



Ulcerative colitis

Unravelling the uncertainty in diagnosis and management

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Key points

- Prompt diagnosis and timely management of people with ulcerative colitis (UC) are key to good patient outcomes.
- An important role for the GP is to identify symptoms of new or flaring UC and to recognise alarm features that should trigger urgent referral to a gastroenterologist.
- Mild to moderate UC can be safely managed in the primary care setting.
- Patients with features of severe UC should be urgently discussed with a gastroenterologist.

Prompt diagnosis of ulcerative colitis (UC) and timely management of flares are crucial to good patient outcomes. GPs play an important role in identifying new cases, the ongoing management of mild to moderately active UC and also in identifying and referring acutely unwell patients for specialty care.

Ulcerative colitis (UC) is a chronic inflammatory disorder of the colon characterised by a relapsing–remitting pattern in most patients (Figure 1). Although the exact cause remains unknown, dysregulation of the gut mucosal immune response results in chronic inflammation of the colon. Many patients with UC can be successfully identified and managed in the primary care setting, with intermittent or regular gastroenterology input.

This article provides a guide for GPs to aid in the prompt diagnosis of potential new cases of UC and the appropriate treatment of flares in people with established UC. It also highlights circumstances that warrant urgent referral of patients with UC to hospital and/or direct communication with a gastroenterologist.

EPIDEMIOLOGY AND PATHOGENESIS

UC is most common in industrialised and Western countries, with the highest reported incidence in Canada (19/100,000) and Northern Europe (24/100,000).¹ The incidence in Australia is similar at 11/100,000.² UC affects men and women equally, and the age at onset is between the ages of 15 and 30 years in most patients, with a smaller second peak in patients over 50 years.

Although the cause of UC remains unknown, the pathogenesis is likely to be multifactorial with interplay between genetic, microbial and other environmental factors. Ultimately it is a dysregulation of gut mucosal immune responses that leads to the development of UC and its clinical manifestations.

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Genome-wide association studies have identified over 160 susceptibility loci for inflammatory bowel disease (IBD; UC and Crohn's disease), with many of these relevant to barrier function and microbial exposure.³

The increasing number of new cases being identified worldwide, especially in regions such as Asia that traditionally have a low incidence of UC, emphasise that genes alone cannot be responsible, and the interplay between genes and other factors is likely to be central. Several environmental factors continue to be studied in depth. Smoking has repeatedly been shown to have a protective effect against UC. Previous gastrointestinal infections have been associated with an increased risk of developing UC, specifically with *Salmonella* spp., *Shigella* spp. and *Campylobacter* spp., although this may be an ascertainment bias. As the 'hygiene hypothesis' suggests, although with limited data, exposure early in life to exogenous antigens may promote tolerance to UC whereas later exposure increases the risk of developing UC.⁴

The role of diet in UC has attracted much interest recently. Studies suggest a trend towards an association between certain dietary fats, fibre and meat intake with the development of UC. However, the data are conflicting and ultimately no true causal association has yet been demonstrated.⁵

APPROACH TO A PATIENT WITH A SUSPECTED NEW CASE OF UC

An important role in general practice is to suspect and subsequently identify potential new cases of UC. This can be challenging, especially as the clinical symptoms of early or mild UC can be similar to those of two much more common conditions, infectious gastroenteritis and irritable bowel syndrome (IBS). Distinguishing between these three conditions involves considering the time course of the symptoms – shortest with infection, longest with IBS – and also whether any alarm features for IBD are present. When considering UC, it is important to remember that a patient's symptoms will depend on the disease extent and severity, and the presence or absence of extraintestinal manifestations. Furthermore, it should be remembered that symptoms alone do not always accurately reflect disease activity.

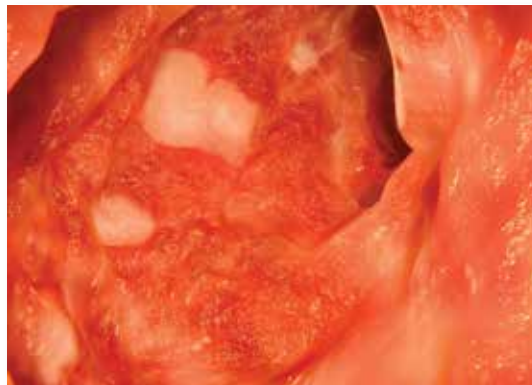


Figure 1. Colon affected by severe ulcerative colitis.

