Non-operative management of ruptured liver in Bougainville

Group Captain John E Payne; Major Peter Tralaggan, RAAMC; Major Mark Harris, RAAMC; Captain Phillip Miller, RAAMC; Sergeant Derek Davis, RAAMC; and Lieutenant Colonel David Scott, RAAMC

THE COMBINED HEALTH ELEMENT (CHE) provided a field surgical capability in support of the Peace Monitoring Group in Bougainville from 1997 to 2001. Surgical capability consisted of one surgical team supported by basic pathology and medical imaging, one high dependency bed and a six-bed ward. The Australian Red Cross supplied blood on a fortnightly basis.

In such circumstances, where diagnostic and therapeutic support is at a bare minimum, management of severely injured patients follows basic pathways determined by clinical and pathophysiological criteria, and limited by available resources.

Non-operative management of ruptured liver resulting from blunt injury has become popular after recognition that similar management for splenic injuries generally had a successful outcome and modern imaging technologies improved the assessment of liver and associated injuries.[1-4]

Non-operative management is suitable for the stable patient who has no associated injuries requiring operative surgical therapy, regardless of the severity of the liver injury on computed tomography, or the degree of blood loss.[1,4]

Case report

AK, a previously well 15-year-old male, discharged a weapon (said to have been taken from a World War II Japanese aircraft) at home in Buin at 1400 hours on 26 August 2001. The breech block recoiled into the patient's lower sternum and xiphisternum.

The local medical officer in Buin was away. A request to the CHE was received from Buin Hospital for assistance at about 1800 hours. At the time there was torrential rain, and with the onset of nightfall aeromedical evacuation of the patient was not possible. At about 1900 hours the officer commanding was advised that the patient was "stable", his pulse rate was 120 per minute, blood pressure 110/70mmHg and his haemoglobin level was 50g/L. He had considerable abdominal pain. We advised Buin to attempt to transport the patient by road to Loloho.

Op Bel Isi, Combined Health Element, Loloho, Bougainville.
Group Captain John E Payne, Surgeon; **Major Peter Tralaggan, RAAMC**, Anaesthetist; **Major Mark Harris, RAAMC**, Emergency Specialist; **Captain Phillip Miller, RAAMC**, Radiographer; **Sergeant Derek Davis, RAAMC**, Pathology Technician; **Lieutenant Colonel David Scott, RAAMC**, Officer Commanding.
Correspondence: Group Captain John E Payne, 22 Squadron, RAAF Richmond, NSW 2755.

Logistic Support Element and Combined Health Element, Loloho, Bougainville (located in what was previously a copper concentrate warehouse owned by Bougainville Copper P/L).

The next morning, at about 0830, further advice from Buin Hospital was that the patient remained stable and that they had been unable to transport him because of the poor weather and road conditions. An Iroquois helicopter was sent, and the patient arrived at the CHE at about 1015 hours. Volunteers who were blood group O negative were readied for blood donation.

AK complained of abdominal pain and difficulty breathing. His blood pressure was 130/90mmHg, pulse 110 per minute, respiratory rate 30 per minute and temperature 38.3°C. He had a markedly distended abdomen, which was uniformly tender. There were abrasions over the lower sternum. Oxygen saturation was 92% and an electrocardiogram was normal. He was given oxygen and stabilised by the resuscitation team. A urinary catheter and nasogastric tube were placed. Chest and abdominal x-ray films were taken and blood was collected.

At the same time as AK arrived, a woman at full term with obstructed labour and fetal distress was admitted for urgent caesarean section.

X-rays showed clear lung fields with no evidence of free gas below the diaphragm. The bowel gas pattern was normal. The principal radiological findings were a one-third reduction in the thoracic volume by upward displacement of both hemidiaphragms. The haemoglobin level on admission was 50g/L, with a haematocrit of 18%. The blood group was A positive. Malaria screening was negative.

On re-examination there was no focal abdominal tenderness, and it was presumed that there was no active bleeding or hollow viscus perforation. Because of the marked distension

and pain, it was thought that the anaemia was caused by intra-abdominal bleeding from rupture of the left lobe of the liver (possibly by incomplete avulsion of segments I and II). The blood loss was estimated to be about four units.

Non-operative management continued; intravenous morphine for pain, Hartmann's solution and two units of cross-matched O negative blood were given. From the time of admission the patient was febrile. Although this may have been caused by the systemic inflammatory response to trauma, intravenous chloramphenicol was given as prophylaxis.

