Cystitis glandularis: Management and challenges in a renal transplant recipient

Himanshu Agarwal*, Mukund G. Andankar, Viswanath Billa

Departments of Urology and 1Nephrology, Bombay Hospital Institute of Medical Sciences, Mumbai, Maharashtra, India

*E-mail: dr.himanshu.mnh@gmail.com

ABSTRACT

Cystitis cystica or glandularis is a clinical and pathological entity of the bladder mucosa occurring secondary to inflammation or chronic obstruction. Its premalignant nature remains controversial, especially in an immunocompromised transplant recipient. We present a rare case where a chronic kidney disease patient was found to have cystitis glandularis while being worked up for living-related donor renal transplant and describe its subsequent management.

INTRODUCTION

Cystitis glandularis (CG) has been defined as an inflammatory bladder condition usually secondary to irritation of the mucosa or bladder obstruction. The condition has been suggested to be potentially premalignant for bladder cancer. We present a rare case of a chronic kidney disease (CKD) patient, diagnosed to have CG who subsequently underwent a living-related donor renal transplant.

CASE REPORT

A 56-year-old man, a known case of CKD secondary to hypertensive nephropathy, was on thrice-weekly maintenance hemodialysis for the past 2 years. During work up for living-related donor renal transplant, he was found to have occasional terminal hematuria for the past 2 years. There was no history of previous urological intervention, urolithiasis, or lower urinary tract symptom (LUTS) before the onset of CKD. There was no history of smoking or industrial exposure to carcinogens. The pre-procedure urine cultures were sterile. A micturating cystourethrogram revealed a contracted bladder with irregular walls and capacity of nearly 50 cc. Cystoscopy revealed multiple bullous lesions involving the posterior bladder wall, mostly the trigone [Figure 1]. The ureteric orifices were visualized and appeared normal. The anterior urethra and the prostatic lateral lobes were normal. Biopsy was taken from the lesions and sent for histopathological examination. The bladder was hydrodistended to a capacity of 250 cc.

The histopathology revealed CG [Figure 2]. The patient was started on antibiotics, alpha-blockers, and anticholinergics for 6 weeks. A repeat cystoscopy after 6 weeks showed persistent lesions. A complete resection of bladder lesions was done transurethrally with postoperative intravesical Mitomycin C instillation. The biopsy was consistent again with CG.

After counseling, the patient underwent a living-related donor, ABO-compatible renal transplant from his sibling. The postoperative period was uneventful. The patient was maintained on triple drug immunosuppression and had a nadir creatinine level of 1.1 mg/dl. On catheter removal, he had an initial diurnal frequency of 15–20 times which reduced to 8–10 times subsequently. Follow-up cystoscopy 6 weeks postoperatively at the time of stent removal showed resolution of all mucosal lesions except a few at bladder neck which were fulgurated. The bladder capacity was nearly 300 cc. The transplant kidney orifice was normal. At
12-month follow-up, the patient is doing well, maintained on alfuzosin and solifenacin with an acceptable diurnal frequency and no nocturia. The graft is functioning well with baseline creatinine levels of 1.4 mg/dl.

DISCUSSION

Cystitis cystica or glandularis has been described as a clinical and pathological entity of the bladder mucosa occurring secondary to inflammation or chronic obstruction.[1] Cystitis cystica occurs when von Brunn's nests invade the lamina propria forming cysts, while CG describes the metaplastic transformation into goblet cells. Microscopically, two types of CG are recognized, the usual type and intestinal type.[2] The condition is usually asymptomatic and often incidentally diagnosed; however, a few cases have been reported where it may present as acute renal failure, obstructive LUTS, or as ureteric obstruction causing hydronephrosis.[3]

The premalignant nature of CG was first described in 1954 by Imergut and Cottler. Chen et al.[4] described high-risk and low-risk varieties of CG on the basis of expression of proliferating cell nuclear antigen. They concluded that although both high risk and low risk are benign lesions, high-risk lesions have a greater tendency of malignant transformation. Yi et al.,[5] however, retrospectively evaluated 166 cases of CG and concluded that CG does not increase the risk of carcinoma of the bladder, and follow-up in the form of repeated cystoscopies is not warranted. The premalignant nature has not been evaluated in prospective comparative trials and still remains controversial.

CONCLUSION

CG is a potentially treatable condition. One should proceed with renal transplantation in patients with CG after transurethral resection of bladder lesions, though long term data are lacking. To the best of our knowledge, this is first ever clinical scenario of a patient with CG undergoing renal transplant.

REFERENCES

1. Semins MJ, Schoenberg MP. A case of florid cystitis glandularis. Nat Clin Pract Urol 2007;4:341-5.
2. Rau AR, Kini H, Pai RR. Morphological evaluation of cystitis glandularis. Indian J Pathol Microbiol 2009;52:203-5.
3. Matsumura N, Hashimoto K, Katoh Y, Iguchi M, Yamasaki D. Three cases of proliferative cystitis causing hydronephrosis. Hinyokika Kyyo 2014;60:323-8.
4. Chen ZQ, Wei ZF, Ye ZQ, Yang WM. Expression of cancer-related indices in different types of cystitis glandularis and clinical significance thereof. Zhonghua Yi Xue Za Zhi 2005;85:1842-4.
5. Yi X, Lu H, Wu Y, Shen Y, Meng Q, Cheng J, et al. Cystitis glandularis: A controversial premalignant lesion. Oncol Lett 2014;8:1662-4.

How to cite this article: Agarwal H, Andankar MG, Billa V. Cystitis glandularis: Management and challenges in a renal transplant recipient. Indian J Urol 2017;33:249-50.