Injection of alcohol to control bleeding from ruptured hepatomas

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Spontaneous haemoperitoneum is an uncommon but dramatic surgical emergency. In areas with a high incidence of hepatoma it often results from rupture of the tumour.[1] Control of bleeding in such patients is often difficult, and the mortality is high.[1,2] We report on three patients with ruptured hepatoma who were successfully treated by injection of alcohol.

Case reports

Case 1—A 60 year old woman had a laparotomy for severe abdominal pain and shock, at which 2 litres of blood was drained from the peritoneal cavity. The liver was cirrhotic with an extensive nodular tumour encasing the porta hepatis. The tumour had ruptured with brisk bleeding from a 3 cm crater. Absolute alcohol (20 ml) was injected directly into the crater. The area blanched immediately, and the bleeding stopped. The postoperative period was uneventful except that she had a swinging fever for five days. She was discharged on the eighth postoperative day. She remained well for 10 months but died 14 months after surgery of extensive hepatoma.

Case 2—A 27 year old man presented with severe epigastric pain, shock, and peritonism. Laparotomy showed a 10 cm tumour at the dome of the right lobe of the liver. It had ruptured and was bleeding; 2·5 litres of blood was drained from the peritoneal cavity. There was also a 5 cm tumour in the left lobe. Alcohol (50 ml) was injected into the ruptured tumour. Bleeding was controlled. He had low grade fever for five days postoperatively and was discharged on the eighth postoperative day. He died seven weeks later of disseminated carcinomatosis and liver failure.

Case 3—A 53 year old man who was known to have a hepatoma suddenly developed severe abdominal pain and shock. At laparotomy an 8 cm ruptured hepatoma with profuse bleeding was found. The liver was severely cirrhotic. Ligation of the hepatic artery slowed but did not stop the bleeding. Absolute alcohol (40 ml) was injected into the lesion, and the haemorrhage stopped. He had swinging fever and a tachycardia for two days after the operation. He was discharged 21 days after operation. There was no evidence of rebleeding. He returned with disseminated carcinomatosis four weeks later and died seven weeks after the episode of bleeding.

Comment

Resection of the affected lobe of the liver has been recommended as the treatment of choice for a ruptured hepatoma whenever possible.[1] Unfortunately, few patients are suitable candidates for resection because either the tumour is too extensive or the liver is too cirrhotic. Ligation of the hepatic artery stops the bleeding but is associated with a mortality from liver failure of 50%.[2] In suitable patients angiographic embolisation can stop the bleeding without an operation.[3] This, however, requires a highly skilled radiologist, who may not be readily available at all hours in all hospitals.

Endoscopic injection of alcohol has been used to stop bleeding from oesophageal varices and peptic ulcers.[4] Absolute alcohol stops bleeding by a process of dehydration and fixation of the tissues followed by thrombosis of the blood vessels. Percutaneous injection of alcohol under ultrasonographic control has been used successfully to destroy hepatocellular carcinomas.[5]

In case 1 resection was not possible because the tumour straddled both lobes and ligation of the hepatic artery was not feasible because of infiltration of the porta hepatis. As no effective alternative was available we injected alcohol. Encouraged by the outcome in this patient we used the same method for the two other patients. In case 2 the bilobar tumour was not suitable for resection, and in case 3 the bleeding was not controlled after ligation of the hepatic artery. Injection of absolute alcohol into the lesion stopped the haemorrhage in both patients.

Intralesional injection of alcohol is cheap, does not require any special skill, and should be possible in any operating theatre. It is a useful treatment for this challenging condition.

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Adjustment of insulin doses of diabetic patients during long distance flights

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Travelling across several time zones changes a patient's schedule for insulin injections. Changes in their insulin requirement may be a concern for travelling patients as only rough guidelines for such changes have been published.[1] We advised patients on a regimen in which they increased their dose of insulin when travelling west and decreased it when travelling east. We report on the success of this regimen in terms of their control of blood glucose concentration and hypoglycaemic episodes.

Patients, methods, and results

We studied 27 patients with type I diabetes. Twenty patients were studied on one round trip, three on five eastward flights only, and four on four westward flights only. The average flight time and shift in time zone were 10·6 and seven hours respectively.

When travelling westwards the patients used their normal insulin regimen until departure and then followed it according to the local time after landing. The additional hours due to the shift in time zone were covered with one or two injections of short acting insulin with meals on the plane. When travelling eastwards the patients caught flights that left between 5 pm and 9 pm. After departure they took their normal

Mean (SD) number of injections and insulin dose during days at home and during westward and eastward travel

	Injections per day	Insulin dose (units)		
		Intermediate acting†	Short acting	Total
Westward travel:				
Normal day at home	3·3 (1·1)	23·7 (9·9)	11·3 (9·2)	35·0 (11·1)
Day of flight	4·8 (1·1)	25·1 (9·6)	17·5 (8·5)*	42·6 (11·1)*
Eastward travel:				
Normal two days at home	6·4 (1·8)	48·5 (19·2)	23·4 (6·8)	71·9 (22·1)
Day of flight and next day	6·3 (1·4)	40·7 (14·4)*	24·2 (13·2)	64·9 (19·0)*

*p<0·001 Compared with value on normal days at home (unpaired *t* test).
†Includes basal insulin dose in two patients using insulin pumps.

dinnertime or bedtime dose of intermediate acting insulin or a slightly reduced dose. In addition, they took an extra dose of short acting insulin (2-6 units) before the late evening meal on the plane. The subsequent morning dose of intermediate acting insulin was reduced because it was taken later than usual, according to the local time. Two patients, who used insulin pumps, maintained their normal basal infusion rate and took boluses of 4-6 units of insulin before the meals on the plane.

