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Case Report

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Pediatric upper gastrointestinal bleeding: a case series and review

C. B. Eke[1,2], J. O. T. Onyia[2], A. L. Eke[3]

[1]Department of Pediatrics, College of Medicine, University of Nigeria, Ituku/Ozalla Campus, Enugu;

[2]Gastroenterology Firm, Department of Paediatrics, University of Nigeria Teaching Hospital, Ituku/Ozalla, Enugu;

[3]Department of Medical Biochemistry, College of Medicine, University of Nigeria, Enugu Campus, Enugu, Nigeria

Corresponding author: J. O. T. Onyia**,** Department of Pediatrics, University of Nigeria Teaching Hospital, Ituku/Ozalla, Enugu, Nigeria.

Tel. +234-8063810681.

E-mail: onyiajot@gmail.com

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Abstract

Significant upper gastrointestinal bleedings are uncommon in children and potentially life-threatening. The etiology varies from one pediatric age group to the other, with some overlaps, and the presentation is usually in the form of hematemesis and or passage of melena stools. The key priorities in the assessment are to determine the severity of bleeding, degree of systemic upset, the site and cause, to enable application of adequate treatment protocols. However, wide gaps still exist in our setting with regard to the care of children with upper gastrointestinal bleeding.

We present three cases managed in our center that highlight available treatments that work to sensitize our pediatricians to current treatment modalities.

Introduction

Significant pediatric upper gastrointestinal bleedings, though uncommon, are potentially life-threatening.[1] The incidence of Upper Gastrointestinal Bleeding (UGIB) in children has been estimated at about 1-2/10,000 per year,[2] while among those admitted to an Intensive Care Unit in one study has been reported to be 0.4% to 1.6%.[3] Children aged 11-15 years are known to have the highest incidence, while those under one year are the least affected.[3]

The etiology varies from one pediatric age group to the other, with some overlaps. Among neonates and infants: swallowed maternal blood/amniotic fluid containing blood, esophagitis/gastritis, stress ulcers (in the critically ill), duplication cyst/vascular malformation, vitamin K deficiency, maternal Idiopathic Thrombocytopenic Purpura (ITP) or Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) use, trauma (nasogastric suctioning) are among the leading causes.[4,5]

The common causes observed in older children include Mallory-Weiss tear, hemorrhagic tonsillitis, *Helicobacter pylori*-associated gastritis, varices, esophagitis/gastritis, peptic ulcer disease, pulmonary hemosiderosis while in adolescents- Mallory-Weiss tear, pulmonary hemosiderosis, medication-induced ulceration (NSAIDs, aspirin, iron, doxycycline), button battery/foreign body, factitious bleeding among others have been implicated.[4,5] Overall the lists cannot be exhausted.

However, known risk factors for UGIB vary and include NSAIDs use and *Helicobacter pylori* infection, peptic ulcer disease, portal hypertension or varices, bleeding disorders, among others.[4,5]

Portal vein thrombosis as a known cause of extrahepatic portal vein obstruction, has been identified as a common cause of UGIB in older infants and younger children. Notable risk

factors for portal vein thrombosis among neonates include umbilical vein cannulation, dehydration, bacterial infection/sepsis, omphalitis, and thrombophilia.[6] These call for caution, particularly in our locale where there is still a dearth of facilities, including endoscopy and surgery to handle the associated long-term complications of variceal bleeding.

Also, some disease conditions have been associated with UGIB in children and include hematologic disorders such as hemophilia A and B and von Will brand disease, biliary atresia, portal vein thrombosis (extra-hepatic portal vein obstruction), primary sclerosing cholangitis, autoimmune hepatitis, Budd Chiari Syndrome, cystic fibrosis, among others.[4,7,8]

Furthermore there are some reported atypical causes of UGIB particularly ectopic varices, vasculitis (Henoch-Scholein purpura), Dieulafoy lesion (isolated submucosal vascular anomaly without ulceration protruding via mucosa causing serious or recurrent bleeding), Burkitt's lymphoma, Gastrointestinal Stromal Tumor (GIST), hemobilia-non-traumatic, some vascular malformations, such as Klippel Trenaunay syndrome, Osler Weber Rendu disease, blue rubber bleb nevus syndrome, progressive systemic sclerosis with telangiectasia.[4] However, these conditions are generally rare but could occur, and there is a need to be aware of them.

