Risk factors predicting prognosis in upper gastrointestinal bleeding

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Abstract: Background: Upper gastrointestinal bleeding is a disastrous problem worldwide and particularly in Egypt and considered the most prevalent gastrointestinal emergency. Like other common medical conditions, risk scores have been developed to try and identify those at lower or higher risk of poor outcome. Numerous prognostic factors have been described in literature to be associated with a lethal outcome; however, to date, it remains unclear whether a single or a combination of these factors is associated with poor outcome of the patient with upper GI bleeding. **Aim:** This prospective study evaluates the role of various risk factors in predicting the prognosis of patients with upper gastrointestinal bleeding. **Patients and Methods:** Data was obtained at the time of presentation in the Emergency Department. The following were noted: history: (Demographic, personal, occupational, present, past medical, habits & drug history). Complete clinical examination includes *ABCDE* priorities of advanced life support "ALS" protocol was done. The presence of hematemesis and its appearance (coffee grounds or fresh blood), melena and hematochezia were also noted. Blood pressure was measured at presentation, as well as heart rate and then close monitoring of all these data was carried out. Laboratory investigations including Hemoglobin level, Platelet count, Prothrombin time and international normalized ratio, Urea and creatinine. Patients underwent ultrasound examination Esophagogastroduodenoscopy (EGD) was performed in all patients by expert endoscopist. **Results:** Two hundred (200) patients were included in this study. One hundred thirty-one patients were males (65.5%). The age of our patients ranged from 18 to 85 years with a mean (+SD) of 58.46+12.15 years (Median age 58.5). 168 patients (84%) had associated comorbidities (e.g. chronic kidney disease, cardiac diseases, liver diseases, etc) Hypotension was present in the initial presentation of sixty seven (67) patients (33.5%) while abdominal examinations of eighty three (83) patients (41.5%) revealed ascites. The hemoglobin level of our patients ranged from 3 to 16 g/dl with a mean (+SD) of 8.54+2.20 (Median 8.35) while INR ranged from 1 to 7 with a mean (+SD) of 1.50+1.02 (Median 1.2). Endoscopic examination in one hundred nine (109) patients (54.5%) showed variceal lesions; other seventy two (73) patients (36.5%) were non-variceal while nine (9) patients (4.5%) showed both variceal and non variceal lesions. Endoscopic examination of another nine (9) patients (4.5%) was normal. Favorable outcome (Improvement) was present in one hundred and fourteen (114) patients (57%); Unfavorable outcome (Recurrence, Need for surgery & Death) was present in seventy six (76) patients (38%) while ten (10) patients (5%) were missed. **Conclusion:** Age, associated comorbidities as hepatic diseases, initial pulse, initial blood pressure, initial investigations as urea, hemoglobin & INR, ultrasonography findings and endoscoping timing can be used effectively to predict prognosis in upper gastrointestinal bleeding.

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1. Introduction:

Upper gastrointestinal bleeding is a disastrous problem worldwide and particularly in Egypt and considered the most prevalent gastrointestinal emergency. Upper GI bleeding represent about 85% of all GI bleeding and originate above ligaments of Treitz [(1,2)]

Peptic ulcer bleeding is the most common cause of Upper GI bleeding across the world, however in Egypt Upper GI bleeding due to variceal bleeding is the most common etiology and represent about 51 % of the cases followed by bleeding duodenal ulcer and represent about 15 % of the cases [(3-5)].

Upper GI bleeding presented to Emergency department by variable clinical picture like dizziness, syncope, hematemesis, melena, hematochezia and generalizes weakness [(6)].

Like other common medical conditions, risk scores have been developed to try and identify those at lower or higher risk of poor outcome. Numerous prognostic factors have been described in literature to be associated with a lethal outcome; however, to date,

it remains unclear whether a single or a combination of these factors is associated with poor outcome of the patient with upper GI bleeding [7, 8].

This prospective study evaluates the role of various risk factors in predicting the prognosis of patients with upper gastrointestinal bleeding.

2. Subjects and Methods

This prospective observational cohort study was conducted on two hundred patients with upper gastrointestinal bleeding who were admitted to Tanta University Hospital, Egypt, over one year period from January 2017 to January 2018.

Study setting and population.

