Europe at the end of the 20th century. *Ann Oncol* 2003;**14**(suppl 5):v128–49.
5 **Hemminki K**, Li X, Plna K, *et al*. The nation-wide Swedish family-cancer database—updated structure and familial rates. *Acta Oncol* 2001;**40**:772–7.
6 **Bhatia S**, Sklar C. Second cancers in survivors of childhood cancer. *Nat Rev Cancer* 2002;**2**:124–32.
7 **Hemminki K**, Dong C. Primary cancers following squamous cell carcinoma of the skin suggest involvement of Epstein-Barr virus. *Epidemiology* 2000;**11**:94.
8 **Lindor NM**, Greene MH. The concise handbook of family cancer syndromes. Mayo Familial Cancer Program. *J Natl Cancer Inst* 1998;**90**:1039–71.
9 Cancer risks in BRCA2 mutation carriers. The Breast Cancer Linkage Consortium. *J Natl Cancer Inst* 1999;**91**:1310–16.
10 **Brose MS**, Rebbeck TR, Calzone KA, *et al*. Cancer risk estimates for BRCA1 mutation carriers identified in a risk evaluation program. *J Natl Cancer Inst* 2002;**94**:1365–72.
11 **Pharoah PD**, Guilford P, Caldas C. Incidence of gastric cancer and breast cancer in CDH1 (E-cadherin) mutation carriers from hereditary diffuse gastric cancer families. *Gastroenterology* 2001;**121**:1348–53.

Percutaneous gall bladder aspiration as an alternative to laparoscopic cholecystectomy in Child-Pugh C cirrhotic patients with acute cholecystitis

Cholelithiasis is a common disease in patients with cirrhosis due to intravascular haemolysis and functional alterations of the gall bladder.[1] Acute cholecystitis (AC) results from obstruction of the cystic duct, usually by a gall stone, followed by distension and subsequent chemical or bacterial inflammation of the gall bladder. Approximately 95% of people with AC have gall stones (calcolous cholecystitis) and 5% lack gall stones (acalcolous cholecystitis). Severe AC may lead to necrosis of the gall bladder wall and to perforation, more commonly in patients with chronic diseases such as diabetes mellitus, renal failure, or cirrhosis.[2] Early cholecystectomy is the standard treatment for AC in the general population and laparoscopic cholecystectomy (LC) is generally a safe and effective procedure even in cases of acute inflammation of the gall bladder.[3 4] Although there is increasing evidence that patients with early cirrhosis may undergo LC with low morbidity and no mortality even when presenting with AC, patients with advanced cirrhosis are at high risk for surgery.[3 4] Those with AC who have multiple comorbid conditions and relative contraindications for surgery may be treated conservatively with antibiotics and drip infusion and, more recently, by radiological interventional procedures in order to decompress the gall bladder and to interrupt the pathogenic mechanism of cystic duct obstruction, distension, and infection of the bile which may lead to gangrenous cholecystitis.[5 6]

Gall bladder decompression by a sonografically guided percutaneous cholecystostomy was firstly reported by Radder in 1980 for gall bladder empyema and later on several studies confirmed the safety and efficacy of this procedure in AC.[7 8] Gall bladder aspiration (GA), introduced in 1985 as a diagnostic procedure, is becoming therapeutic in high risk patients with AC.[9] Although both procedures are performed under local anaesthesia and under sonographic guidance they are technically different. Contrary to percutaneous cholecystostomy which enables continuous drainage by deployment of a pigtail catheter, GA provides only one time decompression and drainage of the contents by a needle. Percutaneous cholecystostomy and GA have been reported as valid alternative procedures to cholecystectomy in patients at high risk for surgery and the clinical outcome with both procedures is similar.[10–13] To our knowledge, only a few studies have reported the use of GA in the treatment of AC in patients at high risk for surgery and no report has focused on the particular subset of cirrhotic patients. Moreover, in a series of high risk patients with AC treated with GA or percutaneous cholecystostomy, advanced liver diseases was very rarely reported.[11–13]

