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ORIGINAL ARTICLE

Non-variceal gastrointestinal bleed in children: surgical experience with emphasis on management challenges

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Abstract

Purpose This exclusively surgical series on pediatric non-variceal gastrointestinal bleed (NVGIB) defines three levels of bleed site and describes etiology, bleed severity, diagnostic algorithm, and surgical management for each bleed site. Management challenges are detailed.

Methods Patients aged ≤ 18 years treated surgically for NVGIB were analysed.

Results Bleed site ($n=87$) was classified as: upper gastrointestinal bleed (UGIB; $n=11$); small bowel bleed (SBB: $n=52$); and lower GIB ($n=24$). Four etiology-based groups were identified: lesions with ectopic gastric mucosa (EGM; $n=33$), tumours ($n=23$), ulcers ($n=21$), and vascular pathology ($n=8$). Bleed severity spectrum was: acute severe bleed ($n=12$); subacute overt bleed ($n=59$); and occult GIB ($n=16$). Preoperative diagnosis was obtained in all UGIB and LGIB lesions. Eighty-two percent of surgical SB lesions were diagnosed preoperatively on Tc^{99m} pertechnetate scan, computed tomography enterography–angiography, and capsule endoscopy; remaining 18% were diagnosed at laparotomy with intra-operative enteroscopy (IOE). Surgical management was tailored to bleed site, severity, and etiology. Indications of IOE and approach to management challenges are detailed.

Conclusions The commonest site-specific bleed etiologies were duodenal ulcers for UGIB, EGM lesions for SBB, and tumours for LGIB. SBB presented diagnostic challenge. Diagnostic algorithm was tailored to bleed site, age-specific etiology, bleed severity, and associated abdominal/systemic symptoms. Management challenges were acute severe bleed, occult GIB, SBB, obscure GIB, and rare etiologies. IOE has a useful role in SBB management.

Keywords Non-variceal gastrointestinal bleed · Tc^{99m} pertechnetate scan · Computed tomography enterography–angiography · Capsule endoscopy · Intra-operative enteroscopy

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Abbreviations

ATD	Alimentary tract duplication
AVM	Arteriovenous malformation
BTL	Blunt trauma liver
CTEA	Computed tomography enterography–angiography
CTA	Computed tomography angiography
CE	Capsule endoscopy
CD	Crohn's disease
DU	Duodenal ulcer
DJF	Duodenojejunal flexure
EGD	Esophago-gastro-duodenoscopy
EGM	Ectopic gastric mucosa
FOB	Fecal occult blood
GIB	Gastrointestinal bleed
GEJ	Gastro-esophageal junction
GIST	Gastrointestinal stromal tumour
HAP	Hepatic artery pseudo-aneurysm
IOE	Intra-operative enteroscopy
ICJ	Ileocaecal junction
ICS	Ileo-colonoscopy
JPC	Juvenile polyposis coli
MD	Meckel's diverticulum
NVGIB	Non-variceal gastrointestinal bleed
PGITL	Primary gastrointestinal tract lymphoma
RPC	Restorative proctocolectomy
SBB	Small bowel bleed
SB	Small bowel
SRUS	Solitary rectal ulcer syndrome
SBE	Small bowel endoscopy
TcPS	Tc^{99m} pertechnetate scan
UGIB	Upper gastrointestinal bleed
USG	Ultrasonography
UC	Ulcerative colitis
VM	Vascular malformation

Introduction

This is a series of 87 pediatric cases presenting with non-varical gastrointestinal bleed (NVGIB) that warranted surgical intervention. The manuscript classifies cases according to the site of GIB. For each site of GIB, the following aspects are described: etiology, bleed severity, diagnostic methods, surgical approach, and follow-up.

Algorithmic approach to management challenges has been highlighted. These were: acute severe bleed, occult GIB, small bowel bleed (SBB), obscure GIB, and bleed from infrequently reported etiologies. The role of intra-operative enteroscopy (IOE) has been defined.

There is paucity of published surgical experience on the above-mentioned aspects of pediatric NVGIB.

Materials and methods

Study group comprised of pediatric patients ≤ 18 years of age treated surgically for NVGIB (March 2000 to May 2018) and retrospectively analysed for bleed site, etiology, bleed severity, diagnostic methods, surgical approach, and post-operative follow-up.

Definitions

UGIB GIB source originating until the ampulla of vater.
SBB bleed source distal to the ampulla of vater and proximal to the ileocaecal junction (ICJ) [1].
LGIB bleed source distal to the ICJ until the anal verge.
Obscure GIB bleed source not identified on standard upper and lower GI endoscopic examinations and small bowel evaluation using small bowel imaging and endoscopy [1].
Occult GIB bleed not perceived on gross examination of stool, presenting as iron deficiency anaemia and detected to have positive fecal occult blood (FOB) [1].

Diagnostic methods

UGIB Initial diagnostic modality was esophago-gastro-duodenoscopy (EGD) with/without endoscopic biopsies. Computed tomography enterography–angiography (CTEA) or CT angiography (CTA) was performed selectively based on clinical presentation and endoscopic findings.

LGIB Initial diagnostic modality was ileo-colonoscopy (ICS) with/without endoscopic biopsies followed by CTEA or CTA if indicated.

An SBB source was suspected for bleed per rectum if esophago-gastro-duodenoscopy (EGD) and ileo-colonoscopy (ICS) were normal after which these patients were classified as having "potential small bowel bleeding" as per American College of Gastroenterology (ACG) guidelines [1].

SBB The diagnostic methods used were: Tc^{99m} pertechnetate scan (TcPS), computed tomography enterography–angiography (CTEA), CT angiography (CTA), capsule endoscopy (CE), and intra-operative enteroscopy (IOE).

CTEA entailed SB distension with neutral contrast of near water density together with intravenous contrast and image acquisition in multiple phases of enhancement. This facilitated delineation of enhancing mural/mucosal/

intraluminal pathology, mass lesion, vascular pathology, and anatomical abnormalities [1].

CE was used selectively to detect mucosal ulcerative and vascular lesions.

Diagnostic laparotomy with IOE The stepwise approach to diagnostic laparotomy with IOE was as follows: visual inspection for grossly visible bowel pathology, palpation, and trans-illumination of entire SB to detect intraluminal pathology followed by an IOE.

