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Severe Upper Gastrointestinal Hemorrhage from Linear Gastric Ulcers in Large Hiatal Hernias: a Large Prospective Case Series of Cameron Ulcers

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Abstract

Background and study aims—Cameron ulcers are a rare but clinically significant cause of severe upper gastrointestinal hemorrhage (SUGIH). Our aims were to describe (1) the diagnosis, treatment and outcomes of patients with Cameron ulcers causing hospitalization for SUGIH, (2) the differences between patients with occult vs. overt bleeding and (3) between patients treated surgically and medically.

Patients and methods—Over the past 17 years, all consecutive patients hospitalized in our two tertiary referral medical centers for severe UGIH or severe obscure GIH and entered into our large prospective databasis were screened for Cameron ulcer diagnosis.

Results—Cameron ulcers were diagnosed in 25 patients of 3960 patients with SUGIH (0.6%). 21 patients had follow-up (median [IQR] time of 20.4 months [8.5–31.8]). Patients were more often elderly females with chronic anemia, always had large hiatal hernias, and were usually referred for obscure SUGIH. Twelve (57.2%) patients were referred to surgery for rebleeding and recurrent blood loss while treated with high dose of proton pump inhibitors (PPI). 9 (42.8%) other patients continued PPI without any rebleeding during the follow-up. Patients with overt bleeding had significantly more prior hospitalizations for SUGIH, more often stigmata of hemorrhage on ulcers, and more red blood cell transfusions than patients with occult bleeding. However, there was no difference in rebleeding and mortality rates between the two groups.

Conclusions—Cameron ulcers in large hiatal hernias are an uncommon cause of SUGIH. Most of patients are referred for obscure GIH. The choice of medical vs. surgical therapy should be individualized.

Keywords

Cameron ulcers; hiatal hernia; gastrointestinal bleeding; anemia; fundoplication

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INTRODUCTION

Linear gastric ulcers in hiatal hernias (e.g. Cameron ulcers) can be associated with overt or occult gastrointestinal hemorrhage (GIH), either severe acute or chronic [1]. In 1986, Cameron and Higgins first described these lesions as either erosions or ulcerations in the gastric mucosa located at the diaphragmatic hiatus on endoscopy (EGD) and usually appearing as multiple linear lesions on the crests of gastric folds [2]. Cameron lesions were reported to be seen in 5 % of patients with hiatal hernias who had routine panendoscopy and the prevalence depended upon the size of the hernia sac, increasing in prevalence with larger hernia sacs [3].

The pathogenesis of Cameron lesions is not known. Published hypotheses include: mechanical trauma to mucosa as the hernia slides through the diaphragmatic hiatus, pressure-induced mucosal ischemia at the diaphragmatic hiatus, gastric acid exposure, and lymphatic obstruction [4].

The relative rarity of these Cameron ulcers as a cause of severe upper gastrointestinal hemorrhage (SUGIH) makes them a diagnostic challenge, since identification of these lesions requires a high index of suspicion, a meticulous endoscopic examination, and an experienced endoscopist.

In a large cohort of patients prospectively evaluated after hospitalization for SUGIH or severe obscure GIH, our purposes were:

1. to describe the endoscopic diagnosis, treatment and outcomes of consecutive patients with Cameron ulcer;
2. to contrast patients with a clinical presentation of occult vs. overt bleeding; and
3. to compare the outcomes of patients treated surgically and medically.

METHODS

Patients

All consecutive patients hospitalized at UCLA Ronald Reagan Medical Center and the VA West Los Angeles Medical Hospital for the management of severe UGIH or obscure GIH between January 1995 and February 2012 had push enteroscopy, were assessed by the CURE Hemostasis Research Team, and were prospectively evaluated. If no diagnosis was made by push enteroscopy, patients also underwent colonoscopy, capsule endoscopy, technetium labeled red blood cell scan, or angiogram to identify the source of bleeding. The medical records were prospectively reviewed and patients were followed-up from the date of diagnosis. The outcomes data were prospectively entered onto standard CURE research forms by the investigators and a research coordinator. All data were de-identified and entered onto computer data files by experienced data managers in a large CURE prospective database of SUGIH.

