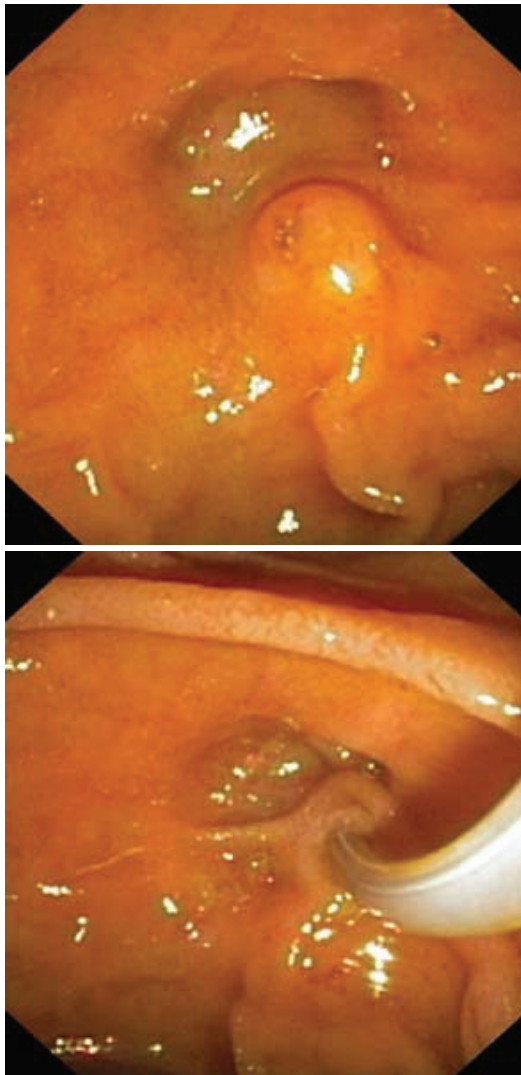


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## CASE 1

A 56 years old female, presented with high grade fever, jaundice and right upper quadrant pain for 3 days.

**ERCP was done and endoscopic views were showed as Figure 1-2.**



**Figure 1-2**

The endoscopic showed extraluminal mucosal outpouching of the duodenum arising adjacent to the ampulla of Vater caused upward facing of the ampulla. Successful cannulation was achieved with swing-tip cannula. Cholangiogram found filling defects in common bile duct and gallbladder and dilated biliary tree. Endoscopic biliary sphincterotomy and balloon sweeping were done.

**Diagnosis:** Periapillary duodenal diverticulum type II, gallstone and acute cholangitis

## Discussion

Periapillary duodenal diverticulum (PDD) was defined as the diverticula arising within 2-3 cm. radius of the ampulla<sup>(1)</sup>. PDD were classified according to the position of the ampulla in 3 types<sup>(2)</sup>, type I: the ampulla located inside the diverticulum, type II: the ampulla located in the margin of the diverticulum and type III: the ampulla located near the diverticulum. About 70-75% of all duodenal diverticulum are periampullary. Prevalence of PDD at ERCP ranged from 5-33% and increased with age<sup>(1-3)</sup>. PDD was a major cause of failure of ERCP, but success rates of more than 90% have been achieved in specialist centers<sup>(1,4)</sup>.

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## CASE 2

A 64 years old female, presented with fever with chills, jaundice and right upper quadrant pain for 2 days. She had been undergone laparoscopic cholecystectomy because of symptomatic gallstone for 3 years.

ERCP was done and endoscopic views were showed as Figure 3-7.