Anatomically, the upper Gastrointestinal (GI) tract extends from the esophagus to the ligament of Trietz, therefore UGIB is defined as bleeding derived from a source proximal to the ligament of Treitz.[9] The upper GI tract consists of the mouth, pharynx, esophagus, stomach and duodenum. The exact demarcation between the upper and lower tracts is the suspensory muscle of the duodenum which differentiates the embryonic borders between the foregut and midgut. This muscle attaches the superior border of the ascending duodenum to the diaphragm and shows the formal division between the duodenum and the jejunum.[9,10]

Often, the presentation of UGIB is in the form of hematemesis and or passage of melena stools.

Categorically, upper gastrointestinal bleeding is divided into variceal and non-variceal bleeds,[11] and the bleeding can be overt or occult/obscure in nature.[12]

Generally, overt gastrointestinal bleeding may occur as part of acute or chronic illnesses, while covert or obscure GI bleeding is often diagnosed in the course of investigating chronic anemias.[12]

In addition, the presentation of UGIB can be acute (in which case, the onset is sudden and without any previous signs or symptoms) or chronic (visible with recurrent melena/anemia); or non-visible with in-apparent loss but positive occult blood in stools and anemia.[12]

There is a paucity of epidemiological data on UGIB in the pediatric population in Nigeria.[13-15] Gastrointestinal bleeding is one of the most serious presenting complaints that any pediatric gastroenterologist could be faced with.

Therefore, a proper understanding of the various causes of upper gastrointestinal tract bleeding and the available effective treatments which are available is pivotal to assure successful outcomes.[4,12]

Also, proper assessment requires a balance between urgency when necessary and reassurance when the probable cause is less serious scenarios. The key priorities in the assessment are to determine the severity of bleeding, the degree of systemic upset, the site and cause, and then apply the treatment protocols.[4,12]

The objectives of the current study are to highlight some presentations and available treatments that work for upper GI bleeding encountered in our practice with a review of the literature in order to sensitize paediatricians in our resource limited setting.

Case Reports

Case 1

Baby A. N. is a 5-month old female who presented to the children's emergency unit with complaints of nasal discharge x 8/7, cough 8/7, vomiting of blood x 1/7 and fever of 12 hours, all prior to presentation.

The vomiting was provoked by feed, consisted of freshly ingested feeds mixed with copious amount of blood which appeared in clots. She had two episodes on the day of onset (less than an hour apart), was not forceful, no associated retching, and was non-projectile in in nature. The estimated volume was 30 mls per episode.

No prior ingestion of medications, including herbs or foods that could mimic the color of blood on vomitus. This necessitated presentation to the emergency unit, where she developed a low-grade fever and had an episode of dark, tarry stools.

She was born preterm at a gestational age of 34 weeks and was managed in the nursery, during which time she had umbilical catheterization on two occasions.

The past medical history was not revealing.

Physical examination showed a hemodynamically stable female child, who is not pale, and afebrile to touch. Other findings were hepatomegaly (2 cm), and splenomegaly (6 cm). She weighed 6.9 kg (98.6% of expected), length was 52 cm. Laboratory results, including full blood count, showed mild anemia with a hemoglobin level of 7.3g/dL, mild leukocytosis with normal differential counts, platelet count $226{,}000 \times 10^{-9}$/L, malarial parasite (+), while abdominal ultrasound revealed moderately enlarged spleen. The initial assessment was severe malaria to rule out bacteremia. She was triaged as per institutional protocols and started on intravenous omeprazole 7 mg 12 hourly, i.v. ceftriaxone 700 mg once daily, intravenous artesunate 17 mg at 0 hours, 12 hours, and 24 hours respectively, and thereafter changed to oral

artemether/lumefantrine (120/20 mg) given at 0 hours, 8 hours, 24 hours, 36 hours, 48 hours, and 60 hours respectively.