In this database of patients hospitalized for SGIH, we retrospectively screened for Cameron ulcers as the final diagnosis of the UGIH or obscure GIH. Cameron ulcer diagnosis was coded in database for any benign linear gastric ulcers located at the site of the diaphragmatic constriction in patients with hiatal hernia recognized as the final diagnosis of the index

hemorrhage (after all appropriate explorations). We did not include focal ulcers located above the diaphragmatic constriction in the herniated portion of the stomach or at the gastroesophageal (GE) junction, since these ulcers were due to peptic ulceration or gastroesophageal reflux disease (GERD) respectively and they had a different physiopathology. A large hiatal hernia was defined by hiatal hernia longer than 50 mm, as endoscopically determined as the difference in the distance from the incisors by pulling back the endoscope from diaphragmatic pinch distally to the squamocolumnar GE junction, proximally. Severe UGIH was defined as decrease in hemoglobin from baseline of ≥ 2 g/dl and/or transfusions of ≥ 2 units of packed red blood cell (PRBC). Obscure GIH was defined as severe GIH with no bleeding source found on panendoscopy and colonoscopy performed by referral gastroenterologists in outside hospitals. .

Every report and all endoscopic pictures and/or videos of Cameron ulcer cases were reviewed by the PI (D.J.) and the co-investigators of the CURE Hemostasis Research Group who performed the push enteroscopy at UCLA or WLA VA Medical Centers to confirm the endoscopic diagnosis of Cameron ulcers.

Data collected

The database provided key baseline information such as patients demographics, history of prior bleeds and transfusions, medications (including NSAIDs, aspirin and anticoagulants), symptoms and signs of GI hemorrhage at presentation, co-morbidities, routine laboratory results (hematocrit, hemoglobin and platelets count), endoscopic findings (mean size and number of Cameron ulcers, SRH, associated lesions, and endoscopic hemostasis if performed). Also prospectively gathered routine short and long-term outcomes after diagnosis were available such as surgery, rebleeding, transfusions, and death. Short term outcomes were considered until 30 days post-entry and long term outcomes between 30 days to the last follow-up in several months. The length of ulcers was estimated with open biopsy forceps. GI bleeding was described as overt when patient's presentation to the hospital for admission included melena, hematochezia and/or hematemesis or occult when patient presented with severe iron deficiency anemia and occult blood positive stools. We classified patients on an intention to-treat basis in either the "surgical group" or the "medically treated group". In this prospective study, referral for surgery was at the discretion of the treating gastroenterologist.

Data management and statistical analysis

SAS software, version 9.1, (SAS Institute, Cary, NC) was used for data management. P values were calculated by the Fisher exact test for discrete variables and by the Wilcoxon rank-sum test for continuous variables (GraphPad Prism version 4.00, GraphPad Software, San Diego, CA). $P < 0.05$ was considered significant in all these analyses.

RESULTS

Patients, demographics, and prior investigations

Twenty-five patients with Cameron ulcers were identified among 3960 patients hospitalized with severe UGIH or obscure GIH. The overall prevalence of bleeding Cameron ulcers was 0.6 %. The specific prevalence in overt UGIH and obscure GI bleeding groups were estimated to be 0.2 % (9/3543) and 3.8 % (16/417), respectively ($p < 0.0001$). The prevalence of patients with Cameron ulcers at UCLA Ronald Reagan Medical Center and the VA West Los Angeles Medical Hospital were different and were 0.8 % (23/2856) and 0.2 % (2/1104), respectively.

Short-term follow-up (until discharge or 30 days) was available for 21 patients and long-term follow-up (after 30 days) was available for 20 patients (one patient died at 18 days). The median time of long-term follow-up was 20.4 months (IQR 8.5–31.8).

