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Surviving Sengstaken

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ABSTRACT

Aim of the Study: To report the outcomes of children who underwent Sengstaken–Blakemore tube (SBT) insertion for life-threatening haemetemesis.
Methods: Single institution retrospective review (1997–2012) of children managed with SBT insertion. Patient demographics, diagnosis and outcomes were noted. Data are expressed as median (range).
Main Results: 19 children [10 male, age 1 (0.4–16) yr] were identified; 18 had gastro-oesophageal varices and 1 aorto-oesophageal fistula. Varices were secondary to: biliary atresia (n = 8), portal vein thrombosis (n = 5), alpha-1-anti-trypsin deficiency (n = 1), cystic fibrosis (n = 1), intrahepatic cholestasis (n = 1), sclerosing cholangitis (n = 1) and nodular hyperplasia with arterio-portal shunt (n = 1). Three children deteriorated rapidly and did not survive to have post-SBT endoscopy. The child with an aortooesophageal fistula underwent aortic stent insertion and subsequently oesophageal replacement. Complications included gastric mucosal ulceration (n = 3, 16%), pressure necrosis at lips and cheeks (n = 6, 31%) and SBT dislodgment (n = 1, 6%). Six (31%) children died. The remaining 13 have been followed up for 62 (2–165) months; five required liver transplantation, two underwent a mesocaval shunt procedure and 6 have completed endoscopic variceal obliteration and are under surveillance.
Conclusions: SBT can be an effective, albeit temporary, life-saving manoeuvre in children with catastrophic haematemesis.

The causes of upper gastrointestinal (GI) bleeding in children include gastro-oesophageal ulcers and erosions (often related to drugs), Mallory–Weiss tears, vascular malformations and, less commonly, gastro-oesophageal varices (GOV) [1]. Geographical variation in the epidemiology is well recognised. An American study (n = 167) reported that the most common findings noted on endoscopy for upper GI bleed included gastric erosions/ulcers (10.8%), erosive oesophagitis (9.5%)and duodenal ulcers/erosions (8.2%). Varices were seen in only 7% of this series [2]. The commonest cause in an endoscopic series reported from Hong Kong (n = 76) was duodenal ulceration (75%) [3]. By contrast, varices were the most common cause (95%) of an acute upper GI bleed reported in a series (n = 139) reported from India [4] and accounted for 26% in a small Canadian study (n = 27) [5].

Direct methods to control variceal bleeding were first described by Westphal in 1930 (reported by Read) with the use of an oesophageal sound placed to apply direct intraluminal pressure. Inflatable oeosphageal tubes were then described [6] and these culminated in the definitive description of the double balloon tube in 1946 by Sengstaken and Blakemore in a series of 30 patients. The tube allowed independent inflation of a gastric and oesophageal balloon with a distal lumen to aspirate the stomach [7]. The current version of this device comes in various sizes and is depicted in Fig. 1.

The definitive management of oesophageal varices in children depends on the nature of the underlying liver disease. In those with portal vein thrombosis (PVT), portosystemic shunting or the Meso-Rex bypass [8] may be possible. Liver transplantation may be indicated in those with cirrhotic livers. Emergency treatment for bleeding from GOV bleed, however, is predominantly endoscopic and designed to control acute haemorrhage, and then thrombose and obliterate the variceal columns. In suitable patients, this may be achieved by repeated injection sclerotherapy [9] or banding [10]. In some children however the bleeding is so severe that these interventions are not feasible, safe or effective and the SBT is required to control blood loss and allow the patient to be resuscitated and stabilised.

There is a paucity of data regarding the use of SBT in children [11,12]. Our aim was to report our experience with the device and review the outcomes of a recent cohort of children who underwent this intervention at our centre.

2. Methods

Following institutional approval (CG ID: 3093) a retrospective review of children (≤16 years) managed with SBT insertion between January 1997 and December 2012 was undertaken. Patient demographics, details of SBT insertion, management and outcome were noted. The degree of underlying liver failure was assessed; a paediatric end stage liver disease (PELD) score for children ≤12 years was calculated from age of patient, serum bilirubin, albumin and INR using an online tool (MD calc) in SI

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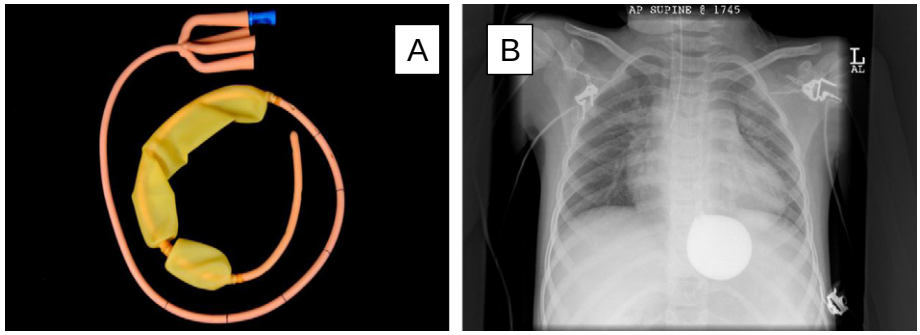

Fig. 1. A: Picture of SBT used at our institution (RÜSCH © 2013 Teleflex Incorporated), B: Xray with contrast instilled in gastric balloon after SBT insertion in one patient.

units [13]. MELD (model for end stage liver disease) was calculated for children aged >12 years from serum bilirubin, creatinine and INR, using the same online tool. Endoscopy was offered to patients with established PHT. Management for those presenting with UGI bleeding included blood product and pharmacological support including the use of Octreotide. Endoscopic sclerotherapy or banding was carried out as required. Our preferred approach to intervention is banding. However, if the child is too small (weight < 10 kg) sclerotherapy is performed instead. At multidisciplinary meetings, decisions are made regarding further management based on the underlying disease; urgent liver transplantation/shunt surgery/TIPS and endoscopic variceal ablation.

