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TOPIC HIGHLIGHT

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Trends on gastrointestinal bleeding and mortality: Where are we standing?

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Abstract

Bleeding from the gastrointestinal tract and its management are associated with significant morbidity and mortality. The predisposing factors that led to the occurrence of these hemorrhagic instances are largely linked to the life style of the affected persons. Designing a new strategy aimed at educating the publics and improving their awareness of the problem could effectively help in eradicating this problem with no associated risks and in bringing the mortality rates down to almost zero.

Key words: Gastrointestinal bleeding; Peptic ulcer; Esophageal varices; Helminthic infestation; Bowel cancer; Mortality; Morbidity; Predicting factors; Age; Sex

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INTRODUCTION

Gastrointestinal (GI) bleeding involves any bleeding in the GI tract from the mouth, oesophagus, stomach, small intestines, large intestines, to the anus. The degree of bleeding can range from microscopic levels detected only by lab tests, to perceptible amounts of bleeding that can be seen in the stool or vomit. However, any level of bleeding can lead to serious problems. Microscopic levels of bleeding can lead to anaemia over time, and more massive amounts of bleeding can lead to death.

How to manage these instances more effectively and to prevent the happening of the co-incident adverse would be discussed by colleagues whom I invited to carry out this task. But in this review, I am going to assess how practically these haemorrhagic instances could be avoided.

TRENDS ON GASTROINTESTINAL BLEEDING AND MORTALITY

Upper GI bleeding involves bleeding from the mouth to the duodenum (common causes of upper GI bleeding are listed in Table 1). But lower GI bleeding involves bleeding from the small intestines to the anus and can be caused by haemorrhoids, cancer, polyps and colitis, among other causes (Table 2). Upper GI bleeding has been estimated to account for up to 20 000 deaths annually in the United States (international records are not available). The overall incidence of acute upper GI haemorrhage has been estimated to be 50 to 100 per 100 000 persons per year. The trends of hospitalization for GI bleeding in the United States in 1998 and in 2006 have

Table 1 Causes of acute upper gastrointestinal bleeding

Common	Gastric ulcer
	Duodenal ulcer
	Esophageal varices
	Malory-Weiss tear
Less common	Gastric erosive/gastropathy
	Esophagitis
	Cameron lesions
	Dieulatory lesion
	Telangiectasias
	Portal hypertensive gastropathy
	Gastric antral vascular ectasia (watermelon stomach)
	Gastric varices
	Neoplasms
Rare	Esophageal ulcer
	Erosive duodenitis
	Aortoenteric fistula
	Hemobilia
	Pancreatic disease
	Crohn's disease

Table 2 Causes of acute lower gastrointestinal bleeding

Common	Colonic diverticula
	Angioectasia
Less common	Colonic neoplasms (including post polypectomy bleeding)
	Inflammatory bowel disease
	Colitis
	Ischemic
	Raditation
	Unspecified (infectious or non specific)
	Haemorrhoids
	Small bowel source
	Upper gastrointestinal source
Rare	Dieulatory lesion
	Colonic ulcerations
	Rectal varices

been summarised in Table 3.

And the hospitals' discharge rates of the admitted subjects for different causes of GI haemorrhage in 1998 and 2006 in the United States have also been listed (Table 4).

The incidence rate for upper GI bleeding appears to be, in general, decreasing (Table 3). This may be due to the prescription of proton pump inhibitors and the skilled efforts to eradicate *Helicobacter pylori* infections (Table 4). But, the risk of upper GI bleeding appears to be increasing in particular groups of patients, such as those with a history of oesophageal varices (Table 5).

Regarding bleeding from the lower GI tract, it appears that haemorrhage from rectum and anus and the incidences of diagnosis of occult blood in stool are increasing (Table 5).

When the total number of discharges for cases of GI bleeding was investigated per age of the discharged patient, it appeared that incidences of GI bleeding are increasing in certain subgroups. The incidences of GI haemorrhage was, for example, found increasing in those who were less than 20 years old (Table 3).