All adult patients (>18 years) presented to the emergency department with *hematemesis and/or melena.* Exclusion criteria were unfit patients for endoscopy, adolescent < 18 years and patients who refused endoscopy.

Study protocol

Data was obtained at the time of presentation in the Emergency Department & at the Gastroenterology Unit. The following were noted: Demographic & personal history: (e.g. age, gender, marital & occupational history), present & past medical history, personal habits: (e.g. alcohol consumption & smoking). Relevant drug history: (e.g. NSAIDs, antiplatelet drugs and anticoagulants). Complete clinical examination includes *ABCDE priorities of advanced life support "ALS" protocols.* The presence of hematemesis and its appearance (coffee grounds or fresh blood), melena and hematochezia were also noted. The presence of clinical signs of liver cirrhosis was noted: angiomas, palmar erythema, gynecomastia, hepatomegaly, splenomegaly, edema, ascites and hepatic encephalopathy. Blood pressure was measured at presentation (hypotension was defined as BP <90/60mmHg), as well as heart rate (tachycardia was defined as heart rate >100 beats/min) and then close monitoring of all these data was carried out.

Laboratory investigations including

Laboratory investigations including Hemoglobinlevel, Platelet count, Prothrombin time and international normalized ratio, Urea and creatinine. Patients underwent ultrasound examination (performed recently or when presenting at the emergency department). The following were considered as diagnostic criteria for liver cirrhosis: abnormal hepatic contour, splenomegaly, ascites, recanalization of round ligament, pericholecystic, perigastric, or in the splenic hilum collateral circulation.

Esophagogastroduodenoscopy (EGD) was performed in all patients by expertendoscopist (The patient was placed in left lateral position after appropriate sedation and endoscopy was done. Gastrointestinal bleeding causes were divided into two categories: variceal (esophageal or gastric varices, portal hypertensive gastropathy) and non-variceal (peptic ulcer, erosive gastritis, tumors, reflux esophagitis, Mallory-Weiss syndrome, Dieulafoy's lesion and angiodysplasia)

Statistical analysis:

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) [117] Qualitative data were described using number and percent. The Kolmogorov-Smirnov test was used to verify the normality of distribution Quantitative data were described using range (minimum and maximum), mean, standard deviation and median. Significance of the obtained results was judged at the 5% level.

Outcome measures

Favorable prognosis referred to patients who were improved without any adverse outcomes. An adverse outcome was defined as death, the need for surgery, recurrent hematemesis/ melena after initial clearing or a hematocrit falling despite transfusion.

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3. Results

Two hundred (200) patients were included in this study. One hundred thirty-one patients were males (65.5%), while other sixty nine (69) patients (34.5%) were females. The age of our patients ranged from 18 to 85 years with a mean (+SD) of 58.46+12.15 years (Median age 58.5). 65% of them were smoker while 35% were nonsmokers.

Thirty two (32) of our patients (16%) had no history of significant major comorbidity while other 168 patients (84%) had associated comorbidities (e.g. chronic kidney disease, cardiac diseases, liver diseases, etc) (Table 12). 74 of our patients (37%) had no drug history and 89 patients (44.5%) had previous history of upper gastrointestinal endoscopy.

After initial examination and resuscitation, 166 patients (83%) admitted in regular wards while 34 patients (17%) needed ICU admission. 69 of patients (34.5%) received blood product transfusion.

Hypotension was present in the initial presentation of sixty seven (67) patients (33.5%) while abdominal examinations of eighty three (83) patients (41.5%) revealed ascites.

The hemoglobin level of our patients ranged from 3 to 16 g/dl with a mean (+SD) of 8.54+2.20 (Median 8.35) while INR ranged from 1 to 7 with a mean (+SD) of 1.50+1.02 (Median 1.2). Ninety-two of our patients (46%) were thrombocytopenic at their initial presentation.

Majority (145 patients) of our cases (72.5%) underwent early endoscopy while fifty five (55) of patients (37.5%) underwent later endoscopy.

Endoscopic examination in one hundred nine (109) patients (54.5%) showed variceal lesions; other seventy two (73) patients (36.5%) were non-variceal while nine (9) patients (4.5%) showed both variceal and non variceal lesions. Endoscopic examination of another nine (9) patients (4.5%) was normal.