Demographics and characteristics of patients at the time of diagnosis were summarized in Table 1. The mean age of patients at the time of diagnosis was 70.9 years $\pm$ 10 (range 36–86) and 64 % were women. Sixteen patients (64 %) had severe overt GI bleeding, whereas 9 (36 %) had occult hemorrhage. Most of patients-22 (88 %)-had chronic anemia on presentation and more than half of them were already being treated with oral iron (14/22–56 %). Thirteen patients (52 %) were on proton pump inhibitors (PPI's) and five (20 %) took H₂-receptors antagonists (H₂RA) drugs. Only 24 % of the patients took non-steroidal anti-inflammatory drugs (NSAID's) and 12 % aspirin, prior to the index SGH before our diagnosis. The mean hemoglobin (Hb) was 9 g/dl $\pm$ 2.1 at the time of diagnosis.

Twenty patients (80 %) had a history of prior bleeding episodes and the mean units of RBC transfusion was 6.4 $\pm$ 6.6 units per patient before our diagnosis of Cameron ulcers. Prior to our diagnosis, the mean number of prior bleeding episodes requiring hospitalization was 2.4 $\pm$ 2.5 per patient. For only 6 patients (30 %), often potential causes of bleeding were identified as the possible cause of these prior bleeding episodes. Diagnoses on endoscopy include UGI angiomata in 3 patients, gastric ulcer in 2 patients, and esophagitis in 1 patient. However, for most patients (14/25–56 %) the diagnosis was still coded as obscure by their referring physicians. Because of recurrent GI bleeding and a final definitive diagnosis of Cameron ulcer by us, the GI bleeding was documented to be secondary to Cameron ulcers that were not seen or recognized by outside gastroenterologists on their endoscopies.

Endoscopic diagnoses and treatment

All patients had large hiatal hernias (>50mm). The mean $\pm$ SD (and median size [IQR]) of the hiatal hernias was 78.3 mm $\pm$ 13.3 (80 [72.5–80]) with a range of 50 to 100. Multiple Cameron ulcers were seen in 88 % of patients. The mean $\pm$ SD (median [IQR]) number of Cameron ulcers per patient was 2.7 $\pm$ 1.3 (3 [2–3.75]). The mean $\pm$ SD (and median [IQR]) length were 13.3 $\pm$ 9.0 mm (10 mm [7.25–20]) (See Figures 1a and 1b as examples). Additional non-Cameron lesions were seen at EGD in 13 patients (52 %) but were not considered to be the bleeding site. These included gastric polyps in 5, mild esophagitis in 3, gastric erosions in 2, esophageal ulcer in 1, small esophageal varices in 1, angiomata in 1, and a gastric ulcer in 1. None of these lesions had stigmata of recent hemorrhage (SRH). For the Cameron ulcers, 4 (16 %) patients had oozing, 2 (8 %) an adherent clot, while 19 (76 %) had clean ulcers. Only one patient with oozing and visible vessel underwent endoscopic treatment with hemoclip (see Figure 2a and 2b). The 3 other patients with oozing had no visible vessel and hemorrhage was minimal. There were all treated by PPI, and they did not have rebleeding, surgery, or death at last follow-up.

Short term treatment and outcomes

For 30 day outcomes, the mean length of initial hospital stay was 2.2 $\pm$ 3.5 days (range 1–18). Four (16 %) patients required intensive care unit (ICU) stays with a mean length of 1 $\pm$ 3.7 days. Fourteen (56 %) patients were transfused, with a mean number of units of RBC transfused of 1.9 $\pm$ 2.2 units. All patients were treated with daily PPI's and oral iron sulfate after diagnosis. For patients already on acid suppressive drugs, the dose was increased. Two (9.5 %) of the 21 patients had rebleeding within 30 days after the endoscopy (including one who had hemoclipping for an oozing lesion). This patient suffered seizures refractory to multiple anti-epileptic medications and died after 18 days in the ICU without rebleeding. No other patients died within 30 days of diagnosis.

