—Report on Experiments and Clinical Cases—

Acute Cholecystitis Caused by a Cholesterol Polyp

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Abstract

A 39-year-old man hospitalized with upper abdominal pain had been found to have a 3 mm polyp in the body of the gallbladder 3 years previously. Laboratory tests on admission showed mild liver dysfunction. Ultrasonography depicted a dilated gallbladder with increased wall thickness; the polyp could no longer be seen. Computed tomography with drip infusion cholangiography again showed a dilated gallbladder, and also stenosis of the distal cystic duct. The resected specimen obtained by laparoscopic cholecystectomy showed disappearance of the polyp from the body of the gallbladder. A cholesterol stone was incarcerated in the cystic duct, representing an impacted detached cholesterol polyp causing acute cholecystitis. Spontaneous detachment of a cholesterol polyp from the gallbladder mucosa, then, can result in acute cholecystitis. (J Nippon Med Sch 2001; 68: 259—261)

Key words: acute cholecystitis, cholesterol polyp, cholesterol stone, impaction

Introduction

Most patients with cholesterol polyps of the gall-bladder note no symptoms. An impacted detached cholesterol polyp has been reported to cause obstructive jaundice¹.

On the other hand, acute cholecystitis, the most common complication of cholelithiasis, is a form of chemically induced inflammation that usually requires cystic duct obstruction and supersaturation of bile. We treated a patient with acute cholecystits caused by an impacted detached cholesterol polyp.

Case Report

A 39-year-old man was admitted to the Nippon Medical School Hospital with upper abdominal pain. Three years before, the patient had been shown to have a 3 mm polyp in the body of the gallbladder (**Fig. 1**).



Fig. 1 Ultrasonography 3 years prior to admission revealed a 3 mm polyp in the body of the gall-bladder.

On admission, physical examination confirmed upper abdominal tenderness and distension. Routine



Fig. 2 Computed tomography with drip infusion cholangiography (3 dimensional image) disclosed a distended gallbladder and stenosis of the distal cystic duct, while the common bile duct was not dilated.



Fig. 3 Intraoperative cholangiography showed the posterior bile duct branching from the distal cystic duct.

laboratory tests performed on serum showed mild liver dysfunction: aspartate aminotransferase activity was 40 IU/L (normal, 10 to 28); alanine aminotransferase, 46 IU/L (normal, 5 to 33); lactate dehydrogenase, 299 IU/L (normal, 180 to 460); total bilirubin concentration, 1.2 mg/dL (normal, 0.2 to 1.2); and direct bilirubin, 0.4 mg/dL (normal, 0.1 to 0.4). The Creactive protein concentration was normal (less than 0.4 mg/dL).

Ultrasonography depicted a dilated gallbladder with mural thickening; the previously noted polypoid lesion had disappeared. The cystic duct was dilated, while the common bile duct was not. Computed to-



Fig. 4 No polyp could be seen in the body of the resected gallbladder. A cholesterol stone (arrow) of the same size as the previously noted cholesterol polyp was impacted in the cystic duct

mography with drip infusion cholangiography (3 dimensional image) indicated dilation of the gallbladder, stenosis of the distal cystic duct, and no dilation of the common bile duct (**Fig. 2**). Laparoscopic cholecystectomy was performed. Intraoperative cholangiography indicated that the posterior bile duct arose from the distal cystic duct (**Fig. 3**). In the resected specimen, the polyp had disappeared from the body of the gallbladder. A cholesterol stone of the same size as the polyp was impacted in the cystic duct (**Fig. 4**). Pathologic examination demonstrated chronic cholecystitis with Rokitansky-Aschoff sinuses.

Discussion

Widespread use of ultrasonography has led to the identification of increasing numbers of polypoid lesions of the gallbladder.

Koga et al.² reported that cholesterol polyps accounted for most benign lesions; in cholecystectomy specimens, all malignant lesions proved to be adenocarcinomas. Sixty-nine percent of patients with benign lesions were under 60 years, old while 75% of those with malignant lesions were over 60. Ninety-four percent of benign lesions were under 1.0 cm in diameter, while 88% of malignant lesions exceeded this size. Kubota et al.³ found 57% of cholesterol polyps to have diameters of less than 10 mm; however, 75% of adenomas and 13% of cancers also measured less than 10 mm.

Collett et al.⁴ described the natural history of gall-bladder polyps, finding a prevalence of 6.7% in the population they studied as well as a marked male predominance (odds ratio, 2.3). During the follow-up period no changes suggestive of malignant transformation were observed. These investigators concluded that gallbladder polyps were relatively common and that few significant changes occurred over a 5-year period.

Generally, cholesterol polyps of the gallbladder are readily detached from the resected specimen. Takii et al.¹ reported a case of obstructive jaundice caused by a cholesterol polyp of the gallbladder. At the distal end of the common bile duct, intraoperative cholangioscopy demonstrated an impacted cholesterol polyp that presumably had become detached from the gallbladder. Most likely, minimal force from apparently trivial causes could detach a cholesterol polyp, which then undergoes changes to become a cholesterol stone.

In conclusion, we encountered a case of acute chole-

cystitis caused by an impacted detached cholesterol polyp. Occasionally, cholesterol polyps of the gallbladder can become detached and produce complications.

References

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