Figure 2 shows an algorithm for differentiating between IBS and IBD.

Patients with UC typically present with a gradual onset of symptoms. Commonly patients experience a change in bowel habit manifesting as increased stool frequency and looser stool consistency with or without mucus, before the onset of rectal bleeding, tenesmus, urgency (with or without incontinence), feeling of incomplete evacuation secondary to rectal inflammation and nocturnal stool motions. Colicky abdominal pain can also be present and generally represents an alarm feature when bleeding or nocturnal stools are also present. Associated extraintestinal manifestations may occur concurrently or before gut symptoms, and can include conditions involving the eye (uveitis, iritis, episcleritis), joints (axial or peripheral arthropathies), skin (aphthous ulcers, erythema nodosum, pyoderma gangrenosum) and hepatobiliary tree (primary sclerosing cholangitis).

Recognising alarm features in a patient is important not only for raising the suspicion of UC but also because alarm features indicate the urgency for seeking prompt gastroenterologist assessment (hours or days rather than weeks). Alarm features include:

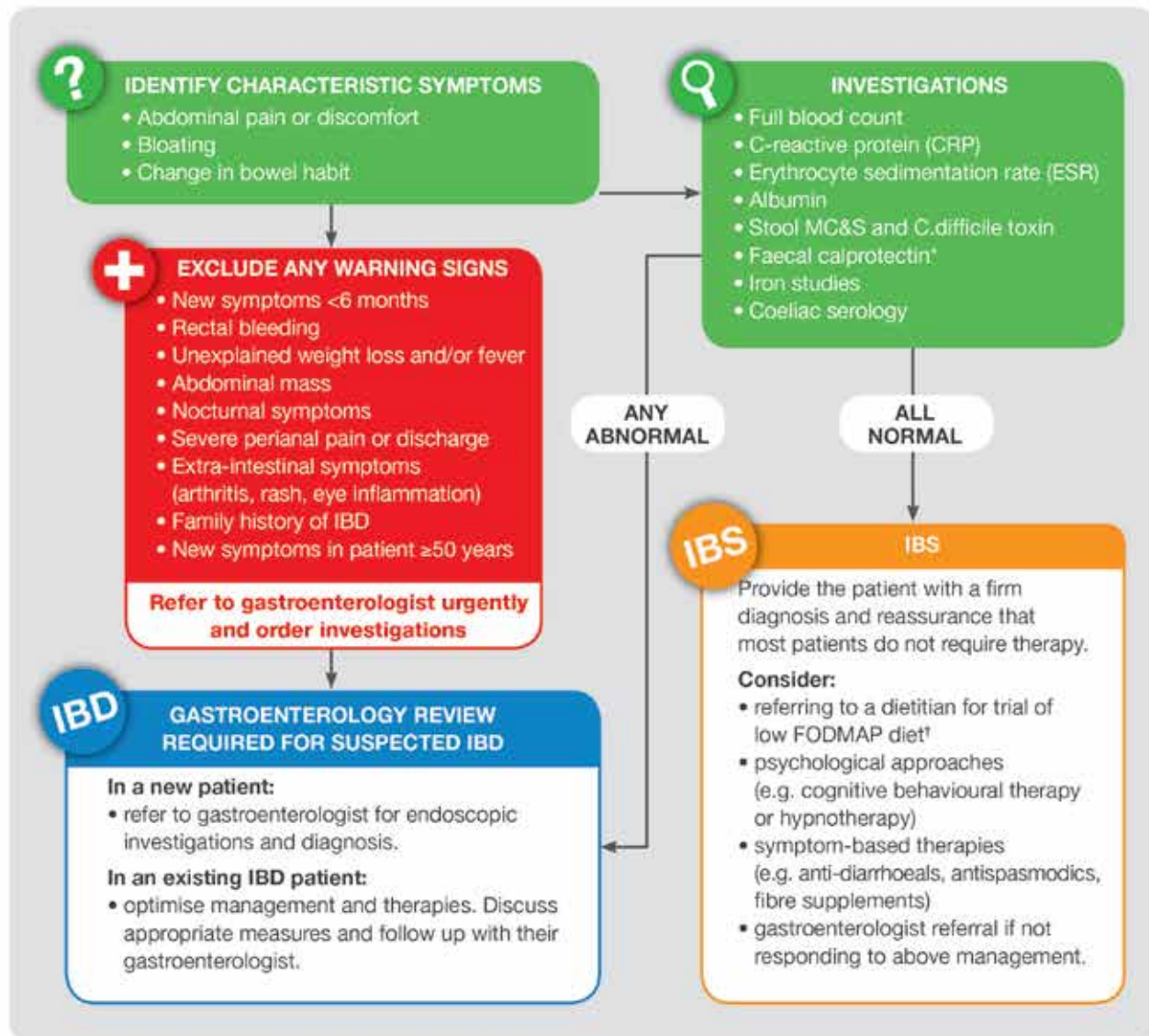
- unexplained weight loss
- rectal bleeding (especially if six or more bloody stools a day)
- nocturnal stools (awaking patient from sleep) or incontinence
- fever and/or tachycardia
- extraintestinal manifestations suggestive of UC
- new symptoms of less than six months in duration, or in a patient aged 50 years or older
- abdominal mass.

