Acute gastroenterology

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Acute gastroenterological problems are encountered frequently on the medical assessment unit. This review focuses on the management of alcoholism, acute diarrhoea and upper gastrointestinal (GI) bleeding.

The alcoholic patient

Alcohol-related admissions are increasing in the UK. Alcohol causes death from accidents, violence and poisoning, while long-term use increases the incidence of cirrhosis and several cancers. One-quarter of the UK adult population drink alcohol at levels which may cause harm and 2.9 million people show evidence of alcohol dependence. The annual cost to the NHS is estimated at £1.4–1.7 billion.[1] Mortality from alcoholic cirrhosis has increased 10-fold in the last 35 years. Acute physicians play a major role in the care of patients with alcoholism by providing screening, assessment, education and early specialist referral.

Identifying the problem

Symptoms that raise the possibility of alcohol abuse are outlined in Table 1. Co-existing depression, cigarette smoking and other drug abuse are all common. Four steps should be used to identify alcohol-related problems:

- detailed personal and family history of alcohol abuse
- quantity, type and frequency of alcohol abuse
- screening questionnaires, and
- search for complications of alcohol abuse.

Screening questionnaires are more effective than laboratory tests for detecting problem drinking in unselected populations. The best known is the CAGE questionnaire (Table 2). Abnormal examination findings include withdrawal (eg tremor, tachycardia), signs of chronic liver disease, peripheral neuropathy, malnourishment and physical injuries. Signs of alcoholic hepatitis (fever, hepatomegaly, ascites, jaundice, anorexia) indicate potentially high mortality.

Managing acute alcohol withdrawal

Prompt identification of patients at risk is important. Benzodiazepines are the most effective pharmacological management of the symptoms of withdrawal which range from mild tremor to seizures, delirium tremens and death. In a randomised controlled trial (RCT) symptom-triggered administration of chlordiazepoxide significantly reduced the mean duration of treatment (9 h *v* 68 h) and dose (100 mg *v* 425 mg) of chlordiazepoxide compared with fixed dose regime, without increasing complications or withdrawal symptoms.[2] Patients require experienced nursing with close monitoring; those with alcoholic hepatitis need supportive therapy, often in an intensive care unit.

Strategies for maintaining abstinence

Counselling. Many alcoholic patients fail to acknowledge a link between alcohol and their presenting problem. This can be addressed by screening and counselling. Even brief counselling interventions can be effective. A randomised study involving over 900 patients across 47 UK general practices demonstrated that simple advice was associated with a significant reduction in the number of drinks consumed per week by both men and women compared with no advice.[3]

Pharmacotherapy. Alcohol interacts with numerous neurotransmitter systems in the brain. Drugs can also be used to maintain abstinence and reduce relapse in patients with alcohol dependence, but they should serve as adjuncts to psychosocial therapies. Several treatments have

Table 1. Comorbid conditions and symptoms associated with excess alcohol use.

Comorbid condition	Symptoms
Gastrointestinal	Dyspepsia, peptic ulcer disease, gastritis, oesophagitis, varices, alcoholic liver disease, pancreatitis
Cardiovascular	Hypertension (especially resistant), cardiomyopathy
Respiratory	Tuberculosis
Neurological	Withdrawal seizures, delirium tremens
Psychiatric	Insomnia, depression, anxiety, hallucinosis, affective disorders
Withdrawal symptoms	Tremor, anxiety, headache, palpitations, seizures
Malignancy	Increased risk of oral, pharyngeal, laryngeal, oesophageal, liver and breast cancer
Social/work problems	Missed work due to hangovers
Other	Decreased libido, impotence, injuries

Table 2. The CAGE questionnaire.

One positive response: need for closer evaluation suggested

Two positive responses: 60–95% sensitivity for identifying lifetime alcohol problems and 40–95% specificity from a wide range of studies

- Have you ever felt you should cut down on your drinking?
- Have people annoyed you by criticising your drinking?
- Have you ever felt guilty about your drinking?
- Have you ever had an alcoholic drink on awakening (eye-opener)?

CAGE = **C**utdown, **A**nnoyed by criticism, **G**uilty about drinking, **E**ye-opener drinkers.

been developed aimed at maintaining abstinence including:

- disulfiram (acetaldehyde dehydrogenase inhibitor)
- acamprosate (decreases glutamergic neurotransmitters)
- naltrexone (opioid antagonist).

