measurement in the investigation and diagnosis of gastro-oesophageal reflux disease.

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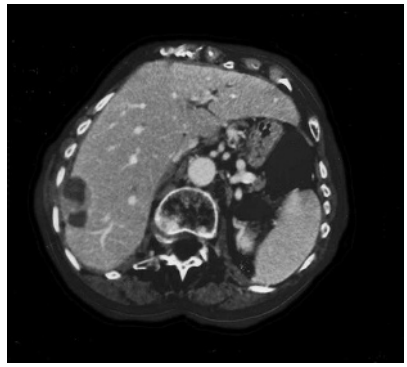
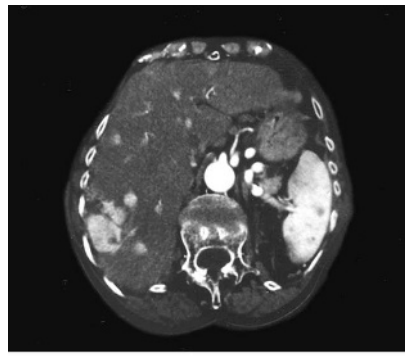
Complications of radiofrequency thermal ablation in hepatocellular carcinoma: what about ''explosive'' spread?

Surgery (resection or transplantation) is the treatment of choice for early hepatocellular carcinoma (HCC) but unfortunately it is feasible in only a minority of patients.[1]





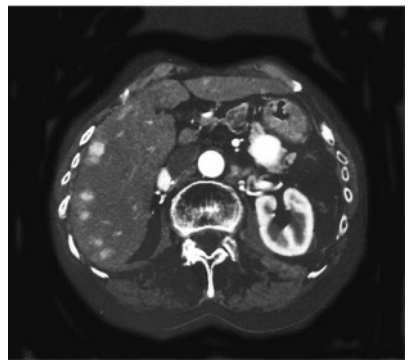


Figure 1 Computed tomography (CT) scan, one month after radiofrequency thermal ablation. The lesion appears to be partially treated with no clearcut residual activity in the arterial CT phase.

Figure 2 After five months, complete reactivation of the original lesion and 13 new additional lesions were evident.

Local percutaneous ablation has therefore dramatically increased in importance and radiofrequency thermal ablation (RFTA) has been shown in recent reports to have global or disease free survival better than that reported for percutaneous ethanol injection (PEI).[2] The recent report in *Gut* apparently also supports the superiority, albeit small, of RFTA over PEI or acetic acid injection in a randomised prospective controlled study (*Gut* 2005;**54**:1151–6). Nevertheless, not all agree on the efficacy of RFTA[3] and major complications have been described.[4] In Lin *et al*'s study (*Gut* 2005;**54**:1151–6), the incidence of major complications was significantly higher than that with the ethanol or acetic acid injection procedures.

We have conducted a multicentre prospective study on the efficacy and complications of RFTA in HCC in North-East Italy, involving 399 HCC patients and a centralised radiological assessment. Overall, 133 patients (33%) experienced some complications, five of which were severe (1.3%). There were two deaths (0.5%), one being procedure related (intestinal perforation) and the other a cerebral infarction.

Some authors have reported rapid intrahepatic progression after RFTA,[5 6] a fact that deserves special attention. We documented nine cases of rapid unexpected spread of HCC after RFTA in our multicentre study. The nine cases (2.3% of the series) are reported in table 1 and examples are given in fig 1 (computed tomography (CT) scan, one month after RFTA; the lesion appears to be partially treated with no clearcut residual activity in the arterial CT phase) and fig 2 (after five months, complete reactivation of the original lesion and 13 new additional lesions were evident).

This rapid unexplained and biologically unclear progression of HCC following RFTA treatment is most unusual considering the natural history of the disease. It may be prompted by increased intratumoral pressure with intravascular spread, seeding due to arterovenus fistula, or to the expandable hooks needles. Suggested risk factors have been high α fetoprotein levels, location near major portal branches, and poor tumour differentiation.[4 5] Also, increased liver concentrations of growth factors (transforming growth factor β1 and β fibroblast growth factor) have been documented in rat liver after thermal coagulation, with increased HCC growth.[7]

Our data show that side effects are relatively frequent following RFTA, with a

Table 1 Nine cases of rapid unexpected spread of hepatocellular carcinoma (HCC) after radiofrequency thermal ablation (RFTA)