Although some of these features can also be present with infectious gastroenteritis, the finding of these in a patient with infectious diarrhoea warrants prompt specialist assessment.

Differentiating between IBS and IBD

IRRITABLE BOWEL SYNDROME (IBS) and **INFLAMMATORY BOWEL DISEASE (IBD)** often present with similar symptoms, making the distinction challenging. IBS symptoms in patients with IBD are around four times more common than in the general population, with slightly higher rates in Crohn's disease (46%) than ulcerative colitis (36%). In IBD patients, symptoms can only be attributed to IBS if their IBD is known to be in remission. There are good therapies (including dietary and psychological) available for IBS. Incorrectly treating IBS symptoms as active IBD will not only be ineffective but also exposes patients to side effects from escalation of IBD medications.

IBS has a complex pathophysiology with contributions from gut inflammation, dietary intolerances and psychological factors. There are simple tests that can be employed to differentiate between IBS and IBD.



*Not currently covered by Medicare. †For more information on the FODMAP diet, visit <http://www.med.monash.edu/ceacs/gastro/fodmap/> or <http://www.gesa.org.au/consumer.asp?td=180>.

References: 1. Barrett JB, Gibson PR. Ther. Adv Gastroenterol 2012;5(4):261-8. 2. Gibson PR, Isler J. Aust Fam Physician 2005;34(4):233-7. 3. Halpin SJ, Ford AC. Am J Gastroenterol 2012;107:1474-82. 4. Spiller R, Aziz Q, Creed F et al. Gut 2007;56:1770-85.

This resource has been authored by the 2013 Clinical Insights Steering Committee: Professor Jane Andrews (Chair), Ms Stephanie Buckton, Professor Ian Lawrence, A/Prof Rupert Leong and Dr Gregory Moore. It has been adapted from the outputs of the multidisciplinary Clinical Insights meeting held in Sydney on 16-17 March 2013. Financial support for the meeting and this educational guide was provided by Shire Australia Pty Limited, ABN 29 128 941 819, Level 6, Avaya House, 123 Epping Road, North Ryde NSW 2113, Australia. Tel: 1800 612 812. Email: enquiriesaustralia@shire.com. AU5/LD/MEZ/14/0065. Clara Lynne of Elixir Healthcare Education Sydney was funded by Shire to provide editorial support to the authors.

Figure 2. 'Differentiating between IBS and IBD', a GP resource from Crohn's and Colitis Australia (www.crohnsandcolitis.com.au/research/clinical-insights-tools).

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1. DIFFERENTIAL DIAGNOSIS FOR ULCERATIVE COLITIS

- Irritable bowel syndrome*
- Infectious diarrhoea*
- Other inflammatory bowel disease – Crohn’s disease, microscopic colitis
- Coeliac disease
- Ischaemic colitis
- Diverticulitis
- Colorectal cancer
- Medication-induced diarrhoea

*Most common.