All the above drugs reduce heavy drinking and increase abstinence rates in alcohol-dependent patients.[4]

Acute diarrhoeal illnesses

Diarrhoea is a common cause of presentation to the medical assessment unit. Causes include infective gastroenteritis, inflammatory bowel disease (IBD), ischaemic colitis, drug-induced (antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs)) and overflow diarrhoea secondary to faecal impaction.

Infection

Infection is the commonest cause of acute gastroenteritis (Table 3). One in five people are afflicted each year, accounting for about 60,000 admissions to English NHS hospitals in 2005–06.[5] Acute gastroenteritis is usually a mild, self-limiting illness. Dehydration can occur in developed countries, requiring hospital admission. Death is uncommon, with the elderly and babies at greatest risk.

Enterohaemorrhagic *Escherichia coli* O157:H7 has been responsible for serious outbreaks of gastroenteritis in the UK, with high mortality. It can cause haemorrhagic colitis, haemolytic uraemic syndrome and thrombotic thrombocytopenic purpura. Rotavirus is the commonest cause of acute diarrhoea in infants, causing about one million deaths worldwide per year. Norwalk virus is spread by the faeco-oral route as well as direct person-to-person contact and is typically associated with sporadic outbreaks affecting large groups (eg cruise ships or hospitals). Antibacterial-associated diarrhoea has a high morbidity and places heavy demands on healthcare resources. *Clostridium difficile* is the most common pathogen, causing a spectrum of disease from mild self-limiting diarrhoea to a severe pseudomembranous colitis.

Inflammatory bowel disease

IBD includes ulcerative colitis (UC) and Crohn's disease. New cases of IBD, particularly UC, may present with bloody diarrhoea.

Ulcerative colitis. 30% of patients with UC will relapse each year and some will have severe colitis requiring hospital admission. The incidence is about 14 per 100,000 per year.

Crohn's disease. 25% of patients with Crohn's disease have colitis predominantly which clinically behaves similarly to UC. The incidence is about eight new cases per 100,000 per year.

Ischaemic colitis

Ischaemic colitis usually presents as sudden onset abdominal pain followed by bloody diarrhoea or rectal bleeding. It is a segmental colitis limited to the splenic flexure or rectosigmoid area where there is watershed between intestinal blood flow derived from the superior and inferior mesenteric arteries. Patients are usually elderly with high cardiovascular risk and vascular disease. If only mucosal blood flow is compromised, a segmental mucosal inflammation develops which may be self limiting and can be managed expectantly. Otherwise, full thickness bowel ischaemia may ensue requiring urgent laparotomy and resection.

Approach to the patient with acute diarrhoea

Patients typically present with a combination of diarrhoea, vomiting, abdominal pain and sometimes fever. There are often pointers to diagnosis within these presenting syndromes (Table 4). Acute

Table 3. Infective causes of acute gastroenteritis.

Bacteria	Shigella	Traveller's diarrhoea, food poisoning
	Salmonella	
	Campylobacter jejuni	
	Staphylococcus aureus	Toxin-related food poisoning
	Bacillus cereus	
	Clostridium botulinum	
	Clostridium difficile	Antibiotic-associated diarrhoea, pseudomembranous colitis
	Enterohaemorrhagic *Escherichia coli* O157:H7	Haemorrhagic colitis, haemolytic uraemic syndrome, thrombotic thrombocytopenic purpura
Viruses	Rotavirus	Infants and young children
	Norwalk virus	Sporadic outbreaks
	Enteric adenoviruses	
	Astrovirus	
	Caliciviruses	
Protozoa	Entamoeba histolytica	Occasionally fulminant colitis
	Giardia intestinalis	Usually chronic diarrhoea
	Cryptosporidium parvum	
	Cyclospora cayetanensis	

Table 4. Presenting syndromes in patients with acute diarrhoea.

Symptom	Suggested cause
Bloody diarrhoea	Colonic inflammation (colitis; see below)
Non-bloody diarrhoea	Viral infections, *Escherichia coli*, toxin-related diarrhoea
Vomiting	More common in viral gastroenteritis and food poisoning
Abdominal pain/cramps	Common in all causes
Fever	Suggestive of infective cause or severe colitis
Very sudden onset	Toxin-related food poisoning

bloody diarrhoea indicates an inflammatory condition of the colon (acute infective colitis, IBD). It can occur on a background of known IBD (eg ulcerative colitis or *de novo* IBD). Any acute colitis has potential to progress to fulminant colitis, toxic megacolon and perforation, therefore all patients need to be monitored closely for signs of toxicity and deterioration.