All patients were advised to take an extra injection of 2-4 units of short acting insulin if their blood glucose concentration increased above 15 mmol/l. They measured their blood glucose concentrations with a glucose reflectometer a mean of six (range four to 11) times on the day of the flight and, for comparison, a mean of five (three to six) times during a total of three normal days at home before travel (one for westward travel and two for eastward travel).

During westward travel the mean increase in the daily insulin dose above the normal value was 1·1 (SD 0·5) units or 3·1 (1·9)% of the total daily dose expressed per time shift hour (table). During eastward travel the mean reduction in the total insulin dose over two days was 1·0 (1·1) units or 2·6 (2·7)% of the total daily dose expressed per time shift hour. The mean blood glucose concentration during the day of the flight was similar during westward (8·6 (2·3) mmol/l) and eastward (9·4 (2·5) mmol/l) travel, but higher (p<0·01, paired *t* test) than the value during the days at home (7·1 (1·4) mmol/l). Altogether 2% and 5% of the measurements of blood glucose concentrations indicated hypoglycaemia (<3 mmol/l) and hyperglycaemia (>15 mmol/l) respectively; these proportions did not differ from those during days at home.

Comment

Our data show that an increase or decrease of 2-4% of their daily dose of insulin per hour of time shift during westward or eastward flights, respectively, was appropriate in our patients, though we did not study any other regimens. The average blood glucose concentration was 1·5 mmol/l higher during westward travel and 2·3 mmol/l higher during eastward travel than that during days at home. Hypoglycaemic and hyperglycaemic concentrations were no commoner during travel than at home.

For westward travel the shift in time zone should be covered by one or two extra injections of short acting insulin on the plane. The additional dose is about 20-30% (2-4% per time shift hour) of the total daily dose. For eastward travel the late evening meal on the plane is covered with an extra dose of short acting insulin (2-6 units). The subsequent breakfast dose of intermediate acting insulin should be reduced by 20-40% (3-5% per time shift hour), but the usual dose of short acting insulin can be injected. The late timing (in local time) of the morning injection means that an injection before lunch is not necessary in patients who normally have one.

In conclusion, careful planning of an insulin regimen together with monitoring of blood glucose concentration maintains good glycaemic control and increases the patient's safety during travel.

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Decreased salivary epidermal growth factor in rheumatoid disease: a possible mechanism for increased susceptibility to gastric ulceration

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The association between non-steroidal anti-inflammatory drugs and gastric ulceration is well recognised. Some workers have suggested that patients with rheumatoid disease are at greater risk of gastric ulceration than those with other forms of arthritis taking the same drugs.[1,2] Epidermal growth factor is a polypeptide secreted into saliva and present in gastric juice. It protects gastric mucosa from injury induced by aspirin and stimulates cellular proliferation, so promoting healing of mucosal ulceration.[3,4] Patients with rheumatoid disease are prone to disease of the salivary glands, and we wondered whether this might affect production of epidermal growth factor, so impairing mucosal defence and the response to injury. We examined this by measuring salivary secretion of epidermal growth factor in patients with rheumatoid disease and assessed the effect of the sicca syndrome on this secretion.

Patients, methods, and results

We recruited patients from the rheumatology outpatient department. Depending on the results of a standard Schirmer's test for the sicca syndrome, patients were allocated to one of four groups: rheumatoid arthritis (n=20), rheumatoid arthritis with the sicca syndrome (n=9), primary sicca syndrome (n=5), and controls (patients with musculoskeletal disorders other than rheumatoid arthritis or primary sicca syndrome) (n=20). The age of the groups was similar. Saliva was collected for 15 minutes, the volume measured, and an aliquot frozen for assay of epidermal growth factor. Immunoreactive epidermal growth factor was measured by radioimmunoassay (Biomedical Technologies, United States). Statistics were calculated with the Mann-Whitney U test and results expressed as medians (ranges).

The salivary concentration of epidermal growth factor was lower in the patients with rheumatoid arthritis (1·3 ng/ml, 1·0-4·0), rheumatoid arthritis plus the sicca syndrome (1·4 ng/ml, 0·46-2·7), and primary sicca syndrome (1·6 ng/ml, 1·2-2·5) than in the controls (3·0 ng/ml, 1·45-10) (p<0·001). The volume of saliva collected in 15 minutes was considerably lower in the patients with primary sicca syndrome (0·7 ml, 0·2-1·2) and with rheumatoid arthritis plus the sicca syndrome (1·2 ml, 0·8-2·2) than in the controls (5·7 ml, 2·6-9·4) (p<0·001), but the volume collected in the patients with rheumatoid arthritis alone (4·1 ml,