Over the next 24 hours the patient's abdomen remained painful and distended. There were few bowel sounds and no focal signs. He remained febrile (38.5°C), looked unwell and at one point his blood oxygen saturation fell to a Po_2 of 85mmHg. Hourly chest physiotherapy with a peak flow meter was enforced. The urine output was maintained.

An erect chest x-ray on Day 2 showed patchy changes in the left lower zone, elevation of both hemidiaphragms, a small right-sided pleural effusion, and again no gas under the diaphragm. The results of haematology and biochemistry tests are shown in the Table. A further two units of blood were transfused.

Because of persistent high fever and abdominal pain, chloramphenicol was replaced with intravenous metronidazole and ampicillin. On Day 3, the fever slowly resolved and bowel function returned. The patient was given "Sustagen" and then "Ensure" (Abbott) by nasogastric tube. By Day 7 he was comfortable enough to walk. During the next two days he was afebrile, became more mobile and began oral feeding. His abdomen remained distended, but pain free.

Results of haematology and biochemistry tests for a patient with intra-abdominal bleeding caused by rupture of the left lobe of the liver

	Days after injury			
	1	2	5	6
Haemoglobin (g/L)*	94	94	96	102
Haematocrit	0.29	0.30	0.30	3.2
White cell count (10^6/L)	16.5	13.6	—	12.8
Platelets (10^9/L)	94	126	208	299
Granulocytes (10^9/L)	12.7	10.7	—	9.5
Lymphocytes (10^9/L)	3.8	2.9	—	3.3
Bilirubin (μmol/L)	10.3	9.1	19.7	13.0
γ-Glutamyl transferase (U/L)	10.5	14.9	40.3	40
Alanine aminotransferase (U/L)	757	226	291	242
Alkaline phosphatase (U/L)	194	163	226	214
Aspartate aminotransferase (U/L)	378	123	—	37
Creatine kinase (U/L)	251	321	—	—
Uric acid (mmol/L)	269	140	—	—
Urea (mmol/L)	3.40	—	—	—
Creatinine (mmol/L)	81	63.2	—	73
Amylase (U/L)	84	175	744	721
Pancreatic amylase (U/L)			(556)	(595)

* After transfusion of 4 units of blood

Ballistics

Investigations suggested that the weapon may have been a 20mm aircraft cannon. It was known that the Mauser MG151 was developed in the 1930s and used by the Germans and Japanese in the Second World War. The Bougainvilleans had a large number of weapons which were left after the Japanese withdrawal. If this was the weapon, then (according to *Jane's Ammunition Handbook, 2000–2001*[5]) the muzzle velocity was 720m/s and the projectile may have weighed 110g. If the patient was struck by a cycling breech block whose mass was about 2kg and the weapon was positioned close to his chest, the force could have been sufficient to rupture the liver between segments III and I–II.

Discussion

The CHE was confronted simultaneously with three patients, all of whom required intensive care for an indefinite time, two of whom might have required all the available reserves of blood.

With the benefit of hindsight, it might be claimed that the patient's management was predetermined by the delay in transport to appropriate care, and that from the time of arrival there should not have been much difficulty in deciding what to do. Nevertheless, he was a small adolescent with a very large intraperitoneal bleed, and no computed tomography was available. The massive abdominal distension caused respiratory difficulty.

The main risk was intraperitoneal or extraperitoneal gut perforation. The absence of free gas on radiography of the chest and abdomen suggested intraperitoneal gut perforation was unlikely. Occult perforation, which is accompanied by a gradual onset of subtle changes in physical and vital signs, is even more difficult to diagnose early when signs from the rupture of solid viscera are so dominant.

The probability of gut perforation in the literature is 1%–7%.[6,7] Patients with liver injuries of this magnitude frequently develop bile peritonitis, which requires drainage. Furthermore, rupture of an enlarged malarial spleen was a possibility, but less likely, as the patient had no evidence of recent or active malaria. Rupture of a non-malarial spleen would be managed non-operatively.[8-11]

The decision not to operate was justified by the lack of a further fall in haemoglobin level and absence of peritonitis. Other contingencies which reinforced this decision were the relatively short supply of A positive blood, as the banked

blood was Group O. Fresh blood from ADF volunteers was available, and could have been used to compensate for a potential coagulopathy from platelet consumption (Table). Judicious laparotomy may not have caused hepatic bleeding, but, if there had been significant haemorrhage, it would have been necessary to resort to a "damage control" approach,[10] undesirable when there were no resources outside the CHE for protracted management of its various sequelae, such as respiratory insufficiency and abdominal compartment syndrome.