Technique of IOE A pediatric upper GI endoscope was introduced through SB enterotomy made in the centre of purse string suture. The enterotomy site was mid-SB or just proximal to the site distal to which the bowel was visualized to be filled with intraluminal blood. SB mucosa was examined for ulcerative or vascular lesions proximal and distal to enterotomy site both during antegrade advancement and withdrawal of endoscope. As the endoscope was advanced, the surgeon guided the bowel over the endoscope taking care to avoid serosal or mesenteric tears. The serosal surface at the site of mucosal pathology detected endoscopically was identified by a superficial serosal suture, thus guiding the site/extent of surgical resection. Endoscopic mucosal biopsies were taken if indicated [1, 2].

Results

Eighty-seven cases (age range 03 months–18 years) were treated surgically for NVGIB (March 2000–May 2018).

Bleed site (Table 1)

The site of bleed was classified as UGIB ($n=11$), SBB ($n=52$), and LGIB ($n=24$).

Table 1 Non-variceal GI bleed: bleed site and etiology

	Upper GI bleed ($n=11$)	Small bowel bleed ($n=52$)[a]	Lower GI bleed ($n=24$)
Lesions with EGM ($n=33$)	Alimentary tract duplication (ATD) ($n=1$) (age: 03 years)	1. Meckel's diverticulum ($n=25$) (age: range: 03 months–11 years, median: 2.5 years) 2. ATD ($n=7$) (age: range: 05 months–11 years, median: 02 years)	
Ulcerative lesions ($n=21$)	1. Duodenal ulcers ($n=4$) [age (years): 1, 3.5, 8, 13] 2. Corrosive induced gastric antral ulcers ($n=1$) [age (years): 13]	1. Tubercular ulcer($n=1$) (age: 15 years) 2. Enteric ulcer ($n=1$) (age: 10 years) 3. Tropical Jejunoileitis ($n=1$) (age: 13 years) 4. Crohn's disease ($n=3$) [age (years): 15, 16, and 18] 5. Indeterminate ulcer/stricture ($n=5$) (age range: 8–17 years and median: 15 years) 6. Blind loop syndrome ($n=1$) (age: 10 years)	1. Ulcerative colitis ($n=1$) (age: 10 years) 2. Crohns' colitis ($n=2$) (age: 09, 10 years) 3. Solitary rectal ulcer syndrome ($n=1$) (age: 09 years)
Tumours ($n=23$)	Gastric teratoma ($n=1$) (age: 03 months) Histology: mature gastric teratoma	Primary GIT lymphoma ($n=2$)[a] [a][Jejunal (1), ileal (1)] [age (years): 10,11] Histology: diffuse large B-cell lymphoma	1. PGIT lymphoma, Left colon; ($n=1$, Age: 09 years) Histology: diffuse large B-cell lymphoma 2. Colonic GI stromal tumour ($n=1$; age: 09 years) Histology: mitosis: < 1/50 hpf 3. Juvenile polyposis coli ($n=18$) (age range: 04–18 years, median: 11 years)
Vascular pathology ($n=8$)	1. Gastric arteriovenous malformation ($n=01$; age: 12 years) 2. Hepatic artery pseudo-aneurysm ($n=03$) [age (years): 7, 8, 16]	Lymphovenous malformation ($n=04$)[a] [a][Duodenal (1), jejunal (3)] [age (years): 3, 3, 5, 12]	

[a]Two cases were eventually diagnosed to have a platelet function disorder

For each site of GIB, the following aspects are described in the subsequent text: (a) etiology, (b) severity of bleed, (c) diagnostic methods, (d) surgical approach, and (e) post-operative follow-up.

Bleed site and etiology (Table 1)

For each site of GIB, the etiology was classified into four major groups: (1) lesions with EGM [Meckel's diverticulum (MD), alimentary tract duplication (ATD)] ($n=33$), (2) tumours ($n=23$), (3) ulcerative lesions ($n=21$), and (4) vascular pathology ($n=8$). Table 1 shows the etiology at each GIB site.

The commonest site of NVGIB in this series was SB ($n=52$). The commonest surgical etiology at each bleed site was as follows: (1) UGIB: ulcers; (2) SBB: lesions with EGM; and (3) LGIB: tumours.

Bleed site and bleed severity with corresponding etiology (Table 2)

Bleed severity has management implications. Hence, for each site of GIB, bleed severity with corresponding etiology in defined in Table 2.

Bleed severity was classified and defined as follows:

(1) Acute severe bleed, hemodynamic instability and hence warranting emergency surgery or angioembolization ($n=12$): Defined as hypovolemic shock (signifying 25–30% blood volume loss) or significant blood loss indicated by an orthostatic increase in pulse rate by 20 beats/min. or a decrease in systolic blood pressure of 10 mmHg or more or an acute (< 12 h) decrease in Hemoglobin of more than 2 g/dl [3, 4].

In acute severe bleed, there were two clinical scenarios with therapeutic implications: (a) etiology and site diagnosed on pre-intervention investigations ($n=10$) and (b) etiology and site undiagnosed preoperatively, because hemodynamic instability precluded detailed preoperative SB evaluation. The diagnosis was obtained at emergency laparotomy and IOE ($n=2$).

(2) Subacute overt bleed ($n=59$): overt persistent or recurrent slow bleed at a rate not severe enough to cause acute hemodynamic instability or an acute drop in hemoglobin unlike "acute severe bleed" [1].

(3) Occult GIB ($n=16$) as defined in "Materials and methods".

UGIB ($n=11$): etiology, diagnostic methods surgical approach: (Tables 1, 2)

Details of clinical presentation, diagnosis, and management specific to each etiological group presenting as UGIB (Tables 1, 2) are as follows.