Favorable outcome (Improvement) was present in one hundred and fourteen (114) patients (57%); Unfavorable outcome (Recurrence, Need for surgery & Death) was present in seventy six (76) patients (38%) while ten (10) patients (5%) were missed.

Table (1): Shows significant correlations between various risk factors and outcome:

	Outcome				Test of sig.	P
	Unfavorable (n= 68)		Favorable (n= 114)			
	No.	%	No.	%		
Age (years) (SIG)						
<60	28	41.2	66	57.9	X^2= 4.767*	0.029*
≥60	40	58.8	48	42.1		
Associated comorbidity						
Hepatic diseases	46	67.6	57	50.0	5.40*	0.020*
Initial pulse						
Normal (≤100)	46	67.6	112	98.2	χ^2=34.218*	MCp <0.001*
Tachycardia (>100)	19	27.9	2	1.8		
Undetected	3	4.4	0	0.0		
Initial BL/PR						
Normal	30	44.1	85	74.6	χ^2= 27.933*	<0.001*
Hypotension	37	54.4	20	17.5		
Hypertension	1	1.5	9	7.9		
Urea (mg/dl)						
Normal (≤50)	41	60.3	102	89.5	21.540*	<0.001*
Increase (>50)	27	39.7	12	10.5		
Min. – Max.	13.0 – 122.0		16.0 – 123.0		U= 2552.00	<0.001*
Mean ± SD.	53.34 ± 29.48		35.63 ± 19.36			
Median	43.0		30.50			
INR						
Min. – Max.	1.01 – 6.30		1.0 – 7.0		U= 3025.00*	0.013*
Mean ± SD.	1.49± 0.86		1.49±1.09			
Median	1.30		1.20			
Hemoglobin (g/dl)						
Min. – Max.	3.0 – 11.40		5.0 – 14.30		t= 5.876*	<0.001*
Mean ± SD.	7.48±1.89		9.25± 2.0			
Median	7.65		8.90			
Endoscopic findings					5.648*	FEp=0.017*
Normal	0	0.0	9	7.9	7.532*	0.006*
Variceal	47	69.1	55	48.2	8.780*	0.003*
Non variceal	14	20.6	48	42.1	6.608*	FEp=0.014*
Both	7	10.3	2	1.8		
Blood Transfusion						
No	34	50.0	91	79.8	17.614*	<0.001*
Yes	34	50.0	23	20.2		

Table (2): Shows insignificant correlations between various risk factors and outcome:

	Outcome				Test of sig.	P
	Unfavorable (n= 68)		Favorable (n= 114)			
	No.	%	No.	%		
Smoking						
No	50	73.5	70	61.4	2.788	0.095
Yes	18	26.5	44	38.6		
Drugs						
Non Steroid Anti-Inflammatory Drugs	13	19.1	19	16.7	0.177	0.674
Anti-coagulants	3	4.4	11	9.6	1.645	0.200
Anti-platelets	1	1.5	6	5.3	1.657	FEp=0.260
Platelets						
Thrombocytopenia (≤150.000)	35	51.5	51	44.7	χ^2= 0.775	0.379
Normal (>150.000)	33	48.5	63	55.3		
Min. – Max.	55.0 – 412.0		45.0 – 431.0		U= 3225.50	0.058
Mean ± SD.	165.46 ± 93.54		188.68 ± 94.84			
Median	142.0		165.0			
Creatinine (mg/dl)						
Normal (≤1.4)	54	79.4	100	87.7	χ^2= 2.258	0.133
Increase (>1.4\)	14	20.6	14	12.3		
Min. – Max.	0.50 – 3.80		0.50 – 4.50		U= 3541.50	0.329
Mean ± SD.	1.21 ± 0.60		1.14 ± 0.63			
Median	1.10		1.10			

4. Discussion

Several risk scoring systems had been developed based on clinical, laboratory and endoscopic parameters; however, no ideal scoring system is worldwide accepted. The factors influencing the outcome of acute upper gastrointestinal hemorrhage have been the focus of much research and debate since the 1940s. Different researchers have put a different emphasis on each of risk factors according to their experiences. [(9)]

The age of our patients ranged from 18 to 85 years with a mean (+SD) of 58.46+12.15 years (Median age 58.5). This means that it affects mainly elderly patients who are a special population prone to have more complications from any illness because of their physiological instability, comorbidities and medications giving another importance to this topic.