Oesophageal varices form less than 10% of the all causes of GI haemorrhages. However, patients with variceal haemorrhage have a mortality rate of at least 30% during their initial hospitalization, with a one year mortality rate approaches 60%[1]. Patients who have bled once from oesophageal varices have a 70% chance of rebreeding, and approximately one third of further bleeding episodes are fatal[2]. The risk of death is maximal during the first few days after the bleeding episode and decreases slowly over the first 6 wk. Oesophageal varices are present in approximately 40% of patients with cirrhosis and in as many as 60% of patients with cirrhosis and ascites[3]. In cirrhotic patients who do not have oesophageal varices at initial endoscopy, new varices will develop at a rate of approximately 5% per year. In patients with small varices at initial endoscopy, progression to large varices occurs at a rate of 10%-15% per year and is related chiefly to the degree of liver dysfunction[4]. On the other hand, improvement in liver function in patients with alcoholic liver disease who abstain from alcohol is associated with a decreased risk, and sometimes even disappearance of the varices[5]. It has been estimated that up to 25% of the patients with newly diagnosed varices would bleed within two years[4]. The risk of bleeding in patients with varices less than 5 mm in diameter is 7% by two years, and in patients with varices greater than 5 mm in diameter is 30% by two years[4]. Mortality rates in the setting of surgical intervention for acute variceal bleeding are high[6]. Associated abnormalities in the renal[7], pulmonary[7], cardiovascular[8], and immune systems in patients with oesophageal varices contribute to 20%-65% of mortality[9]. In Western countries, alcoholic and viral cirrhosis are the leading causes of portal hypertension and oesophageal varices. Thirty percent of patients with compensated cirrhosis and 60%-70% of patients with decompensate cirrhosis have gastroesophageal varices at presentation[9]. The *de novo* rate of development of oesophageal varices in patients with chronic liver diseases is approximately 8% per year for the first 2 years and 30% by the sixth year[9]. A recently published survey[10] on consumption of alcohol by teenagers in the North West of England revealed that almost 90% of the participant school children (aged 15 and 16) drink alcohol at least occasionally. Of those, 38.0% usually binge drink (5+ drinks in one session), 24.4% are frequent drinkers (drinking two or more times a week) and 49.8% drinks in public settings (such as bars, clubs, streets and parks). It is worth to note that excessive drinking by young people, for example, has seen a 20% rise in hospital admissions in England over the last five years. The number of people taken to Accident and Emergency with alcohol-related injuries has also doubled to 148 477 a year. Alcohol-related conditions such as liver disease have doubled in less than a decade, to 262 844 a year as well.

But in developing countries, hepatitis B is endemic in the Far East and Southeast Asia, particularly, as well as South America, North Africa, Egypt and other countries in the Middle East. Schistosomiasis is an important cause of portal hypertension in Egypt, Sudan and other African countries[8]. Those that have been affected with bilharsiasis, they almost have additional complications from hepa-

Table 3 Trends of hospitalization for gastrointestinal bleeding in the United States in 1996 and 2006

	Total number of discharges per 100 000 persons (principal diagnosis)			Total number of discharges per 100 000 persons (all diagnosis)		
	1998	2006	Percent changes (%)	1998	2006	Percent changes (%)
	189	182	-3.8	390	375	-3.7
By bleeding site						
Upper	96	82	-14	170	146	-14
Lower	43	44	+2	75	82	+8
Unspecified	50	56	+11	156	158	+1
By age (yr)						
< 20	7.4	7.5	+1.5	23	25	+8.6
20-29	25	23	-7	55	59	+6.1
30-44	65	59	-8.3	139	140	+0.6
45-64	187	181	-3.4	399	396	-0.9
65-84	859	806	-5.6	1731	1596	-7.8
> 85	2207	1871	-15.2	4257	4375	-18.4
By sex (%)						
Female	259 808 (51)	276 663 (51)				
Male	252 060 (49)	268 589 (49)				

