

Inflammatory bowel disease

The GP's role in shared care of patients

BRITT CHRISTENSEN MB BS, BSc; BELINDA HEADON RN; PETER R. GIBSON MD, FRACP

Key points

- Inflammatory bowel disease is becoming more prevalent in Australia and is associated with significant morbidity.
- Ideal management involves shared care between GPs and specialist gastroenterologists.
- The role of the GP in the management of patients with IBD includes early diagnosis, patient support, preventing disease complications, monitoring for disease relapse and medication side effects and providing nutritional, smoking cessation and psychological support.

Shared care between GPs and specialist gastroenterologists is the ideal model of care for patients with inflammatory bowel disease. GPs play important roles in recognising the symptoms early, supporting the patient, screening for long-term complications and treatment.

Crohn's disease and ulcerative colitis are the two main disorders that make up the collective term, inflammatory bowel disease (IBD). The cause of IBD is unknown, and the clinical illness results from chronic inflammation of the gastrointestinal tract. It is characterised by intermittent exacerbations of disease activity followed by episodes of remission.

Treatment of the inflammation is complex but effective in improving quality of life and altering the natural history of the disease. Hence, although IBD is a chronic illness that is not cured by therapy, its early diagnosis leads to better outcomes for patients, including remission induction and prevention of disease-associated complications. Multidisciplinary management that includes active involvement of the GP is crucial to these outcomes.

This review will address the core information required by GPs to understand the illness and its treatment, and their recommended position in the management algorithm.

CORE KNOWLEDGE OF EPIDEMIOLOGY AND PATHOGENESIS

Incidence and prevalence in perspective

The annual incidence rate for IBD in Australia is approximately 30 per 100,000, implying that more than 6000 patients are first diagnosed in any one year.¹ This is similar to the incidence of rheumatoid arthritis and schizophrenia. The incidences of Crohn's disease and ulcerative colitis are approximately equivalent at 17 per 100,000 and 11 per 100,000, respectively. In about 10% of patients the distinction is unclear, and these patients are classified as

Dr Christensen is an Advanced Trainee in Gastroenterology, Ms Headon is an IBD Clinical Nurse Consultant, and Professor Gibson is Director of Gastroenterology, Department of Gastroenterology, Alfred Hospital and Monash University, Melbourne, Vic.

having ‘indeterminate colitis’. Many patients with indeterminate colitis have the diagnosis clarified over time, and about 5% of patients with IBD have an initial diagnosis of ulcerative colitis changed to Crohn’s disease, or vice versa, as the illness evolves.

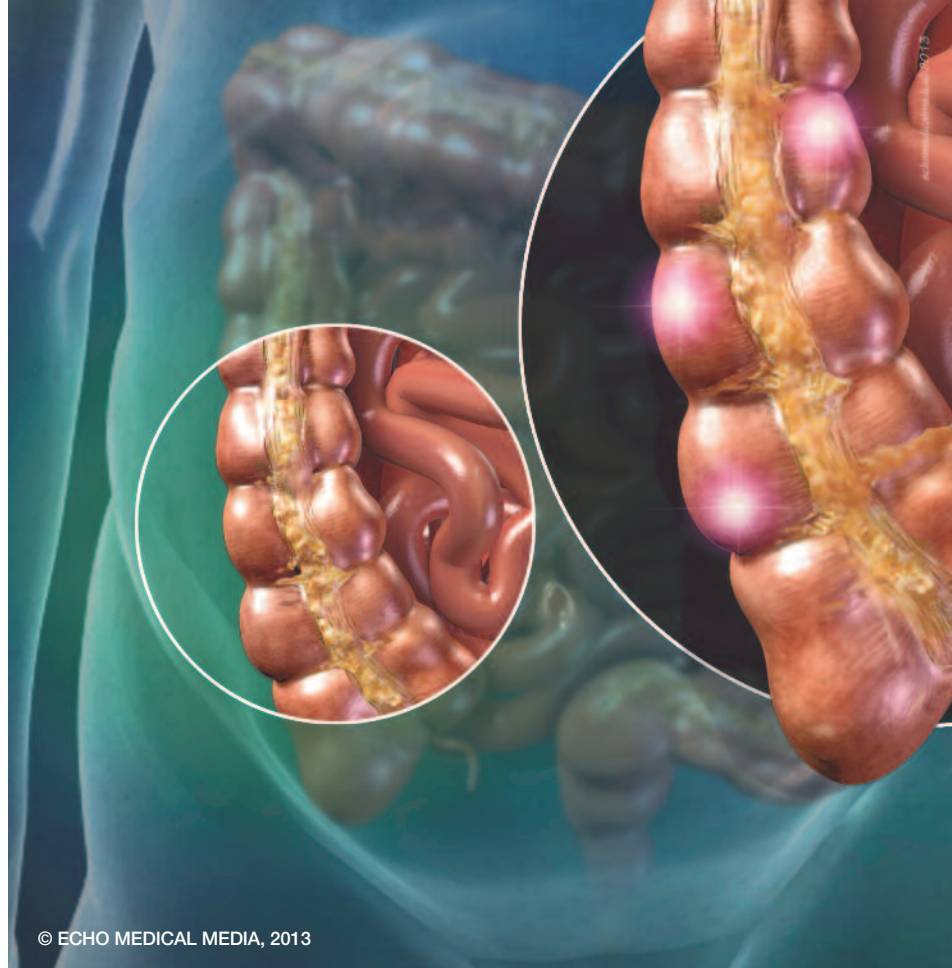
Demographics

Men and women are equally affected by IBD and the condition primarily presents between the ages of 15 and 30 years, with a small second peak of incident cases after age 50 years. The incidence is higher in people of Jewish background and it is rarely seen in Indigenous Australians. The incidence and prevalence of IBD is much lower in Asia than in Australia, but a rising trend has been identified over the past two decades. In addition, IBD is increasingly common in Asian migrants to the west, with incidence often equivalent or even greater to that in the local population.