Long term treatment and outcomes

Twelve (57 %) of the 21 patients with available short-term follow-up were referred for surgery. These 12 patients were considered as the “surgical group” (Table 2). Actually, 10 of these 12 patients underwent surgery and two did not: one died before (at 18 days) and one refused surgery. Surgery was scheduled after failure of medical treatment (double dose of PPI’s) with rebleeding requiring hospitalization in three (27 %) patients and recurrent or chronic blood loss in 8 (73 %) other patients. Eight of the 10 patients (80 %) patients had Nissen fundoplication and two (20 %) had laparoscopic gastropexy. The time between the diagnosis and surgery was $240.5 \text{ days} \pm 345.9$ (range 30–1003). The delay in surgery was because all patients were on double dose PPI’s and the managing gastroenterologists and patients were waiting to see whether they had another episode of severe bleeding before they finally deciding whether to have surgery.

All 9 patients medically treated (“medically treated group”) received PPI therapy and 44.4 % also received iron supplementation for chronic anemia. On our intention to treat analysis, there was no significant difference between medically treated group (9 patients) and surgical group (11 patients) in baseline age ($71.4 \text{ years} \pm 5.9$ vs. 69.8 ± 15.7 , $p=0.59$); overt vs. occult bleeding; presence of chronic anemia; presence of severe co-morbidities; hematocrit (27.2 ± 5.3 vs. 26.4 ± 8.8 , $p=0.73$); platelet count ($314,800/\text{mm}^3 \pm 130,900$ vs. $323,100/\text{mm}^3 \pm 111,700$, $p=0.07$); presentation with syncope, hypotension or shock ($p=0.48$); recent use of NSAID’s, aspirin or anticoagulants before the GIH; or prevalence of major SRH at initial EGD. There was no difference in short-term (up to 30 days) outcomes (RBC transfusions, length of hospitalization, and rebleeding or mortality rates).

The median long-term follow-up (IQR) of the surgical group vs. medically treated group was similar (12.3 months [3.2–31.8] vs. 25.9 months [12.7–31.6]). None of the patients treated medically rebled, had recurrent or chronic blood loss, or died because of Cameron ulcers or GI hemorrhage. After surgery, no patients had rebleeding or blood loss anemia.

Overt versus occult bleeders

Statistically significant differences were seen at baseline in patients hospitalized with overt vs. occult bleeding (Table 3). The mean age in both groups was similar. Patients in the occult group had significantly more prior hospitalizations for bleeding of unknown source than patients of the overt group (100 % vs. 58.3 %, $p<0.05$). SRH were significantly more prevalent in the overt group than the occult group (41.7 % vs. 0 %, $p<0.05$). However, prevalences of NSAID’s, aspirin, any anticoagulant agent, and baseline hematocrits were similar.

Thirty-day outcomes of Cameron ulcer patients with overt bleeding such as rebleeding and death were similar to patients with occult bleeding, despite significantly more number of units of RBC transfusion in the overt group than the occult group at baseline for resuscitation (Table 4).

The median long-term follow-up of overt (15.7 months [6.4–34.9]) vs. occult (25.2 months [11.9–28.1]) UGIB was similar. After diagnosis, there was no difference in long-term outcomes between overt and occult groups in terms of surgery, rebleeding, and death during prospective follow-ups.

DISCUSSION

Various hypotheses have been proposed to explain the pathophysiology of Cameron ulcers. In our study, the proportion of patients with NSAID’s and aspirin was low, suggesting a different etiology of Cameron ulcers with severe hemorrhage than classic gastric ulcers.

Also, for *H. pylori*, Weston *et al.* [3] reported that only a minority of patients with Cameron ulcers were positive for *H. pylori* (32 %). Therefore, *H. pylori* is unlikely to be involved in the pathogenesis of Cameron ulcers. All the patients with SGIH in our study had large hiatal hernias and this is a necessary condition and plays a key role in the pathogenesis of Cameron lesions. The prevalence of Cameron lesions has been reported to correlate with the size of the hiatal hernia, with a risk of 10–20 % for hiatal hernias larger than 5cm [5]. It was previously estimated that Cameron lesions (hemorrhagic or not) are present in 5.2 % of patients with hiatal hernias who had routine EGD's examinations [3]. Ischemic or mechanical factors as the pathogenesis Cameron ulcers seem to be the most likely explanation for development of Cameron lesions. Moskovitz *et al.* [6] biopsied several Cameron lesions and found that histopathologically many of these lesions had focal ischemic gastropathy consisting of coagulation necrosis.