As regard to demographic data, age had a significant correlation with outcome of our study population while gender and smoking had no significant correlation. 58% of adverse outcomes occur in patients aged ≥60 years old.

Bae and colleagues had reported that the age-specific incidence rate of mortality increased with advanced age. Incidence rate of mortality was three times more in men than women. (10) On the other hand; Kaplan *et al* reported that current smokers had a higher risk of hospitalization for upper GIB (but not lower GIB) than nonsmokers, and that this relationship was characterized by an increasing dose-response pattern. (11)

Thirty-two of our patients had a past history of NSAIDs ingestion. Favorable outcome occurs in 19 patients while unfavorable outcome occurs in 13 patients. On the other hand; history of anticoagulants and antiplatelet ingestion presents in 14 and 7 patients respectively with adverse outcome occurs in 3 patients with past history of anticoagulant intake and only one patient with antiplatelet ingestion.

Neither NSAIDS nor anticoagulant or antiplatelet usage was linked to outcome of bleeding attack in our study.

These results are in agreement with a Retrospective cohort included 584 patients with non-variceal upper gastrointestinal bleeding, 43 % using anti thrombotic agents, the cause of death was not attributable to bleeding episode in 64.3% of cases. (12)

According to A. Lanas et al., the non variceal UGB relative risk is about 1.55 times higher with low-dose acetylsalicylic acid compared to non-use whereas when used in combination with clopidogrel or anticoagulants this increased the risk (OR = 1.86 and OR = 1.93, respectively). (13)

Lewis did not observe a positive correlation between NSAIDs and mortality from GI bleeding. The absence of correlation between mortality from GI complications and NSAIDs could also be due to the effect of a reduction in the duration of therapy. (14)

In our study, 103 patients (56.6%) had a past medical history of chronic liver diseases. 57 patients of them (55.3%) had favorable outcome while the other 46 patients (44.7%) had unfavorable outcome. Also; 67.6% of adverse outcome occurred in patients with past medical history of chronic liver diseases while 14.7% and 10.3 % of adverse outcome occurred in patients with past medical history of malignancy and cardiac diseases respectively. So, among the co-morbid conditions, only liver disease had a significant correlation with outcome of gastrointestinal bleeding episode in this study.

This is in agreement with Schemmer et al who found that Liver cirrhosis is the only risk factor which shows a significantly more frequent association with a fatal course after UGI bleeding in 121 patients. (15)

Never to deny the role of co-morbidity as a predictor of outcome had been confirmed in previous studies. (16,17) However, in others although co-morbidity was a predictor of poor outcome this association was lost on multivariate analysis. (18)

Clinical guidelines published in 2008 in Scotland cited a mortality rate of 4% in GI bleeding patients without comorbidities, with the mortality rate increasing 1.8 times in cases with heart failure, 3.8 times in cases with malignancy, and 2.0 times in cases with liver disease. (19)

According to the National Institute for Health and Clinical Excellence 2012 guidelines, patients with GI bleeding who also have chronic diseases are at a higher risk of death. (20)

Blood pressure was measured at presentation (hypotension was defined as BP <90/60mmHg), as well as heart rate (tachycardia was defined as heart rate >100 beats/min) and then close monitoring of all of these data was carried out. In this study, initial vital signs has a significant correlation with of outcome of our study population.54.4% of adverse outcome occurred in patients who were hypotensive at initial presentation.

Several studies reported that hypotension and tachycardia at time of admission for upper gastrointestinal bleeding is linked to mortality. (21)

As regard to laboratory investigations and its relation to outcome of GIT bleeding in our study population; elevated Urea, INR and low Hemoglobin percentage were only linked to unfavorable outcome of bleeding attack. Increased urea level (>50mg/dl) was present in 39.7% of patients with unfavorable outcome. The median INR level in patients with

favorable outcome was 1.2 which increased to 1.3 in patients with adverse outcome while the median hemoglobin level in patients with favorable outcome was 8.9 g/dl which decreased to 7.65 g/dl in patients with unfavorable outcome.