Management

The essential principles of management of acute diarrhoea are:

- resuscitation
- rehydration
- supportive care (including prophylactic anticoagulation with a low molecular weight heparin).

Patients should be isolated in case they are infectious. Useful investigations in the first 24 hours include stool culture and abdominal X-ray to rule out toxic dilatation. Further diagnostic tests include flexible sigmoidoscopy or colonoscopy to obtain mucosal biopsies, and computed tomography to rule out an intra-abdominal collection.

Specific treatment. Antibiotics may be used in probable infective diarrhoea when symptoms are severe or there are signs of bacteraemia. Quinolones are recommended for traveller's diarrhoea. Ciprofloxacin is our preferred empiric treatment for infective diarrhoea.

Treatment for acute inflammatory bowel disease. 70% of patients with acute UC respond to high-dose intravenous (iv) corticosteroids (hydrocortisone 100 mg 6 hourly) within 5–7 days. Other rescue treatments (which should be prescribed only by IBD specialists) include ciclosporin and infliximab.[6] Urgent subtotal colectomy and ileostomy, with a view to interval reconstructive surgery, will be needed by 20–30% of patients with severe colitis. Early referral to a gastroenterologist is mandatory.

Antidiarrhoeal drugs (loperamide, opioids) and smooth muscle relaxants (buscopan) may provoke acute colonic dilatation and should be avoided. NSAIDs can worsen colonic inflammation.

Key Points

The alcoholic patient

- **Identificaton of the problem**
- **Use of a suitable screening questionnaire (eg CAGE)**
- **Be vigilant for systemic symptoms and comorbidities that may be attributable to alcohol misuse**
- **Manage acute withdrawal in the appropriate environment**
- **Consider strategies for maintaining abstinence in willing patients, including counselling and/or pharmacotherapy**

Acute diarrhoea

- **Bloody diarrhoea indicates colonic inflammation which can progress to fulminant colitis whatever the underlying cause**
- **The elderly and babies are at greatest risk**
- **Supportive treatment and fluid resuscitation are more important than specific treatments for infection or inflammatory bowel disease**
- **Patients should have prophylactic low molecular weight heparin (even if bleeding)**

Acute upper gastrointestinal bleed

- **Risk can be stratified using the Rockall score**
- **Fluid resuscitation is more important than endoscopy**
- **Endoscopy should be undertaken when the patient is stable**

KEY WORDS: alcoholism, diarrhoea, gastroenteritis, upper gastrointestinal bleeding

Upper gastrointestinal haemorrhage

Upper GI bleeding is the commonest gastroenterological emergency and carries high morbidity and cost. The annual UK incidence is about 50–150 per 100,000 adults.[7] Mortality has not changed significantly in the last 50 years (10–14%). It is highest in the elderly and in patients with significant comorbidity. Causes are shown in Table 5.

Approach to the patient with an upper gastrointestinal bleed

Upper GI bleeding usually presents with haematemesis and/or melaena. Several facts must be elicited in the history, including use of NSAIDs or aspirin, history of ulcers, alcohol intake, known liver disease or varices, weight loss, dysphagia and known abdominal aortic aneurysm.

On examination, an abdominal mass may suggest an intra-abdominal malignancy. Signs of chronic liver disease may indicate variceal haemorrhage. Urgent investigations include full blood count, coagulation screen, urea and electrolytes, and urgent blood cross-match.

Management

Resuscitation. Adequate fluid resuscitation takes priority over endoscopy. Two large-gauge iv cannulae should be sited. Patients with signs of haemodynamic instability or active bleeding should be monitored in a high dependency

Table 5. Causes of upper gastrointestinal haemorrhage.