Duodenal ulcers (DU, $n=4$)

All were diagnosed on EGD. A concomitant anterior wall perforation was documented on EGD and subsequently confirmed at laparotomy in two cases. Preoperative endoscopic

Table 2 Bleed severity and its etiology at various sites of GIB

	Acute severe bleed, hemodynamic instability ($n=12$)		Subacute overt GI bleed ($n=59$)	Occult GI bleed ($n=16$)
	Etiology/site diagnosed pre-intervention ($n=10$)	Etiology/site undiagnosed preoperatively ($n=2$)		
Upper GI bleed ($n=11$)	Duodenal ulcer ($n=4$) Gastric AVM ($n=1$) HAP ($n=03$)		Corrosive induced gastric antral ulcers ($n=1$) Gastric teratoma ($n=1$)	Esophago-gastro-duodenal ATD ($n=1$)
Small bowel bleed ($n=52$)		Enteric ulcer ($n=1$) Tubercular ulcer ($n=1$)	MD ($n=25$) ATD ($n=5$) Tropical jejunoileitis ($n=1$) Primary GIT lymphoma ($n=1$) Lymphovenous malformations ($n=03$) Platelet function disorder ($n=2$)	ATD ($n=2$) Crohns' disease ($n=3$) Indeterminate ulcer/stricture ($n=5$) Blind loop syndrome ($n=1$) Primary GIT lymphoma ($n=1$) Lymphovenous malformation ($n=1$)
Lower GI bleed ($n=24$)	Ulcerative colitis ($n=1$) Crohns' colitis ($n=1$)		SRUS ($n=1$) Crohns' colitis ($n=1$) JPC ($n=18$)	Primary GIT lymphoma ($n=1$) GIST ($n=1$)

HAP hepatic artery pseudo-aneurysm, *AVM* arteriovenous malformation, *JPC* juvenile polyposis coli, *SRUS* solitary rectal ulcer syndrome, *GIST* GI stromal tumour, *ATD* alimentary tract duplication, *MD* Meckel's diverticulum

grading was as follows: Forrest grade 1 ($n=1$) and Forrest grade IIA ($n=3$). Preoperative endoscopic local adrenaline injection was attempted in two cases.

A history of prior NSAID intake was present in all ($n=4$). None documented H. Pylori on histopathology.

All warranted emergency laparotomy for the following indications: (1) recurrent acute severe bleed, hemodynamic instability, failed medical, and endoscopic therapy ($n=4$) and (2) associated anterior wall perforation ($n=2$).

The surgical procedures performed were: suture transfixation of the bleeding vessel, antrectomy, and Billroth-2 gastro-jejunostomy. In addition, concomitant anterior wall perforations ($n=2$) were managed as follows: (1) tube duodenostomy through the duodenal perforation site (Case 1) and (2) repair of anterior wall perforation re-inforced with an omental patch (Case 2).

Mature gastric teratoma ($n=1$)

Initial EGD detected an intra-gastric wide-based lobulated mass lesion located 3 cm distal to the gastro-esophageal junction (GEJ) onto the greater curvature. A subsequent CTEA abdomen showed fat densities with chunky calcification within an intra and extragastric mass lesion, thereby corroborating diagnosis of a Teratoma. Total excision of mass with wedge resection of stomach was performed.

Gastric arteriovenous malformation (AVM, $n=1$)

This 12 year girl presented with recurrent acute severe UGIB. EGD at first bleed showed an actively bleeding arterial spurt at lesser curvature with mucosal hypertrophy and diffuses erythema from the GEJ junction until Incisura. Subsequent CTEA showed an AVM extending from the GEJ until the Incisura further documented at conventional angiography.

Initial bleed episodes were managed with endoscopic variceal ligation for the arterial spurt followed by sequential steel coil angioembolization of left gastric artery and right gastro-epiploic artery.

After a 1 year bleed-free interval post-angioembolization, the patient presented in hypovolemic shock following recurrent acute severe UGIB. Repeat CTA documented collateralization of embolized vessels.

Hence, an emergency "Total Gastrectomy with Roux-en-Y Esophago-jejunostomy" was performed for recurrent life-threatening UGIB with failed prior angioembolization. A total gastrectomy was warranted, because the lesion extended from GEJ junction until Incisura. Post-surgery, she is maintaining good nutrition on small volume low carbohydrate frequent meals with no features of dumping syndrome and is being supplemented and closely monitored for serum vitamin B12 levels.

Hemobilia: post-traumatic hepatic artery pseudo-aneurysm (HAP, $n=3$)

The salient presenting features were: (1) acute severe UGIB on day 15 ($n=1$), day 20 ($n=1$), and day 32 ($n=1$), respectively, for 03 cases following non-operative management (NOM) of blunt trauma liver (BTL); (2) associated jaundice ($n=1$); and (3) prior surgical repair of duodenal injury day 3 post-BTL ($n=1$).

The investigative modalities contributing to diagnosis were: (1) EGD: documented blood through papilla ($n=1$); (2) abdominal ultrasonography (USG): intrahepatic biliary dilatation, blood clots in the extrahepatic biliary tree ($n=1$); and (3) the definitive diagnosis was obtained on CTA documenting a pseudo-aneurysm arising from segment 5 ($n=02$) and segment 8 ($n=1$) branch of right hepatic artery.

Emergency selective coeliac angiogram with super selective cannulation of donor artery to pseudo-aneurysm and steel coil embolization distal and proximal to pseudo-aneurysm achieved complete exclusion of the pseudo-aneurysm from the circulation by preventing collateral circulation backflow. This resulted in immediate bleed control with minimum post-procedure liver function impairment.

Esophago-gastro-duodenal ATD ($n=1$)

An initial USG showed an upper abdomen cystic lesion with positive "gut signature sign". The diagnosis was substantiated on CTEA chest and abdomen and a positive TcPS.

The extent of ATD was as follows: Proximally, the lesion started from T1 thoracic vertebra and extended all along the esophagus distally and then through the hiatus along the lesser curvature of stomach until the second part of duodenum. Two-staged surgical excision was performed with right posterolateral thoracotomy for excision of esophageal ATD first followed by excision of gastro-duodenal ATD 7 weeks later.

Small bowel bleed ($n=52$): etiology, diagnostic methods, and surgical approach

Diagnostic challenges specific to SBB

The diagnostic methods used were: $TC^{99m}PS$, CTEA, CE, and IOE.

Table 3 depicts diagnostic yield of each investigative modality for various etiological groups.

From diagnostic perspective, SBB was categorized into two groups with implications on management:

Table 3 Small bowel bleed: diagnostic yield of various preoperative investigative modalities ($N=48$)

Investigative modality	Etiology	Positive result (diagnostic yield)
Tc^{99m} pertechnetate scan	MD	19/25 (76%)
	ATD	6/7 (86%)
CTEA	ATD	1/1
	Ulcerative lesions with/without strictures	9/10
	Vascular malformations	4/4
	Primary GIT lymphoma	2/2
CE[a] (age: 8 years, 9 years, 16 years, 12 years)	Ulcerative lesions	3/3
	Vascular malformation	1/1

Two cases: hemodynamic instability precluded preoperative SB imaging

Two cases: eventually diagnosed to have platelet function disorder

[a]CE corroborated CTEA findings in these four cases

1. *SBB with etiology and site undiagnosed on preoperative investigations (n = 09)* this group presented a challenge, wherein diagnosis was obtained at laparotomy with IOE. There were two clinical scenarios in this group:
   1.1. Acute severe suspected SBB, normal EGD and ICS ($n=02$). Hemodynamic instability, however, precluded detailed preoperative SB imaging/endoscopy: Diagnosis at emergency laparotomy with IOE was: ileal enteric ulcer ($n=1$) and ileal tubercular ulcer ($n=1$) bleed.
   1.2. Obscure GIB ($n=07$): subacute overt GIB with etiology and site undiagnosed despite detailed preoperative investigations (EGD, ICS, and SB evaluation by TcPS and CTEA): diagnostic laparotomy with provision for IOE revealed the following: (1) Meckel's diverticulum (MD) with negative preoperative TcPS ($n=06$) and (2) tropical jejuno-ileitis ($n=1$).
2. *SBB with etiology and site diagnosed on preoperative investigations (n = 41)* (Table 3).