At our Department of Surgery, up to February 2004, all cirrhotic patients presenting with AC not responding to conservative treatment were treated with early LC (n = 19). All 15 Child-Pugh A and B patients underwent successful LC with low morbidity and no mortality. A poor outcome was observed in the first four Child-Pugh C cirrhotic patients laparoscopically treated (two deaths occurred for severe liver failure). All Child-Pugh C patients who presented with AC after February 2004 (n = 4), not responding to conservative treatment, underwent gall bladder decompression by ultrasound guided aspiration using an 18 gauge echogenic tip needle. Coagulopathy was corrected with fresh frozen plasma before the procedure in all patients. The procedure was performed in the interventional suite under aseptic conditions using local anaesthesia and a transhepatic route. An 18 gauge echogenic tip needle was used under sonographic guidance to puncture and aspirate the gall bladder. The needle was removed immediately after aspiration of the gall bladder contents and patients remained of their right flank for one hour to press on the punctured liver. GA was technically successful in all cases. A positive clinical response within 72 hours was seen in all patients and cholecystectomy was also avoided after resolution of symptoms. No complications were reported after the procedure. Two patients had recurrence of symptoms after one and two months, respectively, and both were again successfully treated by GA. At the last follow up (December 2005), two patients have been successfully transplanted, one patient died on the waiting list for severe liver failure, and one patient is still awaiting liver transplantation.

In our opinion, the effectiveness of GA can be explained on the basis of the pathogenesis of AC. Of the three factors involved in the development of AC (increased intraluminal pressure, chemical injury of the mucosa from bile salts, and bacterial infection), infection is not present in all cases, as demonstrated by negative bile cultures in 16–48% of patients in different studies.[6] Therefore, one time aspiration of bile from the obstructed gall bladder removes the irritant luminal contents and reduces intraluminal pressure. In most surgical high risk patients, as reported by Chopra *et al*, even those with infected bile GA is enough to determine resolution of AC.[6] Although the mortality rate for the great majority of Child-Pugh A and B cirrhotic patients who undergo LC is low, even in the case of AC, the rate for patients with Child-Pugh C cirrhosis may be as high as 83%.[3 4 10 14] LC is the treatment of choice in Child-Pugh A and B liver transplant candidates, with its associated fewer postoperative adhesions and fast recovery.[1] Treatment of symptomatic cholelithiasis in Child-Pugh C cirrhotic patients needs to be cautious because there is evidence of a high rate of morbidity and mortality in this group of patients.[1] The presence of portal hypertension, liver failure, and ascites correlates with the risk of haemorrhage and sepsis. This is the reason why we chose to use a relatively small needle (18 gauge) and the transhepatic route. Leaving gall stones in the gall bladder is not advisable in the general population but in Child-Pugh C cirrhotic patients, it is necessary because of the high surgical risk. However, this did not correlate in our series or in those of others authors[6 13] with major complications, except recurrence of AC. In our opinion, in Child-Pugh C cirrhotic patients, it is better to decompress minivasively, once or twice, rather than put the patient at risk of death. The goal of GA in such cases is palliation until definitive treatment such as cholecystectomy or liver transplantation are possible.

GA may reverse the progression of inflammation in cirrhotic patients with AC and often provides symptomatic relief. It allows immediate decompression of the acute inflamed gall bladder and can serve as a temporary measure or a definitive treatment. The advantages of GA in Child-Pugh C cirrhotic patients presenting with AC are several. GA does not require general anaesthesia and can be performed at the bedside, with a complication rate significantly lower than that of cholecystectomy. In experienced hands, GA is an easy, quick, and repeatable procedure. Although GA may not be technically feasible in patients with viscous bile, particularly using a 21 gauge needle,[13] it never occurred in our four patients using an 18 gauge needle. Needles thicker than 18 gauge in diameter may facilitate aspiration but in our opinion are not advisable because of the risk of increased bleeding.

We believe that percutaneous gall bladder aspiration, although not without risk, is an effective emergency treatment for Child-Pugh C cirrhotic patients with AC not responding to conservative treatment. It is also recommended in all patients with AC at high risk for surgery as palliative treatment before LC. With the same indication it is also advisable in Child-Pugh A and B cirrhotic patients with AC who have multiple comorbid conditions and relative contraindications for surgery. Early LC remains the treatment of choice when patients are not at high risk for surgery, even in cases of mild cirrhosis. Further prospective comparative randomised trials are needed to establish the best therapeutic option in Child-Pugh C cirrhotic patients.

G Currò, E Cucinotta
Department of Human Pathology, University of Messina, Messina, Italy

Correspondence to: Dr G Currò, Strada Panoramica, 30/A, 98168 Messina, Italy; jose.c@tiscali.it

doi: 10.1136/gut.2005.087841

Conflict of interest: None declared.

References

1 **Silva MA**, Wong T. Gallstones in chronic liver disease. *J Gastroint Surg* 2005;**9**:739–46.
2 **Indar AA**, Beckingham U. Acute cholecystitis. *BMJ* 2002;**325**:639–43.
3 **Puggioni A**, Wong LL. A metaanalysis of laparoscopic cholecystectomy in patients with cirrhosis. *J Am Coll Surg* 2003;**197**:921–6.

