http://www.zju.edu.cn/jzus; E-mail: jzus@zju.edu.cn
ISSN 1009-3095 *Journal of Zhejiang University SCIENCE* V.4, No.5, P.620-622, Sep.-Oct., 2003

Relationship between cholecystolithiasis and polypoid gallbladder*

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Received Oct.21, 2002; revision accepted Feb.20, 2003

Abstract: Objective: To study the relationship between cholecystolithiasis and polypoid gallbladder(PLG), 260 patients with polypoid gallbladder were investigated. The patients were divided into 2 groups: group A (PLG combined with cholecystolithiasis) and group B(without cholecystolithiasis). The clinical pathological characteristics were analyzed. The intestinal epithelium metaplasia and atypical hyperplasia of the gallbladder mucosa were observed under light microscope. Results: Intestinal epithelium metaplasia and atypical hyperplasia of gallbladder mucosa were found in 47 of the 260 cases. The pathological lesions included 16 gallbladder carcinoma, 11 adenomatosis polyp, 5 myoadenoma, 7 cholesterol polyp, 4 inflammatory polyp and 4 adenomatosis hyperplasia, which occurred in 26 and 21 patients in group A and group B, i.e. 44.0% and 10.3% respectively. The difference between group A and group B was statistically significant ($P<0.01$). Conclusion: Cholecystolithiasis and the succeeding inflammatory reaction is a risk-factor for the polypoid gallbladder to develop tumour.

Key words: Polypoid gallbladder(PLG), Cholecystolithiasis, Atypital hyperplasia
Document code: A **CLC number:** R544.1

INTRODUCTION

The term polypoid gallbladder(PLG) is used generically to describe any mucosal projection into the lumen. It may be classified as either non-neoplastic lesions, which comprise 95% of all gallbladder polyps, or neoplastic lesions, of which adenomas comprise the vast majority. A number of reports have demonstrated associations of some PLG with development of gallbladder cancer. At least 80% of patients with cancer of gallbladder have gallstone. But it is not clear whether a causal relationship exists between PLG and gallstone. From January 1997 to October 2001, 260 cases with PLG were treated surgically in our hospital, 62 cases were combined with cholecystolithiasis. This study was aimed to probe the clinical and pathological characters of PLG, especially to investigate if the cholecystolithiasis is a risk factor in the carcinogenesis of PLG.

1. Clinical Data

(1) Two hundred and sixty patients with PLG were included in this study. The diagnosis was confirmed by operation and pathology. The patients were divided into group A (PLG combined with cholecystolithiasis) and group B (PLG without cholecystolithiasis). Group A included 62 patients: 35 male and 27 female, average age 47.5 years old (28-78 years old). Group B included 198 patients: 96 male and 102 female, average age 42.8 years old (15-76 years old). In the two groups, among 212 patients whose polyp's diameter was below 5 mm, 179 patients had multiple polyps; 33 patients had a single polyp. Among 48 patients whose polyp diameter was 5 mm to 12 mm, 31 patients had multiple polyps (12 patients in group A and 19 patients in group B) and 17 patients had a single polyp.

* Project (No.2002ZX019) supported by the Zhejiang Province Health Bureau, China

Table 1 Pathological types of groups A and B

Pathological type	Group A	Group B
Cholesterol polyp	29	118
Inflammatory polyp	4	27
Adenomatosis hyperplasia	4	13
Adenomatosis polyp	8	26
Myoadenoma	8	7
Carcinoma	9	7
Total	62	198

(2) Pathological characters: There were 47 cases of intestinal metaplasia and atypical hyperplasia, 16 cases of gallbladder carcinoma, 11 cases of adenomatosis polyp, 5 cases of myoadenoma, 7 cases of cholesterol polyp, 4 cases of inflammatory polyp, 4 cases of adenomatosis hyperplasia.