Subsequently she was found to be having worsening pallor with no new bleeding episodes. Repeat hemoglobin estimation was 6g/dL and was then transfused with 140 mL of fresh whole blood and discharged 3 days into the admission.

She was re-admitted 3 days after discharge, on account of increasing paleness of the body and generalized body weakness as well as further three episodes of bloody stools.

On examination she was found to be moderately pale (++) but not in shock. Urgent Packed Cell Volume (PCV) 18%; Hemoglobin (Hb) 6.1g/dL, platelet count 79,000mm/L^3, stool analysis revealed blood (+), protein (+). The child was reviewed by the gastroenterology team. A Doppler abdominal ultrasonography was done, which showed a moderately enlarged spleen, with visualization of the formation of new vessels around the thrombus (cavernoma), splenic vein, and normal liver architecture. The diagnosis was reviewed as acute upper gastrointestinal bleeding secondary to portal vein thrombosis (possibly from neonatal umbilical catheterization).

She was started on intravenous vitamin K 10 mg stat, omeprazole 28 mg daily, and octreotide continuous infusion @ 1 mcg/kg/day (given with normal saline infusion using solutes), which was tapered off after 72 hrs following cessation of further vomiting of blood or passage of melena stools.

She had a session of upper gastrointestinal endoscopy using 9 mm Olympus gastroscope under general anesthesia, and two large columns of esophageal varices in the mid-esophagus and several flattened varices around the cardia were found. See Figures 1 and 2, respectively

Further work was carried out, including liver function tests (within normal limits), coagulation screen (normal), repeat full blood count (normal limits), and serum protein C &S, which were all within normal limits.

She has had two sessions of endoscopic sclerotherapy three weeks apart using histoacryl glue with good outcomes. She is booked for a surveillance endoscopy in four weeks' time. The use of beta-blocker (propranolol) at the dose of 0.6 mg/kg/day was started after the first session of sclerotherapy but discontinued after 5 days owing to the development of bradycardia in the child.

Case 2

S. A. is an 8-year-old girl with a history of recurrent abdominal pain of 6 years duration and paleness of the body that started about a year ago.

She has had several blood transfusions (x4 since the onset of the illness). Her PCV ranged between 9-14% prior to the transfusions.

On evaluation by the referring doctor, she was found to have features consistent with iron deficiency anemia and was initially managed as such by hematologists.

Following the persistence of the anemia, she was referred to the pediatric gastroenterologists for further evaluation. Physical examination revealed mild pallor, no jaundice, with no peripheral edema.

Growth parameters (weight for age, height for age) were within normal limits.

Abdominal examination was not revealing, with no epigastric tenderness and no palpable abdominal masses. The musculoskeletal system examination showed no joint lesions/swellings. The cardiovascular system revealed a mild ejection systolic murmur, grade

2/6 (maximal at the aortic), no heaving, the apex beat was not displaced, and the peripheral pulses were normal.

Further investigations showed positive fecal occult blood, and screening for celiac disease (serum IgA anti-tissue transglutaminase, anti-endomysial antibody) was negative.

A working diagnosis of possible occult gastrointestinal bleeding was made. The following investigations were further done:

An upper gastrointestinal series (barium swallow, meal, and follow-through) showed an abnormal looking stomach especially in the antral pyloric portions and apparently ectatic first part of the duodenum.

The hemoglobin level was 7.2 g/dL while liver function tests (ALP 156, ALT 16, AST 19, bilirubin (total = 5.4, conjugated = 0.8 umol/L, GGT=12u/L), serum albumin = 37.8 g/L (globulin 28 g/L), and serum electrolytes were all within normal limits.