4 **Cucinotta E**, Lazzara S, Melita G. Laparoscopic cholecystectomy in cirrhotic patients. *Surg Endosc* 2003;**17**:1958–60.
5 **Lai BS**, Kwong KH, Leung KL, *et al*. Randomized trial of early versus delayed laparoscopic cholecystectomy for acute cholecystitis. *Br J Surg* 1998;**85**:764–7.
6 **Chopra S**, Dodd GD, Mumbower Al, et al. Treatment of acute cholecystitis in non-critically ill patients at high surgical risk: comparison of clinical outcomes after gallbladder aspiration and after percutaneous cholecystostomy. *AJR Am J Roentgenol* 2001;**176**:1025–31.
7 **Radder RW**. Ultrasonically guided percutaneous catheter drainage for gallbladder empyema. *Diagn Imaging* 1980;**49**:330–3.
8 **Patel M**, Miedema BW, James MA, *et al*. Percutaneous cholecystostomy is an effective treatment for high-risk patients with acute cholecystitis *Am Surg* 2000;**66**:33–7.
9 **McGahan JP**, Walter JP. Diagnostic percutaneous aspiration of the gallbladder. *Radiology* 1985;**155**:619–22.
10 **Curro G**, Iapichino G, Melita G, *et al*. Laparoscopic cholecystectomy in Child-Pugh class C cirrhotic patients. *JSLS* 2005;**9**:311–15.
11 **Hultman CS**, Herbst CA, McCall JM, *et al*. The efficacy of percutaneous cholecystostomy in critically ill patients. *Am Surg* 1996;**62**:263–9.
12 **Tazawa J**, Sanada K, Sakai Y, *et al*. Gallbladder aspiration for acute cholecystitis in average-surgical-risk patients. *Int J Clin Pract* 2005;**59**:21–4.
13 **Ito K**, Fujita N, Noda Y, *et al*. Percutaneous cholecystostomy versus gallbladder aspiration for acute cholecystitis: a prospective randomized controlled trial. *AJR Am J Roentgenol* 2004;**183**:193–6.
14 **Corway JD**, Russo MW, Shresda B. Endoscopic stent insertion into the gallbladder for symptomatic gallbladder disease in patients with end-stage liver disease. *Gastrointestinal Endoscopy* 2005;**61**:32–6.

Wireless capsule endoscopic finding in Cronkhite-Canada syndrome

As a new exciting non-invasive diagnostic technology, wireless capsule endoscopy (WCE) has demonstrated its power in examining the small bowel. There is growing acceptance globally not only in the investigation of obscure gastrointestinal bleeding, but in several uncommon diseases such as Behcet's disease,[1] Whipple's disease,[2] even hereditary polyposis syndromes, and so on. In 2005, several articles in *Gut* discussed its increasing role in the diagnosis of Crohn's disease.[3–5]

Cronkhite-Canada syndrome (CCS) is a rare non-hereditary gastrointestinal polyposis with ectodermal changes, first reported in 1955. Intestinal polyposis is one of the common features but there are few published studies showing the direct view in vivo, which has been confirmed mainly by radiological studies previously, partly because of the limitations of the technique. Hence WCE was performed to learn more about the small bowel mucosa in CCS.

A 72 year old Chinese man was admitted with a three month history of fatigue, anorexia, hypogeusia, cutaneous hyperpigmentation, onychodystrophy, as well as oedema of the lower limbs. There was no significant family history. Biological tests showed anaemia, obvious hypoalbuminaemia, and reduced serum Ca (1.71; normal 2.15–2.55 mmol/l), Zn (10.57; normal 12.68–18.56 μmol/l), and Cu (7.81; normal 10.22–25.18 μmol/l) while serum gastrin and CEA were negative. Except for those in the duodenum, which were purplish in colour, numerous reddish, sessile, and oedematous polypoid structures from the stomach to the rectum with friable and haematose mucosa were found on endoscopy. Endoscopy revealed hyperplastic polyps of the antrum and adenomatous proliferous polyps of the colon histopathologically, while the oesophagus was normal, which was concordant with the radiological investigations. Protein losing enteropathy was also confirmed by ^{99m}Tc-HSA. A diagnosis of CCS was established after other relevant examinations.