Table 4 Death rates for gastrointestinal bleeding inpatients

	1998	2006	Percentage change (%)
Inpatient death number	20 013	16 344	-18
Inpatient death number/100 000	7	5	-26
Inpatient death rate (%)			
By bleeding site			
Upper	3.5	2.7	-23
Lower	3.5	2.9	-17
Unspecified	5	3.6	-28
By sex			
Male	4	3	-25
Female	3.8	3	-21
By age (yr)			
< 20	-	-	-
20-29	-	-	-
30-44	1.6	1.1	-31
45-64	2.7	2.2	-19
65-84	4.1	3	-27
> 85	6.4	5.2	-19

titis B and C[11-14]. The instances of acute lower GI bleeding are mainly self-limited and affected patients do not require hospitalization care, approximately 21 per 100 000 adults in the United States require hospitalization for severe lower GI bleeding every year[15] (international records are not available). The hospitalization rate for lower GI bleeding is approximately one third of that for upper GI bleeding[16] and in a survey by the American College of Gastroenterology, lower GI haemorrhage accounted for 24% of all GI bleeding occasions[17]. It has been estimated that detection of occult blood in stools formed 7% of all instances of GI bleeding in the United States in 2007. But it is expected that the incidence rates of detection of occult blood in the stools of patients in developing countries exceed this figure by many times. Helminthic infestation is a common cause for occult blood in stools in developing countries (Table 6). It has been estimated that 80% of the population of most countries in Asia, Africa and south America are infected with helminths, such as Ascaris and widespread infection has been demonstrated throughout Europe, Particularly, Romania, Hungry, Portugal and Turkey[18]. When 312 children in the age group of 4-15 years were examined for different intestinal helminths in three schools located in rural areas in Kupwara, Kashmir, India[19], 222 of 312 (71.15%) tested positive for various intestinal helminths[20]. The various helminth parasites included Ascaris lumbricoides, Trichuris trichiura, Enterobius vermicularis and Taenia saginata. The highest frequency of 69.23% (216/312) was noted for Ascaris lumbricoides followed by Trichuris trichiura 30.76% (96/312), Enterobius vermicularis 7.69% (24/312) and Taenia saginata 7.69% (24/312). Single infection was found in 33.65% (105/312) and mixed infection was seen in 37.5% (117/312) children. Again, Chandrasekhar MR and others in 2003[21] collected faecal samples from 1000 children below 6 years of age. Six hundred and eighty children (68.0%) were detected to have intestinal helminthic infection. The incidence of intestinal helminthiasis in urban group of children was 56.8% (284 out of 500 tested) while in rural group of children was 79.2% (396 out of 500 tested) both in rural and urban population Ascaris lumbricoides was the single predominant species, whereas a combination of A. Lumbricoides and Trichuris trichiura was common multiple infection. All cultures of faecal samples were positive for hook worm ova. In Pakistan, out of 200 children examined, 132 (66%) were found positive for various intestinal helminths infestation[22]. There were 6 different types of helminths found in the specimens examined.

It has become visible from the above review that the main causes for the occurrence of haemorrhage from the GI tract are strongly linked to the life style of the affected persons. Educating the public is thus expected to solve this problem. However, when the effect of health education in the control of bilharzias is assessed the results were disappointing. In 2001, Garba *et al*[23] carried out a survey on two groups of endemic villages in the Niger. In one group of villages, there were health educa-

Table 5 Underlying conditions of gastrointestinal in 1998 and 2006 *n* (%)

