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Modified Hassabs Procedure for control of Esophageal Varices in Portal Hypertension - A Prospective Study.

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ABSTRACT

Background: Modified Hassab's surgery is one of the Devascularisation procedure for controlling hemetemesis due to oesophageal varices. It has less chances of encephalopathy and mortality. We have studied 20 patients of oesophageal varices with portal hypertension and operated for Modified Hassabs Procedure. **Aim:** To determine the efficacy of Modified Hassab's procedure for portal hypertension, due to causes other than Schistosomiasis, to determine the short term outcome and long term follow up of patients treated with this procedure regarding oesophageal varices, rebleeding, liver function, ascitis, encephalopathy and mortality following Modified Hassab's procedure. **Methods:** This study was conducted at a single teaching hospital in Mumbai over a period of six years. 20 patients were studied for immediate and long term complications. **Results:** Oesophageal varices resolved in 11 patients (55%), regressed in 7 patients (35%), Fundic varices completely resolved in all the 4 patients. There was rebleed in two patients and was treated by sclerotherapy. There was no encephalopathy or mortality. **Conclusion:** We conclude that Hassabs Procedure is effective, and good control of varices can be achieved in portal hypertension patients due to Portal Vein Thrombosis and Portal Cavernoma, Cirrhosis of liver, Non Cirrhotic Portal Fibrosis.

Keywords: Hassabs Surgery, Modified Hassabs Surgery, Portal Hypertension.

INTRODUCTION

Devascularisation surgery is one of the most effective way of arresting hemorrhage from oesophageal varices. While the bleeding is effectively controlled, there is no incidence of hepatic encephalopathy and metabolic complication because of maintenance of splanchnic and systemic haemodynamics in these patients. The present study has been conducted on 20 patients of portal hypertension of various etiologies, in whom Modified Hassab's procedure was done for bleeding oesophageal varices. Various aspects in the follow-up of these patients have been discussed to establish the effectiveness of the procedure.

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Hassabs procedure consists of Devascularisation of the upper half of the stomach and oesophagus with Splenectomy. The whole proximal stomach is then devascularised from the terminal two branches of the left gastric artery at the incisura angularis upwards by ligation and division of the lesser and greater omentum, and of the posterior gastric adhesions. After division of the oesophagogastric reflection of peritoneum and mobilization of the vagus nerve, the distal 7 to 8cm of oesophagus is mobilized and all feeding vessels are ligated and divided. The distal 3cm of oesophagus and proximal 5cm of the stomach may then be open longitudinally, thus displaying the varices and allowing obliteration of each variceal column by underscoring from as high as possible within the oesophagus with an absorbable suture. After positioning of a nasogastric tube, the oesophago -gastrostomy is carefully closed by suturing or stapling.[1] In our study Modified Hassab's Procedure was used.

Modified Hassab's Procedure:[2] This consists of:

1. Splenectomy
2. Perhiatal Devascularisation of the lower 3 – 4 inches of the oesophagus
3. Ligation of ascending branch of left gastric artery and vein.
4. Devascularisation of the proximal half and separation of stomach from its bed.

In Modified Hassab's Procedure, the oesophagus and stomach are not opened to undersew the varices from inside the lumen.

MATERIALS AND METHODS

This study was conducted at a single teaching hospital in Mumbai over a period of six years. Twenty patients underwent surgery. Patients were investigated for portal hypertension on the basis of history, clinical examination, biochemical tests for hepatic function, radiological examination and endoscopic examination.

Inclusion Criteria: All male and female,Age group – all, Haemetemesis due to Oesophageal and fundal Varices, Patients with failed endotherapy (sclerotherapy/Bands ligation), Patients with portal hypertension, splenomegaly and hypersplenism, Patients in childs criteria A and B.

Exclusion Criteria: Haemetemesis due to causes other than Oesophageal and fundal varices, Patients with Child C criteria and patients with gross ascites.

The records were maintained in the form of proforma below:

