HEPATIC ARTERY LIGATION IN LIVER TRAUMA

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INTRODUCTION

HAEMORRHAGE is the main cause of death in patients with liver trauma. Presently, treatment of ruptured liver is mainly done in large General Hospitals where surgeons and anaesthesiologists are easily available and where blood is plentiful. But nearly all accident victims are transported to the nearest and smaller hospitals. By the time these patients are transfered to the bigger hospitals, these patients are more often than not, dead.

Hepatic hemostasis, a life saving procedure, can be obtained in a number of ways. Hepatic Artery Ligation (HAL) is the easiest, most rapid and simplest technique for treating hepatic trauma described in this century. It requires no special instruments and any qualified surgeon can do HAL in any operating theatre. (May, 1977).

In this paper, we make a report, the first from Sabah. of two cases of HAL done on patients who came in with ruptured livers.

CASE REPORTS

Case I

In early October 1977, a 25 years old Bugis male labourer was brought to the General Hospital Tawau, with a history of being hit on the left side of the face and the upper abdomen by a log occuring about 1 hour previously. On admission he was pale and had a poor volume pulse of 130 per minute and a blood pressure of 90/50 mm Hg. Abrasions were present on the left cheek and upper abdomen. There was guarding on abdominal palpation. Blood was aspirated from the peritoneum.

At operation shortly after admission, about 1500 c.c. of fresh blood was present in the peritoneum. a large transverse rupture across the right lobe, extending to the porta hepatis was found. Active bleeding was present. Control was easily affected by occluding the right hepatic artery with vascular clamps. The artery was then tied and no further bleeding occurred on removal of the clamps. A cholecystectomy was then done. A total of 5 pints of blood was transfused. A liver function test ordered the next day gave the following result:-
Serum bilirubin 4.6 mg/dl, SGPT: 220 SFU/ml; Total protein:- 6.8 gm/dl, Serum Albumin:- 3.5 gm/dl, Globulin:- 3.3 gm/dl and Alkaline Phosphatase:- 1.3 su/ml. About 3 weeks later serum bilirubin was 1 mg/dl, SGPT:- 43 sfu/ml, Total proteins:- 6.3 gm/dl, Serum albumin:- 2.6 gm/dl and Serum globulin 3.7 gm/dl, alkaline Phosphates:- 4.3 su/ml, The patient was discharged after 7 weeks.

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Case 2

A 23 years old male Toraja was admitted to the Tawau General Hospital in July 1978, after a road traffic accident occuring at about 8 hours previously. He was refered to us from the Lahad Datu District Hospital, about 150 miles from Tawau. His pulse was 110 per minute and the Blood Pressure was 110/60 mm/Hg. There was generalised abdominal tenderness and guarding.

At operation the patient was found to have ruptured the right lobe of the liver, a tear in the right dome of the diaphragm, a small rupture in the upper jejunum and the peritoneal cavity contained about 4.5 litres of blood. Bleeding was controlled by ligating the common hepatic artery. Part of the right lobe of the liver was resected and the remaining part sutured. The tear in the diapragm and the bowel was repaired. The gall bladder was removed. During the operation 6 pints of blood was transfused.

The patient's convalescence was complicated by the development of renal failure. This was successfully dealt with. 3 days after the operation the serum bilirubin was 2 mg/dl, SGPT 180 sfu/ml, total proteins 7.7 gm/dl, serum albumin 2.4 gm/dl, serum globulin 5.3 gm/dl, alkaline phophatase 4.9 su/dl, serum globumin 5.3 gm/dl, alkaline phophatase 4.9 su/ml. When repeated 1 month later, serum bilirubin was 0.6 mg/dl, SGPT 9 sfu/ml, total proteins 6.7 gm/dl serum albumin 1.9 gm/dl, serum globulin 4.8 gm/dl, Alkaline phosphatase 2.5 su/ml.

DISCUSSIONS

]Intentional] HAL has been considered from time immemorial, to be radical depature from

acceptable medical practice. These unfounded fears were based on extrapolation from experimental work on animals, the early work of Graham and Connell (1933) and the errorneous belief that hepatic arteries were end arteries.

Hepatic surgery requires precise knowledge of anatomy and a clear understanding of hepatic metabolism. (Adson and Beart, 1977). The location, configuration and the complexities of the hepatic vasculature are added risks in liver resections.

There are many indications for HAL. The most common and most important is to stop bleeding from a traumatised liver. HAL is a quick, simple and highly effective means of stopping haemorrhage from the ruptured liver (Aaron *et al,* 1975). HAL, besides being simpler than applying absorbable haemostatic agents and major hepatic resections, is also most useful when bleeding points are deep in the substance of the liver (Madding *et al.,* 1977). There is virtually no place in modern surgery for gauze packing of the liver as sepsis and recurrent bleeding are almost inevitable sequelae (Walt, 1969). It must no doubt be emphasized that in the event of a massive destruction of liver tissue, there remains no reasonable alternative to a hepatic lobectomy.

The right or left hepatic artery may be ligated if the lesion if situated in an unequivocally definable anatomical division, but ligation of the proper hepatic artery or common hepatic artery is also acceptably safe (Walt, 1969).

The gall bladder should be removed when any hepatic artery is ligated, for it produces stasis of the bileflow or varying degree of obstruction and inflammation (Mays, 1977).

As the liver is the main metabolic centre of the body, metabolic disturbances invariably occurs after hepatic resections. Following large resections, metabolic deficiencies may results, namely, hypoglycemia, hypoalbuminaemia and bleeding diathesis. Basically, the postoperative care of patients with hepatic injury is supportive in nature (Madding *et al.,* 1977). When the hepatic artery is ligated, hepatic hypoxia occurs. To avoid this, blood flow and oxygen content of the portal blood must be sustained at optimal levels for the first few post-operative days (May, 1977; Walt, 1969).

Though liver functions tests are available to detect and evaluate disorders of the liver, many of these tests measure activities not confined to the liver (Rosoff and Rosoff, 1977). Four laboratory tests, namely blood glucose, serum albumin, serum bilirubin and blood ammonia, reflect with moderate accuracy the overall state of liver function (Stone, 1977).

Intravenous glucose supplementation, which may be required for as long as three weeks, corrects the hypoglycaemia. Stone (1977) believes oral alimentation will usually provide sufficient free carbohydrate by the tenth postoperative day.

As the liver is the only endogenous source for human albumin, any disturbance to the metabolic activity of the liver, will result in hypoalbuminaemia. It may take four to six months before the serum albumin passes into the normal range. This, Walt (1969) views, reflects on the regenerative capacity of the liver.

Transient mild elevations in serum bilirubin are of little concern (Adson and Beart, 1977). This may reflect the swelling of the regenerating hepatic cells (Walt, 1969).

The liver will regenerate completely in about six months, provided the remaining of the liver parenchyma is viable (Stone, 1977, Walt 1969).

Although we acquire yearly new and more sophisticated aids to surgery, they are but additional means to apply these principles which, if one simplify even further, rest upon nothing more complicated than a sound knowledge of anatomy and the ability to apply an adequate modicum of good, honest common sense (Smith, 1978). We tend to agree.

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