Pathogenesis – genes and environment

The cause of IBD is unknown. However, it appears to be the result of a genetic predisposition that leads to dysregulation of the immune system directed against bacteria or their products found in the intestinal lumen that do not usually elicit an immune response.

Many environmental factors have been postulated to increase or decrease the risk of developing IBD. Of these, three in particular – smoking, having been breastfed and diet – impact on management or are queried by patients. Smoking is the one factor that has been demonstrated to modify the risk of developing IBD. Although current smokers are about 40% less likely to develop ulcerative colitis, smoking increases the risk of developing Crohn’s disease by twofold, and also increases disease recurrence and is associated with a more aggressive disease course.² There is some evidence that breastfeeding an infant for more than three months may protect the infant against later development of IBD, with some figures suggesting that the risk may be halved.³ Regarding diet, there is a suggestion that a western diet increases the risk of IBD. Diets rich in meat, dairy products and sugar,

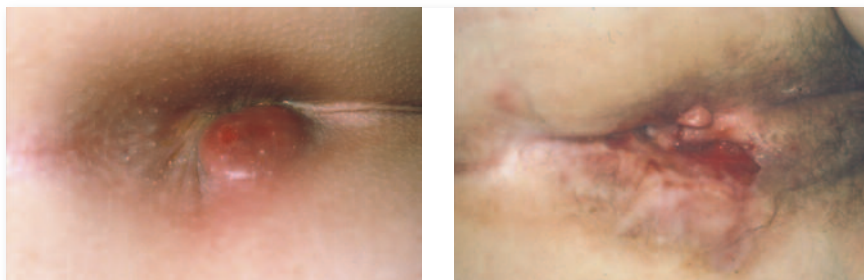


and low in fibre and other plant constituents, have been associated with a higher risk of IBD. In addition, as Asian countries adopt western lifestyles, their rates of IBD are increasing.⁴ This may be explained by the increasing availability of western food, increasing antibiotic use, improved hygiene, greater use of vaccinations or changes in the gut microbiota.

Although no one gene has been conclusively shown to cause IBD, genetically determined factors contribute to IBD susceptibility. Siblings and family members of those affected with IBD are at increased risk of developing the condition, with 10 to 20% of people with IBD having one or more affected family members. In particular, siblings of patients with Crohn’s disease are 17 to 35 times more likely to develop the disease than the general population.⁵ Also, children whose parents both have IBD (Crohn’s disease or ulcerative colitis) have up to a one in three chance of developing IBD by age 28 years.⁶

MAKING THE DIAGNOSIS

A key to the diagnosis of IBD is to suspect it. It is not uncommon for patients with IBD to describe a long period of symptoms (months to years) before the correct diagnosis is made. The GP’s role is to assess the clinical



Figures 1a and b. Perianal manifestations of Crohn's disease that are readily visible by simple inspection. a (left). Perianal abscess. b (right). Inflamed distorted perianal region with skin tag and fistula that is draining pus (which has been wiped away).

presentation and to perform sufficient initial investigations to arouse the suspicion of IBD and then to refer the patient to secondary care for further diagnostic investigations.

Suspecting the diagnosis

In Crohn's disease, inflammation most commonly causes ulceration in the distal ileum and right colon but can result in patchy pathology anywhere from the mouth to the anus. In ulcerative colitis, the distribution is confined to the large bowel and is continuous, extending from the rectum proximally to varying degrees. The gastrointestinal pathology of IBD can manifest in many ways, including diarrhoea, blood in the stool, abdominal pain, weight loss and fever.

In ulcerative colitis, patients often have loose bloody stools and increased frequency of defaecation as their predominant symptom. They may, however, present with constipation, particularly if the inflammation is restricted to the rectum. Other symptoms include urgency, urge incontinence, tenesmus and colicky lower abdominal pain.

The predominant clinical manifestations of Crohn's disease are more variable and patients can present with many non-specific symptoms, including fatigue, prolonged diarrhoea with abdominal pain and weight loss. Pain is much more common in Crohn's disease. Unlike in ulcerative colitis, the inflammation in

Crohn's disease is transmural and, therefore, deep ulceration can lead to the development of fistulas (enterovesical, enterocutaneous, enteroenteric or entero-vaginal) and abscesses. Fibrosis associated with the chronic inflammation can lead to intestinal stenosis and consequent intestinal obstruction. One-third of patients with Crohn's disease have some form of perianal disease, including pain, abscesses, fissures or anorectal fistulas (Figures 1a and b).

IBD is also associated with many extra-intestinal manifestations (see the box on this page). Because these sometimes precede the onset of the intestinal manifestations, the possibility of underlying IBD should be at least considered when these conditions occur in isolation.

Despite this, most patients who present in general practice with diarrhoea and/or chronic abdominal pain will have irritable bowel syndrome (IBS) rather than IBD. The 'alarm' features that should raise the suspicion of a diagnosis of IBD are shown in the box on page 19.

Investigating a patient with suspected IBD

The appropriate first-line investigations for a patient with suspected IBD and what they might contribute are listed below.