Uremic bleeding is a well-recognized complication in patients with renal failure, and it affects platelet aggregation and/or the coagulation cascade. (22)

In patients with chronic kidney disease, GI bleeding is also a common complication. (23) In addition, elevated BUN level in patients with GI bleeding can be due to ingested blood protein. (24) Therefore, bleeding and uremia affect the occurrence of one another.

Anand et al, showed that elevated serum creatinine levels are associated with increased rates of mortality and re-bleeding. (25) In addition, hypovolemia causes acute renal failure in patients with severe bleeding. (26)

On the other hand, a prospective study, conducted to examine the role of serum albumin upon admission in relation to clinical course and in-hospital mortality in patients with non variceal UGB with no related chronic liver disease, end stage renal disease, or neoplasia. Authors concluded that hypoalbuminemia appears to be an important surrogate marker of poor clinical condition, which subsequently suggests a poor outcome in non variceal UGB, with an overall performance for identifying mortality similar to that of the Rockall score. (27)

Twenty three patients with favorable outcome (20.2%) received packed RBCs transfusion, however; we do not set a target Hb level at discharge.

Adequate packed RBC transfusion and correction of anemia are important in some severe disease states, and are related to outcome not only during admission but also after discharge.[28-29] Low Hb is associated with morbidity and mortality in patients with coronary artery disease.[30] However, it was recently reported that excessive transfusion of packed RBCs for the purpose of correcting Hb has no merit, even in critical disease states such as septic shock. (31,32) Minimizing unnecessary transfusions lowers costs and the risk of adverse effects.

Although blood transfusions should be administered to patients with an Hb level of <7g/dL, there is no international consensus about the minimum acceptable Hb that guarantees patient safety before discharge. Moreover, there is variation in the approach to transfusion for patients with upper gastrointestinal bleeding. (34)

A recent study indicates that a transfusion strategy with a minimum acceptable discharge Hb of 8g/dL is at least as effective as a threshold of 10g/dL. Although a discharge Hb of 8g/dL seems too low, it showed no significant difference in the outcomes after discharge. (35)

Regarding to the treatment received, all included patients underwent endoscopic examination and treatment with proton pump inhibitors and while 143 patients (71.5%) underwent endoscopic treatment. Only 8 patients (4%) underwent surgery for bleeding peptic ulcer.

In a large Canadian study, endoscopic treatment and treatment with proton pump inhibitors decreased re-bleeding and mortality in high-risk patients such as old patients with severe co-morbidities and history of NSAIDS or anticoagulants use. (36)

A study by Bor et al, demonstrated that bleeding stopped in 66.9% of patients upon receipt of medical treatment, with only 3.7% of those patients undergoing surgery. (37)

In our study, 138 patients underwent Endoscopy in first 12 hours of presentation while 29 patients underwent Endoscopy in the time limit between 12 hours and 24 hours of presentation and another15 patients underwent endoscopy after 24 hours of presentation. 88.6% of patients with favorable outcome underwent early endoscopy (In the first 12 hours).

A general consensus has emerged that, in acute gastrointestinal bleeding, endoscopy should be performed within 24 h of presentation. Although the outcomes of these studies have provided a framework and resulted in guidelines for the general management of gastrointestinal bleeding, they cannot provide the answers to all specific problems that arise in clinical decision making centred on individual patients. Also, the availability of endoscopy with a team trained in treatment of bleeding is an important factor.

In a study of 210 patients with acute variceal bleeding and hemodynamic stability, performing endoscopic treatment at 4, 8, and 12 hours after arriving at the hospital did not significantly affect the mortality rate. (38) However, in another study, performing endoscopic therapy after more than 15 hours after hospital arrival significantly increased the mortality rate. (39)

Conclusion

Age, associated comorbidities as hepatic diseases, initial pulse, initial blood pressure, initial investigations as urea, hemoglobin & INR, ultrasonography findings and endoscoping timing can be used effectively to predict prognosis in upper gastrointestinal bleeding.

Limitations

Our study had some limitations, as it included both patients presented with variceal bleeding and non-variceal bleeding so comparing outcome and

mortality with other studies may be not accurate. Also, not all bleeding treatment modalities was feasible in our unit beside other factors could influence the outcome and not studied as endoscopist skills, use of vasoactive drugs or coagulants before endoscopy.