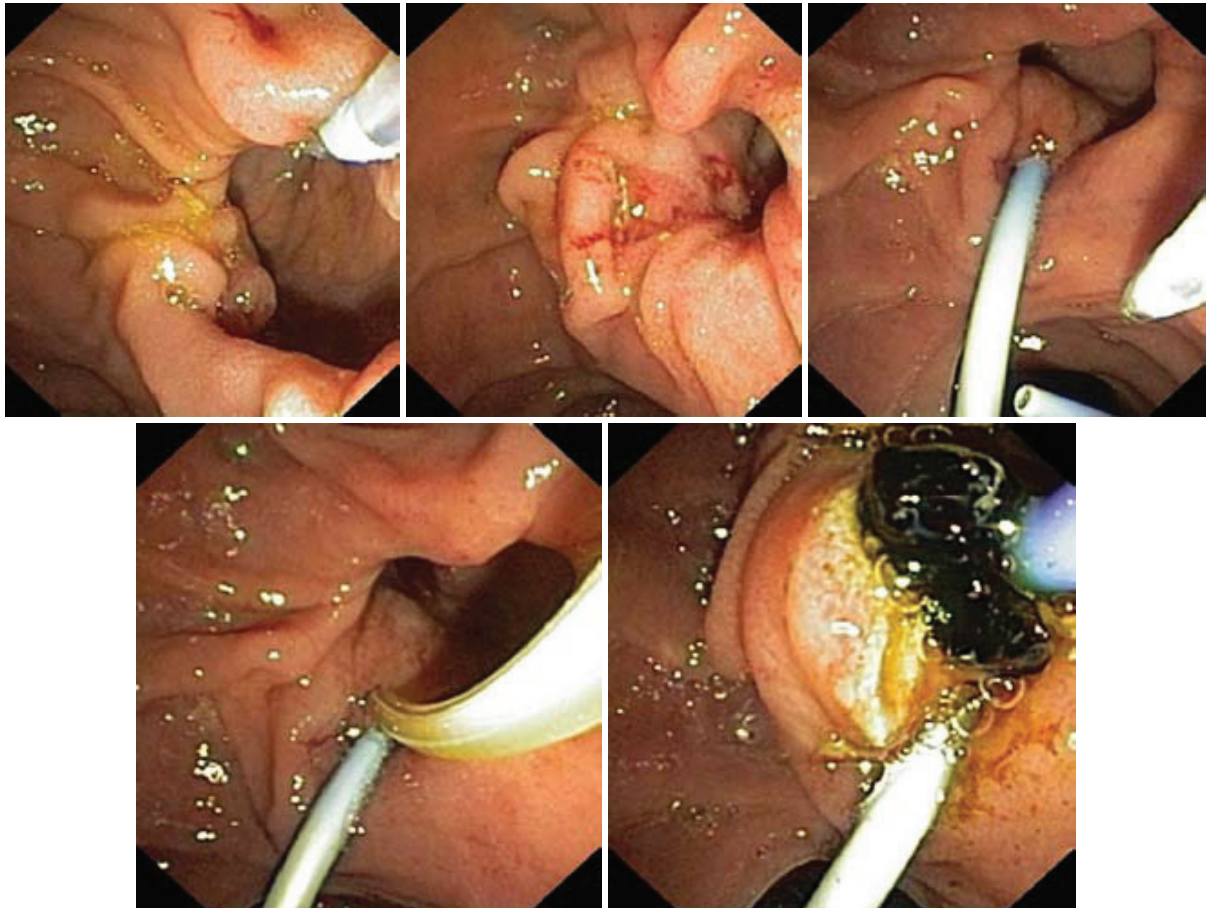


Figure 3-7

The endoscopic showed extraluminal mucosal out pouching of the duodenum containing the ampulla of Vater caused awkward facing of the ampulla. The cannulation was successful after submucosal saline injection and pancreatic duct stenting. Cholangiogram found filling defect in common bile duct and dilated biliary tree. Common bile duct stone was removed after endoscopic biliary sphincterotomy.

