Two female and 6 male patients were evaluated. Mean age was 48 years (28 to 70). The urinary cytology findings available in 4 were normal. No bacterial growth was established in none of the patients. All cases were diagnosed incidentally by radiologic and cystoscopic examinations in the evaluation of different conditions, such as hematuria, ovarian abscess, bladder carcinoma, erectile dysfunction, neurogenic bladder, benign prostate hyperplasia and unexplained dysuria. Patients were followed for 6 months to 2 years after first diagnosis. No recurrence has been established during followup.

Conclusions: Polypoid cystitis is a benign lesion and should be considered in the differential diagnosis of transitional cell carcinoma of the bladder.

Key words: Bladder, Polypoid cystitis, Papillary cystitis, Bullous cystitis.

Kalıcı Kateterlere Bağlı Olmayan Polipoid Sistit: Sekiz Olgunun Sunumu

Amaç: Kateterizasyon öyküsü olmayan polipoid sistitli hastaları değerlendirmek ve ilgili literatür ışığında tartışmak amaçlandı.

Materyal ve Metot: Turgut Özal Tıp Merkezi'nin kayıtlarının retrospektif analizi ile koşulları uygun 8 hasta belirlendi.

Bulgular: İki kadın ve 6 erkek hasta değerlendirildi. Ortalama yaş 48 idi (28-70). Dördünde mevcut olan üriner sitoloji bulguları normaldi. Hiçbir hastada bakteriyel üreme tespit edilmedi. Bütün olgular hematüri, over absesi, mesane karsinomu, erektil disfonksiyon, nörojenik mesane, benin prostat hiperplazisi ve açıklanamayan dizüri gibi farklı durumların değerlendirilmesinde radyolojik ve sistoskopik incelemelerle tesadüfen teşhis edildi. Hastalar ilk tanıdan sonra 6 aydan 2 yıla kadar izlendi. İzlem süresince hiç rekürrens tespit edilmedi.

Sonuçlar: Polipoid sistit benin bir lezyondur ve mesanenin transizyonel hücre karsinomunun ayırıcı tanısında düşünülmelidir.

Anahtar kelimeler: Mesane, Polipoid sistit, Papiller sistit, Bullöz sistit.

Polypoid cystitis is a reversible, exophytic inflammatory lesion of the bladder mucosa and is characterized histologically by normal or mildly hyperplastic urothelium overlying a congested, chronically inflamed and markedly edematous stroma.¹ The polypoid, papillary and bullous cystitis are essentially identical in their inflammatory reactive patterns. They are different on account of the gross morphologic characteristics of exophytic lesions. Histologically the basic difference between them is the relative amount of stromal edema within the lamina propria that varies in intensity to produce this morphologic spectrum. Minimal stromal edema is associated with a filiform papillary configuration. Progressively greater amounts of stromal edema result in polypoid excrescences, or at the extreme end of the spectrum, broad-based mucosal excrescences, wider than they are tall, described as bullous cystitis.²

Polypoid cystitis is recognized frequently in patients with indwelling catheters and is seen mostly on the dome and posterior wall of the bladder which corresponds to the localization of the tip of the catheter. It may be difficult to distinguish it from transitional cell carcinoma macroscopically at cystoscopy because of exophytic nature of the lesion, especially in patients with no history of a catheter. But they can be easily distinguished histologically as an inflammatory pseudopolyp.¹ There are several reports of polypoid cystitis in patients who are unrelated to an indwelling catheter.³⁻¹⁰ In this report, we present the clinical and pathologic features of 8 patients with polypoid cystitis who did not have an indwelling catheter and were confused with carcinoma at initial radiologic and cystoscopic evaluation.