Recommended investigations

Performing the following investigations can help GPs further evaluate the possibility of UC in a patient, identify an unwell patient requiring urgent referral to a gastroenterologist and also simultaneously exclude potential differential diagnoses (Box 1):

- full blood count (FBC)
- C-reactive protein (CRP) and/or erythrocyte sedimentation rate (ESR)
- electrolytes, renal function and albumin
- liver function tests (LFTs)
- iron studies
- coeliac serology, including IgA level (if diarrhoea without blood)
- stool microscopy, culture and sensitivity, ova/cysts/parasites, *Clostridium difficile* toxin
- faecal calprotectin (if diarrhoea without bleeding, as blood will always give an elevated faecal calprotectin level).

Faecal calprotectin is a useful test that is available in many pathology centres. Faecal calprotectin is a stable protein found in all leucocytes; when there is inflammation from any cause, leucocytes are released into the lower gut and faecal calprotectin is thus raised. An elevated faecal calprotectin level is not specific for UC but is highly sensitive for inflammation (between 93 and 99%).⁶ This noninvasive faecal test is not currently MBS funded, and the cost

ranges between \$40 and \$70. It is useful for differentiating between the presence of organic disease and functional disorders (especially IBS), and thus can help determine whether colonoscopy is required for further evaluation. A faecal calprotectin level below 50 µg/g represents a very low likelihood of bowel inflammation, and a level above 100 µg/g indicates a need for further evaluation by a gastroenterologist.

When to refer a patient with a suspected new case of UC

Any patient in whom UC is suspected based on symptoms, with or without abnormal investigation results, should be referred to a gastroenterologist for further assessment. Patients with alarm features should be referred urgently and it is advisable to have direct phone contact with the nominated gastroenterologist or centre in addition to the usual referral pathway.

Acute severe ulcerative colitis (ASUC) is a serious form of UC with a high risk of significant morbidity and mortality and of a need for colectomy. ASUC should be diagnosed in patients with six or more bloody stool motions daily and any feature of systemic illness including:⁷

- tachycardia (heart rate >90 beats per minute)
- fever (temperature >37.8°C)
- anaemia (haemoglobin <105 g/L)
- raised CRP (>30 mg/L) or ESR (>30 mm/h).

It is strongly advised that GPs directly contact a gastroenterologist or centre and refer a patient with suspected ASUC immediately to hospital for urgent assessment and management.

The main adverse outcomes that we seek to avoid in the management of UC are ASUC, persistent active disease, poor bone health, colorectal cancer and potentially avoidable colectomy.

MANAGEMENT

The goals of therapy when treating patients with UC are to:

- induce remission in active disease
- maintain disease control by preventing

2. KEY CONCEPTS IN MANAGEMENT OF ULCERATIVE COLITIS

- Severity and extent of disease guide the choice of agent and route of administration. They also enable individualisation of treatment for each patient

Severity

- S1 Mild = ≤4 stools/day without blood
- S2 Moderate = >4 stools/day ± blood
- S3 Severe = ≥6 bloody stools/day with any systemic feature of toxicity

Extent

- E1 = rectum only
- E2 = left-sided
- E3 = extensive UC (beyond splenic flexure)
- Goals of therapy are to
 - induce remission in active disease
 - maintain disease control, prevent relapse and reduce need for corticosteroids

- Preventive strategies should be routinely practised to reduce the risk of relapse, complications of disease and complications of therapy
- Recognise alarm features and/or severe disease as this necessitates urgent referral to a gastroenterologist (including direct phone contact) ± hospital admission

relapses, and also reduce the need for corticosteroids.

The severity and extent of disease dictate the choice of agent and modality of administration (Box 2 and Figure 3).

Active disease in established UC

The initial step in managing active disease is to establish disease severity, based on stool frequency, presence/absence of blood in the stool and systemic features of toxicity (fever, anaemia, tachycardia, raised CRP/ESR, low albumin) – see Box 2.

Clinician's guide to ulcerative colitis (UC) management



Disease severity*

SYMPTOMS:

- Diarrhoea
- Urgency of defecation or loss of control
- Nocturnal bowel movements
- Rectal bleeding – assess
- Abdominal pain or discomfort