**Diagnosis:** Periampullary duodenal diverticulum type I, primary choledocholithiasis and acute cholangitis

### Discussion

Periampullary duodenal diverticulum (PDD) was associated with primary choledocholithiasis<sup>(1-3)</sup>, and increased risk of recurrent choledocholithiasis even in cholecystectomised patient<sup>(3)</sup>. The proposed pathogen-

esis was a combination of incompetent sphincter of Oddi and duodenal bacterial overgrowth in and around PDD<sup>(3)</sup>. Presence of PDD did not alter the success rate of cannulation or increase postendoscopic retrograde cholangiopancreatography<sup>(2)</sup>, but some special techniques may required to facilitate the cannulation in some patients.

### REFERENCES

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### CASE 3

A 44 year-old-female presented with fever, jaundice and right upper quadrant abdominal pain for 2 days. Ultrasound showed common bile duct dilatation with CBD stone. Acute cholangitis was diagnosed.

**ERCP was done. After passed scope to ampulla finding was showed as Figure 8.**

The arrow showed the post bloody meal by a hook worm near major papilla parasite.

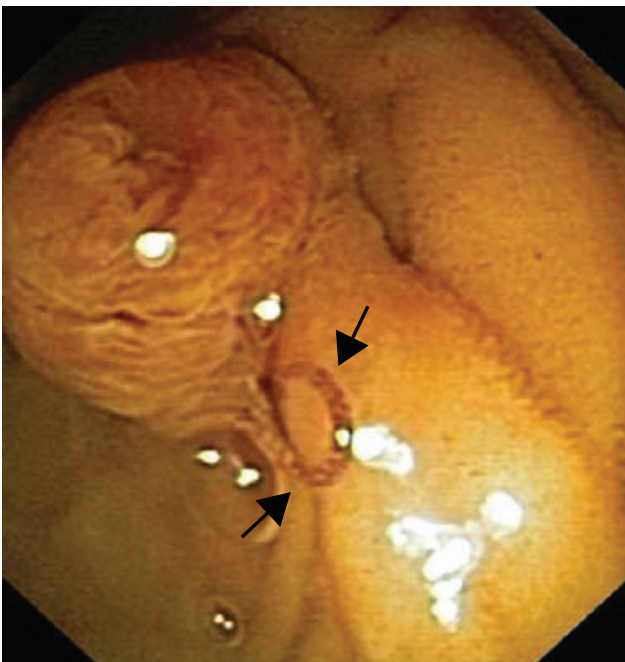


Figure 8

### Discussion

Hookworm infections are common in the tropics and subtropics. The species of hookworm which produce human disease vary geographically<sup>(1)</sup>: *Ancylostoma duodenale* causes infection in Mediterranean countries, Iran, India, Pakistan, and the Far East. *Necator americanus* infects humans in North and South America, Central Africa, Indonesia, islands of the South Pacific, and parts of India.

Hookworm eggs hatch in the soil to release larvae that mature into infective larvae. Percutaneous lar-

val penetration constitutes the principal mode of human infection, although infections with *A. duodenale* may also be acquired by the oral route<sup>(2)</sup>. From the skin, larvae pass to the lungs. At about 8 to 21 days after infection, larvae, like the larvae of *Ascaris*, cross from the pulmonary vasculature, enter the airways, ascend the tracheobronchial tree to the pharynx, and are swallowed. In the small intestine, the larvae mature into adult worms. Adults attach to the mucosa and feed, continually consuming blood and serum proteins.

The potential manifestations reflect the four phases of hookworm infection<sup>(3)</sup>: dermal penetration by infecting larvae, transpulmonary passage, acute gastrointestinal symptoms and chronic nutritional impairments.

Hookworm infections should be treated with mebendazole (100 mg orally BID for three days or 500 mg once). Alternative agents include pyrantel pamoate (11 mg/kg per day for three days, not to exceed 1 g/day) or albendazole (400 mg once). These two drugs are available in the United States but not approved for hookworm treatment by the Food and Drug Administration. In comparison, ivermectin, which is effective for many helminthic parasitic infections, is ineffective for hookworm<sup>(4,5)</sup>.

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