In addition, extensive preoperative investigations (EGD, ICS, Tc^{99m}PS, and CTEA) and IOE excluded an anatomical cause in two cases. These were eventually diagnosed to have a platelet function disorder.

Role of IOE in SBB

IOE ($n=9$) guided surgical management in the following clinical scenarios:

(1) Acute severe suspected SBB, normal EGD and ICS. Hemodynamic instability, however, precluded detailed preoperative SB imaging/endoscopy [enteric (Case 1) and tubercular (Case 2) ulcer bleed].
(2) Subacute persistent SBB, negative TcPS, and CTEA: IOE detected extensive jejunal mucosal ulcers and permitted diagnostic endoscopic mucosal biopsies [tropical Jejunoileitis ($n=1$)].
(3) Subacute GIB because of multiple ulcers with strictures ($n=3$) detected on preoperative CTEA: IOE permitted assessment of site and extent of ulcers and morphology of ulcers and intervening mucosa. IOE, thus, guided appropriate extent of resection to prevent leaving behind gross residual ulcers as a potential source of ongoing post-operative bleed.
(4) Jejunal venous vascular malformation (VM) with ill-defined serosal margins ($n=1$): mucosal extent of lesion on IOE-guided extent of resection.
(5) In addition, IOE excluded a surgically correctable anatomical cause in two cases with persistent overt GIB, thereby prompting work up for a coagulation disorder. These were eventually diagnosed as rare platelet function disorder [Glanzmann Thrombasthenia ($n=2$)].

Details of clinical presentation and management specific to each etiological group presenting as SBB are as follows:

Lesions with EGM ($n=32$)

Meckel's diverticulum (MD; n = 25) The diagnostic yield of preoperative TcPS was 76% (19/25) (Table 3). With high index of clinical suspicion, a diagnostic laparotomy revealed an MD in remaining six patients despite a negative TcPS. Before diagnostic laparotomy, an EGD, ICS, and CTEA were performed in patients with negative TcPS.

Alimentary tract duplications (ATD, $n=7$)

The clinical presentation was: overt SBB ($n=5$), occult SBB ($n=2$), associated diarrhoea, and failure to thrive ($n=2$) and localized perforation ($n=1$).

All were long tubular ATD with a bowel communication. The diagnostic challenge was that long tubular ATDs were missed on abdominal ultrasonography (USG) in 6/7 cases because of marked morphological resemblance to native bowel. However, TcPS documented large area of radio-isotopic uptake in 6/7 patients giving a diagnostic yield of 86%.

The site and extent of ATDs was as follows: (1) proximal end starting at 20 cm from duodenojejunal flexure (DJF) and extending until 15 cm proximal to ileo-caecal junction (ICJ) distally ($n=1$); (2) proximally starting from the third part of duodenum and extending until 30 cm proximal to ICJ distally ($n=1$); (3) mid-ileum-to-mid-transverse colon

($n=1$); (4) proximally from the second part of duodenum and extending until 15 cm proximal to ICJ distally ($n=1$); and (5) along the following length of ileum: 75 cm ($n=1$), 70 cm ($n=1$), and 10 cm ($n=1$), respectively.

Surgical excision was aimed at removing all acid secreting EGM while preserving adequate length of well-vascularized native bowel. This was technically facilitated by: (1) using the "Principle of Bianchi's Procedure", wherein the posterior arcade preserves vascularity of native bowel ($n=5$) and (2) mucosectomy of ATD segment inseparable from native bowel ($n=2$). A wide unhealthy communication with the second part of duodenum was repaired over T-tube duodenostomy with distal-feeding jejunostomy ($n=1$).

Ulcerative lesions ($n=12$)

The etiology (Table 1), clinical presentation, and management details of SB ulcerative lesion are as follows:

Crohn's disease (CD, n = 3) All presented with occult GIB with associated bowel obstruction in two cases.

The number of strictures ranged from 5 to 18 and length of resected bowel, guided by IOE, ranged from 40 to 150 cm. Other salient points were: (1) strictures at multiple sites warranting multiple concomitant resections ($n=1$) and (2) redo-resection for recurrent strictures with bleeding ulcers ($n=1$).

Enteric ulcer bleed (n = 1) The diagnosis was substantiated by a history of fever of 2 week duration preceding bleed onset, a positive Blood culture and histopathology of resected specimen. Emergency laparotomy was indicated for acute severe SBB with hemodynamic instability which did not permit detailed preoperative small bowel imaging. IOE identified briskly bleeding multiple ileal ulcers and guided extent of resection.

Tubercular ulcer bleed (n = 1) Emergency laparotomy was indicated for acute severe SBB with hemodynamic instability which precluded detailed preoperative small bowel imaging. IOE identified brisk arterial spurt from an ileal ulcer. Diagnosis of Tubercular ulcer was documented by caseating granulomas reported on histopathology of resected ileum and mesenteric lymph nodes.

Blind loop syndrome (n = 1) was a consequence of a side-to-side anastomosis performed to bypass an ileal stricture in infancy. Stasis within the blind loop resulted in mucosal ulceration, pseudo-diverticulae, and bacterial overgrowth with consequent occult SBB.

Indeterminate ulcers/strictures (n = 5) SB ulcers with no definite etiology identified on preoperative investigations (inflammatory markers, serology, and radiological findings) and stricture histology were categorized as "indeterminate".

All presented with occult GIB with associated bowel obstruction in two cases.

The number of strictures ranged from 1 to 26 (median: 03) and length of resected segment, guided by IOE, ranged from 50 to 100 cm.

Strictures with indeterminate histology merit long-term follow-up to assess for evolution of the natural course of disease.