- Full blood examination: assists in judging severity (anaemia, leucocytosis, thrombocytosis) as well as clues that

EXTRAIESTINAL MANIFESTATIONS OF IBD

Arthropathy

- Peripheral large joint arthropathy
- Sacroiliitis
- Ankylosing spondylitis

Ocular symptoms

- Uveitis
- Iritis
- Conjunctivitis
- Episcleritis

Dermatological manifestations

- Pyoderma gangrenosum
- Erythema nodosum
- Aphthous ulcers

Abnormal liver function tests

- Primary sclerosing cholangitis
- Gallstones

Venothromboembolism

inflammation might be present.

- Electrolytes and renal function: can be disturbed if there is significant diarrhoea; useful for urgent management decisions.
- Liver function tests: decreased albumin may be a clue to chronic inflammation (which reduces albumin synthesis), protein loss and/or malabsorption; can rule out liver pathology; gives a useful baseline.
- Iron studies: iron deficiency is common in chronic inflammation, mainly due to poor absorption.
- Vitamin B₁₂ and/or folate levels measurement: help plan management.
- C-reactive protein level, with or without erythrocyte sedimentation rate, measurement: an increased value is a clue that inflammation is present, although a normal level does not rule out IBD.
- Coeliac serology: rules out coeliac disease (provided the patient is

consuming gluten).

- Stool culture, including *Clostridium difficile* culture and toxin: infection can precipitate relapse; *C. difficile* infection is more common in patients with IBD.
- Faecal calprotectin level measurement, if available: an increased value is an indicator of intestinal inflammation; gives a useful baseline.

The newly available test, faecal calprotectin, is potentially valuable in assessing patients with possible intestinal inflammation. Calprotectin, a normal protein constituent of neutrophils, is resistant to enzymatic degradation and therefore is not destroyed when white cells migrate into the intestinal lumen at the site of inflammation; it is thus readily detected in the faeces. A calprotectin level below 50 µg/g stool indicates a very low likelihood of inflammation in the bowel and is very reassuring that IBD is not being missed. A level greater than 100 µg/g stool warrants colonoscopy in a patient with suspected IBD. It can also be elevated in association with colorectal cancer. There is no PBS subsidy for the faecal calprotectin test, and the patient would be about \$50 to \$75 out of pocket. Many pathology companies and tertiary hospitals are now offering this service.

Investigations are also directed towards excluding other possible diagnoses. The tests ordered should be chosen in light of the clinical scenario, as outlined in the box on differential diagnoses on this page.

REFERRAL TO A GASTROENTEROLOGIST

When IBD is suspected in a patient, even if blood test results are normal, referral to a gastroenterologist is appropriate so that the definitive diagnosis can be made. Clear communication is important in the referral so that patients can be triaged appropriately. Referral options are discussed later in the section titled 'Models of care in IBD' (see page 20).

A key to diagnosis is the demonstration of chronic inflammation in the

ALARM FEATURES FOR IBD

History

- Rectal bleeding (particularly mixed with stools)
- Nocturnal symptoms (patients wake frequently at night to open their bowels)
- Symptoms of bowel obstruction
- Extraintestinal manifestations

Examination

- Weight loss
- Abdominal mass
- Fevers
- Pallor or tachycardia
- Perianal abscess, fissure or fistula
- Mouth ulcers

Investigations

- Evidence of systemic inflammation (elevated C-reactive protein, white cell count and/or platelet count or erythrocyte sedimentation rate)
- Iron deficiency with or without anaemia
- Blood or white cells on stool microscopy and culture in absence of pathogens
- Elevated faecal calprotectin

gastrointestinal tract. In most patients, this is achieved by colonoscopy, which should include ileoscopy with multiple biopsies of the ileum and colon (Figures 2a and b). Gastroscopy is frequently performed and is considered best clinical practice in paediatrics, where the additional information obtained can contribute to both the diagnosis of IBD and its phenotyping. The value of gastroscopy in adults has not been as overwhelming, but it is often performed for good reason. In the minority of patients with disease not within the reach of upper and lower endoscopy, clinical suspicion dictates imaging of the small intestine, which is now performed by CT or MR

DIFFERENTIAL DIAGNOSES FOR IBD

Infectious diarrhoea

- Acute onset of (usually) bloody diarrhoea, often with pain
- Before referral, consider faecal microscopy, culture and *Clostridium difficile* toxin

Diverticulitis

- Older patients with fever and left lower quadrant pain
- Before referral, consider abdominal CT scan

Coeliac disease

- Fatigue, iron deficiency and non-bloody diarrhoea with or without weight loss
- Before referral, consider coeliac serology

Irritable bowel syndrome (IBS)

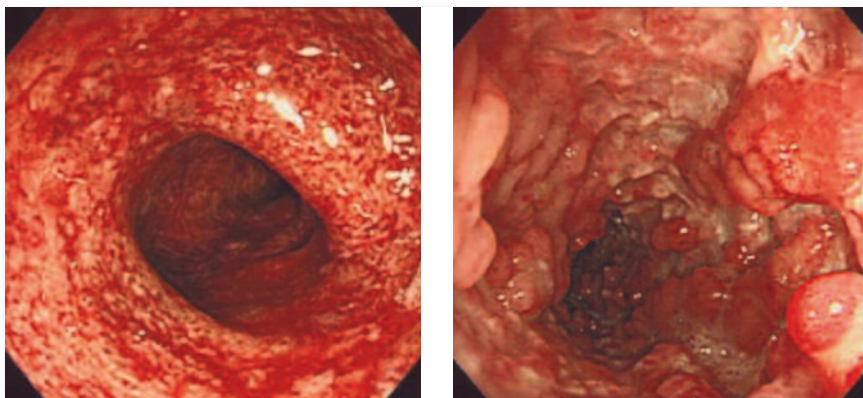
- Combination of abdominal pain with change in bowel habits; bloating often present; not unwell
- No specific positive investigation before referral, but consider C-reactive protein and faecal calprotectin levels (both normal in IBS)

Colorectal cancer

- Older patient; significant family history
- Refer for colonoscopy

enterography or enteroclysis (CT or MR imaging of the small intestine after distension of the intestine with fluid, administered by drinking [enterography] or by tube [enteroclysis], and possibly also the use of intravenous contrast). Barium studies have been superseded.

