Table 1 Endoscopic findings and localisation of cytomegalovirus (CMV) inclusion bodies in patients with CMV enterocolitis

	Colonoscopic findings				Localisation of CMV inclusion bodies							CMV antigenaemia
	Erosion	Ulceration	Oozing	Other	T/I	Caecum	A/C	T/C	D/C	S/C	Rectum	
Case 1	NA	NA	1/3	0/2					1/2‡	0/1	0/2	50
Case 2	1/2	NA	NA	0/5		1/2‡	0/1‡	0/1	0/1	0/1	0/1	14
Case 3	1/4	NA	NA	0/3	0/1	0/1	0/1	1/1‡	0/1‡	0/1‡	0/1‡	8
Case 4	NA	NA	1/5	NE						0/2	1/3	296
Case 5	NA	NA	NA	3/3*	1/1	1/1					1/1	61
Case 6	5/5	NA	NA	NE				3/3	2/2			932
Case 7	1/1	0/3†	NA	NE	0/1			1/4‡				871
Case 8	5/7	NA	NA	0/2	0/2	2/2‡	0/2	3/3‡				108

Each column represents total CMV inclusion bodies per biopsy specimens.

*Biopsy specimens were obtained from the edge of the exfoliation of the mucosa.

†Punched out ulceration.

‡Presence of erosion.

NA, not applicable; NE, not examined; T/I, terminal ileum; A/C, ascending colon; T/C, transverse colon; D/C, descending colon; S/C, sigmoid colon.

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Hyperhomocysteinaemia and vascular disease in liver patients

We read with interest the case report by Buchel *et al* (*Gut* 2005;**54**:1021–3). The authors reported on a patient with portal hypertension, nodular regenerative hyperplasia, and portal thrombosis that presented an avascular hip necrosis after liver transplantation (OLT). The authors suggested that the whole clinical picture might stem from hyperhomocysteinaemia (high Hcy) and 677C→T heterozygosity for the common gene of methylene tetrahydrofolate reductase (MTHFR).

Although the history is suggestive of this hypothesis, high Hcy might also be a secondary phenomenon. It is not clear whether high Hcy was demonstrated in a sample obtained before (as suggested in table 1) or after (as suggested in the text) OLT. In both cases, Hcy might be elevated as a consequence of either liver disease or drug treatment. Liver disease per se raises Hcy due to the multiple metabolic problems generated by a failing liver, including low folate levels, altered transsulphuration/transmethylation pathway, and decreased renal function. High Hcy is present in 50% of liver disease patients,

and values as high as 30 µmol/l are not uncommon in end stage disease,¹ independently of MTHFR polymorphism. After OLT, a specific effect of tacrolimus on Hcy metabolism and concentration is well documented. Both calcineurin inhibitors (cyclosporin and tacrolimus) are known to interfere with the folate dependent remethylation of Hcy, thus raising plasma levels to values in the pathological range. The problem is well documented in patients following renal,² cardiac,³ or liver transplantation.¹ In a recent analysis of 230 patients subjected to OLT, we found 26 cases with fasting Hcy ≥30 µmol/l in the late post-transplant phase, independently of MTHFR polymorphism.⁴

Also, the relationship between high Hcy and MTHFR polymorphism deserves comment. The MTHFR gene independently and unfavourably influences homocysteine metabolism but high Hcy levels are mainly observed in subjects with low folate levels,⁵ indicating a low phenotypic expression. Therefore, the presence of the genetic variant, mainly in its heterozygous form, might be an occasional phenomenon without clinical significance.

In conclusion, although the clinical history of the patients raises the suggested pathogenic role of Hcy and genetic predisposition, no clues can be made on the “egg and chicken” sequence, particularly in a patient with positive markers for hepatitis B infection.

Adequate folate intake or pharmacological supplementation may certainly overcome genetic predisposition to high Hcy, but may also decrease Hcy, independently of any gene defect, and remains a suitable therapeutic option to prevent additional vascular problems.

