Percutaneous ethanol injection in the treatment of hepatocellular carcinoma in cirrhosis: a simple, effective and cheap procedure for percutaneous ablation

To the Editor,

The new 2010 AASLD practice guidelines for the management of hepatocellular carcinoma have been recently published [1]. Reading the 2010 guidelines text, one issue immediately emerges: the disappearance of percutaneous ethanol injection (PEI) from the new therapeutic diagram.

The writers of the 2010 AASLD guidelines clearly state that "alcohol injection and radiofrequency (RF) are equally effective for tumors < 2cm" and "although RF is currently indicated as first choice for local ablation", still state that "PEI remains an important therapeutic tool" [1]. Therefore, why is PEI not mentioned in the diagram together with RF as illustrated in the 2005 edition? On the other hand, only four months before, one of the two writers of the latest 2010 guidelines felt compelled to question PEI itself by correctly arguing that "the assumption of RF as first line techniques is not incorrect, but this does not mean that ethanol injection is to be dismissed" suggesting the use of PEI most of all – and correctly," in emerging economies" [2].

And furthermore, Bruix added "some tumors are located at risk sites and RF can incur severe complications. In addition, in tumors larger than 2 cm in size, initial RF may leave a tiny nest of viable tissue, that can be ablated by ethanol with a relevant saving of resources" [2].

Therefore, why delete PEI in the 2010 guidelines and not keep it as it was in the 2005 guidelines? By doing this it paves the way for further scientific publications all over the world stating that solely RF is the adequate tool for ablation. And if this does happen, it will result in the necessary economic consequences which can only mean – goodbye, emerging economies!

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Although the last four meta-analysis studies up to now favored RF over PEI because RF improves 3 year survival with respect to PEI, Bruix and Sherman state in their latest 2010 guidelines that "large RCTs comparing RF and PEI in tumors > 2 cm and primarily designed to assess survival are needed", because they are well aware that only a few RCTs have been published and all lack a sufficient sample size of patients [2]. Moreover, in the two western RCTs available to date, PEI and RF showed the same 4 year survival, with a great saving of resources by using PEI [3, 4].

Finally, a last comment: the authors paid much attention to a multicentre Italian study including patients with HCC nodules <2 cm treated with RF and who showed 5 year survival comparable to resection [5]. In this uncontrolled study, 5 year survival was indeed 70%, but reference should be made to the same results obtained by Ebara et al [6] in patients of the same type (i.e. cirrhotic Child A patients with a single HCC < 2cm in which 5 year survival was 74%). Please, note that these excellent results were obtained exclusively with simple and very cheap ethanol.

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References

1. Bruix J, Sherman M. Practice Guidelines Committee, American Association for the Study of Liver Diseases. Management of hepatocellular carcinoma. An update. Official website of AASDL - www.asdl.org. 2010, July.
2. Forner A, Bruix J. Ablation for hepatocellular carcinoma: Is there need to have a winning technique? J Hepatol 2010;52:310-312.
3. Giorgio A, de Stefano G, Di Sarno A et al. Comparison between PEI and RF in the treatment of hepatocellular carcinoma in cirrhotic patients with 3 cm or less single nodule: a prospective randomized controlled trial. J Hepatol 2010; 52 S:183
4. Brunello F, Veltri A, Carucci P et al. Radiofrequency ablation versus ethanol injection for early hepatocellular carcinoma: A randomized controlled trial. Scand J Gastroenterol 2008; 43:727-735
5. Livraghi T, Meloni F, Di Stasi M, et al. Sustained complete response

and complications rates of radiofrequency ablation of very early hepatocellular carcinoma in cirrhosis resection: is still the treatment of choice? Hepatology 2008; 47:82-89.