The diagnosis of IBD is made by the gastroenterologist considering all the information together, including the clinical scenario, results of blood tests, endoscopic findings and histopathology, and, in some patients, results of imaging of the small bowel. It is important to emphasise that the histopathology report alone does not determine the diagnosis; clinical correlation is essential.



Figures 2a and b. Endoscopic appearances of inflammatory bowel disease. a (left). Diffuse inflammation with spontaneous bleeding characteristic of severe ulcerative colitis. b (right). Deep ulceration and cobblestone appearance of Crohn's disease.

Patients who need urgent review or hospital admission

Urgent referral or admission to hospital should be considered in patients who present with severe abdominal pain, diarrhoea more than eight times a day, marked weight loss, fever or tachycardia, or a haemoglobin concentration below 100 g/L. These patients require urgent diagnosis and treatment, and a gastroenterologist or hospital should be contacted directly. Observation alone in patients with severe disease carries a risk of bowel perforation and death.

TREATMENT OF GASTROINTESTINAL INFLAMMATORY DISEASE

The pharmacological treatment of IBD is complex, and is summarised in Table 1. Several major changes in approach have occurred recently, as discussed below.

- Aminosalicylates have become the cornerstone of therapy in most patients with ulcerative colitis. Available 5-aminosalicylic acid (5-ASA; also known as mesalazine) preparations are sulfasalazine, coated 5-ASA, balsalazide and olsalazine. Oral and rectal preparations are used in combination for active disease and either or both for maintenance. These drugs, however, have little place in the

management of patients with Crohn's disease, apart from in mild disease and possibly for the prevention of colorectal cancer.

- The role of corticosteroids (prednisolone, hydrocortisone) has been considerably reduced to short-term therapy for temporary control of inflammatory disease.
- The use of older immune modulating drugs has been rejuvenated by the recent availability of thiopurine metabolite measurement and therefore another means of optimising dosages for azathioprine and 6-mercaptopurine (additional to blood monitoring to detect adverse effects such as leukopenia or hepatotoxicity), and by the use of methotrexate subcutaneously rather than orally to ensure a therapeutic dose is achieved.
- The introduction of the biological drugs infliximab and adalimumab, which inhibit the action of tumour necrosis factor alpha (TNF- α), has revolutionised the ability to heal the bowel in many patients, and to keep it healed.
- The goals of treatment have evolved from symptom control to corticosteroid-free clinical remission to

combined long-term clinical and endoscopic remission (mucosal healing), which maximises quality of life and decreases overall health care costs.

The philosophical change in treatment strategy can be best illustrated for Crohn's disease. The current approach is to gain tight control over the disease within the first year after diagnosis by inducing remission (e.g. with corticosteroids) and commencing maintenance therapy with a thiopurine for all but very mild disease at diagnosis. Failure to induce remission within a timely period would lead to switching the thiopurine to methotrexate or to stepping up therapy by introduction of an anti-TNF agent. Ideally, immunomodulator therapy is continued concomitantly with the anti-TNF agent as this leads to better outcomes than with anti-TNF monotherapy. Achieving mucosal healing within the first two years gives the best chance of maintaining healing subsequently.

MODELS OF CARE IN IBD

With the evolving management strategies and complexity of therapeutic decisions for IBD, it is challenging to offer current excellence in care. There is now good evidence that outcomes are dependent on the quality of management, particularly in the first couple of years after diagnosis. In such a setting, the management of the gastrointestinal disease itself should be directed by a gastroenterologist. Hence, all patients should be referred early in their illness, not just for diagnosis but also for designing the management strategies.

The urban GP is faced with three reasonable referral options to offer the patient: an IBD clinic in a tertiary hospital, a private gastroenterologist and a general gastroenterology clinic at the local public hospital. The advantages and disadvantages of each of these options are described in Table 2. Another option for the urban GP may be to refer to a surgeon; although

