PostScript

LETTER

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Bleeding peptic ulcer

We read with interest the paper on prediction of therapeutic failure after adrenaline injection plus heater probe treatment in patients with bleeding peptic ulcer by Wong and colleagues (*Gut* 2002;50:322–5). Even though the authors qualified their generalisation, the statement that "elderly patients often succumb to their concomitant illnesses rather than the bleeding itself" needs to be challenged as being unnecessarily defeatist, given the fact that timeliness of surgical intervention¹ and, as shown below, postoperative management at the intensive care level, may be more crucial to survival than comorbidity as such.

Case report

A 70 year old woman with congestive cardiac failure (including radiographically validated left ventricular failure) and chronic obstructive airways disease experienced an episode of haematemesis and melaena with an associated blood pressure of 78/48 mm Hg on the 1 March 2002, which was the eighth day of her hospital stay. Endoscopy revealed a large actively bleeding duodenal ulcer, which was managed with endoscopic haemostasis, rapidly followed by definitive laparotomy and under running of the bleeding vessel. She was then transferred to a neighbouring hospital for postoperative intensive care management, and this included a 24 hour period of artificial ventilation. On the 11th postoperative day, having been repatriated to our hospital, she was clinically much improved even though

her arterial blood tensions while breathing room air were as follows: partial oxygen tension (paO₂) 4.9 kPa (normal range 10–14), partial carbon dioxide tension (paCO₂) 6.9 kPa (normal range 4.5–6.1), and oxygen saturation 70%, with concurrent transcutaneous oxygen saturation 72% (normal range 95–98%). Her clinical status continued to improve on diuretics, angiotensin converting enzyme inhibitors, bronchodilators, and supplemental oxygen. On her 25th postoperative day, lung function tests revealed a one second forced expiratory volume (FEV₁) of 0.86 litres (40% predicted), forced vital capacity (FVC) of 1.59 litres (61% predicted), and an FEV₁/FVC ratio of 54% (typically less than 70% in airflow obstruction). She could now perform a modified version of the "shuttle" walk² for a distance of 30 m briskly, without stopping for breath, and also without supplemental oxygen. Repeat arterial blood gas tensions on 30 March 2002 showed paO₂ 7.7 kPa and paCO₂ 5.8 kPa while breathing room air.

Comment

On the basis of age, comorbidity, shock at presentation, and endoscopic stigmata of recent haemorrhage, this patient had a high risk of death with or without surgical intervention.³ Only timely intervention and impeccable postoperative care could tip the scales in her favour, hence the successful outcome documented here.

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References

- 1 Wheatley KE, Snyman JH, Brearley S, *et al*. Mortality in patients with bleeding ulcer when those aged 60 or over are operated on early. *Br Med J* 1990;301:272.
- 2 Singh SJ, Morgan MDL, Scott S, *et al*. Development of a shuttle walking test of disability in patients with chronic airways obstruction. *Thorax* 1992;47:1019–24.
- 3 Rockall TA. Management and outcome of patients undergoing surgery after acute upper gastrointestinal haemorrhage. *J R Soc Med* 1998;91:518–23.

CORRECTIONS

The authors of Marchbank *et al* (*Gut* 2002;51:787–92) in the December issue of the journal, have noted a typographical error in their paper. In the results section, it should

state that the pH of the two subjects who were taking proton pump inhibitors who did not show a change to the larger form of TGFα had a pH of less than, and not greater than 4 as published. The authors apologise for the error.

The following errors occurred in the paper "Prospective study of liver dysfunction in pregnancy in Southwest Wales" in the December issue by Ch'ng *et al* (*Gut* 2002;51:876–80) as final author corrections were not included. The published version does not clearly distinguish those patients in whom pre-eclampsia was the sole identifiable cause of liver dysfunction from those in whom pre-eclampsia co-existed with another cause such as HELLP syndrome, obstetric cholestasis, or sepsis. The corrections are to the abstract, tables 1, 2, and 3, and part of the text of the results section on page 878 under the sub-heading "Diagnoses accounting for abnormal liver tests", and to two of the references. The journal apologises for the errors.

In the abstract, under methods, the second sentence should read "Patients with abnormal liver tests were assessed and followed throughout and after pregnancy. Medical advice was provided to obstetric teams."

Pregnancy specific conditions

Pre-eclampsia was a common underlying abnormality seen in 68 patients (48%) but was the sole identifiable cause of liver dysfunction in only 15. Complete⁶ and incomplete⁷ HELLP syndrome occurred in 30 patients of whom 29 were pre-eclamptic making this the commonest diagnosis accounting for abnormal liver tests. In most of those patients with incomplete or partial HELLP syndrome, the platelet count dropped abruptly by more than 50% in parallel with abnormal liver tests. OC was diagnosed in 23 patients (16%); two of whom had pre-eclampsia. Eleven patients (8%) had deranged LFT associated with hyperemesis gravidarum and all responded well to conservative management or oral steroids; none of these developed pre-eclamptic liver dysfunction later in pregnancy. AFLP was diagnosed in five patients (4%), three of whom needed prolonged post-natal hospitalisation because of multi-organ involvement; none of these five patients had pre-eclampsia. Clinical details of these patients have been presented and published recently.^{11 12}

In "Other contributory conditions" the first sentence should read "Seventeen patients had abnormal liver tests in association with sepsis,

Table 1 Aspartate aminotransferase (AST), gamma glutamyl transpeptidase (γGT), and bilirubin values for specific pregnancy related liver disorders

	AST (U/l)	γGT (U/l)	Bilirubin (μmol/l)
HELLP syndrome	66 (41–1123)	24 (6–209)	13 (4–155)
Obstetric cholestasis	210 (30–519)	29 (8–278)	14 (6–34)
Pre-eclamptic liver dysfunction alone	68 (36–210)	18 (7–51)	7 (3–12)
Hyperemesis gravidarum	51 (9–280)	23 (2–64)	25 (4–33)
AFLP	278 (86–542)	50 (22–209)	50 (19–61)

Values are median (range).