6. Ebara M, Okabe S, Kita et al. Percutaneous ethanol injection for small hepatocellular carcinoma: therapeutic efficacy based on 20-year observation. J Hepatol 2005; 43:458-464

Impact of duration of hemodialysis on gastrointestinal symptoms in patients with end stage renal failure

To the Editor,

Gastrointestinal (GI) disorders are common. As in many chronic disorders, patients with end stage renal failure (ESRF) have also been shown to experience multiple GI complaints [1-4]. Autopsy studies have also shown GI pathologies to be fewer with longer durations of renal replacement therapy [5, 6]. However, the impact of duration of hemodialysis (HD) on GI symptoms is not well studied.

Patients (n=123) undergoing HD in a dialysis center were recruited. The duration of HD was categorized into short (≤12 months) and long (>12 months). Symptoms included eight upper and four lower GI symptoms. Patients were also asked if they had any psychosomatic symptoms of depression and those who had been treated for any significant illness in the previous four weeks were excluded. The study was conducted in accordance with the standards set out in the Declaration of Helsinki.

Overall, 70.7% had experienced some GI symptoms during the previous 12 months and this included 65% and 34.1% who had experienced upper GI and lower GI, respectively. A longer duration of HD did not significantly affect the proportion of those who had experienced any GI symptoms (Table I). However, in patients who had experienced some GI symptoms, a longer duration of HD was associated with significantly fewer GI symptoms (p=0.01), specifically less nausea, vomiting and dyspepsia. Interestingly, a longer duration was associated with significantly earlier satiety. There were no significant differences in the other symptoms. However, there was a trend towards significantly more non-epigastrium abdominal pain. Longer duration of HD was only significantly associated with a lower prevalence of anxiety.

This study showed that patients who had experienced GI symptoms had fewer GI symptoms with a longer duration of HD. However, this did not impact on the overall proportion of patients experiencing GI symptoms. It is interesting to note that GI symptoms were affected differently. There was less nausea, vomiting and dyspepsia but earlier satiety. Improvements suggest that with prolonged and stable HD, some factors contributing to GI symptoms can be corrected. Stability in HD can result in less metabolic derangements, less fluid imbalance, less disturbance with daily activities and overall better well-being. The finding of less anxiety in association with longer duration of HD supported this. However, it is very likely that the etiologies of GI symptoms are multi-factorial and a stable HD regime only addresses some of these factors. Other factors such as co-morbid conditions, Helicobacter pylori infection, social factors and medications prescribed are probably important [7]. Diabetes mellitus, which is an important cause of autonomic neuropathy is also an important cause of ESRF and has been shown to be associated with a higher prevalence of GI symptoms [8]. Studies with a longer follow-up and repeated evaluations are required to assess the various factors that can contribute to GI symptoms.

In **conclusion**, a longer duration of HD was significantly associated with fewer GI symptoms in patients who have experienced GI symptoms, but not with the proportion of patients with GI symptoms. Interestingly, not all GI symptoms improved with a longer duration of HD.

Table I. Prevalence of gastrointestinal symptoms and impact of duration of hemodialysis

	Duration (months)		P value
	≤ 12 (n = 67)	>12 (n = 56)	
Mean number of GI symptoms	2.6 ± 1.4	1.9 ± 0.9	0.01
Presence of any GI symptoms			
Yes	51 (76.1)	36 (64.3)	0.15
Upper gastrointestinal symptoms			
Anorexia	9 (13.4)	10 (17.9)	0.49
Nausea	17 (25.4)	5 (9.0)	0.02
Vomiting	9 (13.4)	0 (0.0)	0.01
Odynophagia	0 (0.0)	0 (0.0)	1.00
Dysphagia	2 (3.0)	0 (0.0)	0.19
Early satiety	8 (11.9)	15 (26.8)	0.03
Heartburn	12 (17.9)	6 (10.7)	0.26
Dyspepsia	37 (55.2)	18 (32.1)	0.01
Lower gastrointestinal; symptoms			
Abdominal bloating	13 (19.4)	8 (14.3)	0.45
Non-epigastric pain	5 (7.5)	10 (17.9)	0.08
Irregular bowel habit	3 (4.5)	7 (12.5)	0.11
Bleeding per rectum	11 (16.4)	5 (8.9)	0.22
Presence of any psychosomatic symptoms of depression			
Yes	49 (73.1)	34 (60.1)	0.14
Presence of psychosomatic symptoms of depression			
Anxiety	31 (46.3)	8 (14.3)	<0.001
Backache	19 (28.4)	14 (25.0)	0.68
Depressive symptom	10 (14.9)	10 (17.9)	0.66
Headache	24 (35.8)	23 (41.1)	0.55
Insomnia	35 (52.2)	21 (37.5)	0.10