TABLE 1. CONVENTIONAL MEDICAL AND SURGICAL THERAPIES FOR IBD

Therapy	Application to ulcerative colitis and Crohn's disease	Side effects/monitoring
Oral 5-aminosalicylic acid (5-ASA/mesalazine) preparations: sulfasalazine, coated 5-ASA, balsalazide and olsalazine	<i>Ulcerative colitis:</i> First-line therapy. If moderate disease, start on high dose. Once remission achieved, decrease to maintenance dose slowly as risk of relapse. Should remain on maintenance 5-ASA preparation – able to reduce the risk of relapse by two-thirds and may decrease risk of colon cancer. (PBS-funded) <i>Crohn's disease:</i> 5-ASA preparations largely ineffective; used only in very mild disease	Excellent long-term safety profile. Risk of uncommon but severe side effects with sulfasalazine (Stevens–Johnson syndrome, agranulocytosis, severe hepatitis). Interstitial nephritis in rare cases. Reversible azoospermia with sulfasalazine
Topical 5-ASA (enema, foam, suppository)	<i>Ulcerative colitis:</i> First-line therapy for active distal colitis and proctitis. Additive effect when used with oral 5-ASA. Useful also if tenesmus present. Use suppositories for proctitis, and enemas and foam for more extensive disease. Enemas may reach the splenic flexure but difficult to retain if active disease. (PBS-funded) <i>Crohn's disease:</i> Of no proven benefit	Safe
Topical corticosteroids: prednisolone (enema, suppository), hydrocortisone (foam)	<i>Ulcerative colitis:</i> Less efficacious than rectal 5-ASA. Additive effect when used with rectal 5-ASA <i>Crohn's disease:</i> Of no proven benefit	Significant systemic absorption of corticosteroid
Oral prednisolone (or other systemically acting corticosteroid)	<i>Ulcerative colitis and Crohn's disease:</i> Used to induce remission in moderate to severe disease. Rapid action but low chance of mucosal healing in Crohn's disease. Once beneficial effects observed, taper over at least eight weeks, as rapid withdrawal associated with risk of relapse. No value in maintenance	Associated with many well-known long-term side effects, including osteoporosis, thinning of the skin, hypertension, diabetes, cataracts and weight gain
Coated budesonide	<i>Ulcerative colitis:</i> Of no proven benefit <i>Crohn's disease:</i> Almost as effective as prednisolone for terminal ileal and proximal colonic disease. Coated so released in terminal ileum. Topically active. Considerably reduced systemic corticosteroid effects due to high first-pass metabolism in liver. (TGA-approved for this indication but not PBS-funded)	Well tolerated. No detectable effect on bone health
Antibiotics	<i>Ulcerative colitis:</i> Little evidence for value <i>Crohn's disease:</i> Metronidazole and ciprofloxacin helpful in the short to medium-term for perianal disease	Monitor for Achilles tendonitis and rupture with ciprofloxacin and peripheral neuropathy with metronidazole

general and colorectal surgeons are more than capable of making the diagnosis, planning drug therapy and ongoing management are not in their skill-set, or indeed recommended as part of their role.

Rural GPs have problems of access to the preferred options. The advent of funded telemedicine in Australia may considerably ease patients' access

problems to more cost-effective specialist care. However, barriers have been identified, including time, poor access to the internet and equipment and a preference for traditional consultations.

SHARED CARE – WHO DOES WHAT?

Referral does not transfer all care of the patient to the gastroenterologist. Unfortunately, young patients without

comorbidities sometimes see it as such, and should be actively discouraged from this view. The key to optimal outcomes is shared care. However, the responsibilities of the GP and of the gastroenterologist require better definition. Suggested roles in specific tasks are outlined in Tables 3 and 4.

There is little doubt that most GPs are not equipped to optimally manage the

TABLE 1. CONVENTIONAL MEDICAL AND SURGICAL THERAPIES FOR IBD continued

Therapy	Application to ulcerative colitis and Crohn's disease	Side effects/monitoring
Immunomodulators: thiopurines (azathioprine, mercaptopurine) and methotrexate	<p>Slow onset of action – optimal effects after three months</p> <p><i>Ulcerative colitis:</i> Used in chronically active disease where remission and healing of the mucosa cannot be achieved with 5-ASA alone or relapses occur frequently despite adequate doses of 5-ASA. (Thiopurines and methotrexate not TGA-approved for this indication)</p> <p><i>Crohn's disease:</i> Thiopurines first-line maintenance therapy in all but very mild disease. Use of metabolite measurement to optimise dosage now common. Half of patients intolerant to one thiopurine will tolerate the other. If intolerance to both or inefficacy, use methotrexate subcutaneously. (Thiopurines and methotrexate not TGA-approved for this indication)</p>	<p>Bone marrow suppression (dose-dependent). Abnormal liver function, pancreatitis, fever, nausea. Increased risk of infection, non-melanotic skin cancer and lymphoma. Methotrexate is teratogenic. Regular monitoring of haematology and liver function mandatory for thiopurines and methotrexate – two-weekly until optimal dose reached, then monthly; once on stable dose, three-monthly</p>
Anti-tumour necrosis factor-alpha (TNF- α) monoclonal antibody (anti-TNF agent): infliximab, adalimumab	<p>Infliximab given by intravenous infusions – three induction infusions over six weeks and then one infusion every eight weeks. Adalimumab administered subcutaneously fortnightly</p> <p><i>Ulcerative colitis:</i> Effective as rescue therapy in severe cases with inadequate response to corticosteroids, and for chronically active disease despite immune-modulating therapy. (Infliximab TGA-approved for this indication but not funded by PBS; adalimumab not TGA-approved)</p> <p><i>Crohn's disease:</i> Used for patients failing immune-modulating therapy or for complex fistulising disease as first-line therapy. (PBS funding for these indications for both adalimumab and infliximab)</p>	<p>Well tolerated. Contraindicated in cardiac failure, demyelinating conditions. Risk of opportunistic infection, infusion reactions, lymphoma (rare). All patients to be screened for tuberculosis, hepatitis B and other viruses prior to its use. Vaccinations should be up to date</p>
Surgery	<p><i>Ulcerative colitis:</i> Total proctocolectomy with ileostomy or ileal pouch. Indicated in severe and unresponsive disease, chronically active disease despite adequate immune-modulating therapy, presence of neoplasia (dysplasia or cancer)</p> <p><i>Crohn's disease:</i> Necessary in most patients at some stage. 'Minimal surgery' generally performed, with resection only of what is necessary to achieve symptomatic benefit or resolution of complications and strictureplasty for short strictures. For perianal disease, drainage of abscesses, prevention of abscesses with setons in fistulous tracts</p>	<p>Very low mortality but expected postoperative morbidity. Ileal pouch usually associated with six to eight bowel motions/day but not urgency; 50% patients will have episodes of pouchitis (responsive to antibiotics), 20% will have chronic pouchitis. Prevention of recurrence after 'curative' resection for Crohn's disease required – therapy stratified according to risk</p>

ABBREVIATIONS: IBD = Inflammatory bowel disease; PBS = Pharmaceutical Benefits Scheme; TGA = Therapeutic Goods Administration.

intestinal inflammatory disease itself. A survey of more than 400 GPs in South Australia found that more than one-third were 'uncomfortable' managing patients with IBD in general and that 71% and 91% were uncomfortable with the use of immunomodulators and biologics, respectively.⁷ In addition, most GPs

(92%) reported seeing between none and five IBD patients per month. In other words, decisions regarding anti-inflammatory therapy should largely rest with the gastroenterologist and changes in therapy should not be instituted by the GP without discussion with the gastroenterologist. However, there

are many disease-related issues that should be proactively managed by the GP, such as flare recognition, constipation, pain management and therapy adherence (Table 3).