MATERIALS AND METHODS

The hospital records of Inonu University, Medical Faculty over 8 years were retrospectively reviewed to identify the patients with polypoid cystitis, who did not have a history of recent urinary bladder catheterisation. 8 patients matching this condition were encountered. The ages, characteristics of sex, symptoms and physical findings, blood and urine analyses, radiographic and cytologic findings, the localizations and appearances of the lesions on cystoscopic examinations were taken into consideration. 7 of 8 patients were called for re-examination of recurrence. One of the patients died due to cardiac problems 2 years following diagnosis. Blood levels of urea, creatinine, electrolytes, white cells and haemoglobin were redetermined; the microscopic analysis of urine, urine culture and cytology, ultrasonographic evaluation were done and following all of these examinations, patients were taken to the operating room for cystoscopy under local anaesthesia using the 2% lidocaine gel. Biopsies were taken with cold-cup biopsy forceps from the macroscopically abnormal regions of the bladder mucosa and were examined histopathologically.

RESULTS

1- Clinical Features and Gross Findings

2 female and 6 male patients ranged in age from 28 to 70 years (mean 48). None of them had a recent urethral catheterisation or instrumentation. Serum creatinin levels were normal in all except 2 (1,9 and 2,2 mg/dl, respectively). Electrolytes, white cells and haemoglobin values were within normal levels. The urine cultures of all patients showed no bacterial

growth. Hydronephrosis was not established by ultrasonography or contrast studies such as intravenous pyelography and computerized tomography (CT) in none of them except 2 with bilateral mild to moderate hydronephrosis and hypercreatininemia related to the obstructive nephropathy secondary to the benign prostatic hyperplasia (BPH). The cytologic examinations available in 4 patients were negative for malignancy. The definitive diagnosis was made histopathologically.

One male patient (age 40) had been operated for right inguinal hernia at another clinic. Abdominopelvic CT performed following the operation to investigate the permanent abdominal pain had revealed a tumoral lesion on the left anterior wall of the bladder. Patient had undergone an open bladder biopsy. Transitional cell carcinoma (pT1grade2) had been determined at biopsy specimen. Then he had been referred to our clinic. During cystoscopy, a 2x3 cm in size, edematous, hemorrhagic, sessile, papillary lesion was seen on the right side of the dome. Transurethral resection of the tumour was performed. Histologic diagnosis was polypoid cystitis. In a young female (age 33), a 7 mm papillary lesion was determined over the right ureteral orifice at cystoscopy performed to investigate unexplained frequency, dysuria and urgency of 6 months duration. One male patient (age 49) had a 1 cm lesion on posterior wall of the bladder which had been found incidentally on ultrasonographic study performed for the evaluation of flask neurogenic bladder and erectile dysfunction. The youngest patient (female, age 28) suffered from right ovarian abscess and a transvaginal USG incidentally revealed a 2 cm mucosal lesion on the right upper wall of the bladder, which was mimicking an invasive carcinoma. Transurethral resection specimen defined polypoid cystitis. The lesion was established in 2 patients (ages 66 and 70) with severe symptoms of bladder outlet obstruction and obstructive nephropathy, on the posterior and left lateral wall during transurethral prostatectomy. One male patient (age 60) was diagnosed by USG during evaluation of intermittent hematuria. Cystoscopy and resection were performed for 1 cm lesion on the left upper wall. The last male patient (age 40) had symptoms of chronic prostatitis. A cystoscopy for evaluation of a lesion established incidentally on USG, showed a 1,5 cm edematous papillary lesion on the bladder dome.

All patients have been followed for 6 months to 2 years after conservative excision of the lesions. The

final urologic workup of 7 cases including serum urea, creatinine, electrolyte levels, white cells, haemoglobin and abdominopelvic USG evaluation were normal in all. Urine microscopies were also normal in all except one patient with 7-8 erythrocytes per high-power field, who had an urinary infection due to E.coli, sensitive to ampicilline. Urines of all patients were examined cytologically and all of them were negative for atypical cells. Polypoid cystitis recurrence or transitional cell carcinoma were not seen at cystoscopies in none of the patients.

2- Histologic Findings

Histopathologic examination in all bladder biopsies revealed congested, chronically inflamed and edematous stroma under the urothelium in all cases (Figure-1). Polypoid appearance was very demonstrative in some (Figure-2), the pathologic diagnoses were “polypoid cystitis”.