Mann-Whitney U test, Chi squared and Fisher exact test used where appropriate

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References

1. Murtagh FE, Addington-Hall J, Higginson IJ. The prevalence of symptoms in end-stage renal disease: a systematic review. Adv Chronic Kidney Dis 2007; 14:82-99.
2. Kang JY. The gastrointestinal tract in uremia. Dig Dis Sci 1993; 38:257-268.
3. Strid H, Simrén M, Johansson AC, Svedlund J, Samuelsson O, Björnsson ES. The prevalence of gastrointestinal symptoms in patients with chronic renal failure is increased and associated with impaired psychological general well-being. Nephrol Dial Transplant 2002; 17:1434-1439.
4. Cano AE, Neil AK, Kang JY, et al. Gastrointestinal symptoms in patients with end-stage renal disease undergoing treatment by hemodialysis or peritoneal dialysis. Am J Gastroenterol 2007; 102:1990-1997.
5. Chachati A, Godon JP. Effect of haemodialysis on upper gastrointestinal tract pathology in patients with chronic renal failure. Nephrol Dial Transplant 1987;1:233-237.
6. Vaziri ND, Dure-Smith B, Miller R, Mirahmadi MK. Pathology of gastrointestinal tract in chronic hemodialysis patients: an autopsy study of 78 cases. Am J Gastroenterol 1985; 80:608-611.
7. Nardone G, Rocco A, Fiorillo M, et al. Gastroduodenal lesions and Helicobacter pylori infection in dyspeptic patients with and without chronic renal failure. Helicobacter 2005; 10:53-58.
8. Kim JH, Park HS, Ko SY, et al. Diabetic factors associated with gastrointestinal symptoms in patients with type 2 diabetes. World J Gastroenterol 2010; 16:1782-1787.

Failure in wound healing following percutaneous gastrostomy insertion in patients on corticosteroids

To the Editor,

We present three cases highlighting the risk of failure of wound healing in patients on high dose corticosteroids prior to percutaneous gastrostomy (PEG) placement.

A 62 year old lady with dysphagia secondary to skull base myeloma was referred for PEG. Prednisolone 20mg daily was commenced 22 days previously for concomitant palindromic rheumatoid arthritis. She had also taken i.v. steroids for 3 days (dexamethasone 10mg daily) 7 days prior to PEG. A 15 Fr PEG was inserted, feeding commenced and steroids continued. Thirty six hours later, she developed central abdominal pain and tenderness. Abdominal x-rays revealed distended bowel loops. PEG feed was discontinued, i.v. fluids and antibiotics prescribed, but she developed peritonitis and purulent discharge from the PEG site. Exploratory laparotomy revealed intra-abdominal pus and peri-PEG leakage. After peritoneal lavage and PEG removal, the anterior stomach wall was sutured to the abdominal wall. She was discharged following a prolonged intensive care stay.

A 53 year old lady with severe emphysema awaiting pulmonary transplant was taking 20mg of prednisolone at the time of PEG placement (4 weeks duration). Five days after inserting a 9 Fr PEG, peri-PEG tenderness and surrounding pus was noted. Wound swabs grew methicillin resistant staphylococcus aureus (MRSA), mixed coliforms and enterococci. CT imaging revealed intra-pelvic free fluid and gas around the PEG site. Despite prompt peritoneal lavage and drainage she died. Post mortem necropsy revealed peritonitis with fibrinous exudate over the anterior surface of stomach and small bowel.