An essential part of shared care is that non-IBD-related illness, such as respiratory infections or gynaecological issues,

TABLE 2. PATIENT REFERRAL OPTIONS IN IBD: PROS AND CONS

Advantages	Disadvantages
IBD clinic in a tertiary centre	
<ul style="list-style-type: none"> • Multidisciplinary unit – patients discussed and managed under a team that includes gastroenterologists, specialist nurses and dietitians, surgeons, radiologists, pathologists • Access to IBD nurse who provides a phone service (less than 24-hour turn-round time), educative role, manages immune modulator drug monitoring, able to triage re urgent appointment • Access to dietitians and psychologists, other therapies • Access to latest IBD trials/novel therapies • No out-of-pocket expenses for patient • Ability to incorporate telehealth 	<ul style="list-style-type: none"> • Only available at certain tertiary hospitals • Unable to select location or doctor • Patient can be seen by different doctors on each visit • Some centres may have long initial waiting times
Private gastroenterologist	
<ul style="list-style-type: none"> • Patient has one-on-one relationship with personal service • Patients seen by same doctor each time, increasing continuity of care and closer doctor–patient relationship • Patient or GP can select gastroenterologist and appropriate location 	<ul style="list-style-type: none"> • May rely more on GP to monitor drug levels or provide scripts • Limited access to trials or novel therapies • Patient may have out-of-pocket expenses
Gastrointestinal clinic in a public hospital	
<ul style="list-style-type: none"> • Easy access • No out-of-pocket expenses for patient 	<ul style="list-style-type: none"> • Long waiting times for appointments • Patient often seen by a doctor without a special interest in IBD • Often time constraints for consultations

ABBREVIATION: IBD = Inflammatory bowel disease.

are better managed by the GP than by the gastroenterologist. The gastroenterologist’s advice might be needed if the problem could be related to the disease or its treatment (e.g. chest infection in a patient who is immunosuppressed) or the therapy to be instituted could affect the disease (e.g. treatment of gout with NSAIDs or allopurinol).

IBD and its treatment have potential long-term complications and issues for which preventive strategies may be

effective. As well as the complications of osteoporosis, iron deficiency and increased risks of cancer and cardiovascular disease, these include issues of infection and vaccination in patients taking immunosuppressive agents, increased risk of complications and surgery associated with smoking, body image issues and low psychosocial well-being. The gastroenterologist and GP should work collaboratively in optimising strategies to deal with these issues

INTERNET RESOURCES FOR PATIENTS

- **Crohn’s & Colitis Australia**
<http://www.crohnsandcolitis.com.au>
- **Gastroenterological Society of Australia (GESA)**
An information sheet on inflammatory bowel disease is available at:
http://www.gesa.org.au/files/editor_upload/File/GESA_IBD.pdf
- **Crohn’s & Colitis Foundation of America**
<http://www.ccfa.org>
- **Crohn’s & Colitis UK**
<http://www.nacc.org.uk>

(Table 4). The precise role of each needs to be defined on an individual patient basis, but also depends on the complication itself.

GUIDELINES FOR PATIENT COUNSELLING

Over recent times, significant steps have been taken to address the quality of care provided to patients with IBD in Australia. Specialist centres aim to address all aspects of health in patients with IBD by providing high-quality clinical care via a multidisciplinary IBD team and promoting patient support, education and empowerment.

A wide range of key professionals, including GPs, gastroenterologists, colorectal surgeons, clinical nurse specialists, dietitians, pharmacists, pathologists and radiologists, provide support to patients with IBD. The team should also have access to essential services when possible, including a psychologist or counsellor, rheumatologist, ophthalmologist, dermatologist, obstetrician and nutritional support team. The IBD nurse is recognised as being crucial in improving disease management, compliance and patient satisfaction. Internet resources for patients are listed in the box on this page.