Underlying condition	1998	2006	Discharge percentage change (principal diagnosis)	1998	2006	Discharge percentage change (all diagnosis)
Upper GI: oesophageal varices, ulcer, perforation and other haemorrhages	23 007 (4)	35 058 (6)	52% and 38% after population adjustment	84 382 (8)	103 381 (9)	23% and 11% after population adjustment
Gastric, duodenal ulcers, gastrojejinal uklcers or perforation	156 29 (31)	131 225 (24)	-16% and -24% after population adjustment	215 912 (20)	179 032 (16)	-17% and -25%after population adjustment
Gastritis or duiodenitis and haemorrhage	54 310 (11)	44 104 (8)	-19% and -27% after population adjustment	118 333 (11)	90 635 (8)	-23% and -31%after population adjustment
Angiodysplasia of stomach and dudodenum with haemorrhage	9237 (2)	14 679 (3)	59% and 43% after population adjustment	15 061 (1)	23 032 (2)	53% and 38% after population adjustment
Haematemesis	16 466 (3)	21 230 (4)	29% and 16% after population adjustment	58 955 (6)	72 655 (6)	23% and 11% after population adjustment
Perforation of the large intestine	9117 (2)	10 066 (2)	10% and -0.3% after population adjustment	26 200 (2)	33 246 (3)	27% and 15% after population adjustment
Haemorrhage of rectum and anus	12 084 (2)	21 456 (4)	78% and 60% after population adjustment	52 974 (5)	85 592 (7)	56% and 41% after population adjustment
Diverticulosis and diverticulitis of the colon and haemorrhage	80 007 (16)	83 927 (15)	5% and -5% after population adjustment	101 000 (10)	104 516 (9)	3% and -7% after population adjustment
Diverticulosis and diverticulitis of the small intestine and haemorrhage	15 369 (3)	16 259 (3)	6% and -5% after population adjustment	26 933 (3)	27 433 (2)	2% and -8% after population adjustment
Unspecified GI bleeding (blood in stool)	31 044 (6)	38 284 (7)	23% and 11% after population adjustment	283 440 (27)	325 035 (29)	15% and 4% after population adjustment
Haemorrhage of GI tract (unspecified)	104 991 (21)	129 164 (24)	23% and 11% after population adjustment	283 440 (27)	325 035 (29)	15% and 4% after population adjustment

GI: Gastrointestinal.

Table 6 Causes of occult gastrointestinal bleeding

Mass lesions	Carcinoma (any site)
	Large > 1.5 cm adenoma (any site)
Inflammatory lesions	Erosive oesophagitis ulcer (any site)
	Cameron lesion
	Erosive gastropathy
	Celiac sprue
	Ulcerative colitis
	Crohn's disease
	Non specific colitis
	Caecal ulcer
Vascular lesions	Angiodysplasia (any site)
	Portal hypertensive gastropathy (colonpathy)
	Gastric antral vascular ectasia
	Hemangioma
	Dieulatory lesion
Infection	Hockworm
	Whipworm
	Stronyloidosis
	Ascariosis
	Tuberculous enterocolitis
	Amoebiasis
	Cytomegalo virus

tion campaigns but there were no education campaigns in the second group. The people in the targeted areas received information on Bilharziasis and on how to fight against it. However, 46.6% of interviewed people in the project area couldn't mention any means for controlling bilharziasis. Behaviours that favour the illness were ignored by 1/3 of interrogated people in the project area. Yet, there was an increase in knowledge about the illness in the program zone in comparison with the control area. Despite this increase in knowledge level, changes in behaviour in relation to the illness remained low. Risky behaviour continued in about 2/3 of interrogated people. Only 33% of persons of the project area declared having adopted at least a single good behaviour. This means that changes of behaviour may take time to have effect.

In addition, areas endemic for helminthic infestation worldwide suffer from poor economic growth, poor sanitation and lack of appropriate toilet facilities.

Multiple studies demonstrated that in endemic areas re-infection is exceedingly common and mass chemotherapy alone is insufficient to prevent the spread of these diseases[24].

Relatively little attention has been focused on the impact of personal attitude on the development of haemorrhagic episodes from the GI tract. This review emphasizes on the importance of designing a new strategy aimed at preventing the happening of these episodes.

The occurrence of GI bleeding and its management are associated with significant harm; however, educating the publics through properly designed long term program and improving the general surrounding conditions are free from risk.

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