A 54 year old man had a radiologically inserted 10.5 Fr gastrostomy (RIG) for odynophagia secondary to laryngeal carcinoma. Dexamethasone 8mg three times daily was given for 8 days pre-procedurally for stridor, and continued. By day 5, abdominal distension, generalized tenderness with feed fluid, and oozing around the gastrostomy with new wound gaping was noted. Gastrograffin studies revealed satisfactory RIG position. Antibiotics and parenteral nutrition were commenced, dexamethasone discontinued. CT abdomen revealed intraperitoneal air superior and anterior to the liver, tracking into the lesser sac. Unfortunately, he died.

All patients had normal gastroscopies. Prophylactic antibiotics were not used, as pre-procedural MRSA screening was negative. Patients were swabbed regularly and once MRSA isolated, appropriate antibiotics and nasopharyngeal decontamination commenced.

Corticosteroids interfere with fibroblast proliferation, collagen synthesis, connective tissue deposition, angiogenesis, wound contraction, and re-epithelialization [1]. Abdominal wounds in rat models following treatment with sirolimus and steroids suggested steroids had a synergistic effect on delayed wound healing due to prolonged wound weakness [2].

Delayed fistula closure after PEG removal has been noted in Crohn's patients on corticosteroids and is due to delayed gastric serosal adhesion to the anterior abdominal wall [3]. Our cases highlight this complication. None of our patients had previous problems with post-operative wound healing.

We advocate the reduction/discontinuation of corticosteroids in patients referred for feeding gastrostomies prior to the procedure. We also recommend that after gastrostomy, dosage is kept to a minimum as possible. Neither maximum acceptable doses nor the duration required off or on lower doses have been defined, but the issue is important.

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References

1. Busti AJ, Hooper JS, Amaya CJ, Kazi S. Effects of perioperative antiinflammatory and immunomodulating therapy on surgical wound healing. Pharmacotherapy 2005;25:1566-1591.
2. Gaber MW, Aziz AM, Shang X et al. Changes in abdominal wounds following treatment with sirolimus and steroids in a rat model. Transplant Proc 2006;38:3331-3332.
3. Nightingale J. Gastrostomy placement in patients with Crohn's disease. Eur J Gastroenterol Hepatol 2000;12:1073-1075.

Sexually transmitted infection as a cause of proctitis: asking about sexual orientation is more useful in the diagnosis than endoscopy or histology

To the Editor,

Sexually transmitted infections (STI) are still a significant public health problem both worldwide and in Europe [1], although due to health promotion campaigns in the 1980`s their incidence has been reduced significantly [2]. Current interest is indicated by the recent increasing number of publications on this topic.

STIs such as Chlamydia, gonorrhoea, herpes simplex virus and syphilis commonly present with rectal symptoms and more recently less frequent infections such as lymphogranuloma venerum (LGV) have also occurred in Europe [2]. The publication of Soni et al [3] regarding the diagnosis of LGV proctitis in 12 HIV-positive homosexual men discusses the endoscopic and histological similarities and the difficulties in the differential diagnosis between LGV and inflammatory bowel diseases (IBD) from the view of genito-urinary medicine. However, most of the patients with symptoms of IBD-like proctitis are (perhaps?) HIV-negative and gastroenterologists are not aware of their sexual behaviour, furthermore LGV occurs quite rarely in the rectum. We report a case when the diagnosis of STI was confirmed by positive syphilis serology performed independently from the gastroenterological examinations but this surprising finding assisted in the resolution of the atypical diagnosis.

A 40-year-old Italian man was referred to our gastroenterology clinic because of a four-week history of frequent, urgent, bloody mucous rectal discharge, mild anal discomfort and itching. The patient's medical history was uneventful and the sexual habits were not asked, although he was accompanied by a woman. After excluding the typical causes of gastrointestinal infections, ileocolonoscopy was performed, showing severe oedema and aphthous ulceration involving the lower 5 cm of the rectum. Histology revealed chronic inflammation with MALT acquisition. Topically administered mesalazine resulted in mild clinical improvement; however, after the positive Treponema pallidum (TP) haemagglutination test for syphilis, the introduction of antibiotic therapy promptly diminished the rectal symptoms.