TABLE 3. SHARED CARE IN IBD: MANAGEMENT ISSUES IN PATIENTS WITH ESTABLISHED IBD

Issue and importance	Management of issue	
	Strategy	Division of responsibilities
Suspected relapse		
Important to identify flares or relapses of IBD, and if suspected, to investigate (including full blood examination, urea and electrolytes, liver function tests, CRP and, if diarrhoea present, a stool specimen for <i>Clostridium difficile</i> toxin and, if available, faecal calprotectin) so that appropriate strategies can be formulated with minimal delays	Features such as elevated CRP, anaemia, reduced serum albumin and elevated calprotectin indicate a likely flare. Notify gastroenterologist promptly for rapid assessment and management. Some IBD clinics and gastroenterologists offer a help-line for such situations. If inflammatory markers uniformly normal, change in clinical status might reflect functional gut symptoms, especially as patients with IBD are three times more likely to develop these than those without IBD. Consider constipation in patients with distal ulcerative colitis: plain abdominal x-ray can reveal significant proximal faecal loading and symptoms may be effectively treated with aperients	Both GP and gastroenterologist
Drug therapy monitoring		
Thiopurine or methotrexate treatment (as occurs in more than half of patients with IBD) requires monitoring of leucocyte counts and liver function frequently in the first few months of treatment and three-monthly in the long term	Crucial to ensure safety of medication in first few months of treatment	Gastroenterologist
	Monitoring of patients on stable doses of immunomodulators may be performed by either the GP or gastroenterologist – clarify who is taking responsibility to ensure it is being done	Both GP and gastroenterologist
Biological therapy requires monitoring and paperwork to maintain funding	Prescription and monitoring of biological drugs can only be performed by the gastroenterologist	Gastroenterologist
Therapy adherence		
Nonadherence can be a significant cause of treatment failure, particularly in well patients on maintenance therapy Adherence to 5-ASA maintenance therapy poor in patients with ulcerative colitis, with up to 60% not taking prescribed medications; 90% of adherent patients remain in remission at 30 months, compared with 40% of nonadherent patients	Both the GP and the gastroenterologist play key roles in encouraging therapy adherence, particularly in the ‘well’ phase of the disease and especially by discussing the implications for nonadherence – i.e. increased risk of relapse and ill health, and increased risk of cancer (ongoing active inflammation being an unequivocal risk factor for large and small bowel cancer). Encourage patient participation in support programs developed to assist with compliance	Both GP and gastroenterologist
Pain management		
Abdominal pain generally associated with active disease, but about 20% of patients, especially those with Crohn’s disease, have chronic pain (mainly in abdomen, back and joints) despite good control of inflammation	Treat intercurrent acute painful conditions on their merit, perhaps with avoidance of NSAIDs	GP
	If abdominal pain a problem, look for and treat active disease	Gastroenterologist
Analgesic therapy challenging: <ul style="list-style-type: none"> paracetamol probably safe NSAIDs can worsen disease activity – not preferred opiates cause bowel problems and hyperalgesia – pro-inflammatory, addictive and associated with poorer outcomes 	Chronic joint pains not uncommon in patients with IBD. Treat joint pain clearly related to disease activity with disease-related therapy; manage other joint pain nonpharmacologically if possible, with rheumatologist assistance as necessary. Use NSAIDs with care and only under a gastroenterologist’s supervision. Avoid use of opiates	Both GP and gastroenterologist

ABBREVIATIONS: 5-ASA = 5-aminosalicylic acid; CRP = C-reactive protein; IBD = inflammatory bowel disease; NSAIDs = nonsteroidal anti-inflammatory drugs.

TABLE 4. SHARED CARE IN IBD: LONG-TERM COMPLICATIONS AND ISSUES IN PATIENTS WITH IBD

Issue and importance	Management of issue	
	Strategy	Division of responsibilities
Osteoporosis		
Increased risk of osteoporosis secondary to chronic inflammation, malabsorption, poor diet and corticosteroid use in patients with IBD	Concomitant use of calcium and vitamin D with corticosteroids Check and optimise vitamin D levels. If patient at-risk, perform a bone densitometry scan every two to three years	Both GP and gastroenterologist
Iron deficiency		
Iron poorly absorbed in patients with active inflammation due to an adaptive response of the small intestinal epithelium. Hence, iron deficiency, with or without anaemia, common in patients with IBD who have inadequately controlled disease	Iron repletion mandatory: • oral iron supplementation usually ineffective and can worsen disease activity	Both GP and gastroenterologist
	• intravenous iron supplementation preferred method	Gastroenterologist
Cancer		
<i>Colorectal cancer:</i> Increased risk of bowel cancer in patients with colonic involvement (ulcerative colitis or Crohn’s disease) for more than seven years. Risk level also dependent on disease extent, long-term disease activity control and family history of early onset bowel cancer	Colonoscopic surveillance, looking for dysplasia or mass lesions, recommended; frequency depends on individual’s relative risk	Gastroenterologist
	Encourage adherence to maintenance therapy with mesalazine and/or thiopurine	Both GP and gastroenterologist
<i>Non-melanomatous skin cancers:</i> Increased risk in immunosuppressed patients	Yearly skin checks recommended	GP
Cardiovascular disease		
Two- to three-fold increased risk of venothrombo-embolism. Patients admitted to hospital routinely receive prophylactic heparin, sometimes continued after discharge	Recurrent thrombosis and/or the presence of an inherited thrombophilic state lead to long-term anticoagulation therapy	Gastroenterologist
Arterial disease more common in patients with chronic inflammatory disease in general; same probably true for those with IBD, although standard risk factors may be less common	Identification and management of standard risk factors likely to be important – may be better managed by the GP	GP
Infection and vaccination		
Increased risk of infection in patients taking immunosuppressive medications (many patients with IBD). Risk elevated with increasing immune suppression, greatest risk being in patients taking corticosteroids in combination with other immunosuppressants. It is logical, therefore, that: • there be heightened awareness of the risk of opportunistic infection or more severe clinical manifestations of simple infections, and • preventive strategies be used	If possible, ensure currency of vaccinations before initiation of immunosuppressive therapy. <i>Non-live vaccines:</i> safe to give to patients on immunosuppressive agents, and an effective immune response has been shown to occur. <i>Live vaccines:</i> not safe to give to patients on immunosuppressive agents; they should be given before commencement of such therapy if indicated. If patient already on immunosuppressive therapy, agent(s) must be withheld for a minimum of six weeks before giving the live vaccine	Both GP and gastroenterologist