In the absence of proof, the diagnosis of ruptured liver is supported by what was known of the mechanism of injury, and reports in the literature.[2] The changes in liver enzyme and bilirubin levels are also consistent with hepatic cellular disruption and resorption of blood and bile from the peritoneum. The patient may also have had myocardial contusion, but, as there was no arrhythmia, this possibility did not contraindicate surgery. Serum troponin T measurements were not available.

Treatment with antibiotics in such cases is arguable. The fever present on admission was almost certainly caused by the systemic inflammatory response syndrome,[12] as the pulse rate was greater than 90 per minute, the white blood cell count greater than 12×10^9/L, and the respiratory rate was rapid. We had no capacity to perform blood cultures. There was evidence of pulmonary atelectasis. Without computed tomography, it was not possible to know whether there was a sequestrum of devitalised liver, or other occult visceral damage.

There was some apprehension when the serum amylase level became elevated five days after injury. Pancreatic disruption is possible with this kind of injury, and serum amylase levels are normal in about 45% of cases.[7] The patient improved clinically, with no symptoms or signs of peritonitis at discharge.

References

1. Richardson JD, Franklin GA, Lukan JK, et al. Evolution in the management of hepatic trauma: a 25 year perspective. *Ann Surg* 2000; 232: 324-328.
2. Boone DC, Federle M, Billiar TR, et al. Evolution of management of major hepatic trauma: identification of patterns of injury. *J Trauma* 1995; 39: 344-350.
3. Strong RW. The management of blunt liver injuries. *Aust N Z J Surg* 1995; 69: 609-616.
4. Parks RW, Chrysos E, Diamond T. Management of liver trauma. *Br J Surg* 1999; 86: 1121-1135.
5. Gandex T, Cutshaw C, editors. Jane's Ammunition Handbook, 2000–2001. Jane's Information Group, 2001. ISBN 0710623089.
6. Allen GS, Moore FA, Cox CS Jnr, et al. Hollow visceral injury and blunt trauma. *J Trauma* 1998; 45: 69-78.
7. Nance ML, Peden GW, Shapiro MB, et al. Solid viscus injury predicts major hollow viscus injury in blunt trauma. *J Trauma* 1997; 43: 618-623.
8. Mehall JR, Ennis JS, Saltzman DA, et al. Prospective results of a standardised algorithm based on haemodynamic status for managing paediatric solid organ injury. *J Am Coll Surg* 2001; 193: 347-353.
9. Rotondo M, Schwarb CW, McGonigal M, et al. Damage control: an approach for improved survival in exsanguinating penetrating abdominal injury. *J Trauma* 1993; 35: 375-383.
10. Pachter HL, Guth AA, Hofseteler SR, et al. Changing patterns in the management of blunt splenic trauma: the impact of non-operative management. *Ann Surg* 1998; 227: 708-719.
11. Peitzman AB, Heil B, Rivera L, et al. Blunt splenic injury in adults: multi-institutional study of the Eastern Association for the Surgery of Trauma. *J Trauma* 2000; 49: 177-189.
12. Bone RC, Balk RA, Cerra FB, et al. American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference. Definitions for sepsis and organ failure and guidelines for use of innovative therapies in sepsis. *Chest* 1992; 101: 1644-1655.

(Received 18 Nov 2001, accepted 17 Dec 2002) ❑

ADF Health editorial policy on disclosure of conflicts of interest

Authors who submit articles for publication in *ADF Health* are now asked to make a Conflict of Interest statement with their submission. From the next (September) edition of *ADF Health*, a Conflict of Interest statement will be published with every article.

The purpose of the Conflict of Interest statement is to ensure that any factors — personal relationships, financial connections, sponsorships — that might bias the author of an article are declared so that readers are aware of the potential conflict of interest and can include that knowledge in assessing the information presented to them.

To prevent ambiguity, authors must state explicitly whether potential conflicts do or do not exist. Stating a conflict of interest does not disqualify an author from publication.

For more information about conflict of interest and how it is managed by *ADF Health* and other medical journals, see the *Uniform requirements for manuscripts submitted to biomedical journals,* published by the International Committee of Medical Journal Editors (available at http://www.icmje.org/index.html#conflict).

— Air Vice-Marshal Bruce Short
Editor, *ADF Health*