Clinical symptoms of proctitis can be observed in 15% of the homosexual patients without any proved infection [4], although everyone who searches usually finds STI in some cases. In the study of McMillan et al [5], the rectoscopic finding was pathological in only 12% of the symptoms-free homosexual men, but histology revealed abnormality in every third patient who had previously anal intercourse. Neisseria infection was the second most common followed by TP infection, and secondary syphilis was observed in 5% of the patients, but less than half of these patients had abnormal endoscopic finding, with oedema of the rectum in only 2 of the 100 patients [4]. The histological appearance of secondary syphilis seemed to be more characteristic than any other STI with marked lymphoplasmocytic infiltration with or without microgranuloma. However, the histological finding of the proctitis in homosexual men with only mild symptoms was significantly less severe than that observed in the control group of ulcerative colitis patients [4].

A variety of microorganisms causing proctitis can be transmitted among homosexual men: STI agents (Neisseria gonorrhoea, TP, LGV) and others like Herpesvirus, Shigella, Campylobacter and Entamoeba species [4]. Because the sexual preference of the patient who presents at a gastroenterology outpatient clinic is usually not obvious by his appearance, and as the data showed that clinical, endoscopic and histological alterations are more common among homosexual men, and since the diagnosis may be very difficult because of the lack of knowledge regarding the patient's sexual habits, we should not to be reluctant to ask the appropriate patients the right questions.

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References

1. Uusküla A, Puur A, Toompere K, DeHovitz J. Trends in the epidemiology of bacterial sexually transmitted infections in eastern Europe, 1995-2005. Sex Transm Infect 2010; 86: 6-14.
2. Hamlyn E, Taylor C. Sexually transmitted proctitis. Postgrad Med J 2006; 82: 733-736.
3. Soni S, Srirajaskanthan R, Lucas SB, Alexander S, Wong T, White JA. Lymphogranuloma venereum proctitis masquerading as inflammatory bowel disease in 12 homosexual men. Aliment Pharmacol Ther 2010; 32: 59-65.
4. Andrews H, Wyke J, Lane M, Clay J, Keighley MR, Allan RN. Prevalence of sexually transmitted disease among male patients presenting with proctitis. Gut 1988; 29: 332-335.
5. McMillan A, Lee FD. Sigmoidoscopic and microscopic appearance of the rectal mucosa in homosexual men. Gut 1981; 22: 1035-1041.

Autonomic dysfunction and risk of cholelithiasis in cirrhosis

To the Editor,

Cirrhosis and portal hypertension are frequently associated with dysfunction of the autonomic nervous system. Autonomic neuropathy may contribute to the formation of gallstones in cirrhosis due to impaired gallbladder emptying [1]. The aim of the present study was to determine the risk of gallstones in cirrhosis with and without autonomic dysfunction.

Consecutive patients with cirrhosis (n=50) attending the liver clinic were enrolled. Baseline demographic details, etiology of cirrhosis and severity of liver disease as assessed by Child Pugh score were recorded. Parasympathetic function tests included: heart rate response to Valsalva

maneuver (Max.–Min. R-R interval) and heart rate variation on deep breathing (Expiration: Inspiration ratio - E:I ratio). Sympathetic function included postural fall of blood pressure (BP) and a rise in diastolic BP with sustained hand grip. Autonomic dysfunction was considered when both parasympathetic and one of the two sympathetic tests were abnormal. Patients with recent gastrointestinal bleed, renal and cardio-respiratory dysfunction were excluded. The association between various risk factors for cholelithiasis in cirrhosis was studied using the chi-square, Yates corrected chi-square, Fisher`s exact tests and two sample binomial proportion tests respectively. The study was approved by the institutional review board and written informed consent was obtained from all the patients who participated in the study.