Autoantibodies (Total IgG, anti-nuclear antibody, anti-liver microsomal antibody) were normal. Echocardiography revealed mitral valve prolapse, and mild mitral and aortic valve regurgitation, to rule out rheumatic heart disease. Doppler ultrasound scan of the abdominopelvic region revealed diffuse wall thickening of the stomach involving the fundus and body of the organ especially. The wall thickened measures up to 17 mm with increased blood flow. Magnetic resonance imaging of the abdomen revealed a diffusely thickened stomach wall with prominent rugae, especially at the fundus, suggesting hypertrophic gastropathy.

Upper gastrointestinal endoscopy revealed raised, red mucosal stripes of dilated and tortuous blood vessels involving the cardia, fundus, body, and pyloric antrum. The first part of the duodenum was normal-looking. See Figure 3A and B, respectively. Biopsies were not taken in

order not to provoke further bleeding episodes. Hemostatic spray was applied on the gastric red vessels, which temporarily stopped the oozing of blood from the angiodysplastic vessels in the stomach but did not significantly affect the anemia and the child continued to need more blood transfusions. Attached are the endoscopic views of the stomach with the vascular findings.

A working diagnosis of vascular ectasia/Gastro-Entral Vascular Ectasia (GAVE), probably angiodysplasia was made. She was started on the following medications: lansoprazole 30 mg per oral once daily, sucralfate 500 mg per oral 12 hourly, syrup astyfer (oral iron), folic acid 5 mg po daily, vitamin C, and multivitamins. The child continued to have mild anemia, and a further plan was for her to have an interventional endoscopy with argon laser coagulation application on the bleeding gastric vessels, but it was not available in our center. As a result, her parents arranged for further management at Baptist Medical Centre, Downtown, where she had a Computed Tomography (CT) of the abdomen that revealed entire wall thickening of the stomach (fundus of stomach, greater & lesser curvature, body, & incisura) among other work up .including repeat gastroscopy with biopsy and histology which revealed hemorrhagic gastropathy. The definitive diagnosis is multiple bleeding gastric angiodysplastic lesions with iron deficiency anemia. She underwent Argon Plasma Coagulation (APC) @ 0.4 L/min & 20 watts with significant control.

Currently, she is on sirolimus (rapamycin), an mTOR inhibitor immunosuppressant (target 10 mg daily, now on 5 mg daily), resulting in fewer hospitalizations and no blood transfusion in the current 8 months of therapy.

Case 3

O. C. is a 15-year-old female referred from the Mother of Christ Specialist Hospital, Enugu, 5 months ago with complaints of vomiting of blood and epigastric pain.

Vomitus usually consists of fresh blood estimated to be between 50 mls to 100 mls, often triggered by agitation or anxiety. There is no history of associated blood in stools. She has had similar episodes on about four different occasions, with associated bleeding through the nostrils occurring in the second episode. Often, she has premonitions about the vomiting of blood each time she is agitated. There is a past medical history of a similar illness 4 years ago following agitation by her siblings. Physical examination findings were essentially normal.

A diagnosis of upper gastrointestinal bleeding cause was made. Full blood count was done and the parameters were all within normal limits, fecal occult blood was negative, and coagulation profile and liver function tests were within normal limits. Upper gastrointestinal endoscopy showed no significant findings, and biopsies were taken for histology and *Helicobacter pylori* screening. Histology revealed normal esophageal, gastric, and duodenal tissues. *Helicobacter pylori* infection was excluded.

Otorhinolaryngology review was sought on account of the associated episode of epistaxis, and subsequently, a nasopharyngoscopy was done, which revealed mild bilateral inferior turbinate hypertrophy with intact, healthy mucosa on both nasal cavities. CT scan of the nasopharynx and nose revealed normal findings. The patient was placed on oral rabeprazole 20 mg daily for 2 weeks.