Cameron ulcers are an uncommon cause of severe UGI bleeding. Only a few reports about the clinical and endoscopic features of this lesion have been published [2], [7]. Our case series confirms that Cameron ulcers can be a clinically significant cause of severe GIH. Cameron ulcers should be in the differential diagnosis of all patients with large hiatal hernias and severe anemia or UGIH, whether overt or occult. We confirmed that patients with Cameron lesions are typically elderly females with multiple Cameron lesions in large hiatal hernias [8]. The prevalence of Cameron ulcers in patients with severe UGIH or occult GIH was only 0.6 %. It was clearly higher in obscure GI bleeders (3.8 %) than in patients referred for suspected UGI bleeding (0.2 %). This prevalence for Cameron ulcers causing obscure GIB is similar to a previous study of 206 consecutive obscure GI bleeders referred for push enteroscopy conducted by our group. In that study, the CURE Hemostasis Group reported that almost half patients with obscure GIH had missed UGI lesions within reach of standard EGD that were missed by referral endoscopists. The most common lesions were angiomias, ulcers, Cameron ulcers, GAVE or watermelon stomach, and gastric varices. In that push enteroscopy series of obscure GI bleeders, the prevalence of Cameron ulcers was estimated 6 % (12/206 patients) [9], [10].

The fact that the prevalence of Cameron ulcers in patients hospitalized with SUGIH is low in this report may be due to several explanations. First, this study was conducted in tertiary academic centers. It is possible that our prevalence is low because some patients were diagnosed with Cameron ulcers outside our institution and were not sent to our center for investigation. Secondly, we report the prevalence of Cameron ulcers in patients with severe GIH and not on all patients with large hiatal hernias, some with mild anemia and some with no bleeding, but may have endoscopic erosions or superficial ulcers as previously reported [3]. Third, the usual clinical profile of patients with Cameron ulcers is that of an elderly woman, which is not the profile of patients of a Veteran Administration medical center. The prevalence of Cameron ulcers causing SUGIH is probably closer to the prevalence of patients with Cameron ulcers at UCLA Ronald Reagan Medical Center (0.8 %) than to the prevalence at the VA West Los Angeles Medical Hospital (0.2 %), which has few elderly female patients.

Most of Cameron ulcers in this report were probably missed at the time of initial evaluation by community gastroenterologists during their endoscopies of patients for recurrent GI bleeding and they instead diagnosed them as obscure GI hemorrhage. The higher prevalence of Cameron ulcers in patients with obscure GI bleeding (3.4 %) than in patients with overt UGI bleeding clearly highlights the need for a complete evaluation of the neck of the hernia, including antograde and retrograde views for diagnosing Cameron ulcers. More timely diagnosis, targeted therapy or early referral to a tertiary medical center could have resulted in better patient outcomes including fewer hospitalizations and endoscopies for obscure GI bleeding and quicker treatment of severe anemia. Teaching all endoscopists, internists or

primary care physicians, and surgeons about Cameron ulcerations (which are non-peptic and not related to GERD) is critical in addition to their association with large hiatal hernias and their potential for serious complications.

In this study, of the 21 patients with long-term follow-up, 12 (57.2 %) patients had refractory or recurrent GI bleeding despite double dose PPI's. These patients were referred for surgery. We reported no significant differences for patients treated medically vs. patients referred for surgery in baseline variables or in 30 day outcomes. Therefore, no variable was identified to predict rebleeding or to select which patients may need future surgery. In this prospective cohort series, surgical treatment to repair the hernia sac of Cameron ulcer patients was recommended and usually performed for patients with refractory or recurrent GI bleeding and recurrent Cameron ulcers on repeat EGD. To determine independent predictors of rebleeding or surgery in Cameron ulcers patients, a multivariate analysis would be necessary but the limited sample size of our study did not allow us to perform it and the low prevalence of Cameron ulcers may limit this type of analysis in future studies.