Cause	%
Peptic ulcer	40–50
Gastroduodenal erosions	10
Oesophagitis	5–10
Mallory-Weiss tear	10
Varices	5–10
Vascular malformations	5
Malignancy	4–5
Unidentified	20

Table 6. Rockall score proforma. Reproduced with permission from the BMJ Publishing Group.[8]

Initial Rockall score pre-endoscopy: total maximum 7

Full Rockall score postendoscopy: maximum 11:

- score 2 predicts 0.1% mortality
- score 8 associated with 41% mortality
- initial score of 0 and final <2: potentially consider for early discharge

Points	0	1	2	3	Score
1 **Age**	<60	60–80	>80		
2 **Shock**	None	P >100	SBP <100		
3 **Comorbidity**	None		Cardiac failure IHD Major morbidity	Renal failure Liver disease Malignancy	
4 **Endoscopic diagnosis**	M-W tear or no lesion and no sign of bleeding	All other diagnoses	Malignancy of upper GI tract		
5 **Major stigmata of recent haemorrhage**	None or dark spot only		Visible blood, adherent clot, visible or spurting vessel		
Rockall score					

GI = gastrointestinal; IHD = ischaemic heart disease; M-W = Mallory-Weiss; P = pulse rate; SBP = systolic blood pressure.

environment. Blood transfusion should be given to maintain haemoglobin above 10 g/dl. Patients with bleeding and coagulopathy (international normalised ratio >1.5) or thrombocytopenia (<50 × 10^9/l) should be transfused fresh frozen plasma and platelets.

Risk stratification. Risk stratification is used to triage patients to outpatient management at one end of the spectrum or urgent endoscopy and high dependency care at the other. The Rockall score uses independent clinical, laboratory and endoscopic risk factors which accurately predict mortality (Tables 6 and 7).[8] The Child-Pugh score of severity of cirrhosis can be used to predict mortality in the first 30 days post-variceal haemorrhage.

Endoscopy. Upper GI endoscopy is a safe procedure with low mortality and complication rate. It allows diagnosis, risk stratification and mechanical therapy to achieve haemostasis. Procedural risks (cardiorespiratory effects of sedation, aspiration, bacteraemia, bleeding, perforation) are greater in therapeutic endoscopy (eg variceal band ligation).

Table 7. Predicted mortality according to Rockall score (%). Reproduced with permission from the BMJ Publishing Group.[8]

Rockall score	0	1	2	3	4	5	6	7	8
Pre-endoscopy	0.2	2.4	5.6	11	24.6	39.6	48.9	50	–
Postendoscopy	0	0	0.2	2.9	5.3	10.8	17.3	27	41

Non-variceal bleeding

Medical therapy. A meta-analysis of 21 RCTs (most of which used omeprazole) of the benefit of proton-pump inhibitor therapy for bleeding peptic ulcers found no significant reduction in either risk of rebleeding or need for surgery.[9,10]

Endoscopic therapy. Several endoscopic modalities are available to achieve haemostasis for bleeding ulcers (Table 8). Combination therapy of adrenaline injection with mechanical methods of haemostasis improve the risk of rebleeding compared with a single treatment.[11,12]

Surgery. Most bleeding ulcers can be controlled endoscopically. However, some patients may have refractory bleeding, particularly those with ulcers on the lesser curve of the stomach and posterior wall of the duodenal cap due to ulcer proximity to large underlying arteries. When bleeding persists despite therapeutic endoscopy a surgical procedure is necessary, usually oversewing of the artery or partial gastrectomy.

Table 8. Endoscopic therapies for non–variceal bleeding.

Thermal therapy	Heater probe/argon plasma coagulation
Injection therapy	Adrenaline 1/10,000 Alcohol Fibrin sealants Saline (local tamponade)
Mechanical	Endoclips
Combination treatments	eg Adrenaline + endoclips

Variceal bleeding

Variceal bleeding carries a poor prognosis. Mortality at one year from subsequent variceal bleeding is 5%, 25% and 50% in Child's class A, B and C patients, respectively.

Medical therapy. Terlipressin, a synthetic analogue of vasopressin, is effective at achieving initial haemostasis and reduction in all-cause mortality compared with placebo. Prophylactic antibiotics reduce mortality in cirrhotic patients hospitalised for acute bleeding. Propranolol used as secondary prophylaxis reduces rebleeding.

Endoscopic therapy. Variceal band ligation is the modality of choice in controlling active bleeding. It should be carried out with anaesthetic support and often endotracheal intubation, because of the high risk of aspiration.

Balloon tamponade. If bleeding continues despite terlipressin and endoscopic therapy, balloon tamponade should be used. This controls active bleeding in 90% of patients, although rebleeding occurs in 50% when the balloon is deflated. Serious complications include aspiration and oesophageal perforation. Transfer to a specialist centre should be sought for consideration of transjugular intrahepatic portosystemic shunt insertion or surgery.

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