S1 Mild: ≤4 stools/day without blood

S2 Moderate: >4 stools/day, +/- blood but without systemic toxicity

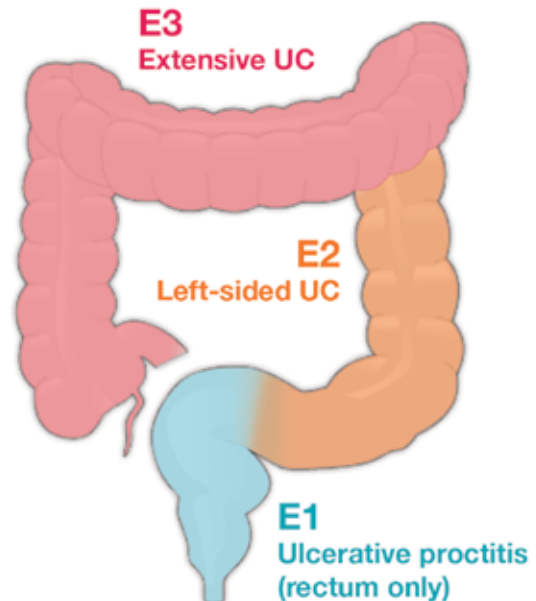
S3 Severe: >6 bloody stools/day with any systemic feature

Red Flags: fever, anaemia, tachycardia, elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), low albumin.

S3 of any extent, contact a gastroenterologist or admit to hospital.



Disease extent*



Investigations

RULE OUT INFECTION:

Stool microscopy and culture (include *Clostridium difficile* toxin).

ASSESS CURRENT LEVEL OF INFLAMMATION:

- Blood tests: full blood count (FBC); liver function test (LFT); albumin; electrolytes, urea, creatinine (EUC); CRP; ESR.
- Faecal biomarker testing: calprotectin and/or lactoferrin.†

ADDITIONAL DIAGNOSTIC TESTS:

Colonoscopy and flexible sigmoidoscopy.



Management

Treatment should be based on disease extent and severity.

For active disease prescribe topical 5-aminosalicylic acid (5-ASA suppository, foam or enema) in addition to recommended induction doses of oral 5-ASA.

E1: 5-ASA rectal suppository (1 g/day)

E2 & E3: Oral 5-ASA (2–4 g/day) + topical 5-ASA enema, foam and/or suppository

- In active disease, combination oral and topical 5-ASA is more effective than either alone.

*Based on the Montreal classification of UC disease severity and extent. †Not currently covered by Medicare.

Maintenance

5-ASA MAINTENANCE THERAPY TO REDUCE THE RISK OF RELAPSE

Medication formulations should be given at ≥ 1.5 g/day. Check individual drug PI for more information (www.tga.gov.au/hp/information-medicines-pi.htm).

- Lifelong therapy is recommended to reduce the risk of colon cancer. Refer to NHMRC guidelines on surveillance colonoscopy (<https://www.nhmrc.gov.au/guidelines/publications/ext0008>).

OTHER CONSIDERATIONS:

- Psychological support (<http://www.ibdclinic.org.au>) and dietetic advice (<http://www.med.monash.edu/cecs/gastro/fodmap>) should be made available.
- When planning a pregnancy, patients should be referred to a gastroenterologist. Refer to ECCO guidelines on IBD in pregnancy (https://www.ecco-ibd.eu/images/6_Publication/6_3_ECCO%20Guidelines/MASTER_JCC_Pregnancy_Guidelines_2010.pdf).
- IBD patients may be at increased risk of osteoporosis. Refer to BSG guidelines (http://www.bsg.org.uk/images/stories/clinical/ost_coe_ibd.pdf).
- Patients may benefit from participating in a patient support group, such as Crohn's & Colitis Australia (<http://www.crohnsandcolitis.com.au>).

Flares

INCREASE ORAL 5-ASA THERAPY TO INDUCTION DOSE AND CONSIDER ADDING RECTAL THERAPY DURING A FLARE

Medication formulations can be doubled to ~ 3 g/day (check individual drug PI for more information; they often recommend dosing up to 4.8 g/day, but most drugs can be safely increased to up to 6 g/day).

- Reassess symptomatic response in 1–2 weeks. If the patient does not respond to treatment, refer to a gastroenterologist.
- If symptoms persist despite 5-ASA, consider systemic corticosteroids. This should be done in consultation with a gastroenterologist, and a strategy for complete withdrawal should be developed.
- Patients who have steroid-dependent disease or have been using steroids more than once a year should be reviewed by a gastroenterologist. They may require azathioprine or 6-mercaptopurine.
- The most common cause of flares is non-adherence. Refer to MARS to assess patient adherence (<http://pub.basecase.com/EvGWaXTPR/>).