Table 2 Degrees of atypical hyperplasia of groups A and B

Pathological Type	Group A*			Group B		
	mild	moderate	severe	mild	moderate	severe
Cholesterol polyp	5	0	0	2	0	0
Inflammatory polyp	1	0	0	2	1	0
Adenomatosis hyperplasia	2	0	0	1	1	
Adenomatosis polyp	1	5	0	3	2	0
Myoadenoma	2	1	0	1	1	0
Gallbladder carcinoma	0	0	9	0	0	7
Total	11	6	9	9	5	7

* There were 41.9%(26/62)atypical hyperplasia in group A and 10.6%(21/198) in group B, ($P < 0.01$)

1) There were 16 cases of gallbladder carcinoma with 9 cases of them in group A. Besides the pathologic manifestation of tumors, the images of typical simple hyperplasia, moderate and severe atypical hyperplasia, and the succeeding papilloadenoma (collen type) were observed through a light microscope.

2) There were 41.9%(26/62)cases of intestinal metaplasia and mild atypical hyperplasia in group A and 10.6%(21/198) in group B. The statistic data showed significant difference between the two groups($P < 0.01$).

2. Discussion

It was reported that 7.3% – 36.17% PLG was accompanied with cholecystolithiasis(Cheng *et al.*, 1997; Wu *et al.*, 2000; Huang *et al.*, 1996). The incidence of cholecystolithiasis was high in gallbladder carcinoma and myoadenoma. In China, about 20% – 82.6% gallbladder carcinoma was combined with cholecystolithiasis(Si *et al.*, 2000; Ju *et al.*, 2000), while 73.7% – 95% in myoadenoma. In our study 23.8%(62/260) of patients with PLG had cholecystolithiasis, 56% (9/16) had gallbladder carcinoma combined with cholecystolithiasis. The incidence of cholecystolithiasis in myoadenoma was much higher than in other types. This result is similar to those in the literature.

The repeated hurt-repair process of gallbladder mucosa could be triggered by cholecystolithiasis and the chronic inflammatory reaction that follows. So the pathological changes such as biliary epithelium metaplasia and atypical hyperplasia can be found. Duarte *et al.*(1997) reported that 16% of 161 cholecystolithiasis cases in their research developed atypical hyperplasia of gallbladder epithelium. The incidence of atypical hyperplasia in China is 25.8% in the cholecystolithiasis group and 2.7% in the non-cholecystolithiasis group(Li *et al.*, 1997). Zhang *et al.* (1994) reported 16.8% of 379 cholecystolithiasis cases were combined with atypical hyperplasia, 1.32% with carcinoma in situ, 2.11% with infiltrate carcinoma. Furthermore it was suggested that the atypical hyperplasia and carcinoma resulted from simple hyperplasia. Carcinoma in situ might accompany various atypical hyperplasia, and infiltrate carcinoma might follow carcinoma in situ and severe hyperplasia.

Many studies identified a close relationship between severe hyperplasia of gallbladder epithelium and gallbladder carcinoma. The development of images from simple hyperplasia of mucosa epithelium in gallbladder to moderate, severe atypical hyperplasia and papillocarcinoma combined with colon metaplasia were observed in our

research. These results were in agreement with those reported before. Forty-four percent of cases in group A were accompanied by intestinal metaplasia and atypical hyperplasia while the percentage was 10.3% in group B. There was obvious difference between the two groups. It was shown that PLG combined with cholecystolithiasis was a high risk factor for malignant change.

It is generally believed that the cholesterol polyp has no tendency to carcinogenesis. But 7 of 147 cholesterol polyps were proved to be mild atypical hyperplasia. It was shown that in the case of patients polyps diameter of 5 mm to 12 mm, inflammatory reaction could usually be proved. Twelve such cases were found in group A and 19 in group B. The reasons may be as follows: 1. Gallbladder polyp is usually accompanied by cholecystolithiasis or inflammation; 2. Carcinogens such as cholanthrene and methylcholanthrene are the results of bile duct infection and chemical reaction; 3. There are deoxycholic acid and lithocholic acid in infectious bile juice, which are the homophylic of polycyclic arcomatic hydrocarbon. These may result in the atypical hyperplasia of gallbladder mucosa epithelium. The cholecystolithiasis and inflammation may be the most important of these factors. So the cholecystolithiasis is the risk factor in carcinoesis of PLG.

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