A repeat gastroscopy was done, and no source of bleeding was identified.

Following no significant findings on examination and work-up, a diagnosis of factitious disorder was made. She has had serial counseling in the clinic.

The last clinic visit was 2 months ago, symptoms are now resolved as the last episode of upper GI bleeding was 2 months prior to the visit, and the patient had no new complaints. Physical examination findings were normal.

Discussion

Gastrointestinal bleeding is one of the most serious presenting complaints that any gastroenterologist may encounter in the course of practice.[1] Children with GI bleeding may present in a stable condition or with different forms of hemodynamic instability, including hypovolemic shock. Hence, the management of GI bleeding requires accurate, prompt diagnosis and treatment of the underlying condition.[4] The initial approach in cases with shock should include an assessment of the airway, breathing, and circulation.[2] An intravenous access is usually secured, and it could be particularly helpful in the event of profound hemorrhage in situations of difficult intravenous access as such line could be lifesaving upon usage for rapid administration of fluids and or blood as well as pharmacological agents needed in resuscitation.

Blood transfusion is appropriate for unstable patients. The hemogram should be determined. The amount of blood to be transfused should be determined and aimed not to overshoot post-transfusion hemoglobin of 8- 9 g/dL. This conservative hemogram target is hinged on the fact that more liberal transfusion can lead to higher variceal pressures, thereby increasing the risk of re-bleeding.[4]

Individuals with active bleeding and coagulopathy should be considered for transfusion with fresh frozen plasma, while those with thrombocytopaenia with platelet count of $30{,}000 \times 10^{-9}$/L will require platelet concentrate transfusion.

In situations as in Case 1 above, where the patient presents with continuing bleeding while performing an initial evaluation, the child should be kept on *nil per os* in order not to provoke further bleeding while preparations for possible diagnostic endoscopy, which may require sedation, go on.

Vasoactive agents, including somatostatin analogs like octreotide, may be needed particularly in variceal bleeding as well as non-variceal bleeding to achieve splanchnic vaso-constriction and ultimately reduce bleeding prior to endoscopy as well as the re-bleeding risk.[1,14]

Studies have shown that cytoprotective agents such as sucralfate and misoprostol have no role in the treatment of clinically significant UGIB in children,[1,16] and were not considered in the cases reported.

The use of parenteral vitamin K (infants, 1-2 mg/dose, children 5-10 mg) and Proton Pump Inhibitor (PPI) should be administered empirically in case of a major UGIB.[17] Such therapy in cases of mucosal bleeds as in Case 1 above is mainly directed at neutralization and/or prevention of gastric acid release. High dose PPIs is often more efficacious than histamins-2 receptor blockers,[17] as PPI reduces the frequency of re-bleeding, need for surgery, and length of hospitalization and overall improves clinical outcomes.[18] Patients with bleeding ulcers should be screened for *H. pylori* infection, which survives in acidic gastric pH and treated if positive.[19]

In all cases of massive GI bleeding, instituting an early endoscopic intervention should be prioritized and performed after initial resuscitation and hemodynamic stabilization.[20] This principle was applicable in Case 1, and the diagnosis of esophageal varices was made, and further treatment, including sclerotherapy, was started to forestall further bleeding.

In children who have a high risk of variceal bleeding, the use of beta-blockers has been suggested as primary prophylaxis for variceal bleeding.[21] Generally, beta-blockers are effective in preventing variceal bleeding in children with portal hypertension. A study that evaluated the use of beta blockers in non-bleeding portal hypertensive children showed a significant decrease in the grade of oesophageal varices, and a reduction in the severity of associated gastro-esophageal varices.[21]

However, caution should be taken in the use of the beta-blockade as some children may develop some adverse effects, including drowsiness, bronchospasm, bradycardia, and hypotension. However, in some cases, failed beta-blockage in variceal bleeding has also been reported.[22,4]

In Case 1 reported above, beta-blocker (propranolol) was introduced at the dose of 0.6mg/kg/day,[4] after the first session of sclerotherapy but was however withdrawn after 5 days due to the development of bradycardia.[23]

Also, it is important to note that possible causes of portal vein thrombosis include neonatal omphalitis, thrombophilia, and umbilical catheterization. In this report, Case 1 had a couple of umbilical catheterizations as a neonate and was not followed up for possible development of portal hypertension with variceal bleeding.