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Second gastric cancers among patients with primary sporadic and familial cancers in Sweden

Gastric cancer ranks as the fourth most common cancer and the second most frequent cause of cancer death worldwide.¹ The aetiology includes causes such as *Helicobacter pylori* infection (for distal gastric cancer but not for cardia cancer),² dietary imbalance, smoking, and genetic factors.^{2–3} Estimation of the incidence of second primary cancers may provide valuable insight into the aetiology and shared risk factors with the initial cancer. However, as gastric cancer patients have poor survival,⁴ a study of second malignancies after primary gastric cancer may not be informative. Instead, we examined the occurrence of second gastric cancers following any first cancers, based on the nationwide Swedish Family Cancer Database. This database has been described in detail previously.⁵ Briefly, it was created by linking information from the Multigenerational Register, censuses, Cancer Registry, and death notifications. The database has an almost complete follow up of registered cancer patients and it provides a unique opportunity to quantify the risks of developing second gastric cancers among all primary cancer patients. Person years at risk were accumulated for each subject from the data of diagnosis of the first malignancy to that of a second gastric cancer, death, emigration, or 31 December 2002, which came first. Standardised incidence ratio (SIR) was used to estimate the risks of second gastric cancers, adjusted for sex, age, period, residence, and socioeconomic level. Confidence intervals were calculated assuming a Poisson distribution. Family history included all first

Table 1 Standardised incidence ratio (SIR) for second gastric cancers following first primary cancers

First cancer	Cancer site	Cases	Second gastric cancer								
			All			Corpus cancer			Cardia cancer		
			O	SIR	95% CI	O	SIR	95% CI	O	SIR	95% CI
Upper aerodigestive tract		19092	105	1.16	0.95, 1.41	61	1.33	1.01, 1.70	9	0.83	0.38, 1.59
Oesophagus		6360	32	3.44	2.35, 4.86	14	3.19	1.74, 5.37	11	10.53	5.23, 18.91
Stomach		34152	36	<u>0.43</u>	0.30, 0.59	15	<u>0.36</u>	0.20, 0.60	6	<u>0.77</u>	0.28, 1.69
Colon		58767	218	1.19	1.03, 1.36	86	1.02	0.81, 1.26	27	1.27	0.84, 1.85
Rectum		33806	128	1.09	0.91, 1.30	51	0.94	0.70, 1.24	16	1.16	0.66, 1.88
Lung		59598	76	0.87	0.68, 1.09	31	0.73	0.50, 1.04	8	0.80	0.34, 1.58
Breast		114333	379	1.44	1.29, 1.59	192	1.59	1.38, 1.84	20	0.94	0.58, 1.46
Cervix		19425	74	1.37	1.08, 1.73	30	<u>1.11</u>	0.75, 1.58	4	1.07	0.28, 2.76
Endometrium		23351	66	0.85	0.66, 1.08	29	0.82	0.55, 1.17	8	1.29	0.55, 2.55
Ovary		22617	54	1.33	1.00, 1.73	30	1.54	1.04, 2.20	6	2.00	0.72, 4.38
Prostate		107644	510	1.02	0.93, 1.11	202	0.91	0.79, 1.04	79	1.13	0.90, 1.41
Testis		6065	20	1.92	1.17, 2.97	10	2.41	1.15, 4.45	5	2.64	0.83, 6.22
Kidney		25948	67	0.90	0.70, 1.14	28	0.78	0.52, 1.12	12	1.38	0.71, 2.42
Urinary bladder		38107	202	1.13	0.98, 1.29	73	0.89	0.70, 1.12	28	1.17	0.78, 1.69
Melanoma		30561	66	0.79	0.61, 1.01	29	0.88	0.59, 1.26	9	0.77	0.35, 1.48
Skin, squamous cell		27090	178	1.48	1.27, 1.71	89	1.70	1.36, 2.09	13	0.84	0.44, 1.44
Nervous system		32287	49	<u>0.84</u>	0.62, 1.11	22	<u>0.82</u>	0.51, 1.24	4	0.62	0.16, 1.62
Thyroid gland		8512	29	1.23	0.82, 1.76	8	0.73	0.31, 1.44	4	1.62	0.42, 4.20
Endocrine glands		15871	45	0.83	0.60, 1.11	19	0.79	0.48, 1.24	3	0.48	0.09, 1.42
Connective tissue		6438	23	1.33	0.84, 2.00	12	1.51	0.78, 2.65	4	1.90	0.49, 4.90
Non-Hodgkin's lymphoma		25874	80	1.36	1.08, 1.70	28	1.12	0.74, 1.62	11	1.44	0.71, 2.58
Hodgkin's disease		6264	21	<u>1.90</u>	1.17, 2.91	8	1.49	0.64, 2.96	2	1.58	0.15, 5.83
Myeloma		11482	41	1.52	1.09, 2.06	25	1.90	1.23, 2.81	1	0.33	0.00, 1.90
Leukaemia		26161	49	0.85	0.63, 1.13	19	<u>0.70</u>	0.42, 1.10	5	0.77	0.24, 1.82
All		824465	2648	1.11	1.07, 1.15	1152	1.05	0.99, 1.11	304	1.10	0.98, 1.23