Vascular malformations [VM;($n=4$)]

With a clinical background of SBB in an otherwise well preserved child, preoperative diagnosis was obtained on CTEA in all.

All were slow flow venous/lymphovenous malformations. The site, extent, and management were as follows:

(1) Jejunal venous malformations ($n=3$): the jejunal length involved ranged from 10 ($n=1$) to 15 cm ($n=2$). Complete resection of the involved segment was done in all. The serosal margins were ill defined in one case, wherein mucosal extent of lesion on IOE guided extent of resection.
(2) Duodenal lymphovenous malformation ($n=1$): the lesion involved entire duodenum from first part to the fourth part with extension along the base of the mesentery until ICJ. There were no clear planes of dissection between the VM and pancreas and superior mesenteric artery and vein pedicle. This precluded surgical excision. A gastrojejunostomy with truncal vagotomy was performed for associated subacute duodenal obstruction. Post-surgery, the child has been on propranolol therapy and on endoscopic argon plasma coagulation therapy which has decreased the frequency of blood transfusions required.

Tumours ($n=2$)

Upfront surgical excision followed by adjuvant chemotherapy was performed in two cases of small bowel primary gastrointestinal tract lymphoma (PGITL) for persistent bleed with resectable anatomy.

Lower GI bleed: etiology, clinical presentation, and management (Tables 1, 2)

Clinical presentation and management details specific to each etiology of LGIB are as follows:

Tumours

The largest etiological group for LGIB was tumours and these were: (1) Juvenile Polyposis Coli (JPC; $n=18$); (2) PGITL of left colon ($n=1$); and (3) transverse colonic gastrointestinal stromal tumour (GIST; $n=1$).

Juvenile Polyposis coli (JPC; $n=18$): The indications for surgery were severe anaemia [hemoglobin: range 2.7–7.7 g%; median (5.6 g%)], hypoproteinaemia [S. albumin: range 1.1–3.3 g%; (median: 1.9 g%)], and failure to thrive.

A staged restorative proctocolectomy (RPC) was performed in 17 cases with the following details: (1) ileal-J-pouch–anal anastomosis ($n=13$) and (2) straight ileo-anal anastomosis ($n=4$) were performed selectively, wherein anatomy did not permit a tension free pouch–anal anastomosis. Surgery was restricted to subtotal colectomy with rectal polypectomy in one case because of concomitant incidentally detected extrahepatic portal vein obstruction with extensive perirectal porto-systemic collaterals.

Histopathology of resected proctocolectomy specimen was reported as hamartomatous juvenile polyps in all with adenomatous change ($n=10$) and dysplasia ($n=08$) in some.

PGITL of left colon (n = 1) Diagnosis was obtained at CTEA followed by Colonoscopy. Management entailed wide surgical excision and adjuvant chemotherapy.

Transverse colonic GIST (n = 1) Diagnosis was obtained at CTEA. Transverse colectomy with sleeve resection of the contiguously involved greater curvature of stomach was performed.

Ulcerative lesions

These were: (1) ulcerative colitis (UC, $n=1$); (2) Crohn's colitis ($n=2$); and (3) solitary rectal ulcer syndrome ($n=1$).

Ulcerative colitis (n = 1) Emergency subtotal colectomy was warranted for fulminant colitis unresponsive to medical therapy followed by an elective completion proctectomy and ileal-J pouch–anal anastomosis.

Crohn's disease with colonic involvement (n = 2) The case details are as follows: (1) extensive Crohns' colitis (Case 1): emergency subtotal colectomy was warranted for pan colonic involvement with acute severe bleed, failed medical therapy and (2) terminal ileal and right colonic Crohns' disease (Case 2): a right hemicolectomy was performed.

Post-operative follow-up For each site of GIB and corresponding etiology, Table 4 depicts post-operative follow-up on the following parameters: (1) early 30-day post-operative mortality; (2) recurrent bleed; and (3) disease recurrence. On long-term follow-up, four cases of Crohns' disease presented with disease relapse and GIB from a new site. These were managed on medical therapy.

Discussion

The discussion would focus on challenges, namely: (1) management challenges related to acute severe bleed and occult GIB; (2) diagnostic considerations and challenges specific

Table 4 Post-operative follow-up

	EGM ($n=33$)	Ulcerative lesions ($n=21$)	Tumours ($n=23$)	Vascular pathology ($n=8$)
UGIB (N = 11; FU duration: range: 1.5–130 months, median: 18 months)				
Early post-operative mortality	0	0	0	0
Recurrent bleed	0	0	0	0
Disease recurrence	0	0	0	0
SBB (N = 50[a]; FU duration: range: 2–128 months; median: 14 months)				
Early post-operative mortality (*etiology)	0	01 (*tubercular ulcer)	0	0
Recurrent bleed (*etiology)	0	02 (*Crohns' disease)	0	01 (*unresectable duodenal vascular malformation)
Disease recurrence (*etiology)	0	02 (*Crohns' disease)	0	0
LGIB (N = 24; FU duration: range: 6–178 months, median: 48 months)				
Early post-operative mortality	0	0	0	0
Recurrent bleed (*etiology)	0	02 (*Crohn's disease)	0	0
Disease recurrence (*etiology)	0	02 (*Crohn's disease)	0	0

[a]Two cases diagnosed to have a platelet function disorder have been excluded

Asterisk indicates the "Etiology" of recurrent bleed and disease recurrence

to each site of GI bleed; (3) proposed diagnostic algorithms for each site of GIB; and (4) key points related to bleed from infrequently reported etiologies.

Management challenges related to acute severe bleed and occult GIB

Acute severe bleed with hemodynamic instability warranting emergency intervention The biggest management challenge was acute severe bleed from SB source undiagnosed preoperatively, because hemodynamic instability precluded detailed preoperative SB imaging/endoscopy. In this scenario, IOE at emergency laparotomy plays a key role in achieving prompt identification of bleed site, immediate bleed control, obtaining specimen for histopathological diagnosis and thus, is lifesaving. Hence, surgical exploration should not be delayed because of inability to obtain preoperative diagnosis.

However, it is noteworthy that preoperative diagnosis was obtained in all cases presenting with acute severe bleed from an UGI or LGI source, by one or a combination of the following investigations: EGD, ICS, CTEA, and CTA.

Overall, the commonest etiology of acute severe bleed was acute ulcerative lesions [DUs, ileal enteric and tubercular ulcer, fulminant UC, and colonic CD] followed by arterial vascular pathology [gastric AVM and HAP] (Table 2).

