The Intestinal Type of Florid Cystitis Glandularis Mimics Bladder Tumor

- A Case Report -

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Cystitis glandularis is a benign metaplastic proliferative lesion of the urinary bladder which usually occurs in the setting of chronic irritation and infection or in some cases as a congenital process. Sometimes it presents as a tumor mass-like florid lesion, grossly mimicking malignancy. We report a case of 59-year-old man with multiple mass lesions around the trigone and the neck portion, which suggested the possibility of malignancy in clinical and radiological evaluations. Final diagnosis was confirmed by transurethral resection. The surface urothelial lining was intact. The submucosa showed von Brunn’s nests, cystitis glandularis and cystitis cystica in the edematous lamina propria. There were numerous glands lined by tall columnar, mucin producing epithelium without atypia, conforming to the appearance of the intestinal variant of cystitis glandularis. The cystitis glandularis may mimic a neoplasm on gross evaluation. The intestinal variant of cystitis glandularis is particularly likely to be problematic when florid.

Key Words: Cystitis glandularis; Intestinal variant; Urinary bladder

Cystitis glandularis (CG) is a relatively common proliferative disorder of the urinary bladder. It is thought to evolve either from von Brunn’s nests in the setting of chronic irritation and infection or in some cases, as a congenital process reflecting a partial origin of the bladder from the embryonal cloaca. The intestinal variant of CG is relatively rare compared to finding cystitis cystica and the nonintestinal pattern of cystitis glandularis.1-3 This intestinal variant sometimes presents as a florid form and it is often confused with bladder tumor.4-5 Herein we report on a case of florid cystitis glandularis of the intestinal type in a 59-year-old man, and the patient presented with multiple bladder tumor lesions.

CASE REPORT

A 59-year-old man visited the outpatient clinic because of persistent microscopic hematuria that was originally found at a local clinic. He had been diabetic for the last 10 years and was managed at a local clinic by oral hypoglycemic agents. He complained of hematuria, nocturia, urgency and a narrow urine stream. On rectal examination, the volume of the prostate was increased; the size was estimated to be up to 25 gm. Routine urine analysis was unremarkable except for slightly increased specific gravity. The intravenous pyelography showed prominent trabeculations at the base of the bladder and large filling defects that measured 3.8 × 2.2 cm in size, which were suggestive of bladder mass lesions (Fig. 1). At cystoscopy, there were multiple elevated mass lesions with a cobble stone appearance around the trigone and the neck portion. The other portion of the bladder wall was trabeculated. Malignancy was clinically suspected.

Transurethral resection was carried out for obtaining the definitive diagnosis and treatment. On microscopic examination, the mucosal surface was lined by intact urothelial epithelium. Many nests of tall columnar epithelial cells were diffusely scattered throughout the edematous stroma. Some of them consisted of urothelial epithelium and the others consisted of glandular elements. The glandular elements were variable in size and shape.
Florid Cystitis Glandularis

and they were lined by a single layer of tall columnar cells with abundant cytoplasmic mucous material; this somewhat resembled intestinal mucosa (Fig. 2). The intracytoplasmic mucin content of the glandular elements showed positivity for PAS, alcian blue and mucicarmine staining. Some glands showed hyperplastic features with prominent nucleoli, but they showed an orderly arrangement and the nuclei of all the lining cells were located at the basal portion without nuclear stratification. His postoperative course was uneventful and there has been no evidence of recurrence for one year.

**DISCUSSION**

CG is a relatively common benign proliferative disorder of the bladder mucosa; it is characterized histologically by submucosal nests of urothelial cells that have undergone glandular metaplasia. Two distinct types exist. The first, most typical type is composed of glands lined by cuboidal to columnar epithelium with overlying layers of urothelial epithelium. The second variant is the intestinal type that is characterized by glands lined with mucinous columnar epithelium with basally located nuclei and frequent goblet cells. The exact frequency of the two subtypes is uncertain, but the intestinal variant appears to be much less common than the typical variant.

CG seems to occur as a result of glandular metaplasia of the urothelial epithelium, and this is due to prolonged irritation. It is believed that this metaplastic change starts from the urothelial hyperplasia to the formation of von Brunn's nests, which subsequently develop a lumen and proceeds to cystitis cystica and cystitis glandularis.

The patients usually present with symptoms related to bladder irritation such as dysuria, urgency, frequency, hematuria or abdominal pain. CG is most common in the trigone and bladder neck. Clinically and radiologically, it is difficult to differentiate from tumorous conditions, especially when they manifest as a mass-like or polypoid lesion, such as our case. It appears that most cases of this type have been of the intestinal variant.

Florid CG is clinically more significant because it usually causes more obstructive symptoms, requires more aggressive treatment and can mimic malignancy.

The relationship of CG with adenocarcinoma has been stressed in some literature, but its exact incidence is hard to measure due to the rarity of cases. Although the natural course of CG is not well established, several features, including the high inci-

Fig. 1. The intravenous pyelography shows prominent trabeculations at the base of the urinary bladder. Large filling defects (arrows) are suggestive of mass lesions.

Fig. 2. (A) The mucosal surface is lined by intact urothelial epithelium. Many cellular nests are scattered in the edematous lamina propria. Some cellular nests undergo central degeneration, replaced by concreted proteinaceous material or cystic dilatation. (B) The glandular elements are lined by a single layer of tall columnar cells with abundant cytoplasmic mucous material, somewhat resembling intestinal mucosa. (Hematoxylin-eosin, × 40)
idence of coexistence of CG and adenocarcinoma, the similarities of their pathogenesis and the occurrence of adenocarcinoma in patients with CG during follow-up period, all support the theory that CG may be a premalignant lesion.9

The main differential diagnosis is a low grade adenocarcinoma of the urinary bladder. The histologic features, including the absence of epithelial cells in the extravasated mucin, the lack of atypicality of the cells lining the glands, the orderly distribution of the glands, and the lack of infiltration of the muscularis mucosa, are useful findings in differentiating CG from low grade adenocarcinoma.1,2

Transurethral resection is the treatment of choice, but this malady has a nasty tendency to recur. If it is refractory to standard treatment, cystoprostatectomy and neobladder construction can be considered.3

In conclusion, cystitis glandularis may mimic a neoplasm on radiologic or cystoscopic evaluation. The intestinal variant of cystitis glandularis is particularly likely to be more problematic, and especially when it presents as a florid form.

REFERENCES