Of the 50 patients (male:female = 36:14) studied, the mean age was 44.8 ± 8.3 years. Cirrhosis was related to chronic ethanol abuse in 25 (50%), hepatitis B in 10 (20%), hepatitis C in 3 (6%), and others in 12 (24%) patients. Severity of liver disease was as follows: Child-Pugh class A: 6, B: 32, C: 12. The prevalence of diabetes was 16%. Forty four % of the patients had autonomic dysfunction. Gallstones were observed in 7 (14%) patients. The prevalence of gallstones increased with increasing severity of liver disease. Autonomic dysfunction in cirrhosis was significantly associated with an increased risk of cholelithiasis (p=0.01). However, age (p=0.34), gender (p=0.97), parity (p=0.97), presence of diabetes (p=0.55) and etiology of liver disease did not increase the risk of the formation of gallstones (Table I).

Table I. Risk factors for cholelithiasis in cirrhosis

		With gallstones (n=7) No (%)	Without gallstones (n=43) No (%)	P value
Autonomic dysfunction	Yes	6 (85.7)	16 (37.2)	P=0.01
	No	1 (14.3)	27 (62.8)	
Severity of liver disease	Child A	0 (0.0%)	6 (14)	P=0.04
	Child B	3 (42.9)	29 (67.4)	
	Child C	4 (57.1)	8 (18.6)	
Diabetes mellitus	Yes	1 (14.3)	7 (16.3)	P=0.55
	No	6 (85.7)	36 (83.7)	

Autonomic neuropathy occurs in 35-80% of patients with cirrhosis [2]. The prevalence of gallstones in cirrhosis ranges from 29 to 59% [3]. There is conflicting data concerning risk factors related to cholelithiasis in cirrhosis. Several studies have identified factors such as age, gender, previous alcohol abuse, high body mass index, severity and duration of cirrhosis, gallbladder wall thickness to be associated with an increased risk of gallstones in cirrhosis [4-6]. Acalovschi et al [7] demonstrated male gender and alcoholic cirrhosis to be negatively associated with cholelithiasis. In the present study, autonomic dysfunction was seen in 44%. Gallstones were observed in 14% of cirrhotics, with 85% of them having autonomic dysfunction. Age, gender, parity, diabetes and etiology of liver disease did not increase the risk of cholelithiasis in cirrhosis.

In **conclusion**, liver cirrhosis with autonomic dysfunction was associated with an increased risk of gallstones.

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References

1. Chawla A, Puthumana L, Thuluvath PJ. Autonomic dysfunction and cholelithiasis in patients with cirrhosis. Dig Dis Sci 2001; 46:495–498
2. Milovanovic B, Milinic N, Trifunovic D et al. Autonomic dysfunction in alcoholic cirrhosis and its relation to sudden cardiac death risk predictors. Gen Physiol Biophys 2009; 28: 251-261.
3. Li CP, Hwang SJ, Lee FY, et al. Evaluation of gallbladder motility in patients with liver cirrhosis: relationship to gallstone formation. Dig Dis Sci 2000; 45:1109-1114.
4. Zhang Y, Liu D, Ma Q, Dang C, Wei W et al. Factors influencing the prevalence of gallstones in liver cirrhosis. J Gastroenterol Hepatol. 2006; 21:1455-1458.
5. Benvegnù L. Incidence of cholecystolithiasis in cirrhosis and relation to the etiology of liver disease. Digestion 1997; 58:293-298
6. Conte D, Fraquelli M, Fornari F, et al. Close relation between cirrhosis and gallstones: cross-sectional and longitudinal survey. Arch Intern Med 1999; 159:49-52.
7. Acalovschi M, Blendea D, Feier C, et al. Risk factors for symptomatic gallstones in patients with liver cirrhosis: a case-control study. Am J Gastroenterol 2003; 98:1856-1860.