A barium enema and small bowel series were performed to look for cancerous changes and to evaluate risk, although this was based on only anecdotal evidence (4th ICCE 2005). Forlax (Beaufour-Ipssen, France) and subsequent Senna (a type of mild Chinese herbal medicine) were taken as purgatives together with enough liquid and electrolytes. Espumisan ($((CH_3)_3Si[\text{-}O\text{-}Si(CH_3)_2]nCH_3+SiO_2$; Berlin-Chemie AG, Menarini, Germany) was taken before examination after six hours of fasting. PillCam SB capsule endoscope (Given Imaging Ltd, Israel) was grasped by a polypectomy snare and transferred endoscopically into the duodenum to prevent the gastric polypi retarding[6] the capsule. Our patient remained on his right side for two hours after ingestion to allow a longer examination time of the small bowel. WCE showed a strange pattern of disease involvement along most of the small bowel mucosa: the mucosa was actinia-like with multiple herpes-like polyps studded in the jejunum, but it was strawberry-like with reddish polyps studded separately in the distal ileum (fig 1).

Some data support the fact that CCS may be a late onset disease,[7] and perhaps some patients are asymptomatic, such that the condition is not as rare as once thought. Furthermore, Samoha and Arber recently noted that juvenile polyposis and/or Peutz-Jegher syndrome could be a form of CCS, or vice versa.[8] Thus more sensitive, safe, and simple methods must be found to scan those patients that perhaps have been missed previously. WCE is a potential candidate. It is also possible that there is mucosal polymorphism, as our findings were different to those of Coumaros (the ''arborescent villosities'' reaching the whole small bowel; 4th ICCE). More studies are necessary. It would be helpful if there were a registry database to allow sharing of information from all physicians which would help correct the problem in studying such a rare disease.

Acknowledgements

We thank Deborah Mutter (Manager Clinical Research and Publications, Given Imaging, Inc) for her contribution in drafting the article, and our colleagues Jie Zhang (Department of Gastroenterology) and Jian Tan (Department of Nuclear Medicine), our clinical investigators, for their work. We are also grateful to our colleagues Naixia Huang, Kui Jiang, and Wentian Liu (Department of Gastroenterology) in caring for the study patients, and Wei Li (Department of Pathology) as participating investigator. We thank the patients and the patients' guardians and families for their support.

X C Cao*
State Key Laboratory of Experimental hematology, Institute of hematology & Blood Disease Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Tianjin, China, and Department of Gastroenterology of Tianjin Medical University Hospital,Tianjin Medical University, Tianjin, China

B M Wang*
Department of Gastroenterology of Tianjin Medical University Hospital,Tianjin Medical University, Tianjin, China

Z C Han
State Key Laboratory of Experimental hematology, Institute of hematology & Blood Disease Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Tianjin, China

Correspondence to: Professor Z Han, State Key Laboratory of Experimental hematology, CAMS&PUMC, 288 Nanjing Rd, Tianjin, China, 300020; tihzchan@public.tpt.tj.cn

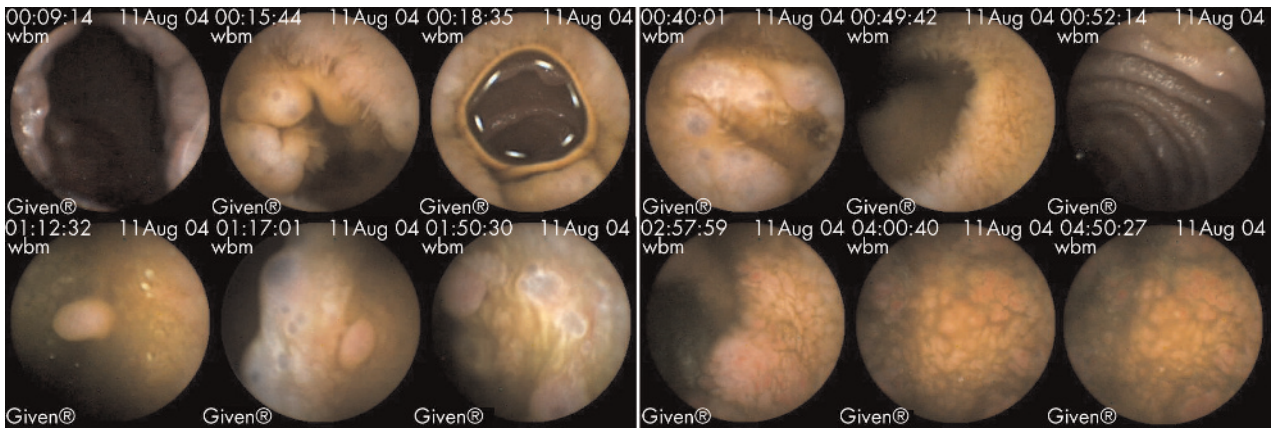

Figure 1 Wireless capsule endoscopy of the small bowel mucosa.