Figure-1: Congested vessels and chronic inflammatory infiltrate are seen within the lamina propria. Urothelium is partially eroded. (H.E x 40)

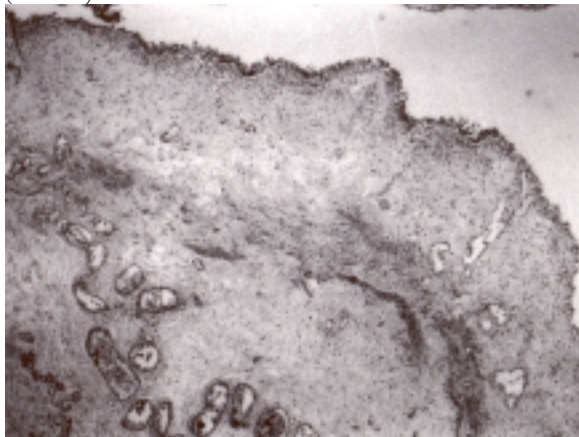
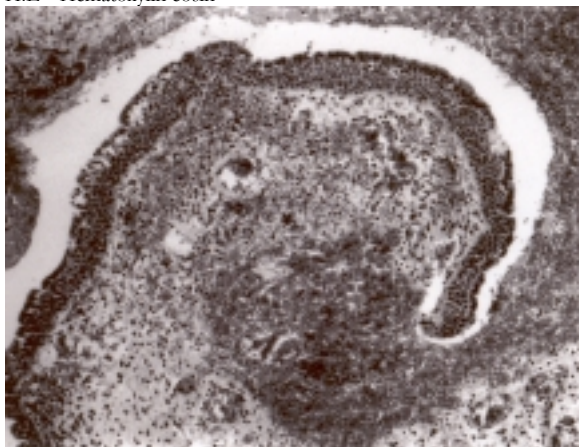


Figure-2: A polypoid protrusion is seen. Lamina propria includes chronic inflammatory infiltrate and dilated vessels. (H.E x 100)
H.E= Hematoxylin eosin



DISCUSSION

The term “polypoid cystitis” was used by Mostofi and by Friedman and Ash for a related process characterized by polypoid mucosal lesions. It is associated usually with the presence of an indwelling catheter. Ekelund and Johansson have found the histologic changes of polypoid cystitis in 41 of 50 geriatric patients treated with bladder catheterisation.¹¹⁻¹³ Most of the lesions (34/50 patients) were on posterior wall which was corresponding to the localization of catheter tip. Frequency of lesions reached to maximum at three months of catheter treatment. In another study, Ekelund et al. found PC in 20 hospitalized patients with bladder catheters.¹⁴ The majority of lesions were located in the posterior wall or dome as in his first report. In that study, almost all lesions disappeared within 28 weeks after catheter removal despite persistent bacteriuria. In a study, statistically significant correlation between the grade of mucosal inflammatory response and the duration of catheterisation; good correlation between the cystoscopic appearances and histological findings; and no correlation between the severity of the mucosal inflammatory response and the presence or absence of associated bacterial infection have been found.¹⁵ In this report, as different from the report of Ekelund et al., the investigators suggested that catheter-associated PC was characterized by eosinophilic infiltrates secondary to toxic, irritative and antigenic effects of urethral catheters.¹⁵ However, Ekelund and Johansson described the inflammatory response as consisting of neutrophils, lymphocytes and inflammatory cells. Norlen et al have established gross and histologic similarity between the bladder and urethral lesions in 20 male patients with indwelling catheters due to benign prostate hyperplasia for 1-12 months.¹⁶ They suggested the term “polypoid urethritis” for urethral lesions.