Occult GIB The diagnosis of occult GIB and its etiology was often missed for long, because these children frequently presented to general pediatricians with persistent anaemia before being referred to our centre. Eventually, a positive FOB prompted referral to pediatric gastroenterology services.

The commonest source of occult GIB was small bowel. The commonest etiology of occult GIB was chronic SB ulcers [CD, indeterminate ulcers with strictures, blind loop syndrome] followed by ATDs, tumours [PGITL, Colonic GIST] and slow flow jejunal lymphovenous malformation (Table 2).

Diagnostic considerations and challenges specific to each site of GI bleed

UGIB The bleed site/etiology was diagnosed preoperatively in all cases by EGD, CTEA, CTA, and conventional angiography. EGD, as the first line key investigation, gave definitive diagnosis in all ulcerative lesions. For vascular pathology and tumours, EGD picked up pointers to the definitive diagnosis. A definitive diagnosis with precise delineation of anatomy was obtained on subsequent CTEA [3] for tumours and CTA followed by conventional angiography for a vascular pathology.

LGIB Again, bleed site/etiology was diagnosed preoperatively in all cases by ICS, histopathology of colonoscopic biopsy, and CTEA [4].

SBB SBB source localization was a greater challenge because of limitation of radiological and endoscopic access.

While ACG [1] recommends CE as first line investigation for SBB in adults, data from current series substantiates that diagnostic algorithm for SBB in children needs to be different from that in adults because of differences in age-specific etiology, deficiency of small size SB endoscopes, inability to swallow a Capsule, and need for sedation/anaesthesia for invasive small bowel endoscopy (SBE).

In adults, mucosal ulcerative and vascular pathology are more common. In children, however, lesions with EGM, tumours, mural inflammatory lesions, vascular malformations, and congenital or acquired structural anatomical lesions were commoner (Table 1).

Data from the current series show that the commonest surgical cause of SBB was lesions with EGM (MD and ATD) and 78% of these were diagnosed on TcPS.

Similarly, the diagnostic yield of a CTEA was 93% (15/16) for SB ulcers with/without strictures, ATD, vascular Malformations, tumours, and structural anatomical pathology (Table 3).

Eventually, 82% (41/50) of surgical SB lesions were diagnosed on two key non-invasive investigations, i.e., TcPS and CTEA.

Hence, TcPS needs to be the first line investigation, preceding ICS and EGD, in a clinical setting of intermittent, brisk, bleed per rectally in an otherwise well child. A CTEA would be indicated in SBB with (1) associated obstructive/systemic symptoms or (2) persistent/recurrent SBB with no obstructive/systemic symptoms but a negative TcPS.

CE was used selectively ($n=4$) to corroborate the diagnosis of a mucosal ulcerative or vascular lesion on a prior equivocal CTEA after excluding bowel obstruction.

It is clear, thus, that improved preoperative diagnosis of surgical SBB lesions with TcPS, CTEA, and CE has narrowed down the spectrum of "true obscure GI bleed" in children [1].

Eventually, preoperative diagnosis could not be achieved in 09/50 (18%) cases with surgical SBB and this group constituted a diagnostic challenge. In 2/9 cases, acute severe bleed and hemodynamic instability from ileal enteric ($n=1$) and tubercular ($n=1$) ulcer bleed precluded detailed preoperative SB imaging/endoscopy, eventually diagnosed at emergency IOE.

Remaining seven patients presented with recurrent, subacute bleed undiagnosed on preoperative TcPS, EGD, ICS, and CTEA. Interestingly, diagnostic laparotomy in six of these cases revealed a pathology as simple as MD. This corroborates the clinical recommendation that "in the presence of high index of clinical suspicion for MD, diagnostic laparoscopy/laparotomy should be considered despite a negative TcPS and an inconclusive SB radiological imaging" [5, 6].

IOE, though infrequently reported in pediatric surgical literature, has a potential for wider application in children because of limited applicability of non-operative enteroscopic techniques such as CE and SBE. IOE, though invasive, is the gold standard to assess entire SB for mucosal ulcerative or vascular pathology.

Based on experience from our series and published literature, the role of IOE in SBB is defined as follows [1, 7]:

(1) Acute severe suspected SBB, wherein hemodynamic instability precludes detailed preoperative SB evaluation.
(2) SBB with negative TcPS and CTEA: CE and SBE contraindicated because of subacute obstruction, prior surgery/adhesions, and small age.
(3) Obscure GIB: GIB source undiagnosed on detailed preoperative imaging and endoscopy.
(4) Guiding extent of resection to prevent leaving behind gross residual disease as a potential source of ongoing post-operative bleed in: (1) multiple SB strictures and ulcers and (2) SB vascular malformations with ill-defined serosal margins.
(5) IOE-guided diagnostic SB mucosal biopsies.

Diagnostic algorithms

Thus, with the background of data presented in this series, our suggested diagnostic algorithm for NVGIB from each site of GI bleed in children is shown in Figs. 1 and 2. The diagnostic algorithms are based on the following considerations: (1) age-specific etiology [8–11]; (2) diagnostic yield of various investigative modalities; (3) bleed severity; (4) associated abdominal and systemic symptoms; and (5) limited scope of using invasive diagnostic methods for SB evaluation in children.