Incidental diagnosis of stromal tumor of the stomach by percutaneous ultrasound-guided biopsy

To the Editor,

Gastrointestinal stromal tumors (GISTs) are the most common mesenchymal neoplasms of the gastrointestinal tract with an estimated incidence of 1.5/100,000/ year [1]. GISTs originate from the interstitial cells of Cajal (ICCs) which regulate gut motility. This hypothesis is based on observations that both GISTs and ICCs express the receptor tyrosine kinase (RTK) KIT which can be detected by immunostaining for the CD117 antigen as a marker of the KIT protein [2]. Physiological activation of RTK KIT leads to controlled stimulation of cell proliferation and enhanced cell survival. Gain-of-function mutations of the genes encoding for RTK KIT cause neoplastic growth and transformation of cells [3]. Histologically, GISTs are further divided in spindle cell (70%), epitheliod cell (10%), or mixed cell (20%) subtypes.

GISTs are often discovered incidentally during upper endoscopy performed for dyspepsia, or because of symptoms such as bleeding, abdominal pain, evidence of abdominal mass, or obstruction. We report the case of a 60 year old woman with gastric GIST diagnosed by percutaneous ultrasound-guided biopsy.

A 60 year old woman was admitted to our Gastrointestinal Division because of nonalcoholic fatty liver disease. An abdominal ultrasonography (US) showed a hypoechoic tumor of gastric wall (Fig. 1A). A specimen was obtained through US-guided biopsy using an 18-gauge cutting needle (semiautomatic Menghini-modified needle, Biomoll 18G-HS). Esophagogastroduodenoscopy (EGD) showed an antral bulge with a diameter of 50 mm between the anterior wall and small curvature of the stomach lined with a smooth, normally appearing mucosa, and, on the top, a deep ulceration with flat spots (Fig. 1B). Histology of biopsy specimens showed H. pylori negative chronic, non active, gastritis. Histology of US-guided biopsy specimens showed a GIST, with prevalence of spindle cells and mild cellular pleomorphism (Fig. 2A), strong positivity for CD117 (i.e. KIT) (Fig. 2B) and approximately 2% positivity for ki-67, marker of cell proliferation (Fig. 2C). CT and ^{18}FDG-PET scans did not show metastatic lesions. The patient underwent gastrectomy with Billroth II gastrojejunostomy. Diagnosis was confirmed by histopathological examination of surgical resection specimen. After approximately eleven months of follow up the patient is doing well.

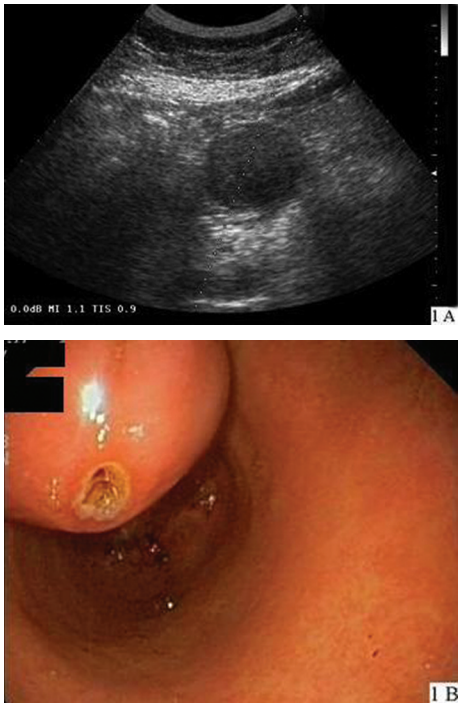

Fig 1 Ultrasonography: A: round, hypoechoic mass on the gastric wall; B: Esophagogastroduodenoscopy: a large antral mass with smooth, normal appearing overlying mucosa and a deep ulceration on the top with flat spots.