The time between the bleeding episode and the surgery was long. The potential of medical therapy to control GI bleeding and anemia was tried first. Indeed, in our study all medically treated patient (9/21–42.8%) followed-up during a median (IQR) of 25.9 months (12.7–31.6) did not rebleed. For patients already on a single dose of PPI at the time of our diagnosis, doubling of the PPI dosage was recommended for healing the Cameron lesions that were diagnosed. Along with increasing the oral iron dosage, this was usually effective for control of symptoms and to increase the hemoglobin level of half of our patients who had been hospitalized for these problems.

Most literature reports about surgical treatment of Cameron ulcers are case series. Small surgical case series from 2 to 5 patients with overt or occult GI bleeding reported successful treatment with surgery but did not report any comparison with medical treatment [11–14]. There is no consensus on the laparoscopic or open type of surgical repair with or without fundoplication, [15], [16]. The largest surgical series to date was published by Boyd *et al.* [8] in 1991. The majority of patients were females with a median age of 67 years. Patients were followed up with a median duration (range) of 24 months (0–90). Among them, 27 of the 49 patients presented with occult or acute GI bleeding. Only four were treated surgically. Among patients who were treated medically and not surgically, 14/42 (33%) in whom follow-up data was available, had documented episodes of rebleeding. However the study of Boyd *et al.* [8] included true Cameron ulcers situated at the site of the diaphragmatic constriction as well as ulcerations above this in the herniated portion of the stomach, which are probably related to GERD and are not true Cameron ulcers. Weston *et al.* [3] only included true Cameron ulcers and reported a rebleeding rate of 6.3 % for patients treated medically. In our study, the rebleeding rate at 30 day was 9.5 %, and during long-term follow-up, surgery was recommended for 11 (55 %) patients because they had refractory or recurrent GI bleeding.

Endoscopic plication, a novel and emerging treatment option for patients with GERD and small hiatal hernias, has not been evaluated for Cameron ulcer treatment. This is probably because the hiatal hernias are too large for effective endoscopic management and reduction in the hiatal hernia and surgical repair of the diaphragm may also be required

Endoscopic hemostasis of Cameron ulcers is rarely reported. Lin *et al.* [17] reported that endoscopic band ligation may be effective. For Cameron ulcers, the application and guidelines for appropriate techniques for endoscopic hemostasis have not yet been established. Most of our patients were not treated endoscopically. However, endoscopic treatment may offer definitive initial hemostasis and a less-invasive alternative to urgent

surgery in case of severe acute UGIH from Cameron ulcers. Hemoclips, which do not cause significant tissue damage compared to thermal coagulation, should be considered whenever hemostasis is warranted for Cameron ulcers with major SRH [18].

Weston *et al.* [3] reported that patients with acute overt bleeding had more severe disease and worse long-term outcomes. This contrasts with our study results where patients with overt GI bleeding had significantly higher baseline units of RBC transfusion and a higher prevalence of Cameron ulcers with SRH than patients with occult GI bleeding, but there was no difference in short and long term clinical outcomes such as rebleeding, surgery, or mortality after diagnosis (Table 4).

This study has several limitations. It is a relatively small study, and even though data were prospectively recorded, the screening for Cameron ulcer diagnosis was retrospective leading to the possibility of missing patients with such lesions. However, we report the largest cohort of Cameron ulcers and provide data about diagnosis, treatment and outcomes of Cameron ulcers. Another limitation is the absence of standardized follow-up. Patients had different durations of follow-up because our database evolved between 1995 and 2012. However, the follow-up duration was similar for medically treated patients and patients referred for surgery and also similar for patients with either overt or occult GI bleeding. One other limitation of this study was that all patients did not undergo a routine second-look endoscopy after the Cameron ulcer diagnosis and treatment, unless they rebled. Therefore the recurrence of Cameron ulcers and/or their healing could not be documented after medical or surgical therapy. In 1996 Weston *et al.* [3] reported that about a third of patients treated medically had an endoscopic recurrence of the Cameron lesions.