Patient No	Sex, age, aetiology	% Efficacy	No of nodules	Size before RFTA (cm)	New nodules (time/months)	Grading	AFP (ng/ml)
1	M, 79, HCV	100%	1	4.6	Satellites + new controlateral HCC node (1 month)	G1	15.7
2	M, 78, HBV	90%	2	2.4–3	3 (3 cm each) (3 months)	G1	32.9
3	M, 61, HBV	100%	1	1.8	Multifocal (>3) (4 months)	G1	32
4	M, 68, HCV	90%	1	4.2	3 (1 month)	G1–G2	10
5	M, 69, HCV	100%	2	4.2–1.5	Multiple (up to 6 cm) (3 months)	–	8
6	M, 73, HCV	100%	1	4	Multiple (>3) (1 month)	–	32
7	F, 73, HCV	70%	1	3.1	13 (5 months)	–	117
8	M, 65, HCV	100%	1	2.0	1 (8 cm) new lesion (7 months)	G1	12.6
9	M, 72, HBV+ETOH	100%	1	2.8	Multifocal (5 months)	G1	4.9

HBV, hepatitis B virus; HCV, hepatitis C virus; ETOH, alcohol; AFP, α fetoprotein.

mortality rate less than 1%. Our series confirmed that ''explosive HCC spread'' may occur after RFTA, with no correlation with AFP, or differentiation or location of the tumour, and frequently even after total ablation.

Why do these patients develop this severe (and dramatic if observed in patients on waiting lists for orthotopic liver transplantation)[8] and unexpected complication and what is its prevalence? Whether or not we can identify patients at risk is open to discussion, which we believe should be exhaustive before abandoning PEI as obsolete in favour of RFTA as the treatment of choice for early disease not eligible for surgery. Let us wait for the end of the honeymoon period of this new procedure!

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Collagenous colitis in adult Sri Lankans: experience from the Indian subcontinent

The literature contains few data on the clinical features and demographics of collagenous colitic patients from the Indian subcontinent. Almost all published articles describe various clinicopathological facets of collagenous colitis in the West, which is now classified as a variant of microscopic colitis, where diagnosis is missed by failure to biopsy the normal or ''near normal'' looking mucosa at endoscopy for histological examination.

The case notes of 210 patients who underwent colonoscopy at Base Hospital-Panadura (Medical Unit), Sri Lanka (equivalent to a district general hospital in the UK, with a bed strength of 350), for various reasons, from 20 July 1999 to 20 January 2002, were retrospectively reviewed. Clinical features and results of other routine investigations of patients who were histologically diagnosed as having collagenous colitis were analysed. All those with chronic colonic symptoms had mucosal biopsies taken from all major segments of the colon, despite normal or ''near normal'' looking mucosa.

The results revealed that 29 patients had histological evidence of collagenous colitis with an approximate equal male and female distribution (15:14). The age distribution was 21–50 years (mean 45 (SD 7) years). Collagen thickening was focal in 17 patients, uniform in eight, and mixed in four. Symptoms included diarrhoea alone in four cases, diarrhoea with abdominal pain in 15, and abdominal pain alone in 10, all having intermittent symptoms for more than a year. No other major symptoms or signs were elicited. Histological changes were mainly seen in the distal colon. The colonic mucosa appeared normal in 21 and mildly erythematous (subjective) in eight patients at endoscopy. Routine haematological, biochemical, and stool tests, and abdomino-pelvic ultrasound examinations were normal. Van-Giesen stain was used to confirm the presence of collagen in biopsies following haematoxylin and eosin.

Collagenous colitis is increasingly recognised as a variant of chronic inflammatory bowel disease, which causes chronic watery diarrhoea, predominantly affecting women in the sixth or seventh decade.[1] In contrast, in our series, a female preponderance was not observed and mean age at presentation was much younger (approximately 35 years of age). Abdominal pain was a notable symptom in 86% (25/29) of cases while diarrhoea was found in only 66% (19/29). Early age at presentation may be due to early diagnosis, a background of a high degree of awareness of its existence, and the enthusiastic approach of biopsy of normal or ''near normal'' looking colonic mucosa at endoscopy in relatively young patients. Autoimmune mechanisms, cytokine pleomorphism, commensal bacteria, infective agents, various proinflammatory mediators, and nitrous oxide have all been implicated in the pathogenesis.[2] Whatever the mechanisms, these were not severe enough to cause significant abnormalities in our routine investigations or on clinical examination. It has been found that biopsy specimens from as proximal as the transverse colon should be obtained to rule out collagenous colitis with certainty.[3]

In our series, the majority of patients showed focal thickening, probably as a result of early diagnosis. Although unpublished, we have repeatedly highlighted the existence of collagenous colitis mimicking irritable bowel syndrome in adult Sri Lankans on many occasions,[4–7] and published data in the *Journal of Ceylon College of Physicians*.[8]

In conclusion, collagenous colitis seems to be more prevalent in adult Sri Lankans than suspected, and at an early age. The three histological types most likely represent various stages of disease progression, with changes being more pronounced in the distal colon. There was no sex preponderance and diarrhoea was not an integral feature in every subject. Collagenous colitis closely mimicked irritable bowel syndrome of relatively young Sri Lankans. An enthusiastic approach is essential for early diagnosis. More studies from the Indian subcontinent and South East Asian regions would be useful to compare with the results obtained in this study.

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