It is known that during umbilical vein catheterization, advances to the inferior vena cava could be made. Following anatomical variants or incorrect placements, different levels can be advanced at the portal sinus level creating susceptibility to PVT through vascular endothelial damage and occlusion with cases of unresolved thrombosis (either spontaneously or managed with anti-coagulation), which may develop portal hypertension and subsequently upper gastrointestinal bleeding.[24] Hence, pediatricians and neonatologists caring for this group of children should ensure that umbilical cannulation should only be applied when absolutely

necessary, and if any suspicion of portal thrombosis, such babies should be investigated using doppler ultrasound and managed accordingly, including follow-up to avert the development of varices, and later variceal bleeding.

According to the American Society of Gastrointestinal Endoscopy (ASGE), esophagogastroduodenoscopy is recommended in the presence of melena and haematemesis, [25] as it could be applied in diagnostic, therapeutic, and surveillance modalities of treatment.[8] Endoscopy is a very useful first-step diagnostic tool in the localization of gastrointestinal bleeding in the pediatric population. In our series, the three reported cases all had an endoscopy, which identified the source of bleeding in two (66.7%) of the cases. If no source of bleeding is found during the first endoscopic procedure, additional procedures are often non-diagnostic. In Case 3 of factitious upper GI bleeding, further investigations by the otorhinolaryngology team, including CT scan and nasopharyngoscopy, were unrevealing. However, some authorities have recommended further additional diagnostic workup in factitious bleeding in cases with ongoing undiagnosed gastrointestinal bleeding requiring blood transfusions.[25]

In addition, endoscopic intervention often reduces the rate of re-bleeding, the need for surgical intervention, and mortality in high risk cases with UGIB.

In Cases 1 and 2 reported above endoscopic interventions were applied. Case 1 was started on endoscopic sclerotherapy using histoacyrl glue as the age and possible small size of the varices precluded the use of variceal band ligation with good results while Case 2 had application of hemostatic spray that temporarily stopped the oozing of blood from the angiodysplasia in the stomach but did not significantly affect the anemia while child continued to need more blood transfusion before parents decision to seek further medical management in abroad where after review was subsequently started on sirolimus (rapamycin), a specific and potent inhibitor of

Mtor, a serine/threonine kinase in the Phosphor-Inositide-3- Kinase (P13K)/Akt pathway which regulates numerous cellular processes including angiogenesis and cell growth.[26] Sirolimus has been used in other vascular anomalies with good results.[27]

Factitious gastrointestinal bleeding is a manifestation of factitious disorder wherein the individual feigns/acts gastrointestinal bleeding in the absence of external gain.[28] It was indeed a very challenging diagnosis to make, as in Case 3. Further studies, including GI contrast studies, endoscopies with biopsies, and a barrage of blood and imaging studies, including ultrasound and CT scan, in addition to multidisciplinary collaboration and, in this instance, with the otolaryngologists as the patient reported epistaxis at some time. All these interventions often lead to increased risks of iatrogenic complications and direct health care costs. This group of patients demonstrates characteristic behaviors that may go unnoticed except when addressed. In our index patient (Case 3), once she is involved in any anxious situation in the school, next she becomes very panicky, and the anxiety is often followed by bleeding episodes. Further history only gave out the diagnosis when extensive workups in the child were all unrevealing. The child and parents were reassured, and she underwent psychotherapy with no further bleeding episodes so far.