1. **History:** Enquiries were made regarding age, sex presenting complaints, presence or absence of gastrointestinal hemorrhage and jaundice. Any addiction to alcohol was noted. These points were verified after the other causes of haematemesis were eliminated namely gastritis, peptic ulcer.
2. **Examination:** Pallor, icterus was noted. On clinical examination of abdomen, the status of liver, spleen and presence of ascitis was looked for and so was evidence of liver cell failure.
3. **Investigations:**
   Biochemical investigation: Haemoglobin, Total count , Prothrombin time (PT), Platelet count, Serum bilirubin / Direct bilirubin, Total proteins/ albumin, BUN / creatinine.
   Radiological investigations: Ultrasonography and liver scan was done for all the patients. Doppler study was carried out in only one patient and only two patients underwent bone marrow biopsy.
   Upper GI Endoscopy: All patients underwent upper GI Endoscopy pre and post operatively.
   Only patients classified as Child's A and B were considered for surgery. Indications for performing Hassab's procedure in portal hypertension patients were portal vein thrombosis, portal cavernoma, and hypersplenism.
   The operative technique we used is the same as that described for Modified Hassab's procedure with minor modifications. The Modified Hassab's procedure includes: 1) Perhiatal devascularization of the lower 3–4 inches of the oesophagus, 2) Ligation of the left gastric artery, 3) Devacularisation of the proximal half of the stomach and separation of the stomach from its bed, 4) Splenectomy.
   Our modifications were:
   1) Upper Midline with connecting Left subcostal abdominal incision.
   2) Placement of over and over muscular sutures over lower 1inch of oesophagus at three different levels and ligation of upper tributary of left coronary vein was done. This causes obliteration of portal veins going and feeding oesophageal plexus of veins.
   3) Intra operatively, somatostatin at the rate of 20µ drops/min.
   There are dense collaterals in a case of portal hypertension so these modification have helped in better operative field exposure, mobilization of spleen and high ligation of oesophageal collaterals. Somatostatin helped to reduce intra operative blood loss.
4. **Intraoperative findings:**
   The intra operative findings considered noteworthy were – Liver size and surface – presence of nodularity, Splenic size, Condition of portal and splenic veins, Other abnormal dilated veins, Presence of ascitis.
5. **Post operative and follow up study:**
   Patients were routinely followed up in the post operative period with clinical and laboratory evaluation after 6 weeks and later after every 6months.
   Rebleeding, encephalopathy were recorded. The complications of the procedure, sequelae and outcome of treatment were noted. Based on the results we were able to assess the efficacy of the Modified Hassab procedure, for control of haematemesis in patients of portal hypertension.

RESULTS

Peak incidence of portal hypertension was seen to be in age group of 11 to 20 yr., constituting 65% of cases. We had patients from age group of 11 to 50 years. Both male and female patients were equal in number. Lump in left side of abdomen was present in 95% of the cases. All patients had history of haemetemesis, 70% cases gave history of loss of appetite,19 (95%) had distension of abdomen, lump in Lt. Hypochondrium (95%), Jaundice in 8 patients, pain in abdomen in 8 patients. Splenomegaly was present in all the patients. About 75% of the patients had evidence of pallor, while icterus was seen in only 15% of the patient, ascitis was found to be present on abdominal examination in 20% of patient's, hepatomegaly was present in only 10% of patients. None of the patients showed presence of asterixis. Hemoglobin was less than 5mg% in 15 patients. White blood count was less than 3,500 / cumm in 17 patients and platelet count was less than 10,0000 in 15 patients. Prothrobin Time was prolonged in all the patients. It was grossly prolonged that is more than 6 seconds. in 35% of patients while it was prolonged for 4 – 6 sec. in 25% of patients. Albumin level was decreased in 75% of the patients. It was less than 3gm% in 20% of our patients, while only 25% of the patients had albumin level more than 3.5gm%. Sr. bilirubin was less than 2mg/100ml in 85% of the patients, while it was between 2 – 3 mg / 100ml in 15% of the patients.

Eleven(55%) of patients in our study belonged to Child's B class, while 45% patients were into Child's A class. Ultrasonography of abdomen revealed that splenomegaly and portal cavernoma was present in all the patients. Non cirrhotic portal fibrosis (NCPF) was present in 60% of the patients. Cirrhosis of liver was seen in 25% patients. Evidence of portal vein thrombosis was present in 15% of patients. Minimal ascitis was present in only 20% of the cases. Liver echotexture was normal in 75% cases [Table 1]. About 30% of the patients in the study group had grade IV oesophageal varices preoperatively as seen on upper GI Endoscopy, 20% had grade III + grade IV oesophageal varices. Another 20% had grade III oesophageal varices, 10% of the patients presented with fundic varices only, 10% of the patients had combination of oesophageal varices and fundic varices. Post Operatively ,early complications (0 – 30 days) like intraperitoneal bleeding leading to hypotension due to slippage of ligature , rebleeding, evidence of sepsis, acute pulmonary oedema, multiple organ failure was not observed. Late complications like post splenectomy opportunistic infection, hepatic encephalopathy, incidence of incisional hernia, mortality post operatively was not observed. Haemoglobin, white blood cells, platelet count which were low pre-operatively normalized in 48 hours post-operatively. Sr. bilirubin level of three patients were raised preoperatively which returned to normal level in first week after surgery. 15 patients had Sr. albumin at lower than normal value preoperatively had improvement in albumin value 2 weeks postoperatively.

Oesophageal varices resolved in 11 patients (55%), regressed in 7 patients (35%). Fundic varices completely resolved in all the 4 patients with isolated fundic varices as well as in combination with oesophageal varices [Table 2].

Among all the 20 patients the maximum duration of follow up was 5 years and recently operated patients had follow up, upto 6 months post operatively. 2 (10%) patients had rebleeding,after 4 year post operatively which was successfully treated by endoscopic sclerotherapy.