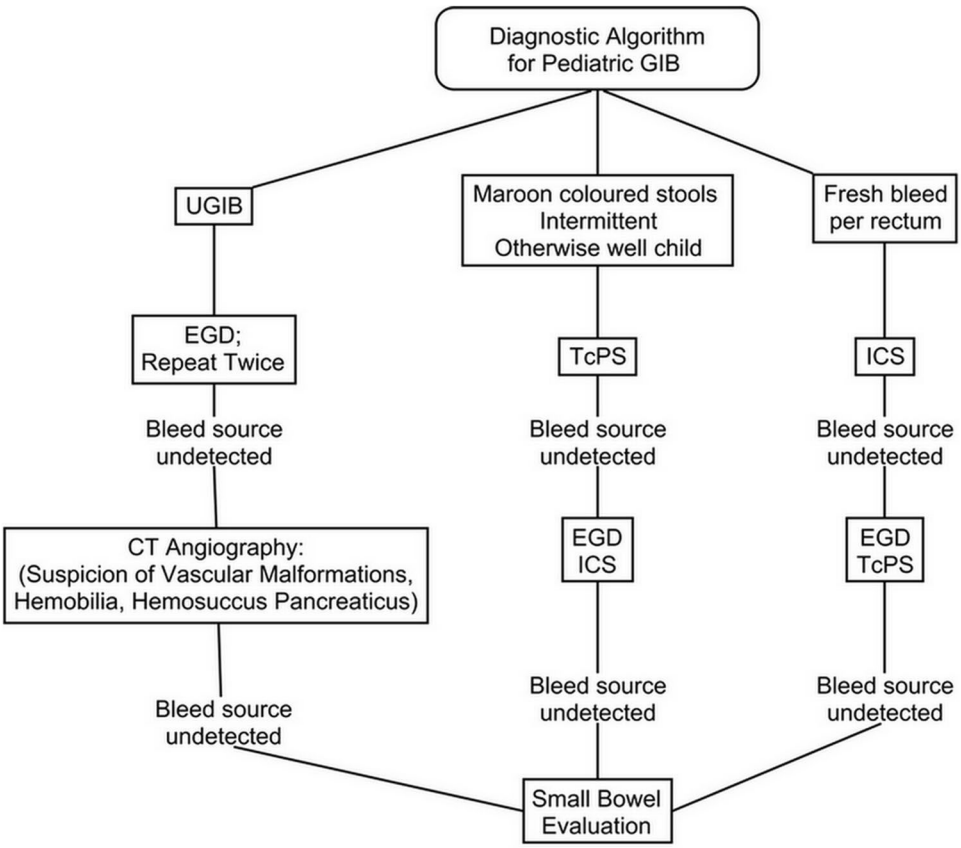

Fig. 1 Diagnostic algorithm for pediatric gastrointestinal bleed

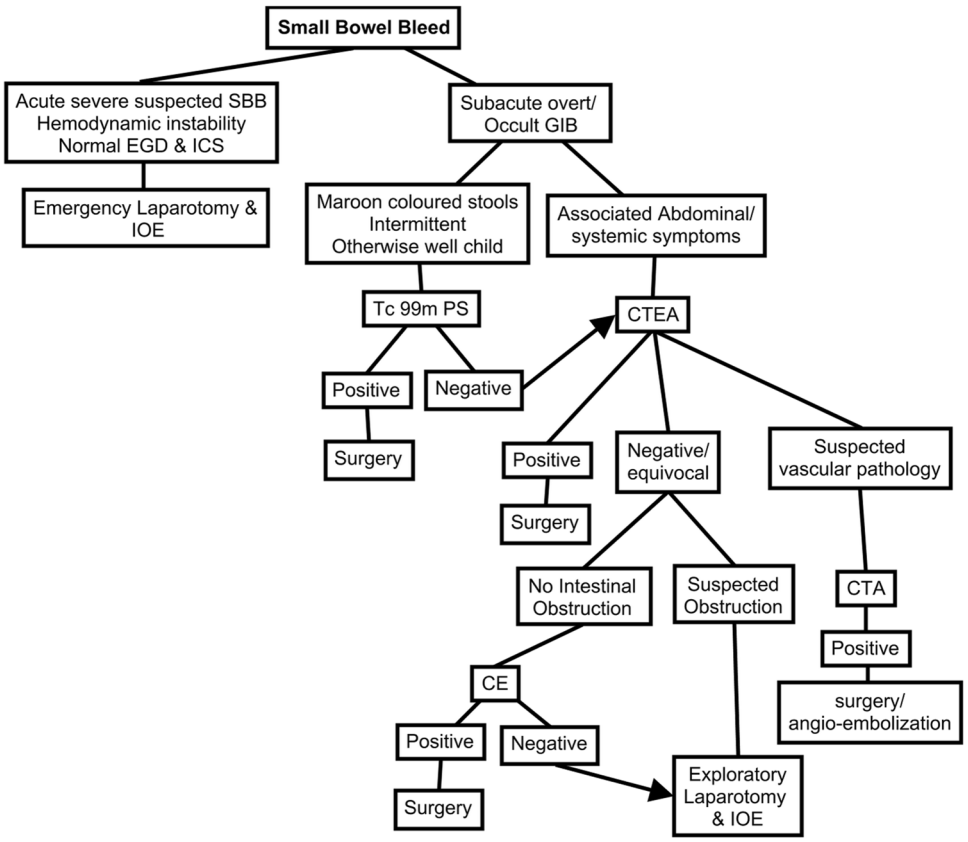

Fig. 2 Diagnostic algorithm for pediatric small bowel bleed

Key points related to bleed from infrequently reported etiologies

Ulcerative lesions

Duodenal ulcers (DU, n = 4) Chronic primary DUs are per se rare in children. Medical and endotherapy have further reduced the need for elective DU surgery. However, limited case series [12] reports "complicated secondary ulcers" in high-risk children with co-morbidities who were receiving ulcerogenic drugs and warranted emergency surgery as corroborated with our experience.

Only 10% of bleeding posterior penetrating DUs have been reported to have an unsuspected anterior wall perforation ("kissing ulcers") with an increased mortality as compared to bleed alone or perforation alone [13, 14].

Inflammatory bowel disease [CD (n = 5), UC (n = 1)] The indications of surgery were: (1) bleed with failed medical therapy ($n = 2$); (2) associated bowel obstruction ($n = 2$); and (3) obtaining definitive diagnosis before instituting medical therapy ($n = 5$).

Surgical considerations specific to CD were as follows: (1) risk of relapses and, hence, surgical approach has to judiciously balance between preservation of bowel length on one hand and not leaving behind residual gross disease/ulcers as a potential source of ongoing post-operative bleed on the other hand. IOE had a useful role in guiding appropriate extent of resection. (2) Multiple strictures/ulcers ($n = 5$) and synchronous multiple site resections ($n = 1$) (3) redo-resection for recurrent strictures with bleeding ulcers ($n = 1$); (3) severe hypoproteinaemia and anaemia ($n = 2$) warranting a post resection SB stoma; and (4) gross disparity of bowel lumen proximal and distal to long standing stricture, wherein a stapled functional end-to-end anastomosis was helpful [15].

Enteric ulcer (n = 1) and tubercular ulcer (n = 1) bleed Literature review yielded scant case reports of life-threatening bleed from enteric ulcers and tubercular ulcers in today's era of early diagnosis and effective medical therapy. Tubercular ulcer bleed without obstructive features has been reported in the setting of immunocompromised host or tuberculosis endemic areas. Preoperative diagnosis is usually not obtained when bleed site is SB ulcers and the patient is hemodynamically compromised. IOE facilitates bleed site localization and diagnosis through histopathology of resected specimen as corroborated by our experience [16–18].