TABLE 4. SHARED CARE IN IBD: LONG-TERM COMPLICATIONS AND ISSUES IN PATIENTS WITH IBD continued

Issue and importance	Management of issue	
	Strategy	Division of responsibilities
Smoking		
Ongoing smoking associated with poorer disease course and increased risk of complications and surgery in patients with Crohn's disease. Therefore, smoking cessation imperative	Smoking cessation a goal of all health professionals managing patients with Crohn's disease. Smoking cessation as effective as a course of corticosteroids but longer lasting	Both GP and gastroenterologist
Sexual health and reproduction		
Patients with IBD often concerned about body image (surgical scars, stoma, perianal disease), symptoms affecting sexual activity (fatigue, dyspareunia) and the effects of disease surgery and drugs used on fertility, pregnancy and fetus	Counselling about these issues and medication safety during pregnancy best delivered by the gastroenterologist, who probably has easier access to up-to-date information in the field than GPs	Gastroenterologist
Psychosocial management		
Patients with IBD at high risk of psychosocial consequences due to often young age and IBD being a chronic illness with significant body image and sexual consequences. Patients have difficulty coping with the diagnosis and consequences of the disease. Depression or anxiety common, and can increase abdominal symptoms and reduce response to IBD-related medication	Prevention of psychosocial consequences important. As first step, work towards engaging patient in their own disease and treatment decisions via education and written materials. Specialist IBD clinics provide wide net of support, counselling and education	Gastroenterologist
	Screen patients and have low threshold for referring to counsellor, psychologist or psychiatrist	Both GP and gastroenterologist
	Refer patients to Crohn's & Colitis Australia (http://www.crohnsandcolitis.com.au/) for written material and general support	Both GP and gastroenterologist

ABBREVIATIONS: DTPa = diphtheria, tetanus and acellular pertussis; HPV = human papilloma virus; IBD = inflammatory bowel disease

CONCLUSION

Many GPs may feel uncomfortable managing patients with IBD and few have a large volume of patients with the disease. Heightened awareness of the increasing prevalence of IBD in Australia and the features that raise clinical suspicion of its presence will help the GP in ensuring an early diagnosis.

Management of the intestinal inflammatory disease itself is complex and requires considerable specialised expertise. However, most management issues involve patients with chronic illness, and shared care is the gold standard. The GP plays a key role in supporting the patient through the diagnosis and beyond, preventing associated disease complications, assisting with adherence to therapy and smoking cessation, promoting

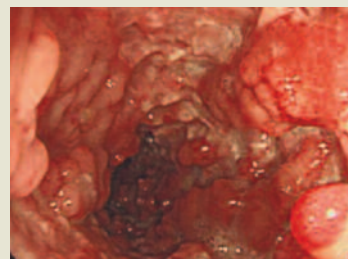
psychosocial wellbeing and managing intercurrent issues. MT

REFERENCES AND FURTHER READING

References and Further reading are included in the pdf version of this article available at www.medicinetoday.com.au.

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Inflammatory bowel disease

The GP's role in shared care of patients

BRITT CHRISTENSEN MB BS, BSc; **BELINDA HEADON** RN; **PETER R. GIBSON** MD, FRACP

REFERENCES

1. Wilson J, Hair C, Knight R, et al. High incidence of inflammatory bowel disease in Australia: a prospective population-based Australian incidence study. *Inflamm Bowel Dis* 2010; 16: 1550-1556.
2. Mahid SS, Minor KS, Soto RE, Hornung CA, Galandiuk S. Smoking and inflammatory bowel disease: a meta-analysis. *Mayo Clin Proc* 2006; 81: 1462-1471.
3. Hansen TS, Jess T, Vind I, et al. Environmental factors in inflammatory bowel disease: a case-control study based on a Danish inception cohort. *J Crohns Colitis* 2011; 5: 577-584.
4. Prideaux L, Kamm MA, De Cruz PP, Chan FK, Ng SC. Inflammatory bowel disease in Asia: a systematic review. *J Gastroenterol Hepatol* 2012; 27: 1266-1280.
5. Fielding JF. The relative risk of inflammatory bowel disease among parents and siblings of Crohn's disease patients. *J Clin Gastroenterol* 1986; 8: 655-657.
6. Russell RK, Satsangi J. IBD: a family affair. *Best Pract Res Clin Gastroenterol* 2004; 18: 525-539.
7. Tan M, Holloway RH, Lange K, Andrews JM. General practitioners' knowledge of and attitudes to inflammatory bowel disease. *Intern Med J* 2012; 42: 801-807.

FURTHER READING

Andrews JM, Mountfield RE, Van Langenberg DR, Bampton PA, Holtmann GJ. Un-promoted issues in inflammatory bowel disease: opportunities to optimize care. *Intern Med J* 2010; 40: 173-182.

Crawford NW, Catto-Smith AG, Oliver MR, Cameron DJ, BATTERY JP. An Australian audit of vaccination status in children and adolescents with inflammatory bowel disease. *BMC Gastroenterol* 2011; 11: 87.

Judd TA, Day AS, Lemberg DA, Turner D, Leach ST. Update of fecal markers of inflammation in inflammatory bowel disease. *J Gastroenterol Hepatol* 2011; 26: 1493-1499.

Grimpen F, Pavli P. Advances in the management of inflammatory bowel disease. *Intern Med J* 2010; 40: 258-264.

Mikocka-Walus AA, Turnbull D, Holtmann G, Andrews JM. An integrated model of care for inflammatory bowel disease sufferers in Australia: development and the effects of its implementation. *Inflamm Bowel Dis* 2012; 18: 1573-1581.

Waters OR, Lawrance IC. Understanding the use of immunosuppressive agents in the clinical management of IBD. *Curr Drug Targets* 2011; 12: 1364-1371.