Table 1: Findings of ultrasonography of abdomen

USG findings	No. of patients	Percentage (%)
Cirrhosis of liver	05	25%
NCPF	12	60%
Normal liver	15	75%
Portal cavernoma	20	100%
Portal vein thrombosis	03	15%
Splenomegaly	20	100%
Ascitis	04	20%

Portal Cavernoma was seen in all the studied patients

Table 2: Post operative status of varices as seen on upper Gastrointestinal Scopy.

No.	No. of patients	Preop. Upper GI scopy findings	Post op. Upper GI scopy findings
1	02	Grade II oesophageal varices	Nil
2	04	Grade III oesophageal varices	Nil
3	04	Grade III + IV oesophageal varices	1 patient had grade I oesophageal varix
4	06	Grade Iv oesophageal varices	1 patient had grade III oesophageal varix. 4 patients had grade II oesophageal varices. 1 patient had grade I oesophageal varix
5	02	Fundic varices	Nil
6	02	Grade III oesophageal varices + fundic varices	Nil

Good regression in grade of varices was seen in almost all patients.

DISCUSSION

Therapy for portal hypertension and variceal bleeding has evolved over the past 100 years. The many treatment modalities available suggest that, no single therapy is entirely satisfactory for all patients, or for all clinical situations. Non-operative treatments like endoscopic sclerotheapry, or band ligation are generally preferred for acutely bleeding patients since they are at high operative risks because of decompensated hepatic function. Therapies that are effective (a low bleeding rate) and minimally alter hepatic physiology are optimal for long-term prevention of recurrent bleeding. The goal of surgical treatment of portal hypertension is the control of variceal bleeding without the induction of hepatic failure. Preservation of portal blood flow to the liver is essential to obtain satisfactory results, which is why selective shunts such as, the Warren shunt are considered the surgical treatment of choice in preventing recurrent variceal haemorrhage.[3,4] So the advantage of selective shunts over non selective shunts was low chance of Encephalopathy. But the effectiveness of preventing recurrent bleeding is same as nonselective shunt. So the goal at controlling variceal bleeding without induction of hepatic failure can be achieved by nonshunt surgeries also. The objectives of nonshunt operations are, either ablation of varices, or more commonly, extensive interruption of collateral vessels connecting the high pressure portal venous system with the varices. Oesophago gastric

devascularization procedures combined with oesophageal transection and splenectomy is most effective nonshunt operation.[5]

Sugiura – Futagawa procedure is the most popular nonshunting procedure to treat bleeding varices since 1973. Oesophageal transection is one of the most important steps in that procedure however, it has high morbidity and mortality if esophageal fistula occurs (about 5 to 8%).[6]

Ours is a heterogeneaous group of patients with portal hypertension. Different etiologies and diverse degrees of liver functioning are present.

In his study Wu Yk, found that post hassabs procedure, oesophageal varices were resolved in 8 patients (62%), diminished in size in 3 patients (23%) and remain unchanged in 2 patients (15%). Gastric varices disappeared in all patients,[7] where as in our study oesophageal varices resolved in 11 patients (55%), regressed in 7 patients (35%), Fundic varices completely resolved in all the 4 patients with isolated fundic varices as well as fundic varices in combination with oesophageal varices.

Hassab in his study of devascularisation surgery had a rebleed rate of 5% in 3 years and & 7 % in 10 years of follow up, whereas the rebleed rate is 17-18 % if hassabs procedure is not done properly.[8] We had two patients presenting with rebleed 4 years after surgery which was managed by sclerotherapy. Wu YK had 23 % of rebleed rate.[7]

Enchephalopathy was mild in Hassabs study, whereas none of our patient had post operative encephalopathy.[8] Gastro-esophageal decongestion surgery with splenectomy(Hassab) is as effective in controlling bleeding as DSRS with better liver, better heart, better life and better survival with nil or minimal encephalopathy. Combined with sclerotherapy it forms the ideal therapy for bleeding varices in all types of pathology.[8] There was no post-operative mortality in our study, similar findings were there in Wu YK's study.

Doppler color flow imaging for demonstrating changes in portal hemodynamic after Hassab procedure showed that the operation can decrease blood flow in the gastrosplenic region and enhance liver perfusion.[8,9]

A modified Hassab procedure in the management of bleeding esophageal varices may be applied with a modified gastroesophageal decongestion, and splenectomy can be performed in patients who need immediate surgical intervention for variceal bleeding.[7]

Hassab's operation is effective in controlling bleeding by portal vein flow restoration and in hepatic encephalopathy correction by ligation of a massive splenorenal shunt with better quality of life and survival and no or minimal encephalopathy.[8]

CONCLUSION

Our studies revealed effective control of oesophageal varices after doing Hassabs Surgery. The total re-bleeding rate was 10% which was effectively controlled by endoscopic sclerotherapy. There was no postoperative encephalopathy or mortality. Hassab's procedure is a simple and effective procedure for control of hemetemesis with oesophageal varices due to portal hypertension, other than due to schistosomiasis.

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