Red Flags

REFER TO A GASTROENTEROLOGIST IF:

- severe (S3)
- family history of colon cancer
- pain
- unexplained weight loss
- symptoms persistent despite therapy.

REFER TO HOSPITAL ADMISSION FOR TREATMENT IF:

- severe (S3) or extensive UC with any of the following signs of systemic toxicity: fever $>37.8^{\circ}\text{C}$, anaemia (haemoglobin <10.5 g/dL), tachycardia (>90 bpm), elevated ESR >30 mm/h or CRP >30 , low albumin.

Additional information

Gastroenterological Society of Australia (GESA): <http://www.gesa.org.au/content.asp?id=239>

ECCO guidelines: <https://www.ecco-ibd.eu>

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Figure 3 (continued). 'Clinician's guide to ulcerative colitis (UC) management', a GP resource from Crohn's and Colitis Australia (www.crohnsandcolitis.com.au/research/clinical-insights-tools).

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Mild to moderately active UC can be safely managed in primary care as indicated in Figure 3. A combination of maximum-dose oral and topical 5-aminosalicylic acid (5-ASA) therapy should be used as first-line management in this setting. The various topical and oral formulations of 5-ASA available and the recommended dosing are summarised in the Table. The following are useful practical hints:

- combined topical and oral therapy is more effective than monotherapy
- topical 5-ASA therapy (suppository, enema or foam) is more effective than topical corticosteroids
- suppositories are best for limited rectal disease (E1) whereas enemas, foams or suppositories can be used in E2 or E3 disease (see Box 2)
- reassess response no later than 10 to 14 days after escalation of 5-ASA therapy
- if a patient is unwell enough to consider oral corticosteroids then their case should be discussed with a gastroenterologist.

GPs should refer a patient with UC to a gastroenterologist in the following circumstances:

- persistent symptoms after 10 to 14 days despite using maximum-dose oral and topical 5-ASA therapy as directed above – these patients should be referred to a gastroenterologist as they warrant further investigation and may require escalation of therapy with corticosteroids and thiopurines. It should be noted that because thiopurines take eight to 12 weeks for maximal efficacy to be reached they are not an appropriate induction agent
- severe disease activity at any time (six or more bloody stools a day with any systemic feature of toxicity) – these patients should all be discussed with a gastroenterologist and referred to hospital for assessment
- corticosteroid-dependent UC (unable to wean and/or cease corticosteroids)

TABLE. 5-AMINOSALICYLIC ACID (5-ASA) FORMULATIONS AND RECOMMENDED DOSING FOR ULCERATIVE COLITIS

| Drug (brand name) | Formulation | Maximum dose | Maintenance dose |
|---|-------------|--------------|------------------|
| Sulfasalazine (Pyralin EN, Salazopyrin) | Oral | 4 g/day | 2 g/day |
| Mesalazine (Mesasal) | Oral | 1.5 g/day | 750 mg/day |
| Mesalazine (Pentasa) | Oral | 4 g/day | 2 g/day |
| | Enema | 1 g/day | 1 g/day |
| | Suppository | 1 g/day | 1 g/day |
| Mesalazine (Salofalk) | Oral | 3 g/day | 1.5 g/day |
| | Enema | 4 g/day | 2 g/day |
| | Foam enema | 4 g/day | 2 g/day |
| | Suppository | 1 g/day | 1 g/day |
| Mesalazine (Mezavant) | Oral | 4.8 g/day | 2.4 g/day |
| Balsalazide (Colazide) | Oral | 6.75 g/day | 3 g/day |
| Olsalazine (Dipentum) | Oral | 2 g/day | 1 g/day |

or regularly requiring one or more courses of corticosteroid a year.

Managing disease in remission

The long-term management of UC includes appropriate maintenance medical therapy for an individual (to prevent flares of new UC activity) and regular monitoring to detect complications, toxicity or newly active disease.

Maintenance therapy

Common agents used in maintenance therapy of UC include 5-ASA and thiopurines. **5-ASA therapy** is available in topical (suppositories, enemas, foams) and oral formulations, as mentioned earlier. Topical 5-ASAs are most effective for distal disease (rectal and left-sided disease) and are more effective than topical corticosteroid therapies. The minimum recommended frequency for topical maintenance therapy is three times per week.