2.1. SBT insertion procedure

The indication for SBT insertion was a life-threatening upper GI bleed not responding to medical management. A standard protocol was followed; insertions were carried out following endotracheal intubation. Sizes of SBT available to use were 12 Fr to 18 Fr (length 65 cm to 100 cm; RÜSCH, ©2013 Teleflex Incorporation) but 14 or 16 Fr was typically used. After insertion through the mouth and to a measured length beyond the oesophago-gastric junction, the gastric balloon (GB) was inflated with 50/50 mixture of 0.9% saline and Omnipaque [(180 mg I/ml) Iohexol™ GE Healthcare, Buckinghamshire, UK] contrast medium. Radiography was used to confirm position of the GB (Fig. 1B) and traction applied to wedge the balloon into the oesophago-gastric junction. Traction was maintained by adhesive trouser leg tape fixation onto Lyofoam® (Mölnlycke Health Care Bedfordshire, UK), carefully applied at the corner of the mouth extending onto the ipsilateral cheek and then the neck. To avoid pressure necrosis we released traction and took down SBT fixation for a few minutes after every 6 h. When reapplying fixation, this was carried out on the contralateral side.

If the child was stable after 24 h the gastric balloon was left inflated but the SBT left off traction for up to 24 h. Exceptionally if the bleeding was not controlled the oesophageal balloon was also inflated. Typically, endoscopic evaluation and commencement of variceal obliteration were undertaken after 48 h. Except where indicated, data are expressed as median (range). For non-normally distributed data, the Mann–Whitney U test was used for statistical comparison (GraphPad Software, ©2013 GraphPad Software, Inc. and Social science statistics ©2013Jeremy Stangroom). A P value of 0.05 was considered significant.

3. Results

19 children [10 male, age 1 (0.4–16) year] were identified within the study period. Gastro-oesophageal varices (n = 18) and an aorto-oesophageal (AO) fistula were the causes for major bleeding. The underlying diagnoses for those with varices are listed in Table 1 but included portal vein thrombosis (n = 5) and cirrhotic liver disease in children previously treated for biliary atresia (n = 8). Prior to the presentation which resulted in SBT insertion, all but 1 patient (with AO fistula) had been offered endoscopy (n = 18). A history of previous haematemesis was noted in 13 children (multiple episodes n = 6, single, n = 7) who underwent 1 (1–4) OGD prior to insertion of SBT. Six of these patients had sclerotherapy and another 6 underwent banding; 1 patient did not require intervention at the time of initial endoscopy. The remaining 5 patients were awaiting endoscopy at the time of the bleeding that resulted in SBT insertion.

Gastric balloon (GB) only was put on traction in 15 children for 24 (1–72) h. Both gastric and oesophageal balloons were inflated in 4 children for 24 (2–24) h. Among the 4 patients who required oesophageal balloon inflation along with gastric balloon inflation, 1 patient had AO fistula, and 2 other patients had profuse bleeding on release of oesophageal balloon and died subsequently within few days without further endoscopic evaluation. The only patient who survived the oesophageal balloon inflation had grade 3 gastro-oesophageal varices along with fundal varices.

Upper GI endoscopy was performed after 48 h of insertion of SBT (Fig. 2). Three children deteriorated rapidly and were too unstable to have post- SBT endoscopy. However, the remaining 15 patients (AO fistula patient excluded) had upper GI endoscopy 2 (0–7) days post-SBT insertion and demonstrated GOV alone (n = 12) and with GOV ulceration (n = 3). Active bleeding was seen during post SBT endoscopy in 9 patients. Three patients had immediate re-insertion of SBT in view of ongoing bleeding and had a further endoscopy at 3, 5 and 7 days post-SBT insertion. Among, the 6 children where no active bleeding was seen on post SBT endoscopy, 3 patients had GOV with gastric mucosal ulceration and 3 patients had GOV only. Endoscopic banding (EB) was performed in 2 (22%) of these patients. Sclerotherapy to GOV was performed in the remaining 4 (44%) patients. The sclerosants used were 5% ethonalamine for oesophageal aspects of GOV and Cyanoacrylate glue (Histoacryl®, B. Braun Surgical company, SA) for gastric components of GOV.

Complications of SBT included gastric mucosal ulceration (n = 3, 16%), pressure necrosis at lips and cheeks (n = 6, 31%) and SBT dislodgment (n = 1, 6%). The SBT dislodgement (gastric balloon migrated into oesophagus) seen in one patient was noted on the x-ray to check the position. The SBT was repositioned by deflating and advancing the GB further into the stomach, and re-inflated with larger volume of contrast. A further x-ray film confirmed a satisfactory position.

Table 1
Underlying diagnosis of portal hypertension (PHT) in our series (n = 18) (FNH=Focal nodular hyperplasia, HCC=Hepatocellular carcinoma).

Underlying diagnosis of PHT	n=
Biliary atresia	8
Portal vein thrombosis	5
Alpha 1 anti-trypsin deficiency	1
Cystic fibrosis	1
Intrahepatic Cholestasis + HCC	1
Sclerosing cholangitis	1
FNH + arterio-portal shunt	1