Thus, tubercular and enteric ulcer bleeds, though rare, need to be considered in the differential diagnosis of SBB in endemic areas.

Vascular pathology

Vascular malformations (VM, n = 4) Noteworthy features in our series were: (1) spectrum ranging from slow flow venous malformations to high flow AVM and hence; (2) presentation ranging from occult GIB ($n = 1$) in slow flow malformation to acute severe bleed in high flow gastric AVM [19] ($n = 1$); and (3) associated duodenal obstruction in an unresectable duodenal lymphovenous malformation ($n = 1$).

VMs should be suspected for brisk, intermittent bleed in an otherwise well child and negative TcPS. The reported diagnostic modalities are Doppler ultrasonography, CTEA, MRI, and diagnostic laparoscopy. The differentiation between slow flow (capillary, lymphatic, and venous malformations) and high flow (arteriovenous) malformations is made by the pattern of enhancement in arterial and venous phase of CTEA and by histopathology of excised specimen [20].

Surgical resection, if technically feasible, is the treatment of choice. Angioembolization has been reported for high flow AVM with well-defined arterial feeders. However, rebleed rates of 5–37% have been reported because of rich collateral blood supply of the stomach, thereby emphasizing upon the need for long-term follow-up and this corroborates with our experience [21–23].

The diagnosis was obtained on CTEA in all our cases. It was corroborated by CE ($n = 1$) and the extent of resection guided by IOE ($n = 1$) in SB vascular malformations and this corroborates with published experience [24].

HAP (n = 3) Our experience emphasizes upon the need to have a high index of clinical suspicion for HAP in the clinical setting of UGIB post-BTL. CTA has a high diagnostic yield. Reported pediatric experience on endovascular management of intrahepatic HAP as described under "Results" is very limited [25–28].

Tumours

While the commonest tumours were JPC, other rare tumours presenting as NVGIB were: gastric teratoma ($n = 1$), primary gastrointestinal tract lymphoma (PGITL, $n = 3$), and transverse colonic GI stromal tumour (GIST, $n = 1$).

Gastric teratoma (n = 1) Gastric teratomas constitute only 1% of all teratomas and presentation as UGIB is even rarer. UGIB is attributed to ulceration on the endophytic component. A polypoidal wide-based mass lesion on UGIE with fat densities and chunky calcification on imaging modalities, characteristically presenting in infants, corroborates the diagnosis of a gastric teratoma. Histologically, most are mature with isolated case reports of immature/malignant gastric teratomas [29–32].

PGITL (n = 3) While chemotherapy forms the mainstay therapy for PGITL, surgery was indicated for GI bleed with a resectable anatomy in our series. While ileo-caecal region is the commonest reported site in children, PGITL of the left colon, as reported in one case in our series, is extremely rare [33–35].

Transverse colonic GIST (n = 1) GIST are rare in children with a reported annual incidence (UK National Registry of Childhood Tumours) of 0.02 per million. The commonest site is stomach; colonic GIST as reported in our series constitutes only 10% of all pediatric GIST.

Complete surgical excision is the mainstay of treatment. Adjuvant therapy (Imatinib) is indicated for incomplete resection, tumour spillage, and high mitotic index. The role of adjuvant therapy, however, is less well defined in children, because Pediatric GIST differ from those in adults in terms of immunohistochemistry and molecular genetics [36–39].

The final key question is: when is surgical intervention warranted for non-variceal GI bleed in children?

This series documents that surgical intervention is warranted in the following clinical situations:

(1) Acute severe bleed causing hemodynamic instability: while the etiology and site is usually diagnosed preoperatively in UGIB and LGIB; life-threatening SBB may be diagnosed at emergency laparotomy and IOE which is lifesaving and should not be delayed.
(2) At the other end of the spectrum is "subacute overt" or "occult GIB" with a definite surgically correctable cause identified on preoperative investigations, wherein an "early elective" surgery may be planned.

    The diagnostic armamentarium described in the text identified a surgically correctable etiology preoperatively in 56/75 (75%) cases in this group of "subacute overt" and occult GIB.
(3) The third clinical case scenario is the true "obscure" GI bleed, wherein the decision to explore and the appropriate timing of surgical exploration is most challenging. This refers to GIB, wherein site and etiology is undiagnosed despite extensive preoperative investigations. While hemodynamic stability is maintained; the

child has persistent or recurrent bleed and, hence, needs frequent blood transfusions to sustain hemodynamic stability and desired hematocrit.

The threshold for diagnostic laparotomy with IOE in "obscure" GIB should be lower in children as compared to adults, because of limited options for preoperative invasive SBE, poorer tolerance for recurrent bleed, and laparotomy identify a surgically correctable cause more often in children as substantiated by data in this series.

Key messages from this study This exclusively surgical series on 87 pediatric cases of NVGIB defines 3 sites of NVGIB, namely, UGI, SB, and LGI, respectively. Small bowel was the commonest site of NVGIB.

For each bleed site, four distinct etiology-based groups were identified which in order of frequency were: lesions with EGM (38%) followed by tumours (26%), ulcerative lesions (24%), and vascular pathology (9%).

With reference to GIB site, the commonest surgical etiology was as follows: (1) UGIB: duodenal ulcers; (2) SBB: lesions with EGM; and (3) LGIB: juvenile polyposis coli.

Bleed severity has management implications. At each site of GIB, bleed severity was classified, as acute severe bleed, subacute overt bleed and occult GIB, and etiology of each grade of bleed severity are presented.

Diagnostic algorithms tailored to bleed site, age-specific etiology, bleed severity, and associated abdominal and systemic symptoms have been presented.

Preoperative diagnosis was obtained in all UGIB and LGIB lesions. SBB presented a diagnostic challenge and diagnostic approach to SBB has been detailed. TcPS and CTEA were two key non-invasive investigations diagnosing 82% of SB surgical lesions preoperatively; the remaining 18% were diagnosed at laparotomy with provision for IOE.

Surgical considerations specific to bleed site, severity, and infrequently reported etiologies are detailed.

Management challenges were acute severe bleed with hemodynamic instability, occult GIB, SBB, obscure GIB, and bleed from infrequently reported etiologies. These were successfully managed by a multimodality and algorithmic approach. IOE has a useful diagnostic role in SBB and its technique and indications have been detailed.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Human rights statement All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent For this type of study, formal consent is not required.

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