HELLP, haemolysis, elevated liver enzymes, low platelets; AFLP, acute fatty liver of pregnancy.

Table 2 Diagnoses accounting for abnormal liver tests

	n	No with ↑ AST	No with ↑ γGT	No with ↑ bilirubin	No with ↑ urate	No with low platelets
Group 1—Pregnancy specific						
HELLP syndrome (5 complete, 25 partial)	30	30	12	10	29	30
Obstetric cholestasis	23	22	10	2	17	0
Pre-eclampsia alone	15	14	4	0	13	0
Hyperemesis gravidarum	11	7	4	5	3	1
Acute fatty liver of pregnancy	5	5	3	4	5	3
Hepatic infarct/haematoma	1	1	1	1	1	1
Group 2—Other conditions						
Postoperative (caesarean section)	22	21	10	3	21	7
Sepsis	17	17	6	2	12	2
Placental pathologies	12	11	4	3	9	3
Diabetes	8	6	5	1	5	4
Drug related	4	1	4	1	N/A	2
Bile duct stones	3	3	2	2	1	0
Hepatitis C	2	2	1	0	0	0
Group 3—Diagnosis obscure	14	8	4	1	6	0

N/A, not available.

Table 3 Timing of liver dysfunction. Onset of specific pregnancy related liver disorders (in weeks' gestation)

HELLP syndrome	36 (25–38)
Obstetric cholestasis	35 (21–39)
Pre-eclampsic liver dysfunction	37 (25–40)
Hyperemesis gravidarum with liver dysfunction	9 (6–14)
AFLP	38 (32–38)

Values are median (range).

HELLP, haemolysis, elevated liver enzymes, low platelets; AFLP, acute fatty liver of pregnancy.

most commonly caused by urinary tract infection (9 patients); 5 of these 17 were also pre-eclamptic."

References 11 and 12 should be as follows:

- 11 **Ch'ng CL**, Morgan M, Kingham JG. Acute fatty liver of pregnancy in South Wales, UK. *J Hepatol* 2002;**36**(Suppl 1):186.
- 12 **Ch'ng CL**, Morgan M, Kingham JGC. Acute fatty liver of pregnancy in South Wales, UK. *Gastroenterology* 2002;**123**(Suppl 1):53.

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17th International Workshop on Therapeutic Endoscopy

This will be held on 3–5 December 2002 in Hong Kong. Further information: Professor SC Sydney Chung, Endoscopy Centre, Prince of Wales Hospital, Shatin, NT, Hong Kong. Tel: +852 2632 2233; fax: +852 2635 0075; email: info@hksde.org

Advances in the Inflammatory Bowel Diseases

This conference will take place on 6–7 December 2002 in New York, USA. Further information: Heather Drew, Imedex, 70 Technology Drive, Alpharetta, GA 30005-3969, USA. Tel: +1 770 751 7332; fax: +1 770 751 7334; email: h.drew@imedex.com; website: www.imedex.com

15th European Intensive Course (SMIER) Digestive Endoscopy

This course will take place on 16–17 December 2002 in Strasbourg, France. Further information: Michele Centonze Conseil, 6 bis Rue des Cendriers, 75020 Paris, France. Tel: +33 1 44 62 68 80; fax: +33 1 43 49 68 58.

The Future of Gastro-entero-hepato-pancreatology is bright

This Academic Farewell Symposium of Guido NJ Tytgat will be held on 12 December 2002 in Amsterdam, the Netherlands. Deadline for registration is 1 November 2002 (no registration fee) and registration should be done via email to: j.goedkop@amc.uva.nl.

Cancer of Oesophagus and Gastric Cardia: from Gene to Cure

This conference will be held on 13–15 December 2002 in Amsterdam, The Netherlands. Further information: European Cancer Centre, PO Box 9236, NL 1006 AE Amsterdam, The Netherlands. Tel: +31 (0)20 346 2547; fax: +31 (0)20 346 2525; email: ecc@ikca.nl

Imaging of the Abdomen: an Update

This will be held on 23–24 January 2003 in Amsterdam, the Netherlands. Further information: visit the website www.epgs.nl or email epgs@amc.uva.nl. Tel: +31 20566 3926/4386.

Surgery of the Foregut

This meeting will be held on 17–18 February 2003 in Florida, USA. Further information: Cleveland Clinic Florida, Office of CME, 2950 Cleveland Clinic Boulevard, Weston, FL 3331, USA. Tel: +1 954 659 5490; (toll free: +1 866 293 7866); fax: +1 954 659 5491; email: cme@ccf.org

NOTICES

The national register of hepatitis C infections with a known date of acquisition.

The register steering group invite clinical and epidemiological researchers to submit proposals to access data held in the register. It is envisaged that a variety of studies might benefit from linkage with or access to the register, and proposals from all specialties and institutions are welcomed. Any researchers interested in applying for access to information held within the national register should contact the register co-ordinator (see below) for a list of available data and an application form. Study proposals should then be submitted to the register co-ordinator by **16 December 2002**.