Our conclusions from this study are:

1. Cameron ulcers occur in large hiatal hernias and can cause severe GI hemorrhage and multiple hospitalizations prior to a specific diagnosis and treatment.
2. The relative rarity of Cameron ulcers makes them a diagnostic challenge.
3. If the endoscopist is unaware of this disorder or does not look carefully for Cameron ulcers, they are often missed at the time of initial upper endoscopic evaluation.
4. The choice of therapy, particularly surgery, should be individualized for each patient. We recommend medical treatment first, and surgery for patients who fail medical therapy, including twice daily PPI and iron supplementation.

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Abbreviations

EGD	esophagogastroduodenoscopy
GERD	gastroesophageal reflux disease

GI	gastrointestinal
Hct	hematocrit
H2RA	H2-receptors antagonist
ICU	intensive care unit
IQR	interquartile range
NSAID	non-steroidal anti-inflammatory drug
PPI	proton pump inhibitor
PRBC	Packed red blood cell
SRH	Stigmata of recent hemorrhage
SUGIH	severe upper gastrointestinal hemorrhage

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Figure 1. Example of Cameron Ulcers

a and b: multiple linear ulcers in a large hiatal hernia on end-on (1a) and retroflexion (1b) at diaphragmatic pinch.

Figure 2. Example of Cameron Ulcers
a and b: Oozing Cameron Ulcer before (2a) and after hemoclippping (2b)

Table 1

Characteristics of Cameron Ulcer Patients

	Cameron ulcer patients n=25
Age (mean $\pm$ SD, range)	70.9 years $\pm$ 10 (36–86)
Sex (no. male/female)	6/19 (24 %/64 %)
Ethnicity, no. (%)	
Caucasian	20 (80 %)
Hispanic	3 (12 %)
Black	1 (4 %)
Asian	1 (4 %)
Occult bleeders, no. (%)	9 (36 %)
Overt bleeders, no. (%)	16 (64 %)
Melena	87.5 %
Hematemesis	31.3 %
Hematochezia	6.3 %
Hypotension, syncope or shock, no. (%)	2 (8 %)
NSAID's, no. (%)	6 (24 %)
Aspirin, no. (%)	3 (12 %)
Anticoagulant, no. (%)	4 (16 %)
Antihypertensive use, no. (%)	7 (28 %)
Acid suppressive drugs	
PPI, no. (%)	13 (52 %)
H2RA, no. (%)	5 (20 %)
Chronic anemia, no. (%)	22 (88 %)
Iron supplementation, no. (%)	14 (56 %)
Co-Morbidities, no. (%)	
Obesity	11 (44 %)
GERD	12 (48 %)
Severe lung disease	1 (4 %)
Severe heart disease	4 (16 %)
Severe renal disease	1 (4 %)
Baseline hemoglobin (mean $\pm$ SD)	9 g/dl $\pm$ 2.1
Baseline hematocrit (mean $\pm$ SD)	27.2 $\pm$ 6.3
Platelet count (mean $\pm$ SD)	315,600/mm ³ $\pm$ 127,400

GERD: Gastroesophageal reflux disease; NSAID's: non-steroidal anti-inflammatory drugs; PPI: proton pump inhibitor; H2RA: H2-receptors antagonist

Table 2

Comparison between Patients Treated Medically and Surgically with Short and Long-term Outcomes