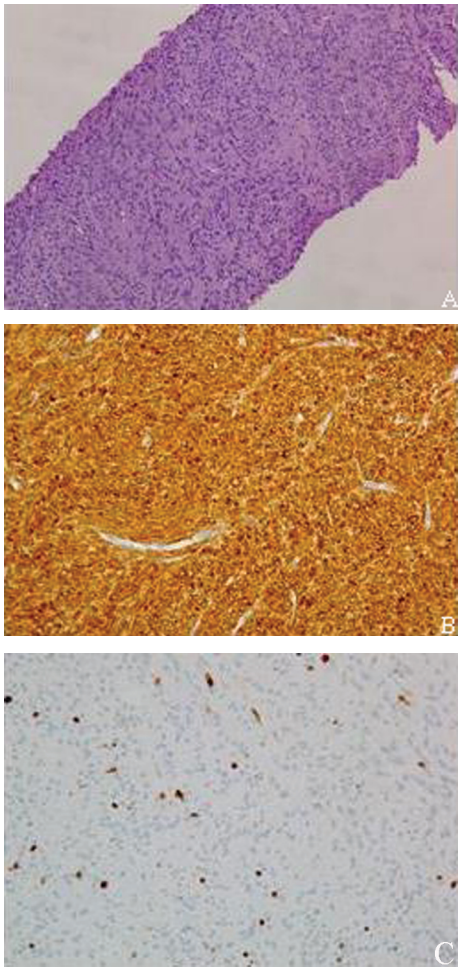

Fig 2. Photomicrographs of histological specimens obtained by US-guided biopsy. A: H&E staining (x40): prevalence of spindle cells with mild cellular pleomorphism; B: intense immunoreactivity for CD117 (c- kit) in both spindle and epithelioid cells (x200); C: nuclear immunoreactivity for ki-67, in neoplastic cells (x200).

Diagnosis of GIST mainly relies on histopathological examinations of specimens obtained by endoscopic ultrasound (EUS)-guided biopsy, surgical resection, or, less frequently, by forceps biopsy during EGD [4, 5]. Some controversy exists regarding use of percutaneous biopsy of potentially neoplastic lesions because of the possibility of neoplastic seeding along the biopsy tract or of spreading tumor cells via peritoneal or mesenteric contamination. However, mounting evidence suggests that neoplastic seeding or spreading following percutaneous biopsy of gastrointestinal tract lesions is highly unlikely [6]. Moreover, the procedure is well tolerated, with only four cases of complications being described in the literature [6].

In conclusion, this report confirms that even large GISTs may be asymptomatic and indicates that specimens suitable for the diagnosis of GIST may safely and accurately be obtained via abdominal US-guided percutaneous biopsy.

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References

1. Casali PG, Jost L, Reichardt P et al. ESMO Guidelines Working Group Gastrointestinal stromal tumors: ESMO clinical recommendations for diagnosis, treatment and follow-up. Ann Oncol 2008;19 suppl 2: ii35-8.
2. Kindblom LG, Remotti HE, Aldenborg F et al. Gastrointestinal pacemaker cell tumor (GIPACT): gastrointestinal stromal tumors show phenotypic characteristics of the interstitial cells of Cajal. Am J Pathol 1998; 152: 1259-1269.
3. Hirota S, Isozaki K, Moriyama Y, et al. Gain-of-function mutations of c-kit in gastrointestinal stromal tumors. Science 1998; 279: 577-80
4. Ha CY, Shah R, Chen J, et al. Diagnosis and management of GI stromal tumors by EUS-FNA: a survey of opinions and practices of endosonographers. Gastrointest Endosc 2009; 69:1039-1044.
5. Sepe PS and Brugge WR. A guide for diagnosis and management of gastrointestinal stromal tumors. Nat Rev Gastroenterol Hepatol 2009; 6:363-371.
6. Marco-Domenech SF, Gil-Sanchez S, Fernandez-Garcia P et al. Sonographically guided percutaneous biopsy of gastrointestinal tract lesions. Am J Roentgenol 2001; 176: 147-151