Conclusions

Upper GI bleeding, though uncommon in children, could be challenging to manage. A proper understanding of the various causes of upper gastrointestinal tract bleeding, and their available effective treatments, are key to assuring successful outcomes in a resource-limited economy.

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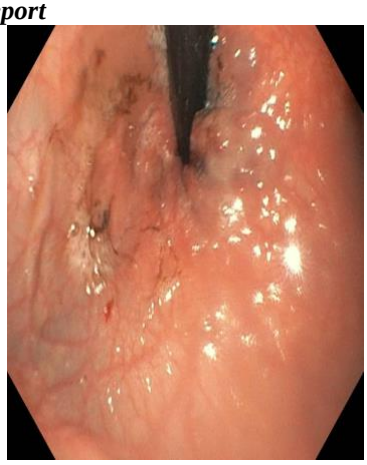


Figure 1. Several flattened varices around the cardia.

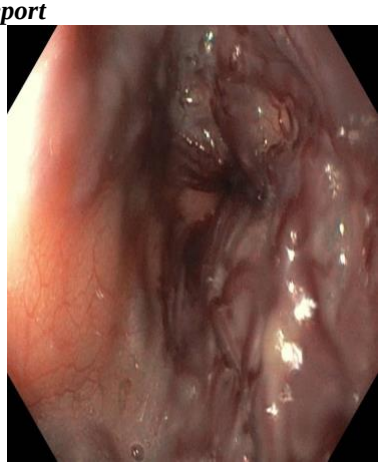
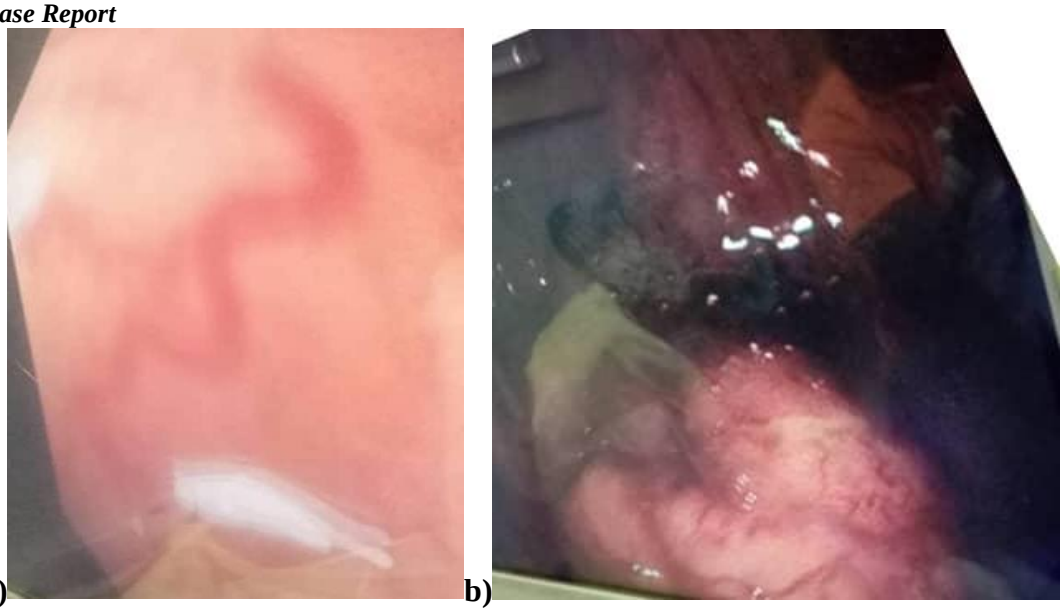


Figure 2. Large columns of esophageal varices on the mid-esophagus.



a) **b)**

Figure 3. Endoscopic views of the stomach show raised, red mucosal stripes of dilated and tortuous blood vessels involving the cardia, fundus, body, and pyloric antrum.