Bold type, 95% confidence interval (CI) does not include 1.00; underline type, 99% CI does not include 1.00.
O, observed.

degree relatives (parents, siblings, and children) of the first cancer patients.

A total of 824 465 patients with first cancer were retrieved from this database, of whom 2648 developed a second gastric carcinoma. Table 1 shows the SIRs of second gastric cancers following all primary cancers. The risks were examined further by follow up time (data not shown). Significant increases after cervical, ovarian, and testicular cancers, and after non-Hodgkin lymphoma and Hodgkin disease were mainly confined to the period >9 years of follow up, which could be partly explained by therapeutic effects because treatment related effects usually occur a decade after the first carcinoma.⁶ The highest SIR (mainly for cardia cancer) was noted after oesophageal carcinoma, but it was confined to the first year after diagnosis which may have been due to observation bias or increased surveillance. Gastric carcinoma was in excess after skin carcinoma throughout the follow up period, except for the first year, which was in agreement with previous studies.⁷ Epstein-Barr virus may be

related to the increase because skin cancer patients can be immunocompromised and thus at an increased risk of virally induced disease.⁷ Also, *H. pylori* may escape from faltering immune surveillance, and the specific increase in corpus cancer would support the role of *H. pylori*.

Patients with a family history of the first cancer were at a higher risk than all patients (table 2). SIR was increased after familial colon carcinoma (3.31) and was very high when colon cancer patients were diagnosed at a young age (10.61). Inherited predisposition, such as hereditary non-polyposis colorectal carcinoma (HNPCC), may contribute towards the occurrence of second gastric carcinomas.⁸ However, gastric cancers were not increased after other HNPCC related cancers (rectal, endometrial, and ovarian cancers). Gastric cancer was also increased among patients with familial breast cancer (2.32), particularly when diagnosed at a young age (3.43). Breast carcinomas are increased in BRCA1 and BRCA2 mutation carriers who also have an increased risk of

gastric carcinoma.⁹⁻¹⁰ Another link between breast and gastric cancer may be germline mutations of the E-cadherin gene, which have been observed in patients with diffuse gastric cancer and breast carcinoma.¹¹ This study suggested that shared genetic factors may be a contributor towards the development of second gastric cancers.

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Table 2 Standardised incidence ratio (SIR) for second gastric cancers in patients with a family history in first degree relatives

Cancer site	Relatives with cancers (all ages) Second cancers				Relatives with cancers (age <45 y) Second cancers			
	Cases	O	SIR	95% CI	Cases	O	SIR	95% CI
Colon	1268	9	3.37	1.53, 6.42	233	6	10.61	3.82, 23.25
Breast	7737	31	2.32	1.58, 3.30	1766	12	3.43	1.76, 6.01
Prostate	3540	12	<u>1.00</u>	0.52, 1.76	12	0		
Skin, squamous cell	275	3	3.75	0.71, 11.09	31	0		
All	19517	67	1.65	1.28, 2.10	3840	20	2.70	1.64, 4.17

Bold type, 95% confidence interval (CI) does not include 1.00; underline type, 99% CI does not include 1.00.
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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 (calculous cholecystitis) and 5% lack gall stones (acalculous 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 sonographically 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 minimally, 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.

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