References

1. Sostres C, Lanas A. Epidemiology and demographics of upper gastrointestinal bleeding: prevalence, incidence, and mortality. Gastrointestinal Endoscopy Clinics. 2011;21(4):567-81.
2. Van Leerdam M. Epidemiology of acute upper gastrointestinal bleeding. Best practice & research Clinical gastroenterology. 2008;22(2):209-24.
3. Atkinson RJ, Hurlstone DP. Usefulness of prognostic indices in upper gastrointestinal bleeding. Best Practice & Research Clinical Gastroenterology. 2008;22(2):233-42.
4. Shalaby SI, Abo-Eldahab M, Abd-Elhafeez H, Awad MAK, Attia M, Gupta N. Clinical and clinico-pathological impacts of upper gastrointestinal bleeding in elderly Egyptian patients. IOSR J Pharm Biol Sci. 2016;11:25-30.
5. Zakaria S, Fouad R, Shaker O, El Akel W, Hashem A, Esmat G. Natural history of the untreated symptomatic acute hepatitis C infection in Egypt: P-185. Liver International. 2006;26:73.
6. Laine L, Shah A. Randomized trial of urgent vs. elective colonoscopy in patients hospitalized with lower GI bleeding. The American journal of gastroenterology. 2010;105(12):2636.
7. Dworzynski K, Pollit V, Kelsey A, Higgins B, Palmer K. Management of acute upper gastrointestinal bleeding: summary of NICE guidance. BMJ: British Medical Journal (Online). 2012;344.
8. Blatchford O, Davidson LA, Murray WR, Blatchford M, Pell J. Acute upper gastrointestinal haemorrhage in west of Scotland: case ascertainment study. BMj. 1997;315(7107):510-4.
9. Katschinski B, Logan R, Davies J, Faulkner G, Pearson J, Langman M. Prognostic factors in upper gastrointestinal bleeding. Digestive diseases and sciences. 1994;39(4):706-12.
10. Bae S, Kim N, Kang JM, Kim D-S, Kim K-M, Cho YK, et al. Incidence and 30-day mortality of peptic ulcer bleeding in Korea. European journal of gastroenterology & hepatology. 2012;24(6):675-82.
11. Kaplan RC, Heckbert SR, Koepsell TD, Furberg CD, Polak JF, Schoen RE, et al. Risk factors for hospitalized gastrointestinal bleeding among older persons. Journal of the American Geriatrics Society. 2001;49(2):126-33.
12. Teles-Sampaio E, Maia L, Salgueiro P, Marcos-Pinto R, et al. Antiplatelet agents and/or anticoagulants are not associated with worse outcome following nonvariceal upper gastrointestinal bleeding. Rev Esp Enferm Dig 2016;108(11):703-708.
13. Lanas A, Wu P, Medin J, Mills EJ. Low doses of acetylsalicylic acid increase risk of gastrointestinal bleeding in a meta-analysis. Clinical Gastroenterology and Hepatology 2011;9(9):762-768.e6.
14. Lewis JD, Bilker WB, Brensinger C, et al. Hospitalization and mortality rates from peptic ulcer disease and GI bleeding in the 1990s: relationship to sales of nonsteroidal anti-inflammatory drugs and acid suppression medications. Am J Gastroenterol. 2002;97:2540–2549.
15. Schemmer P, Decker F, Dei-Anane G, et al. The vital threat of an upper gastrointestinal bleeding: Risk factor analysis of 121 consecutive patients. World J Gastroenterol 2006; 12(22): 3597-3601.
16. Silverstein FC, Gilbert DA, Tedesco FJ and members of the ASGE survey on upper gastrointestinal bleeding II: Clinical prognostic factors. Gastrointest Endosc981; 27(2): 80-93.
17. Kollef MH, O'Brien JD, Zuckerman GR, et al. BLEED: A classification tool to predict outcomes in patients with acute upper and lower gastrointestinal haemorrhage. Crit Care Med1997; 25: 1125-1132.
18. S Z Kalula, G H Swingler, J A Louw. Clinical predictors of outcome in acute upper gastrointestinal bleeding SAM Journal April 2003, Vol. 93, No. 4.
19. Scottish Intercollegiate Guidelines Network Elliott House, Edinburgh, Management of acute upper and lower gastrointestinal bleeding A national clinical guideline. September, 2008.
20. National Clinical Guideline Centre. London, Acute upper gastrointestinal bleeding Management Clinical Guideline Methods, evidence and recommendations. June 2012.
21. Cameron EA, Pratap JN, Sims TJ, et al. Three-year prospective validation of a pre-endoscopic risk stratification in patients with acute upper-gastrointestinal haemorrhage. Eur J Gastroenterol Hepatol,2002, 14(5):497 501.
22. Kaya E, Karaca MA, Aldemir D, et al. Predictors of poor outcome in gastrointestinal bleeding in emergency department. World J Gastroenterol 2016; 22(16): 4219-4225.
23. Hedges SJ, Dehoney SB, Hooper JS, et al. Evidence-based treatment recommendations for