	Surgery Scheduled n=12	Medical Treatment n=9	p
Age (mean $\pm$ SD)	71.4 years $\pm$ 5.9	69.8 years $\pm$ 15.7	0.5938
Overt vs. Occult bleeding, no. (%)	7/5 (58.3/41.7)	5/4 (55.5/44.5)	1.0000
Prior hospitalization for bleeding episodes of unknown source, no. (%)	9 (75 %)	7 (77.8 %)	1.0000
Severe Co-Morbidities, no. (%)			
Severe lung disease	1 (8.3 %)	0	0.2237
Severe heart disease	1 (8.3 %)	3 (33.3 %)	0.5654
Severe renal disease	0	1 (11.1%)	0.4169
Any NSAIDs use, no. (%)	2 (16.6 %)	3 (33.3 %)	0.6108
Aspirin use, no. (%)	2 (16.6 %)	1 (11.1%)	1.0000
Any anticoagulant use, no. (%)	0	2 (18 %)	1.0000
Shock, syncope or hypotension, no. (%)	2	0	0.4857
Baseline hematocrit (mean $\pm$ SD)	27.2 $\pm$ 5.3	26.4 $\pm$ 8.8	0.7284
Major SRH, no. (%)	1 (8.3 %)	3 (33.3 %)	0.2722
Length of ulcer (mean $\pm$ SD)	16.7 mm $\pm$ 10.5	10.6 mm $\pm$ 7.1	0.2698
Short-term outcomes (within 30 days) n=21			
RBC transfusion units (mean $\pm$ SD)	1.5 $\pm$ 2	2.2 $\pm$ 2.0	0.4108
Hospital days (mean $\pm$ SD)	2.7 $\pm$ 4.9	1.7 $\pm$ 1.1	0.6614
Rebleeding, no. (%)	2 (18 %)	0	0.3143
Death, no. (%)	1 (9 %)	0	1.0000
Long-term outcomes (after 30 days to last follow-up) n=20			
	Surgery Scheduled n=11	Medical Treatment n=9	p
Follow-up in months (median, IQR)	12.3 (3.2–31.8)	25.9 (12.7–31.6)	0.4034
Rebleeding before surgery, no. (%)	11 (100 %)	0	p<0.0001*
Rebleeding after surgery, no. (%)	0	0	1.0000

	Surgery Scheduled n=12	Medical Treatment n=9	p
Death, no. (%)	0	0	1.0000

Hct: hematocrit; IQR: interquartile range; NSAID's: non-steroidal anti-inflammatory drugs; NS: not significant; RBC: Red blood cell; SRH: Stigmata of recent hemorrhage.

Table 3

Comparison between Patients with Overt and Occult Bleeding with Short and Long-term Outcomes

	Overt n=12	Occult n=9	p
Age (mean $\pm$ SD)	73.7 years $\pm$ 8.2	66.8 years $\pm$ 13.2	0.2270
Any NSAIDs use, no. (%)	3 (25 %)	2 (22 %)	1.000
Aspirin use, no. (%)	3 (25 %)	0	0.2286
Any anticoagulant use, no. (%)	1 (11.1 %)	1 (11.1 %)	1.000
Baseline hematocrit (mean $\pm$ SD)	25.1 $\pm$ 6.4	29.5 $\pm$ 6.7	0.0884
Prior hospitalization for bleeding episodes of unknown source, no. (%)	7 (58.3 %)	9 (100 %)	0.0451 *
SRH, no. (%)	5 (41.7 %)	0	0.0451 *
Short-term outcomes (within 30 days) n=21			
RBC transfusion units (mean $\pm$ SD)	2.6 $\pm$ 2.2	0.8 $\pm$ 1.2	0.0486 *
Hospital days (mean $\pm$ SD)	3 $\pm$ 4.9	1.2 $\pm$ 0.7	0.3572
Rebleeding, no. (%)	2 (16.7 %)	0	0.3143
Death, no. (%)	1 (8.4 %)	0	1.000
Long-term outcomes (after 30 days to last follow-up) n=20			
	Overt n=11	Occult n=9	p
Follow-up in months (median, IQR)	15.7 (6.4–34.9)	25.2 (11.9–28.1)	1.0000
Rebleeding, no. (%)	6 (54.5 %)	5 (55.6 %)	1.0000
Surgery performed	5 (45.4 %)	5 (55.6 %)	0.6699
Death, no. (%)	0	0	1.0000

Hct: hematocrit; IQR: interquartile range; NSAID's: non-steroidal anti-inflammatory drugs; NS: not significant; RBC: Red blood cell; SRH: Stigmata of recent hemorrhage.

*
p<0.05