According to Petersen, when PC do not resolve, fibrosis of the lamina propria stroma may result in a permanent papillary fibrous polyp.² However, Young does not adopt this opinion, since fibrous polyps of the bladder are very rare.⁴ If Petersen’s idea is true, the fibrous polyp cases should be encountered more frequently. In addition, there are several differences between them. Such as; fibrous polyps are typically solitary, they have a fibrous, less edematous core, devoid of significant numbers of inflammatory cells, lack the metaplastic changes often associated with polypoid cystitis.

There is much debate about whether the polypoid cystitis should be regarded as a premalignant lesion. In Young's series, there was slight urothelial hyperplasia in two cases and there were papillae covered by metaplastic squamous epithelium in other two patients.⁴ Goble et al. established urothelial hyperplasia in 11 patients (36%), moderate urothelial dysplasia in 2 patients (6%) with catheters for >4 months and neither urothelial necrosis nor squamous metaplasia in any of biopsies.¹⁵ They thought that the dysplasia was possibly a coincidental finding and also they suggested that the hyperplasia and dysplasia was probably associated with the development of bladder tumours in patients with long-term indwelling catheters. Murphy showed that the catheters induced the urothelial neoplasia in rats.¹⁷ He suggested that the first stage of irritative urothelial carcinogenesis was papillary hyperplasia (reversible stage). Ekelund et al. found that urothelial hyperplasia related to catheter reaction was reversible.¹⁴ The incidence of squamous cell carcinoma following catheterisation for at least 10 years is 8-10%.¹⁸

There are several points which are useful in distinguishing the polypoid cystitis or papillary cystitis from transitional carcinoma. The fronds of the polypoid cystitis are typically much broader than those of a papillary carcinoma. In contrast, the thin papillae of papillary cystitis are more difficult to distinguish from carcinoma. In papillary cystitis (as in polypoid cystitis) the urothelium may be hyperplastic, but it is usually not as stratified as in carcinoma; additionally, umbrella cells are more often present. The fibrovascular cores of the papillae of a transitional carcinoma typically lack the prominent inflammation seen in papillary cystitis and the edema seen in polypoid cystitis. Large papillae of a transitional carcinoma also often give rise to smaller papillae, a feature not associated with papillary or polypoid cystitis. Finally, the urothelium adjacent to a papillary carcinoma is often hyperplastic. Variations in nuclear size and shape within a papillary lesion or in the adjacent urothelium favor a diagnosis of carcinoma; however, it should be emphasized that inflammatory lesions may be associated with reactive atypia.⁴

Polypoid cystitis may also be established in patients without the history of catheter use. For example the evaluation of gross hematuria initiated by suprapubic trauma in a 10-year-old patient has revealed polypoid cystitis on the left lateral wall of the bladder.⁶ In a case report, a 13-year-old boy presented with a 6 months history of intermittent, painless macroscopic

hematuria occurring towards the end of the micturition has been presented.⁷ Our report is not first, however, it is important from the point of differential diagnosis of polypoid cystitis from the carcinoma of the bladder. In addition, as different from the most of the literature about the association of polypoid cystitis with indwelling catheters, it demonstrates that polypoid cystitis lesions may be localized at different sites of the bladder other than the posterior wall and dome. No catheter was present in our patients and their lesions were established incidentally on radiologic and cystoscopic examinations during the evaluations of various conditions such as bladder carcinoma, unexplained dysuria, frequency and urgency, neurogenic bladder and erectile dysfunction, right ovarian abscess, BPH, intermittent hematuria and chronic prostatitis. All patients who were misdiagnosed as bladder tumour at initial diagnosis, were reevaluated as polypoid cystitis after the histopathologic examination of resection specimens.

CONCLUSIONS

Polypoid cystitis is a benign inflammatory lesion rather than a malignant tumour. It may be confused with transitional cell carcinoma of the urinary bladder at initial diagnosis, especially in patients without an indwelling catheter, because of similar appearances. All polypoid or papillary lesions in patients with or without a catheter should be harvested for microscopic examination to make a confident differential diagnosis. The clinical features and pathologic findings may reliably help the pathologist to distinguish papillary and polypoid cystitis from papillary transitional cell carcinoma.

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