uremic bleeding. Nat Clin Pract Nephrol 2007; 3: 138-153.
24. Kalman RS, Pedrosa MC. Evidence-based review of gastrointestinal bleeding in the chronic kidney disease patient. Semin Dial 2015; 28: 68-74.
25. Bang CS, Lee YS, Lee YH, et al. Characteristics of nonvariceal upper gastrointestinal hemorrhage in patients with chronic kidney disease. World J Gastroenterol 2013; 19: 7719-7725.
26. Anand D, Gupta R, Dhar M, et al. Clinical and endoscopic profile of patients with upper gastrointestinal bleeding at tertiarycare center of North India. J Dig Endosc 2014; 5: 139-143.
27. Lameire N. The pathophysiology of acute renal failure. Crit Care Clin 2005; 21: 197-210.
28. González-González JA, Vázquez-Elizondo G, Monreal-Robles R, et al. Hipoalbuminemia en el desenlaceclínicodepacientes con sangrado de tubodigestivo alto no variceal. Revista de Gastroenterología de México. 2016;81:183- 189.
29. Ferraris VA, Brown JR et al. Society of Thoracic Surgeons Blood Conservation Guideline Task Force; 2011 update to the Society of Thoracic Surgeons and the Society of Cardiovascular Anesthesiologists blood conservation clinical practice guidelines. Ann Thorac Surg 2, 2011; 91: 944–982.
30. Amsterdam EA, Wenger NK, Brindis RG et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014; 130: 2354–2394.
31. Diedler J, Sykora M, Hahn P et al. Low hemoglobin is associated with poor functional outcome after non-traumatic, supratentorial intracerebral hemorrhage. Crit Care 2010; 14: 63.
32. Sabatine MS, Morrow DA, Giugliano RP et al. Association of hemoglobin levels with clinical outcomes in acute coronary syndromes. 2005; 111: 2042–2049.
33. Holst LB, Haase N, Wetterslev J et al. Lower versus higher hemoglobin threshold for transfusion in septic shock. N Engl J Med 2014; 371: 1381–1391.
34. Carson JL, Sieber F, Cook DR et al. Liberal versus restrictive blood transfusion strategy: 3-year survival and cause of death results from the FOCUS randomised controlled trial. Lancet 2015; 385: 1183–1189.
35. Jairath V, Kahan BC, Logan RF et al. Red blood cell transfusion practice in patients presenting with acute upper gastrointestinal bleeding: a survey of 815 UK clinicians. Transfusion 2011; 51: 1940–1948.
36. Lee Jae Min et al. Discharge hemoglobin in patients with acute nonvariceal upper gastrointestinal bleeding… Endoscopy International Open 2016; 04: E865–E869.
37. Coskun F, Topeli A, Sivri B. Patients admitted to the emergency room with upper gastrointestinal bleeding: Factors influencing recurrence or death. Adv Ther 2005;22:453- 461.
38. Bor S, Dağli U, Sarer B, et al. A retrospective study demonstrating properties of nonvariceal upper gastrointestinal bleeding in Turkey. Turk J Gastroenterol. 2011;22:249–254.
39. Hsu YC, Chen CC, Wang HP. Endoscopy timing in acute variceal hemorrhage: perhaps not the sooner the better, but delay not justified. Am J Gastroenterol. 2009;104:2629–2630.
40. De Franchis R. Evolving consensus in portal hypertension. Report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2005;43:167–176.

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