If oral therapy is required because of either extensive disease (E3) or inadequate disease control with topical therapy, it is important to note that combination oral and topical therapy is more effective than monotherapy alone. Oral 5-ASA therapy

should be given at a minimum dose of 1.5 g each day (Table). Given that patient compliance is paramount in maintaining disease control, studies have demonstrated that once daily dosing of oral 5-ASA therapy is as efficacious as split dosing.⁸

The thiopurines azathioprine (AZA) and 6-mercaptopurine (MP) are used in disease refractory to 5-ASA treatment alone or when 5-ASAs are poorly tolerated. Patients requiring these agents should be co-managed with a gastroenterologist. It should be remembered when commencing thiopurines that they can take up to three months to reach their maximal effect. The recommended routine management for patients taking thiopurines includes:

- three-monthly FBC and LFTs to monitor for bone marrow suppression and hepatotoxicity
- annual influenza vaccination and skin checks for basal and squamous cell carcinomas
- three-yearly pneumococcal vaccination
- regular Pap smears and mammograms for women.

Corticosteroid therapy is occasionally required but usually in the context of a flare. A patient who requires a prolonged

course of corticosteroids (six weeks or longer) or two or more courses within any one-year period should be referred to a gastroenterologist. Corticosteroids should not be used as a maintenance management strategy.

Complication prevention

Various management strategies can be used to prevent the development of complications related to UC and its treatment in patients who are otherwise well. These strategies include the following.

- Vaccinations should be kept up to date while patients are well, although live vaccinations must be avoided in patients on any immunosuppressant.
- Routine blood tests (FBC and LFT) should be performed every three months in patients taking a thiopurine and annually in those taking 5-ASA.
- Primary sclerosing cholangitis is an uncommon condition (3 to 5%) that can co-exist in UC patients. Affected patients are at increased risk of developing colorectal cancer and should have annual colonoscopies. LFT should be routinely monitored as recommended above while on thiopurine/5-ASA therapy, or alternatively two-yearly if on no therapy. Suspect primary sclerosing cholangitis if LFT results are increasingly abnormal.
- Vitamin D levels should be checked and supplements used if required as patients with UC are at increased risk of developing osteopenia and osteoporosis. A bone mineral density scan (BMD) should be considered if there has been a history of prolonged or repeated corticosteroid exposure, or significant weight loss.
- Patients with UC of eight or more years' duration and disease extending beyond the rectum (E2 or E3 disease) are at increased risk of developing early colorectal cancer and should be referred to a gastroenterologist for consideration of a screening colonoscopy.

RESOURCES FOR GPs

With an increasing recognition that many people with UC are predominantly managed in primary care, tools have been developed to better support nonspecialist management of patients with UC in Australia. These include guides to diagnosing UC and managing mild to moderate disease (Figures 2 and 3), as well as a template for tailored UC management plans for individual patients to support shared GP-specialist care and patients. Although a pharmaceutical company funded the development of these resources, the content is entirely the work of the 2013 Clinical Insights Steering Committee, who are all experienced gastroenterologists.

The tools are freely available and can be viewed or downloaded from the Crohn's and Colitis Australia website at www.crohnsandcolitis.com.au/research/clinical-insights-tools or within some GP software.

CONCLUSION

UC is increasing worldwide. Prompt diagnosis and management are crucial to good patient outcomes and can be achieved by recognising common clinical features and alarm symptoms. Management of mild to moderately active UC is safe and appropriate in the primary care setting. Referral to a gastroenterologist should be triggered in suspected new cases of UC, especially in the presence of alarm features. In patients with established UC, further input should be sought from a gastroenterologist if there has been a failure to respond to appropriate escalation of 5-ASA therapy, if there is a need for corticosteroids or if there are alarm features or severe disease activity.

Tools to support the management of UC in general practice are available from the Crohn's and Colitis Australia website. **MI**

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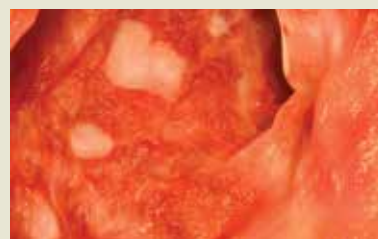
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COMPETING INTERESTS: Dr Ooi: None. Professor Andrews is the chair of the 2013 Clinical Insights Steering Committee, who authored the clinician tools reproduced in this article (Figures 2 and 3).

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